

Guidelines for Environmental Infection Control in Health-Care Facilities

**Recommendations of CDC and the Healthcare Infection Control
Practices Advisory Committee (HICPAC)**

**U.S. Department of Health and Human Services
Centers for Disease Control and Prevention (CDC)
Atlanta, GA 30333**

2003

Suggested Citations:

Available from the CDC Internet Site:

The full-text version of the guidelines appears as a web-based document at the CDC's Division of Healthcare Quality Promotion's Internet site at:
www.cdc.gov/ncidod/hip/enviro/guide.htm

The full-text version of the guidelines should be cited when reference is made primarily to material in Parts I and IV. The print version of the guidelines appears as:

Sehulster LM, Chinn RYW, Arduino MJ, Carpenter J, Donlan R, Ashford D, Besser R, Fields B, McNeil MM, Whitney C, Wong S, Juranek D, Cleveland J. Guidelines for environmental infection control in health-care facilities. Recommendations from CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). Chicago IL; American Society for Healthcare Engineering/American Hospital Association; 2004.

Part II of these guidelines appeared in the CDC's "Morbidity and Mortality Weekly Report:"

Centers for Disease Control and Prevention. Guidelines for environmental infection control in health-care facilities: recommendations of CDC and the Healthcare Infection Control Practices Advisory Committee (HICPAC). MMWR 2003; 52 (No. RR-10): 1–48.

Updates to the Part II recommendations also appeared in the MMWR in 2003 as "Errata: Vol. 52 (No. RR-10)" (MMWR Vol. 52 [42]: 1025–6) on October 24, 2003 and as a "Notice to Readers" scheduled to appear in February 2004. The full-text version of these guidelines (this document) incorporates these updates.

Centers for Disease Control and Prevention Healthcare Infection Control Practices Advisory Committee (HICPAC)

Guidelines for Environmental Infection Control in Health-Care Facilities

Abstract

Background:

Although the environment serves as a reservoir for a variety of microorganisms, it is rarely implicated in disease transmission except in the immunocompromised population. Inadvertent exposures to environmental opportunistic pathogens (e.g., *Aspergillus* spp. and *Legionella* spp.) or airborne pathogens (e.g., *Mycobacterium tuberculosis* and varicella-zoster virus) may result in infections with significant morbidity and/or mortality. Lack of adherence to established standards and guidance (e.g., water quality in dialysis, proper ventilation for specialized care areas such as operating rooms, and proper use of disinfectants) can result in adverse patient outcomes in health-care facilities.

Objective:

The objective is to develop an environmental infection-control guideline that reviews and reaffirms strategies for the prevention of environmentally-mediated infections, particularly among health-care workers and immunocompromised patients. The recommendations are evidence-based whenever possible.

Search Strategies:

The contributors to this guideline reviewed predominantly English-language articles identified from MEDLINE literature searches, bibliographies from published articles, and infection-control textbooks.

Criteria for Selecting Citations and Studies for This Review:

Articles dealing with outbreaks of infection due to environmental opportunistic microorganisms and epidemiological- or laboratory experimental studies were reviewed. Current editions of guidelines and standards from organizations (i.e., American Institute of Architects [AIA], Association for the Advancement of Medical Instrumentation [AAMI], and American Society of Heating, Refrigeration, and Air-Conditioning Engineers [ASHRAE]) were consulted. Relevant regulations from federal agencies (i.e., U.S. Food and Drug Administration [FDA]; U.S. Department of Labor, Occupational Safety and Health Administration [OSHA]; U.S. Environmental Protection Agency [EPA]; and U.S. Department of Justice) were reviewed. Some topics did not have well-designed, prospective studies nor reports of outbreak investigations. Expert opinions and experience were consulted in these instances.

Types of Studies:

Reports of outbreak investigations, epidemiological assessment of outbreak investigations with control strategies, and *in vitro* environmental studies were assessed. Many of the recommendations are derived

from empiric engineering concepts and reflect industry standards. A few of the infection-control measures proposed cannot be rigorously studied for ethical or logistical reasons.

Outcome Measures:

Infections caused by the microorganisms described in this guideline are rare events, and the effect of these recommendations on infection rates in a facility may not be readily measurable. Therefore, the following steps to measure performance are suggested to evaluate these recommendations:

1. Document whether infection-control personnel are actively involved in all phases of a health-care facility's demolition, construction, and renovation. Activities should include performing a risk assessment of the necessary types of construction barriers, and daily monitoring and documenting of the presence of negative airflow within the construction zone or renovation area.
2. Monitor and document daily the negative airflow in airborne infection isolation rooms (AII) and positive airflow in protective environment rooms (PE), especially when patients are in these rooms.
3. Perform assays at least once a month by using standard quantitative methods for endotoxin in water used to reprocess hemodialyzers, and for heterotrophic, mesophilic bacteria in water used to prepare dialysate and for hemodialyzer reprocessing.
4. Evaluate possible environmental sources (e.g., water, laboratory solutions, or reagents) of specimen contamination when nontuberculous mycobacteria (NTM) of unlikely clinical importance are isolated from clinical cultures. If environmental contamination is found, eliminate the probable mechanisms.
5. Document policies to identify and respond to water damage. Such policies should result in either repair and drying of wet structural materials within 72 hours, or removal of the wet material if drying is unlikely within 72 hours.

Main Results:

Infection-control strategies and engineering controls, when consistently implemented, are effective in preventing opportunistic, environmentally-related infections in immunocompromised populations. Adherence to proper use of disinfectants, proper maintenance of medical equipment that uses water (e.g., automated endoscope reprocessors and hydrotherapy equipment), water-quality standards for hemodialysis, and proper ventilation standards for specialized care environments (i.e., airborne infection isolation [AII], protective environment [PE], and operating rooms [ORs]), and prompt management of water intrusion into facility structural elements will minimize health-care-associated infection risks and reduce the frequency of pseudo-outbreaks. Routine environmental sampling is not advised except in the few situations where sampling is directed by epidemiologic principles and results can be applied directly to infection control decisions, and for water quality determinations in hemodialysis.

Reviewers' Conclusions:

Continued compliance with existing environmental infection control measures will decrease the risk of health-care-associated infections among patients, especially the immunocompromised, and health-care workers.

**Centers for Disease Control and Prevention
Healthcare Infection Control Practices Advisory Committee (HICPAC)**

***Guidelines for Environmental Infection Control in
Health-Care Facilities***

Table of Contents

Executive Summary	1
Part I. Background Information: Environmental Infection Control in Health-Care Facilities	3
A. Introduction	3
B. Key Terms Used in this Guideline	5
C. Air	6
1. Modes of Transmission of Airborne Diseases	6
2. Airborne Infectious Diseases in Health-Care Facilities	7
3. Heating, Ventilation, and Air Conditioning Systems in Health-Care Facilities	13
4. Construction, Renovation, Remediation, Repair, and Demolition	21
5. Environmental Infection-Control Measures for Special Health-Care Settings.....	34
6. Other Aerosol Hazards in Health-Care Facilities	40
D. Water	40
1. Modes of Transmission of Waterborne Diseases	40
2. Waterborne Infectious Diseases in Health-Care Facilities	41
3. Water Systems in Health-Care Facilities	46
4. Strategies for Controlling Waterborne Microbial Contamination	53
5. Cooling Towers and Evaporative Condensers.....	57
6. Dialysis Water Quality and Dialysate.....	59
7. Ice Machines and Ice	65
8. Hydrotherapy Tanks and Pools.....	67
9. Miscellaneous Medical/Dental Equipment Connected to Main Water Systems	69
E. Environmental Services	71
1. Principles of Cleaning and Disinfecting Environmental Surfaces.....	71
2. General Cleaning Strategies for Patient-Care Areas.....	74
3. Cleaning Strategies for Spills of Blood and Body Substances	77
4. Carpeting and Cloth Furnishings	78
5. Flowers and Plants in Patient-Care Areas	80
6. Pest Control	81
7. Special Pathogen Concerns.....	82
F. Environmental Sampling	88
1. General Principles: Microbiologic Sampling of the Environment	88
2. Air Sampling.....	89
3. Water Sampling	94
4. Environmental Surface Sampling	95
G. Laundry and Bedding	98
1. General Information	98
2. Epidemiology and General Aspects of Infection Control.....	98
3. Collecting, Transporting, and Sorting Contaminated Textiles and Fabrics.....	99

4. Parameters of the Laundry Process	100
5. Special Laundry Situations.....	102
6. Surgical Gowns, Drapes, and Disposable Fabrics.....	103
7. Antimicrobial-Impregnated Articles and Consumer Items Bearing Antimicrobial Labeling ..	103
8. Standard Mattresses, Pillows, and Air-Fluidized Beds	104
H. Animals in Health-Care Facilities	105
1. General Information	105
2. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animals	106
3. Service Animals	108
4. Animals as Patients in Human Health-Care Facilities	110
5. Research Animals in Health-Care Facilities	111
I. Regulated Medical Waste	112
1. Epidemiology	112
2. Categories of Medical Waste	112
3. Management of Regulated Medical Waste in Health-Care Facilities	113
4. Treatment of Regulated Medical Waste.....	113
5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks.....	116
6. Medical Waste and CJD	116
Part II. Recommendations for Environmental Infection Control in Health-Care Facilities	117
A. Rationale for Recommendations	117
B. Rating Categories.....	117
C. Recommendations—Air	118
D. Recommendations—Water	125
E. Recommendations—Environmental Services	133
F. Recommendations—Environmental Sampling	138
G. Recommendations—Laundry and Bedding	138
H. Recommendations—Animals in Health-Care Facilities.....	141
I. Recommendations—Regulated Medical Waste.....	143
Part III. References.....	145
Part IV. Appendices.....	201
Appendix A. Glossary of Terms	201
Appendix B. Air	210
1. Airborne Contaminant Removal	210
2. Air Sampling for Aerosols Containing Legionellae.....	210
3. Calculation of Air Sampling Results.....	211
4. Ventilation Specifications for Health-Care Facilities.....	212
Appendix C. Water	220
1. Biofilms	220
2. Water and Dialysate Sampling Strategies in Dialysis.....	222
3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae	223
4. Procedure for Cleaning Cooling Towers and Related Equipment	225
5. Maintenance Procedures Used to Decrease Survival and Multiplications of <i>Legionella</i> spp. in Potable-Water Distribution Systems	227
Appendix D. Insects and Microorganisms.....	228
Appendix E. Information Resources.....	229
Appendix F. Areas of Future Research	230
Index—Parts I and IV	231

List of Figures, Boxes, and Tables

Figures

Figure 1. Diagram of a ventilation system	14
Figure 2. Example of positive-pressure room control for protection from airborne environmental microbes (PE)	35
Figure 3. Example of negative-pressure room control for airborne infection isolation (AII)	36
Figure 4. Example of airborne infection isolation (AII) room with anteroom and neutral anteroom	37
Figure 5. Diagram of a typical air conditioning (induced draft) cooling tower	58
Figure 6. Dialysis water treatment system	60

Boxes

Box 1. Environmental infection control: performance measures	2
Box 2. Eight criteria for evaluating the strength of evidence for environmental sources of infection	4
Box 3. Chain of infection components	4
Box 4. Suggested members and functions of a multi-disciplinary coordination team for construction, renovation, repair, and demolition projects	24
Box 5. Construction design and function considerations for environmental infection control	25
Box 6. Unresolved issues associated with microbiologic air sampling	28
Box 7. Construction/repair projects that require barrier structures	32
Box 8. Strategy for managing TB patients and preventing airborne transmission in operating rooms	39
Box 9. Recovery and remediation measures for water-related emergencies	52
Box 10. Contingency planning for flooding	53
Box 11. Steps in an epidemiologic investigation for legionellosis	56
Box 12. General steps for cleaning and maintaining ice machines, dispensers, and storage chests	66
Box 13. Preliminary concerns for conducting air sampling	90
Box 14. Selecting an air sampling device	93
Box 15. Undertaking environmental-surface sampling	95
Box C.1. Potential sampling sites for <i>Legionella</i> spp. in health-care facilities	224
Box C.2. Procedures for collecting and processing environmental specimens for <i>Legionella</i> spp.	225

Tables

Table 1. Clinical and epidemiologic characteristics of aspergillosis	7
Table 2. Environmental fungal pathogens: entry into and contamination of the health-care facility	8
Table 3. Clinical and epidemiologic characteristics of tuberculosis (TB)	10
Table 4. Microorganisms associated with airborne transmission	13
Table 5. Filtration methods	15
Table 6. Engineered specifications for positive- and negative pressure rooms	19
Table 7. Ventilation hazards in health-care facilities that may be associated with increased potential of airborne disease transmission	22
Table 8. Strategies to reduce dust and moisture intrusion during external demolition and construction	30

Table 9. Infection-control measures for internal construction and repair projects	32
Table 10. Summary of ventilation specifications in selected areas of health-care facilities.....	39
Table 11. Clinical and epidemiologic characteristics of legionellosis/Legionnaires disease.....	41
Table 12. <i>Pseudomonas aeruginosa</i> infections in health-care facilities	42
Table 13. Other gram-negative bacteria associated with water and moist environments	43
Table 14. Nontuberculous mycobacteria—environmental vehicles	45
Table 15. Water and point-of-use fixtures as sources and reservoirs of waterborne pathogens	47
Table 16. Water demand in health-care facilities during water disruption emergencies	50
Table 17. Additional infection-control measures to prevent exposure of high-risk patients to waterborne pathogens.....	57
Table 18. Microbiologic limits for hemodialysis fluids.....	62
Table 19. Factors influencing microbial contamination in hemodialysis systems.....	63
Table 20. Microorganisms and their sources in ice and ice machines	65
Table 21. Infections associated with use of hydrotherapy equipment	67
Table 22. Levels of disinfection by type of microorganism	72
Table 23. Air sampling methods and examples of equipment	91
Table 24. Examples of eluents and diluents for environmental-surface sampling.....	96
Table 25. Methods of environmental-surface sampling.....	97
Table 26. Examples of diseases associated with zoonotic transmission.....	105
Table 27. Microorganisms and biologicals identified as select agents	115
Table B.1. Air changes/hour (ACH) and time required for airborne-contaminant removal efficiencies of 99% and 99.9%	210
Table B.2. Ventilation requirements for areas affecting patient care in hospitals and outpatient facilities	212
Table B.3. Pressure relationships and ventilation of certain areas of nursing facilities.....	217
Table B.4. Filter efficiencies for central ventilation and air conditioning systems in general hospitals	219
Table B.5. Filter efficiencies for central ventilation and air conditioning systems in outpatient facilities	219
Table B.6. Filter efficiencies for central ventilation and air conditioning systems in nursing facilities	220
Table B.7. Filter efficiencies for central ventilation and air conditioning systems in psychiatric hospitals	220
Table D.1. Microorganisms isolated from arthropods in health-care settings	228

List of Abbreviations Used in This Publication

AAA	animal-assisted activity
AAMI	Association for the Advancement of Medical Instrumentation
AAT	animal-assisted therapy
ACGIH	American Council of Governmental Industrial Hygienists
ACH	air changes per hour
ADA	Americans with Disabilities Act
AER	automated endoscope reprocessor
AFB	acid-fast bacilli
AHA	American Hospital Association
AHJ	authorities having jurisdiction
AIA	American Institute of Architects
AII	airborne infection isolation
AmB	amphotericin B
ANC	absolute neutrophil count
ANSI	American National Standards Institute
AORN	Association of periOperative Registered Nurses
ASHE	American Society for Healthcare Engineering
ASHRAE	American Society of Heating, Refrigeration, and Air-Conditioning Engineers
BCG	Bacille Calmette-Guérin
BCYE	buffered charcoal yeast extract medium
BHI	brain-heart infusion
BMBL	CDC/NIH publication “Biosafety in Microbiological and Biomedical Laboratories”
BOD	biological oxygen demand
BSE	bovine spongiform encephalopathy
BSL	biosafety level
C	Centigrade
CAPD	continuous ambulatory peritoneal dialysis
CCPD	continual cycling peritoneal dialysis
CMAD	count median aerodynamic diameter
CDC	U.S. Centers for Disease Control and Prevention
CFR	Code of Federal Regulations
CFU	colony-forming unit
CJD	Creutzfeldt-Jakob disease
cm	centimeter
CMS	U.S. Centers for Medicare and Medicaid Services
CPL	compliance document (OSHA)
CT/EC	cooling tower/evaporative condenser
DFA	direct fluorescence assay; direct fluorescent antibody
DHHS	U.S. Department of Health and Human Services
DHBV	duck hepatitis B virus
DNA	deoxyribonucleic acid
DOP	dioctylphthalate
DOT	U.S. Department of Transportation
EC	environment of care (JCAHO)
ELISA	enzyme-linked immunosorbent assay
EPA	U.S. Environmental Protection Agency
ESRD	end-stage renal disease

EU	endotoxin unit
F	Fahrenheit
FDA	U.S. Food and Drug Administration
FIFRA	Federal Insecticide, Fungicide, and Rodenticide Act
FRC	free residual chlorine
ft	foot (feet)
FTC	U.S. Federal Trade Commission
GISA	glycopeptide intermediate resistant <i>Staphylococcus aureus</i>
HBV	hepatitis B virus
HCV	hepatitis C virus
HEPA	high efficiency particulate air
HICPAC	Healthcare Infection Control Practices Advisory Committee
HIV	human immunodeficiency virus
HPV	human papilloma virus
HSCT	hematopoietic stem cell transplant
HVAC	heating, ventilation, air conditioning
ICRA	infection control risk assessment
ICU	intensive care unit
ID₅₀	50% median infectious dose
IPD	intermittent peritoneal dialysis
JCAHO	Joint Commission on Accreditation of Healthcare Organizations
kg	kilogram
L	liter
MAC	<i>Mycobacterium avium</i> complex; also used to denote MacConkey agar
MDRO	multiple-drug resistant organism
MIC	minimum inhibitory concentration
µm	micrometer; micron
mL	milliliter
min	minute
mg	milligram
MMAD	mass median aerodynamic diameter
MMWR	“Morbidity and Mortality Weekly Report”
MRSA	methicillin-resistant <i>Staphylococcus aureus</i>
MSDS	material safety data sheet
N	Normal
NaCl	sodium chloride
NaOH	sodium hydroxide
NCID	National Center for Infectious Diseases
NCCDPHP	National Center for Chronic Disease Prevention and Health Promotion
NCCLS	National Committee for Clinical Laboratory Standards
ng	nanogram
NICU	neonatal intensive care unit
NIH	U.S. National Institutes of Health
NIOSH	National Institute for Occupational Safety and Health
nm	nanometer
NNIS	National Nosocomial Infection Surveillance
NTM	nontuberculous mycobacteria
OPL	on-premises laundry
OSHA	U.S. Occupational Safety and Health Administration
Pa	Pascal
PCP	<i>Pneumocystis carinii</i> pneumonia

PCR	polymerase chain reaction
PD	peritoneal dialysis
PE	protective environment
PEL	permissible exposure limit
PPE	personal protective equipment
ppm	parts per million
PVC	polyvinylchloride
RAPD	randomly amplified polymorphic DNA
RODAC	replicate organism direct agar contact
RSV	respiratory syncytial virus
RO	reverse osmosis
SARS	severe acute respiratory syndrome
SARS-CoV	SARS coronavirus
sec	second
spp	species
SSI	surgical site infection
TB	tuberculosis
TLV®-TWA	threshold limit value-time weighted average
TSA	tryptic soy agar
TSB	tryptic soy broth
TSE	transmissible spongiform encephalopathy
U.S.	United States
USC	United States Code
USDA	U.S. Department of Agriculture
USPS	U.S. Postal Service
UV	ultraviolet
UVGI	ultraviolet germicidal irradiation
VAV	variable air ventilation
vCJD	variant Creutzfeldt-Jakob disease
VISA	vancomycin intermediate resistant <i>Staphylococcus aureus</i>
VRE	vancomycin-resistant <i>Enterococcus</i>
VRSA	vancomycin-resistant <i>Staphylococcus aureus</i>
v/v	volume/volume
VZV	varicella-zoster virus

Note: Use of trade names and commercial sources is for identification only and does not imply endorsement by the U.S. Department of Health and Human Services. References to non-CDC sites on the Internet are provided as a service to the reader and does not constitute or imply endorsement of these organization s or their programs by CDC or the U.S. Department of Health and Human Services. CDC is not responsible for the content of pages found at these sites.

The following CDC staff member and HICPAC member prepared this report:

Lynne Schulster, PhD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases

Raymond Y.W. Chinn, MD
HICPAC Advisor
Sharp Memorial Hospital
San Diego, California

Disclosure of Relationship: Raymond Y.W. Chinn is a private-practice physician and salaried employee of Sharp Memorial Hospital in San Diego, California. Dr. Chinn received no research funds from commercial sources either directly, or indirectly through awards made to the hospital, before or during the development of these guidelines.

Contributions were made by the following CDC staff members:

Matthew Arduino, DrPH
Joe Carpenter, PE
Rodney Donlan, PhD
Lynne Schulster, PhD
Division of Healthcare Quality Promotion
National Center for Infectious Diseases

David Ashford, DVM, Dsc, MPH
Richard Besser, MD
Barry Fields, PhD
Michael M. McNeil, MBBS, MPH
Cynthia Whitney, MD, MPH
Stephanie Wong, DMV, MPH
Division of Bacterial and Mycotic Diseases
National Center for Infectious Diseases

Dennis Juranek, DVM, MSC
Division of Parasitic Diseases
National Center for Infectious Diseases

Jennifer Cleveland, DDS, MPH
Division of Oral Health
National Center for Chronic Disease Prevention and Health Promotion

***In collaboration with the Healthcare Infection Control Practices Advisory Committee
(HICPAC)***

HICPAC Members, February 2002

Robert A. Weinstein, MD
Chair
Cook County Hospital
Chicago, IL

Jane D. Siegel, MD
Co-Chair
University of Texas Southwestern Medical Center
Dallas, TX

Michele L. Pearson, MD
Executive Secretary
Centers for Disease Control and Prevention
Atlanta, GA

Raymond Y.W. Chinn, MD
Sharp Memorial Hospital
San Diego, CA

Alfred DeMaria, Jr., MD
Massachusetts Department of Public Health
Jamaica Plain, MA

Elaine L. Larson, RN, PhD
Columbia University School of Nursing
New York, NY

James T. Lee, MD, PhD
University of Minnesota
VA Medical Center
St. Paul, MN

Ramon E. Moncada, MD
Coronado Physician's Medical Center
Coronado, CA

William A. Rutala, PhD, MPH, CIC
University of North Carolina School of
Medicine
Chapel Hill, NC

William E. Scheckler, MD
University of Wisconsin Medical School
Madison, WI

Beth H. Stover, RN, CIC
Kosair Children's Hospital
Louisville, KY

Marjorie A. Underwood, RN, BSN, CIC
Mt. Diablo Medical Center
Concord, CA

Liaison Members

Loretta L. Fauerbach, MS, CIC
Association for Professionals in Infection
Control and Epidemiology (APIC)
Washington, DC

Sandra L. Fitzler, RN
American Health Care Association
Washington, DC

Dorothy M. Fogg, RN, BSN, MA
Association of periOperative Registered
Nurses (AORN)
Denver, CO

Stephen F. Jencks, MD, MPH
U.S. Centers for Medicare and Medicaid
Services
Baltimore, MD

Chiu S. Lin, PhD
U.S. Food and Drug Administration
Rockville, MD

James P. Steinberg, MD
Society for Healthcare Epidemiology of America,
Inc. (SHEA)
Atlanta, GA

Liaison Members (continued)

Michael L. Tapper, MD
Advisory Committee for the Elimination of
Tuberculosis (ACET)
New York, NY

Expert Reviewers

Trisha Barrett, RN, MBA, CIC
Alta Bates Medical Center
Berkeley, CA

Michael Berry
University of North Carolina
Chapel Hill, NC

Walter W. Bond, MS
RCSA, Inc.
Lawrenceville, GA

Douglas Erickson, FASHE
American Society for Healthcare
Engineering (ASHE)
Park Ridge, IL

Richard Miller, MD
University of Louisville School of Medicine
Louisville, KY

Gina Pugliese, RN, MS
Premier Safety Institute
Oak Brook, IL

James D. Scott, PE
Michigan Department of Consumer and
Industry Services
Lansing, MI

Dale Woodin
American Society for Healthcare Engineering
(ASHE)
Chicago, IL

Judene Bartley, MS, MPH, CIC
Epidemiology Consulting Services, Inc.
Beverly Hills, MI

Col. Nancy Bjerke, BSN, MA, MEd, MPH, CIC
(USAF, Retired)
Infection Control Associates (ICA)
San Antonio, TX

Cheryl Carter, RN
University of Iowa Health Center
Iowa City, IA

Martin S. Favero, PhD
Advanced Sterilization Products, Johnson and
Johnson
Irvine, CA

Col. Shannon E. Mills, DDS
HQ USAF / Surgeon General Detail
Bolin AFB, DC

Craig E. Rubens, MD, PhD
Children's Hospital and Medical Center
Seattle, WA

Andrew J. Streifel, MPH, REHS
University of Minnesota
Minneapolis, MN

Executive Summary

The *Guidelines for Environmental Infection Control in Health-Care Facilities* is a compilation of recommendations for the prevention and control of infectious diseases that are associated with health-care environments. This document a) revises multiple sections from previous editions of the Centers for Disease Control and Prevention [CDC] document titled *Guideline for Handwashing and Hospital Environmental Control*;^{1,2} b) incorporates discussions of air and water environmental concerns from CDC's *Guideline for the Prevention of Nosocomial Pneumonia*;³ c) consolidates relevant environmental infection-control measures from other CDC guidelines;⁴⁻⁹ and d) includes two topics not addressed in previous CDC guidelines — infection-control concerns related to animals in health-care facilities and water quality in hemodialysis settings.

Part I of this report, *Background Information: Environmental Infection Control in Health-Care Facilities*, provides a comprehensive review of the scientific literature. Attention is given to engineering and infection-control concerns during construction, demolition, renovation, and repairs of health-care facilities. Use of an infection-control risk assessment is strongly supported before the start of these or any other activities expected to generate dust or water aerosols. Also reviewed in Part I are infection-control measures used to recover from catastrophic events (e.g., flooding, sewage spills, loss of electricity and ventilation, and disruption of the water supply) and the limited effects of environmental surfaces, laundry, plants, animals, medical wastes, cloth furnishings, and carpeting on disease transmission in healthcare facilities.

Part II of this guideline, *Recommendations for Environmental Infection Control in Health-Care Facilities*, outlines environmental infection control in health-care facilities, describing measures for preventing infections associated with air, water, and other elements of the environment. These recommendations represent the views of different divisions within CDC's National Center for Infectious Diseases (NCID) (e.g., the Division of Healthcare Quality Promotion [DHQP] and the Division of Bacterial and Mycotic Diseases [DBMD]) and the consensus of the Healthcare Infection Control Practices Advisory Committee (HICPAC), a 12-member group that advises CDC on concerns related to the surveillance, prevention, and control of health-care-associated infections, primarily in U.S. health-care facilities.¹⁰ In 1999, HICPAC's infection-control focus was expanded from acute-care hospitals to all venues where health care is provided (e.g., outpatient surgical centers, urgent care centers, clinics, outpatient dialysis centers, physicians' offices, and skilled nursing facilities). The topics addressed in this guideline are applicable to the majority of health-care venues in the United States. This document is intended for use primarily by infection-control professionals (ICPs), epidemiologists, employee health and safety personnel, information system specialists, administrators, engineers, facility managers, environmental service professionals, and architects for health-care facilities.

Key recommendations include a) infection-control impact of ventilation system and water system performance; b) establishment of a multidisciplinary team to conduct infection-control risk assessment; c) use of dust-control procedures and barriers during construction, repair, renovation, or demolition; d) environmental infection-control measures for special care areas with patients at high risk; e) use of airborne particle sampling to monitor the effectiveness of air filtration and dust-control measures; f) procedures to prevent airborne contamination in operating rooms when infectious tuberculosis [TB] patients require surgery; g) guidance regarding appropriate indications for routine culturing of water as part of a comprehensive control program for legionellae; h) guidance for recovering from water system disruptions, water leaks, and natural disasters [e.g., flooding]; i) infection-control concepts for equipment that uses water from main lines [e.g., water systems for hemodialysis, ice machines, hydrotherapy equipment, dental unit water lines, and automated endoscope reprocessors]; j) environmental surface cleaning and disinfection strategies with respect to antibiotic-resistant

microorganisms; k) infection-control procedures for health-care laundry; l) use of animals in health care for activities and therapy; m) managing the presence of service animals in health-care facilities; n) infection-control strategies for when animals receive treatment in human health-care facilities; and o) a call to reinstate the practice of inactivating amplified cultures and stocks of microorganisms on-site during medical waste treatment.

Whenever possible, the recommendations in Part II are based on data from well-designed scientific studies. However, certain of these studies were conducted by using narrowly defined patient populations or for specific health-care settings (e.g., hospitals versus long-term care facilities), making generalization of findings potentially problematic. Construction standards for hospitals or other health-care facilities may not apply to residential home-care units. Similarly, infection-control measures indicated for immunosuppressed patient care are usually not necessary in those facilities where such patients are not present. Other recommendations were derived from knowledge gained during infectious disease investigations in health-care facilities, where successful termination of the outbreak was often the result of multiple interventions, the majority of which cannot be independently and rigorously evaluated. This is especially true for construction situations involving air or water.

Other recommendations are derived from empiric engineering concepts and may reflect an industry standard rather than an evidence-based conclusion. Where recommendations refer to guidance from the American Institute of Architects (AIA), the statements reflect standards intended for new construction or renovation. Existing structures and engineered systems are expected to be in continued compliance with the standards in effect at the time of construction or renovation. Also, in the absence of scientific confirmation, certain infection-control recommendations that cannot be rigorously evaluated are based on a strong theoretical rationale and suggestive evidence. Finally, certain recommendations are derived from existing federal regulations. The references and the appendices comprise Parts III and IV of this document, respectively.

Infections caused by the microorganisms described in these guidelines are rare events, and the effect of these recommendations on infection rates in a facility may not be readily measurable. Therefore, the following steps to measure performance are suggested to evaluate these recommendations (Box 1):

Box 1. Environmental infection control: performance measures

-
- 1. Document whether infection-control personnel are actively involved in all phases of a health-care facility's demolition, construction, and renovation. Activities should include performing a risk assessment of the necessary types of construction barriers, and daily monitoring and documenting of the presence of negative airflow within the construction zone or renovation area.**
 - 2. Monitor and document daily the negative airflow in airborne infection isolation (AII) rooms and positive airflow in protective environment (PE) rooms, especially when patients are in these rooms.**
 - 3. Perform assays at least once a month by using standard quantitative methods for endotoxin in water used to reprocess hemodialyzers, and for heterotrophic and mesophilic bacteria in water used to prepare dialysate and for hemodialyzer reprocessing.**
 - 4. Evaluate possible environmental sources (e.g., water, laboratory solutions, or reagents) of specimen contamination when nontuberculous mycobacteria (NTM) of unlikely clinical importance are isolated from clinical cultures. If environmental contamination is found, eliminate the probable mechanisms.**
 - 5. Document policies to identify and respond to water damage. Such policies should result in either repair and drying of wet structural or porous materials within 72 hours, or removal of the wet material if drying is unlikely with 72 hours.**
-

Topics outside the scope of this document include a) noninfectious adverse events (e.g., sick building syndrome); b) environmental concerns in the home; c) home health care; d) bioterrorism; and e) health-care–associated foodborne illness. This document includes only limited discussion of a) handwashing/hand hygiene; b) standard precautions; and c) infection-control measures used to prevent instrument or equipment contamination during patient care (e.g., preventing waterborne contamination of nebulizers or ventilator humidifiers). These topics are mentioned only if they are important in minimizing the transfer of pathogens to and from persons or equipment and the environment. Although the document discusses principles of cleaning and disinfection as they are applied to maintenance of environmental surfaces, the full discussion of sterilization and disinfection of medical instruments and direct patient-care devices is deferred for inclusion in the *Guideline for Disinfection and Sterilization in Health-Care Facilities*, a document currently under development. Similarly, the full discussion of hand hygiene is available as the *Guideline for Hand Hygiene in Health-Care Settings: Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force*. Where applicable, the *Guidelines for Environmental Infection Control in Health-Care Facilities* are consistent in content to the drafts available as of October 2002 of both the revised *Guideline for Prevention of Health-Care–Associated Pneumonia* and *Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities*.

This guideline was prepared by CDC staff members from NCID and the National Center for Chronic Disease Prevention and Health Promotion (NCCDPHP) and the designated HICPAC advisor. Contributors to this document reviewed predominantly English-language manuscripts identified from reference searches using the National Library of Medicine’s MEDLINE, bibliographies of published articles, and infection-control textbooks. Working drafts of the guideline were reviewed by CDC scientists, HICPAC committee members, and experts in infection control, engineering, internal medicine, infectious diseases, epidemiology, and microbiology. All recommendations in this guideline may not reflect the opinions of all reviewers.

Part I. Background Information: Environmental Infection Control in Health-Care Facilities

A. Introduction

The health-care environment contains a diverse population of microorganisms, but only a few are significant pathogens for susceptible humans. Microorganisms are present in great numbers in moist, organic environments, but some also can persist under dry conditions. Although pathogenic microorganisms can be detected in air and water and on fomites, assessing their role in causing infection and disease is difficult.¹¹ Only a few reports clearly delineate a “cause and effect” with respect to the environment and in particular, housekeeping surfaces.

Eight criteria are used to evaluate the strength of evidence for an environmental source or means of transmission of infectious agents (Box 2).^{11,12} Applying these criteria to disease investigations allows scientists to assess the contribution of the environment to disease transmission. An example of this application is the identification of a pathogen (e.g., vancomycin-resistant enterococci [VRE]) on an environmental surface during an outbreak. The presence of the pathogen does not establish its causal role; its transmission from source to host could be through indirect means (e.g., via hand transferral).¹¹ The surface, therefore, would be considered one of a number of potential reservoirs for the pathogen, but not the “de facto” source of exposure. An understanding of how infection occurs after exposure,

based on the principles of the “chain of infection,” is also important in evaluating the contribution of the environment to health-care–associated disease.¹³ All of the components of the “chain” must be operational for infection to occur (Box 3).

Box 2. Eight criteria for evaluating the strength of evidence for environmental sources of infection* +

1. **The organism can survive after inoculation onto the fomite.**
 2. **The organism can be cultured from in-use fomites.**
 3. **The organism can proliferate in or on the fomite.**
 4. **Some measure of acquisition of infection cannot be explained by other recognized modes of transmission.**
 5. **Retrospective case-control studies show an association between exposure to the fomite and infection.**
 6. **Prospective case-control studies may be possible when more than one similar type of fomite is in use.**
 7. **Prospective studies allocating exposure to the fomite to a subset of patients show an association between exposure and infection.**
 8. **Decontamination of the fomite results in the elimination of infection transmission.**
-

* These criteria are listed in order of strength of evidence.

+ Adapted from references 11 and 12.

Box 3. Chain of infection components*

1. **Adequate number of pathogenic organisms (dose)**
 2. **Pathogenic organisms of sufficient virulence**
 3. **A susceptible host**
 4. **An appropriate mode of transmission or transferal of the organism in sufficient number from source to host**
 5. **The correct portal of entry into the host**
-

* Adapted from reference 13.

The presence of the susceptible host is one of these components that underscores the importance of the health-care environment and opportunistic pathogens on fomites and in air and water. As a result of advances in medical technology and therapies (e.g., cytotoxic chemotherapy and transplantation medicine), more patients are becoming immunocompromised in the course of treatment and are therefore at increased risk for acquiring health-care–associated opportunistic infections. Trends in health-care delivery (e.g., early discharge of patients from acute care facilities) also are changing the distribution of patient populations and increasing the number of immunocompromised persons in non-acute-care hospitals. According to the American Hospital Association (AHA), in 1998, the number of hospitals in the United States totaled 6,021; these hospitals had a total of 1,013,000 beds,¹⁴ representing a 5.5% decrease in the number of acute-care facilities and a 10.2% decrease in the number of beds over the 5-year period 1994–1998.¹⁴ In addition, the total average daily number of patients receiving care in U.S. acute-care hospitals in 1998 was 662,000 (65.4%) – 36.5% less than the 1978 average of 1,042,000.¹⁴ As the number of acute-care hospitals declines, the length of stay in these facilities is concurrently decreasing, particularly for immunocompetent patients. Those patients remaining in acute-care facilities are likely to be those requiring extensive medical interventions who therefore at high risk for opportunistic infection.

The growing population of severely immunocompromised patients is at odds with demands on the health-care industry to remain viable in the marketplace; to incorporate modern equipment, new diagnostic procedures, and new treatments; and to construct new facilities. Increasing numbers of health-care facilities are likely to be faced with construction in the near future as hospitals consolidate to reduce costs, defer care to ambulatory centers and satellite clinics, and try to create more “home-like” acute-care settings. In 1998, approximately 75% of health-care–associated construction projects focused on renovation of existing outpatient facilities or the building of such facilities;¹⁵ the number of projects associated with outpatient health care rose by 17% from 1998 through 1999.¹⁶ An aging population is also creating increasing demand for assisted-living facilities and skilled nursing centers. Construction of assisted-living facilities in 1998 increased 49% from the previous year, with 138 projects completed at a cost of \$703 million.¹⁶ Overall, from 1998 to 1999, health-care–associated construction costs increased by 28.5%, from \$11.56 billion to \$14.86 billion.¹⁶

Environmental disturbances associated with construction activities near health-care facilities pose airborne and waterborne disease threats risks for the substantial number of patients who are at risk for health-care–associated opportunistic infections. The increasing age of hospitals and other health-care facilities is also generating ongoing need for repair and remediation work (e.g., installing wiring for new information systems, removing old sinks, and repairing elevator shafts) that can introduce or increase contamination of the air and water in patient-care environments. Aging equipment, deferred maintenance, and natural disasters provide additional mechanisms for the entry of environmental pathogens into high-risk patient-care areas.

Architects, engineers, construction contractors, environmental health scientists, and industrial hygienists historically have directed the design and function of hospitals’ physical plants. Increasingly, however, because of the growth in the number of susceptible patients and the increase in construction projects, the involvement of hospital epidemiologists and infection-control professionals is required. These experts help make plans for building, maintaining, and renovating health-care facilities to ensure that the adverse impact of the environment on the incidence of health-care–associated infections is minimal. The following are examples of adverse outcomes that could have been prevented had such experts been involved in the planning process: a) transmission of infections caused by *Mycobacterium tuberculosis*, varicella-zoster virus (VZV), and measles (i.e., rubeola) facilitated by inappropriate air-handling systems in health-care facilities;⁶ b) disease outbreaks caused by *Aspergillus* spp.,^{17–19} *Mucoraceae*,²⁰ and *Penicillium* spp. associated with the absence of environmental controls during periods of health-care facility-associated construction;²¹ c) infections and/or colonizations of patients and staff with vancomycin-resistant *Enterococcus faecium* [VRE] and *Clostridium difficile* acquired indirectly from contact with organisms present on environmental surfaces in health-care facilities,^{22–25} and d) outbreaks and pseudoepidemics of legionellae,^{26, 27} *Pseudomonas aeruginosa*,^{28–30} and the nontuberculous mycobacteria (NTM)^{31, 32} linked to water and aqueous solutions used in health-care facilities. The purpose of this guideline is to provide useful information for both health-care professionals and engineers in efforts to provide a safe environment in which quality health care may be provided to patients. The recommendations herein provide guidance to minimize the risk for and prevent transmission of pathogens in the indoor environment.

B. Key Terms Used in this Guideline

Although Appendix A provides definitions for terms discussed in Part I, several terms that pertain to specific patient-care areas and patients who are at risk for health-care–associated opportunistic infections are presented here. Specific engineering parameters for these care areas are discussed more

fully in the text. **Airborne Infection Isolation (AII)** refers to the isolation of patients infected with organisms spread via airborne droplet nuclei $<5\ \mu\text{m}$ in diameter. This isolation area receives numerous air changes per hour (ACH) (≥ 12 ACH for new construction as of 2001; ≥ 6 ACH for construction before 2001), and is under negative pressure, such that the direction of the airflow is from the outside adjacent space (e.g., corridor) into the room. The air in an AII room is preferably exhausted to the outside, but may be recirculated provided that the return air is filtered through a high efficiency particulate air (HEPA) filter. The use of personal respiratory protection is also indicated for persons entering these rooms.

Protective Environment (PE) is a specialized patient-care area, usually in a hospital, with a positive airflow relative to the corridor (i.e., air flows from the room to the outside adjacent space). The combination of HEPA filtration, high numbers of air changes per hour (≥ 12 ACH), and minimal leakage of air into the room creates an environment that can safely accommodate patients who have undergone allogeneic hematopoietic stem cell transplant (HSCT).

Immunocompromised patients are those patients whose immune mechanisms are deficient because of immunologic disorders (e.g., human immunodeficiency virus [HIV] infection, congenital immune deficiency syndrome, chronic diseases [such as diabetes, cancer, emphysema, and cardiac failure]) or immunosuppressive therapy (e.g., radiation, cytotoxic chemotherapy, anti-rejection medication, and steroids). Immunocompromised patients who are identified as **high-risk patients** have the greatest risk of infection caused by airborne or waterborne microorganisms. Patients in this subset include those who are severely neutropenic for prolonged periods of time (i.e., an absolute neutrophil count [ANC] of ≤ 500 cells/mL), allogeneic HSCT patients, and those who have received intensive chemotherapy (e.g., childhood acute myelogenous leukemia patients).

C. Air

1. Modes of Transmission of Airborne Diseases

A variety of airborne infections in susceptible hosts can result from exposures to clinically significant microorganisms released into the air when environmental reservoirs (i.e., soil, water, dust, and decaying organic matter) are disturbed. Once these materials are brought indoors into a health-care facility by any of a number of vehicles (e.g., people, air currents, water, construction materials, and equipment), the attendant microorganisms can proliferate in various indoor ecological niches and, if subsequently disbursed into the air, serve as a source for airborne health-care-associated infections.

Respiratory infections can be acquired from exposure to pathogens contained either in droplets or droplet nuclei. Exposure to microorganisms in droplets (e.g., through aerosolized oral and nasal secretions from infected patients³³) constitutes a form of direct contact transmission. When droplets are produced during a sneeze or cough, a cloud of infectious particles $>5\ \mu\text{m}$ in size is expelled, resulting in the potential exposure of susceptible persons within 3 feet of the source person.⁶ Examples of pathogens spread in this manner are influenza virus, rhinoviruses, adenoviruses, and respiratory syncytial virus (RSV). Because these agents primarily are transmitted directly and because the droplets tend to fall out of the air quickly, measures to control air flow in a health-care facility (e.g., use of negative pressure rooms) generally are not indicated for preventing the spread of diseases caused by these agents. Strategies to control the spread of these diseases are outlined in another guideline.³

The spread of airborne infectious diseases via droplet nuclei is a form of indirect transmission.³⁴ Droplet nuclei are the residuals of droplets that, when suspended in air, subsequently dry and produce

particles ranging in size from 1–5 μm . These particles can a) contain potentially viable microorganisms, b) be protected by a coat of dry secretions, c) remain suspended indefinitely in air, and d) be transported over long distances. The microorganisms in droplet nuclei persist in favorable conditions (e.g., a dry, cool atmosphere with little or no direct exposure to sunlight or other sources of radiation). Pathogenic microorganisms that can be spread via droplet nuclei include *Mycobacterium tuberculosis*, VZV, measles virus (i.e., rubeola), and smallpox virus (i.e., variola major).⁶ Several environmental pathogens have life-cycle forms that are similar in size to droplet nuclei and may exhibit similar behavior in the air. The spores of *Aspergillus fumigatus* have a diameter of 2–3.5 μm , with a settling velocity estimated at 0.03 cm/second (or about 1 meter/hour) in still air. With this enhanced buoyancy, the spores, which resist desiccation, can remain airborne indefinitely in air currents and travel far from their source.³⁵

2. Airborne Infectious Diseases in Health-Care Facilities

a. Aspergillosis and Other Fungal Diseases

Aspergillosis is caused by molds belonging to the genus *Aspergillus*. *Aspergillus* spp. are prototype health-care-acquired pathogens associated with dusty or moist environmental conditions. Clinical and epidemiologic aspects of aspergillosis (Table 1) are discussed extensively in another guideline.³

Table 1. Clinical and epidemiologic characteristics of aspergillosis

		References
Causative agents	<i>Aspergillus fumigatus</i> (90%–95% of <i>Aspergillus</i> infections among hematopoietic stem cell transplant (HSCT) patients; <i>A. flavus</i> , <i>A. niger</i> , <i>A. terreus</i> , <i>A. nidulans</i>)	36–43
Modes of transmission	Airborne transmission of fungal spores; direct inhalation; direct inoculation from environmental sources (rare)	37
Activities associated with infection	Construction, renovation, remodeling, repairs, building demolition; rare episodes associated with fomites	44–51
Clinical syndromes and diseases	Acute invasive: pneumonia; ulcerative tracheobronchitis; osteomyelitis; abscesses (aspergillomas) of the lungs, brain, liver, spleen, and kidneys; thrombosis of deep blood vessels; necrotizing skin ulcers; endophthalmitis; and sinusitis Chronic invasive: chronic pneumonitis Hypersensitivity: allergic bronchopulmonary aspergillosis Cutaneous: primary skin and burn-wound infections	44, 45, 52–58
Patient populations at greatest risk	Hematopoietic stem cell transplant patients (HSCT): immunocompromised patients (i.e., those with underlying disease), patients undergoing chemotherapy, organ transplant recipients, preterm neonates, hemodialysis patients, patients with identifiable immune system deficiencies who receive care in general intensive care units (ICUs), and cystic fibrosis patients (may be colonized, occasionally become infected)	36, 59–78
Factors affecting severity and outcomes	The immune status of the patient and the duration of severe neutropenia	79, 80
Occurrence	Rare and sporadic, but increasing as proportion of immunocompromised patients increases; 5% of HSCT patients infected, <5% of solid organ transplant recipients infected	36, 37, 81–88
Mortality rate	Rate can be as high as 100% if severe neutropenia persists; 13%–80% mortality among leukemia patients	58, 83, 89, 90

Aspergillus spp. are ubiquitous, aerobic fungi that occur in soil, water, and decaying vegetation; the organism also survives well in air, dust, and moisture present in health-care facilities.^{91–93} The presence of aspergilli in the health-care facility environment is a substantial extrinsic risk factor for opportunistic invasive aspergillosis (invasive aspergillosis being the most serious form of the disease).^{69, 94} Site renovation and construction can disturb *Aspergillus*-contaminated dust and produce bursts of airborne

fungal spores. Increased levels of atmospheric dust and fungal spores have been associated with clusters of health-care–acquired infections in immunocompromised patients.^{17, 20, 44, 47, 49, 50, 95–98}

Absorbent building materials (e.g., wallboard) serve as an ideal substrate for the proliferation of this organism if they become and remain wet, thereby increasing the numbers of fungal spores in the area. Patient-care items, devices, and equipment can become contaminated with *Aspergillus* spp. spores and serve as sources of infection if stored in such areas.⁵⁷

Most cases of aspergillosis are caused by *Aspergillus fumigatus*, a thermotolerant/thermophilic fungus capable of growing over a temperature range from 53.6°F–127.4°F (12°C–53°C); optimal growth occurs at approximately 104°F (40°C), a temperature inhibitory to most other saprophytic fungi.⁹⁹ It can use cellulose or sugars as carbon sources; because its respiratory process requires an ample supply of carbon, decomposing organic matter is an ideal substrate.

Other opportunistic fungi that have been occasionally linked with health-care–associated infections are members of the order *Mucorales* (e.g., *Rhizopus* spp.) and miscellaneous moniliaceous molds (e.g., *Fusarium* spp. and *Penicillium* spp.) (Table 2). Many of these fungi can proliferate in moist environments (e.g., water-damaged wood and building materials). Some fungi (e.g., *Fusarium* spp. and *Pseudoallescheria* spp.) also can be airborne pathogens.¹⁰⁰ As with aspergillosis, a major risk factor for disease caused by any of these pathogens is the host’s severe immunosuppression from either underlying disease or immunosuppressive therapy.^{101, 102}

Table 2. Environmental fungal pathogens: entry into and contamination of the health-care facility

Implicated environmental vehicle	References
<i>Aspergillus</i> spp.	
Improperly functioning ventilation systems	20, 46, 47, 97, 98, 103, 104
Air filters ^{*,†}	17, 18, 105–107
Air filter frames	17, 18
Window air conditioners	96
Backflow of contaminated air	107
Air exhaust contamination [†]	104
False ceilings	48, 57, 97, 108
Fibrous insulation and perforated metal ceilings	66
Acoustic ceiling tiles, plasterboard	18, 109
Fireproofing material	48, 49
Damp wood building materials	49
Opening doors to construction site	110
Construction	69
Open windows	20, 108, 111
Disposal conduit door	68
Hospital vacuum cleaner	68
Elevator	112
Arm boards	57
Walls	113
Unit kitchen	114
Food	21
Ornamental plants	21
<i>Mucorales</i> / <i>Rhizopus</i> spp.	
Air filter	20, 115
False ceilings	97
Heliport	115
<i>Scedosporium</i> spp.	
Construction	116

(Table 2. continued)

Implicated environmental vehicles	References
<i>Penicillium</i> spp.	
Rotting cabinet wood, pipe leak	21
Ventilation duct fiberglass insulation	112
Air filters	105
Topical anesthetic	117
<i>Acromonium</i> spp.	
Air filters	105
<i>Cladosporium</i> spp.	
Air filters	105
<i>Sporothrix</i>	
Construction (pseudoe epidemic)	118

- *. Pigeons, their droppings and roosts are associated with spread of *Aspergillus*, *Cryptococcus*, and *Histoplasma* spp. There have been at least three outbreaks linked to contamination of the filtering systems from bird droppings^{98, 103, 104} Pigeon mites may gain access into a health-care facility through the ventilation system.¹¹⁹
- +. The American Institute of Architects (AIA) standards stipulate that for new or renovated construction a) exhaust outlets are to be placed >25 feet from air intake systems, b) the bottom of outdoor air intakes for HVAC systems should be 6 feet above ground or 3 feet above roof level, and c) exhaust outlets from contaminated areas are situated above the roof level and arranged to minimize the recirculation of exhausted air back into the building.¹²⁰

Infections due *Cryptococcus neoformans*, *Histoplasma capsulatum*, or *Coccidioides immitis* can occur in health-care settings if nearby ground is disturbed and a malfunction of the facility's air-intake components allows these pathogens to enter the ventilation system. *C. neoformans* is a yeast usually 4–8 μm in size. However, viable particles of <2 μm diameter (and thus permissive to alveolar deposition) have been found in soil contaminated with bird droppings, particularly from pigeons.^{98, 103, 104, 121} *H. capsulatum*, with the infectious microconidia ranging in size from 2–5 μm , is endemic in the soil of the central river valleys of the United States. Substantial numbers of these infectious particles have been associated with chicken coops and the roosts of blackbirds.^{98, 103, 104, 122} Several outbreaks of histoplasmosis have been associated with disruption of the environment; construction activities in an endemic area may be a potential risk factor for health-care-acquired airborne infection.^{123, 124} *C. immitis*, with arthrospores of 3–5 μm diameter, has similar potential, especially in the endemic southwestern United States and during seasons of drought followed by heavy rainfall. After the 1994 earthquake centered near Northridge, California, the incidence of coccidioidomycosis in the surrounding area exceeded the historical norm.¹²⁵

Emerging evidence suggests that *Pneumocystis carinii*, now classified as a fungus, may be spread via airborne, person-to-person transmission.¹²⁶ Controlled studies in animals first demonstrated that *P. carinii* could be spread through the air.¹²⁷ More recent studies in health-care settings have detected nucleic acids of *P. carinii* in air samples from areas frequented or occupied by *P. carinii*-infected patients but not in control areas that are not occupied by these patients.^{128, 129} Clusters of cases have been identified among immunocompromised patients who had contact with a source patient and with each other. Recent studies have examined the presence of *P. carinii* DNA in oropharyngeal washings and the nares of infected patients, their direct contacts, and persons with no direct contact.^{130, 131} Molecular analysis of the DNA by polymerase chain reaction (PCR) provides evidence for airborne transmission of *P. carinii* from infected patients to direct contacts, but immunocompetent contacts tend to become transiently colonized rather than infected.¹³¹ The role of colonized persons in the spread of *P. carinii* pneumonia (PCP) remains to be determined. At present, specific modifications to ventilation systems to control spread of PCP in a health-care facility are not indicated. Current recommendations

outline isolation procedures to minimize or eliminate contact of immunocompromised patients not on PCP prophylaxis with PCP-infected patients.^{6, 132}

b. Tuberculosis and Other Bacterial Diseases

The bacterium most commonly associated with airborne transmission is *Mycobacterium tuberculosis*. A comprehensive review of the microbiology and epidemiology of *M. tuberculosis* and guidelines for tuberculosis (TB) infection control have been published.^{4, 133, 134} A summary of the clinical and epidemiologic information from these materials is provided in this guideline (Table 3).

Table 3. Clinical and epidemiologic characteristics of tuberculosis (TB)*

Causative agents	<i>Mycobacterium tuberculosis</i> , <i>M. bovis</i> , <i>M. africanum</i>
Mode of transmission	Airborne transmission via droplet nuclei 1–5 µm in diameter
Patient factors associated with infectivity and transmission	<ul style="list-style-type: none"> ▪ Disease of the lungs, airways, or larynx; presence of cough or other forceful expiratory measures ▪ Presence of acid-fast bacilli (AFB) in the sputum ▪ Failure of the patient to cover the mouth and nose when coughing or sneezing ▪ Presence of cavitation on chest radiograph ▪ Inappropriate or shortened duration of chemotherapy
Activities associated with infections	<ul style="list-style-type: none"> ▪ Exposures in relatively small, enclosed spaces ▪ Inadequate ventilation resulting in insufficient removal of droplet nuclei ▪ Cough-producing procedures done in areas without proper environmental controls ▪ Recirculation of air containing infectious droplet nuclei ▪ Failure to use respiratory protection when managing open lesions for patients with suspected extrapulmonary TB¹³⁵
Clinical syndromes and disease	Pulmonary TB ; extrapulmonary TB can affect any organ system or tissue; laryngeal TB is highly contagious
Populations at greatest risk	<ul style="list-style-type: none"> ▪ Immunocompromised persons (e.g., HIV-infected persons) ▪ Medically underserved persons, urban poor, homeless persons, elderly persons, migrant farm workers, close contacts of known patients ▪ Substance abusers, present and former prison inmates ▪ Foreign-born persons from areas with high prevalence of TB ▪ Health-care workers
Factors affecting severity and outcomes	<ul style="list-style-type: none"> ▪ Concentration of droplet nuclei in air, duration of exposure ▪ Age at infection ▪ Immunosuppression due to therapy or disease, underlying chronic medical conditions, history of malignancies or lesions of the lungs
Occurrence	Worldwide; incidence in the United States is 5.6 cases/100,000 population (2001) ¹³⁶
Mortality	930 deaths in the United States (1999) ¹³⁶
Chemoprophylaxis / treatment	Treatment of latent infection includes isoniazid (INH) or rifampin (RIF). ^{4, 134, 137–139} Directly observed therapy (DOT) for active cases as indicated: INH, RIF, pyrazinamide (PZA), ethambutol (EMB), streptomycin (SM) in various combinations determined by prevalent levels of specific resistance. ^{4, 134, 137–139} Consult therapy guidelines for specific treatment indications. ¹³⁹

* Material in this table is compiled from references 4, 133–141.

M. tuberculosis is carried by droplet nuclei generated when persons (primarily adults and adolescents) who have pulmonary or laryngeal TB sneeze, cough, speak, or sing;¹³⁹ normal air currents can keep these particles airborne for prolonged periods and spread them throughout a room or building.¹⁴² However, transmission of TB has occurred from mycobacteria aerosolized during provision of care (e.g., wound/lesion care or during handling of infectious peritoneal dialysis fluid) for extrapulmonary TB patients.^{135, 140}

Gram-positive cocci (i.e., *Staphylococcus aureus*, group A beta-hemolytic streptococci), also important health-care-associated pathogens, are resistant to inactivation by drying and can persist in the

environment and on environmental surfaces for extended periods. These organisms can be shed from heavily colonized persons and discharged into the air. Airborne dispersal of *S. aureus* is directly associated with the concentration of the bacterium in the anterior nares.¹⁴³ Approximately 10% of healthy carriers will disseminate *S. aureus* into the air, and some persons become more effective disseminators of *S. aureus* than others.^{144–148} The dispersal of *S. aureus* into air can be exacerbated by concurrent viral upper respiratory infection, thereby turning a carrier into a “cloud shedder.”¹⁴⁹ Outbreaks of surgical site infections (SSIs) caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients.^{150–153} In these situations, the strain causing the outbreak was recovered from the air in the operating room^{150, 151, 154} or on settle plates in a room in which the carrier exercised.^{151–153} *S. aureus* and group A streptococci have not been linked to airborne transmission outside of operating rooms, burn units, and neonatal nurseries.^{155, 156} Transmission of these agents occurs primarily via contact and droplets.

Other gram-positive bacteria linked to airborne transmission include *Bacillus* spp. which are capable of sporulation as environmental conditions become less favorable to support their growth. Outbreaks and pseudo-outbreaks have been attributed to *Bacillus cereus* in maternity, pediatric, intensive care, and bronchoscopy units; many of these episodes were secondary to environmental contamination.^{157–160}

Gram-negative bacteria rarely are associated with episodes of airborne transmission because they generally require moist environments for persistence and growth. The main exception is *Acinetobacter* spp., which can withstand the inactivating effects of drying. In one epidemiologic investigation of bloodstream infections among pediatric patients, identical *Acinetobacter* spp. were cultured from the patients, air, and room air conditioners in a nursery.¹⁶¹

Aerosols generated from showers and faucets may potentially contain legionellae and other gram-negative waterborne bacteria (e.g., *Pseudomonas aeruginosa*). Exposure to these organisms is through direct inhalation. However, because water is the source of the organisms and exposure occurs in the vicinity of the aerosol, the discussion of the diseases associated with such aerosols and the prevention measures used to curtail their spread is discussed in another section of the Guideline (see Part I: Water).

c. Airborne Viral Diseases

Some human viruses are transmitted from person to person via droplet aerosols, but very few viruses are consistently airborne in transmission (i.e., are routinely suspended in an infective state in air and capable of spreading great distances), and health-care–associated outbreaks of airborne viral disease are limited to a few agents. Consequently, infection-control measures used to prevent spread of these viral diseases in health-care facilities primarily involve patient isolation, vaccination of susceptible persons, and antiviral therapy as appropriate rather than measures to control air flow or quality.⁶ Infections caused by VZV frequently are described in health-care facilities. Health-care–associated airborne outbreaks of VZV infections from patients with primary infection and disseminated zoster have been documented; patients with localized zoster have, on rare occasions, also served as source patients for outbreaks in health-care facilities.^{162–166} VZV infection can be prevented by vaccination, although patients who develop a rash within 6 weeks of receiving varicella vaccine or who develop breakthrough varicella following exposure should be considered contagious.¹⁶⁷

Viruses whose major mode of transmission is via droplet contact rarely have caused clusters of infections in group settings through airborne routes. The factors facilitating airborne distribution of these viruses in an infective state are unknown, but a presumed requirement is a source patient in the early stage of infection who is shedding large numbers of viral particles into the air. Airborne transmission of measles has been documented in health-care facilities.^{168–171} In addition, institutional outbreaks of influenza virus infections have occurred predominantly in nursing homes,^{172–176} and less frequently in medical and neonatal intensive care units, chronic-care areas, HSCT units, and pediatric

wards.^{177–180} Some evidence supports airborne transmission of influenza viruses by droplet nuclei,^{181, 182} and case clusters in pediatric wards suggest that droplet nuclei may play a role in transmitting certain respiratory pathogens (e.g., adenoviruses and respiratory syncytial virus [RSV]).^{177, 183, 184} Some evidence also supports airborne transmission of enteric viruses. An outbreak of a Norwalk-like virus infection involving more than 600 staff personnel over a 3-week period was investigated in a Toronto, Ontario hospital in 1985; common sources (e.g., food and water) were ruled out during the investigation, leaving airborne spread as the most likely mode of transmission.¹⁸⁵

Smallpox virus, a potential agent of bioterrorism, is spread predominantly via direct contact with infectious droplets, but it also can be associated with airborne transmission.^{186, 187} A German hospital study from 1970 documented the ability of this virus to spread over considerable distances and cause infection at low doses in a well-vaccinated population; factors potentially facilitating transmission in this situation included a patient with cough and an extensive rash, indoor air with low relative humidity, and faulty ventilation patterns resulting from hospital design (e.g., open windows).¹⁸⁸ Smallpox patients with extensive rash are more likely to have lesions present on mucous membranes and therefore have greater potential to disseminate virus into the air.¹⁸⁸ In addition to the smallpox transmission in Germany, two cases of laboratory-acquired smallpox virus infection in the United Kingdom in 1978 also were thought to be caused by airborne transmission.¹⁸⁹

Airborne transmission may play a role in the natural spread of hantaviruses and certain hemorrhagic fever viruses (e.g., Ebola, Marburg, and Lassa), but evidence for airborne spread of these agents in health-care facilities is inconclusive.¹⁹⁰ Although hantaviruses can be transmitted when aerosolized from rodent excreta,^{191, 192} person-to-person spread of hantavirus infection from source patients has not occurred in health-care facilities.^{193–195} Nevertheless, health-care workers are advised to contain potentially infectious aerosols and wear National Institute of Occupational Safety and Health (NIOSH) approved respiratory protection when working with this agent in laboratories or autopsy suites.¹⁹⁶ Lassa virus transmission via aerosols has been demonstrated in the laboratory and incriminated in health-care-associated infections in Africa,^{197–199} but airborne spread of this agent in hospitals in developed nations likely is inefficient.^{200, 201} Yellow fever is considered to be a viral hemorrhagic fever agent with high aerosol infectivity potential, but health-care-associated transmission of this virus has not been described.²⁰² Viral hemorrhagic fever diseases primarily occur after direct exposure to infected blood and body fluids, and the use of standard and droplet precautions prevents transmission early in the course of these illnesses.^{203, 204} However, whether these viruses can persist in droplet nuclei that might remain after droplet production from coughs or vomiting in the latter stages of illness is unknown.²⁰⁵ Although the use of a negative-pressure room is not required during the early stages of illness, its use might be prudent at the time of hospitalization to avoid the need for subsequent patient transfer. Current CDC guidelines recommend negative-pressure rooms with anterooms for patients with hemorrhagic fever and use of HEPA respirators by persons entering these rooms when the patient has prominent cough, vomiting, diarrhea, or hemorrhage.^{6, 203} Face shields or goggles will help to prevent mucous-membrane exposure to potentially-aerosolized infectious material in these situations. If an anteroom is not available, portable, industrial-grade high efficiency particulate air (HEPA) filter units can be used to provide the equivalent of additional air changes per hour (ACH).

Table 4. Microorganisms associated with airborne transmission*

	Fungi	Bacteria	Viruses
Numerous reports in health-care facilities	<i>Aspergillus</i> spp.+ <i>Mucorales (Rhizopus</i> spp.) ^{97, 115}	<i>Mycobacterium tuberculosis</i> +	Measles (rubeola) virus ¹⁶⁸⁻¹⁷⁰ Varicella-zoster virus ¹⁶²⁻¹⁶⁶
Atypical, occasional reports	<i>Acremonium</i> spp. ^{105, 206} <i>Fusarium</i> spp. ¹⁰² <i>Pseudoallescheria boydii</i> ¹⁰⁰ <i>Scedosporium</i> spp. ¹¹⁶ <i>Sporothrix cyanescens</i> ¶ ¹¹⁸	<i>Acinetobacter</i> spp. ¹⁶¹ <i>Bacillus</i> spp.¶ ^{160, 207} <i>Brucella</i> spp.** ²⁰⁸⁻²¹¹ <i>Staphylococcus aureus</i> ^{148, 156} Group A <i>Streptococcus</i> ¹⁵¹	Smallpox virus (variola)§ ^{188, 189} Influenza viruses ^{181, 182} Respiratory syncytial virus ¹⁸³ Adenoviruses ¹⁸⁴ Norwalk-like virus ¹⁸⁵
Airborne in nature; airborne transmission in health care settings not described	<i>Coccidioides immitis</i> ¹²⁵ <i>Cryptococcus</i> spp. ¹²¹ <i>Histoplasma capsulatum</i> ¹²⁴	<i>Coxiella burnetii</i> (Q fever) ²¹²	Hantaviruses ^{193, 195} Lassa virus ²⁰⁵ Marburg virus ²⁰⁵ Ebola virus ²⁰⁵ Crimean-Congo virus ²⁰⁵
Under investigation	<i>Pneumocystis carinii</i> ¹³¹	—	—

* This list excludes microorganisms transmitted from aerosols derived from water.

+ Refer to the text for references for these disease agents.

§ Airborne transmission of smallpox is infrequent. Potential for airborne transmission increases with patients who are effective disseminators present in facilities with low relative humidity in the air and faulty ventilation.

¶ Documentation of pseudoepidemic during construction.

** Airborne transmission documented in the laboratory but not in patient-care areas

3. Heating, Ventilation, and Air Conditioning Systems in Health-Care Facilities

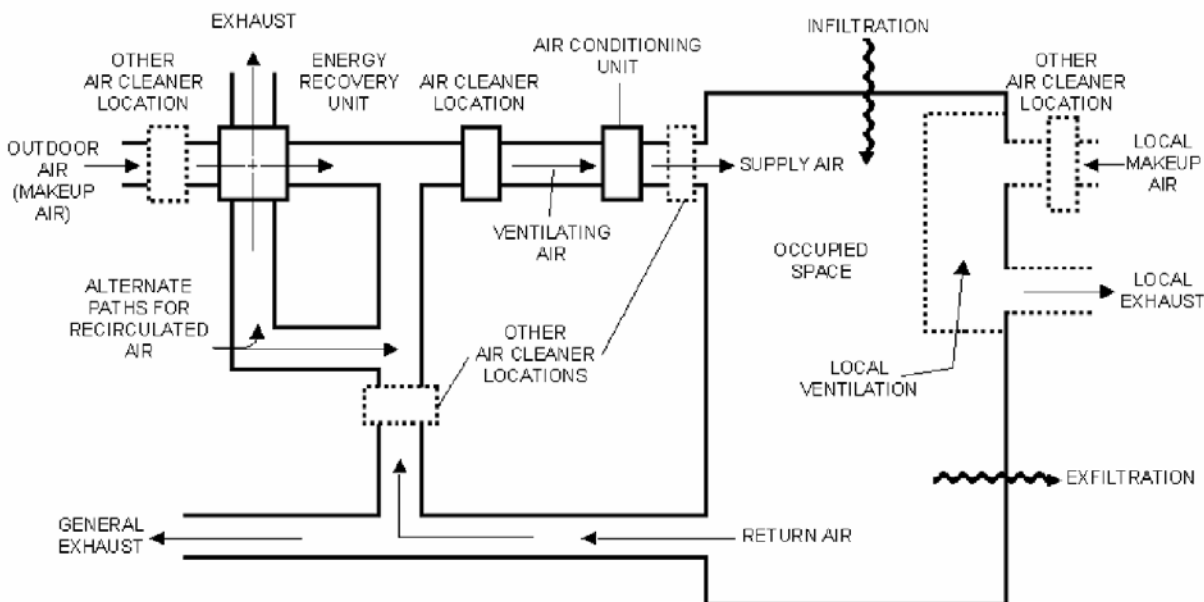
a. Basic Components and Operations

Heating, ventilation, and air conditioning (HVAC) systems in health-care facilities are designed to a) maintain the indoor air temperature and humidity at comfortable levels for staff, patients, and visitors; b) control odors; c) remove contaminated air; d) facilitate air-handling requirements to protect susceptible staff and patients from airborne health-care-associated pathogens; and e) minimize the risk for transmission of airborne pathogens from infected patients.^{35, 120} An HVAC system includes an outside air inlet or intake; filters; humidity modification mechanisms (i.e., humidity control in summer, humidification in winter); heating and cooling equipment; fans; ductwork; air exhaust or out-takes; and registers, diffusers, or grilles for proper distribution of the air (Figure 1).^{213, 214} Decreased performance of healthcare facility HVAC systems, filter inefficiencies, improper installation, and poor maintenance can contribute to the spread of health-care-associated airborne infections.

The American Institute of Architects (AIA) has published guidelines for the design and construction of new health-care facilities and for renovation of existing facilities. These AIA guidelines address indoor air-quality standards (e.g., ventilation rates, temperature levels, humidity levels, pressure relationships, and minimum air changes per hour [ACH]) specific to each zone or area in health-care facilities (e.g., operating rooms, laboratories, diagnostic areas, patient-care areas, and support departments).¹²⁰ These guidelines represent a consensus document among authorities having jurisdiction (AHJ), governmental regulatory agencies (i.e., Department of Health and Human Services [DHHS]; Department of Labor, Occupational Safety and Health Administration [OSHA]), health-care professionals, professional organizations (e.g., American Society of Heating, Refrigeration, and Air-Conditioning Engineers [ASHRAE], American Society for Healthcare Engineering [ASHE]), and accrediting organizations (i.e., Joint Commission on Accreditation of Healthcare Organizations [JCAHO]). More than 40 state agencies that license health-care facilities have either incorporated or adopted by reference these

guidelines into their state standards. JCAHO, through its surveys, ensures that facilities are in compliance with the ventilation guidelines of this standard for new construction and renovation.

Figure 1. Diagram of a ventilation system*



Outdoor air and recirculated air pass through air cleaners (e.g., filter banks) designed to reduce the concentration of airborne contaminants. Air is conditioned for temperature and humidity before it enters the occupied space as supply air. Infiltration is air leakage inward through cracks and interstitial spaces of walls, floors, and ceilings. Exfiltration is air leakage outward through these same cracks and spaces. Return air is largely exhausted from the system, but a portion is recirculated with fresh, incoming air.

* Used with permission of the publisher of reference 214 (ASHRAE)

Engineering controls to contain or prevent the spread of airborne contaminants center on a) local exhaust ventilation [i.e., source control], b) general ventilation, and c) air cleaning.⁴ General ventilation encompasses a) dilution and removal of contaminants via well-mixed air distribution of filtered air, b) directing contaminants toward exhaust registers and grilles via uniform, non-mixed airflow patterns, c) pressurization of individual spaces relative to all other spaces, and d) pressurization of buildings relative to the outdoors and other attached buildings.

A centralized HVAC system operates as follows. Outdoor air enters the system, where low-efficiency or “roughing” filters remove large particulate matter and many microorganisms. The air enters the distribution system for conditioning to appropriate temperature and humidity levels, passes through an additional bank of filters for further cleaning, and is delivered to each zone of the building. After the conditioned air is distributed to the designated space, it is withdrawn through a return duct system and delivered back to the HVAC unit. A portion of this “return air” is exhausted to the outside while the remainder is mixed with outdoor air for dilution and filtered for removal of contaminants.²¹⁵ Air from toilet rooms or other soiled areas is usually exhausted directly to the atmosphere through a separate duct exhaust system. Air from rooms housing tuberculosis patients is exhausted to the outside if possible, or passed through a HEPA filter before recirculation. Ultraviolet germicidal irradiation (UVGI) can be used as an adjunct air-cleaning measure, but it cannot replace HEPA filtration.

b. Filtration

i. Filter Types and Methods of Filtration

Filtration, the physical removal of particulates from air, is the first step in achieving acceptable indoor air quality. Filtration is the primary means of cleaning the air. Five methods of filtration can be used (Table 5). During filtration, outdoor air passes through two filter beds or banks (with efficiencies of 20%–40% and $\geq 90\%$, respectively) for effective removal of particles 1–5 μm in diameter.^{35, 120} The low-to-medium efficiency filters in the first bank have low resistance to airflow, but this feature allows some small particulates to pass onto heating and air conditioning coils and into the indoor environment.³⁵ Incoming air is mixed with recirculated air and reconditioned for temperature and humidity before being filtered by the second bank of filters. The performance of filters with $\leq 90\%$ efficiency is measured using either the dust-spot test or the weight-arrestance test.^{35, 216}

Table 5. Filtration methods*

Basic method	Principle of performance	Filtering efficiency
Straining	Particles in the air are larger than the openings between the filter fibers, resulting in gross removal of large particles.	Low
Impingement	Particles collide with filter fibers and remain attached to the filter. Fibers may be coated with adhesive.	Low
Interception	Particles enter into the filter and become entrapped and attached to the filter fibers.	Medium
Diffusion	Small particles, moving in erratic motion, collide with filter fibers and remain attached.	High
Electrostatic	Particles bearing negative electrostatic charge are attracted to the filter with positively charged fibers.	High

* Material in this table was compiled from information in reference 217.

The second filter bank usually consists of high-efficiency filters. This filtration system is adequate for most patient-care areas in ambulatory-care facilities and hospitals, including the operating room environment and areas providing central services.¹²⁰ Nursing facilities use 90% dust-spot efficient filters as the second bank of filters,¹²⁰ whereas a HEPA filter bank may be indicated for special-care areas of hospitals. HEPA filters are at least 99.97% efficient for removing particles $\geq 0.3 \mu\text{m}$ in diameter. (As a reference, *Aspergillus* spores are 2.5–3.0 μm in diameter.) Examples of care areas where HEPA filters are used include PE rooms and those operating rooms designated for orthopedic implant procedures.³⁵

Maintenance costs associated with HEPA filters are high compared with other types of filters, but use of in-line disposable prefilters can increase the life of a HEPA filter by approximately 25%. Alternatively, if a disposable prefilter is followed by a filter that is 90% efficient, the life of the HEPA filter can be extended ninefold. This concept, called progressive filtration, allows HEPA filters in special care areas to be used for 10 years.²¹³ Although progressive filtering will extend the mechanical ability of the HEPA filter, these filters may absorb chemicals in the environment and later desorb those chemicals, thereby necessitating a more frequent replacement program. HEPA filter efficiency is monitored with the dioctylphthalate (DOP) particle test using particles that are 0.3 μm in diameter.²¹⁸

HEPA filters are usually framed with metal, although some older versions have wood frames. A metal frame has no advantage over a properly fitted wood frame with respect to performance, but wood can compromise the air quality if it becomes and remains wet, allowing the growth of fungi and bacteria. Hospitals are therefore advised to phase out water-damaged or spent wood-framed filter units and replace them with metal-framed HEPA filters.

HEPA filters are usually fixed into the HVAC system; however, portable, industrial grade HEPA units are available that can filter air at the rate of 300–800 ft³/min. Portable HEPA filters are used to a) temporarily recirculate air in rooms with no general ventilation, b) augment systems that cannot provide adequate airflow, and c) provide increased effectiveness in airflow.⁴ Portable HEPA units are useful engineering controls that help clean the air when the central HVAC system is undergoing repairs,²¹⁹ but these units do not satisfy fresh-air requirements.²¹⁴ The effectiveness of the portable unit for particle removal is dependent on a) the configuration of the room, b) the furniture and persons in the room, c) the placement of the units relative to the contents and layout of the room, and d) the location of the supply and exhaust registers or grilles. If portable, industrial-grade units are used, they should be capable of recirculating all or nearly all of the room air through the HEPA filter, and the unit should be designed to achieve the equivalent of ≥ 12 ACH.⁴ (An average room has approximately 1,600 ft³ of airspace.) The hospital engineering department should be contacted to provide ACH information in the event that a portable HEPA filter unit is necessary to augment the existing fixed HVAC system for air cleaning.

ii. Filter Maintenance

Efficiency of the filtration system is dependent on the density of the filters, which can create a drop in pressure unless compensated by stronger and more efficient fans, thus maintaining air flow. For optimal performance, filters require monitoring and replacement in accordance with the manufacturer's recommendations and standard preventive maintenance practices.²²⁰ Upon removal, spent filters can be bagged and discarded with the routine solid waste, regardless of their patient-care area location.²²¹ Excess accumulation of dust and particulates increases filter efficiency, requiring more pressure to push the air through. The pressure differential across filters is measured by use of manometers or other gauges. A pressure reading that exceeds specifications indicates the need to change the filter. Filters also require regular inspection for other potential causes of decreased performance. Gaps in and around filter banks and heavy soil and debris upstream of poorly maintained filters have been implicated in health-care-associated outbreaks of aspergillosis, especially when accompanied by construction activities at the facility.^{17, 18, 106, 222}

c. Ultraviolet Germicidal Irradiation (UVGI)

As a supplemental air-cleaning measure, UVGI is effective in reducing the transmission of airborne bacterial and viral infections in hospitals, military housing, and classrooms, but it has only a minimal inactivating effect on fungal spores.^{223–228} UVGI is also used in air handling units to prevent or limit the growth of vegetative bacteria and fungi. Most commercially available UV lamps used for germicidal purposes are low-pressure mercury vapor lamps that emit radiant energy predominantly at a wave-length of 253.7 nm.^{229, 230} Two systems of UVGI have been used in health-care settings – duct irradiation and upper-room air irradiation. In duct irradiation systems, UV lamps are placed inside ducts that remove air from rooms to disinfect the air before it is recirculated. When properly designed, installed, and maintained, high levels of UVGI can be attained in the ducts with little or no exposure of persons in the rooms.^{231, 232} In upper-room air irradiation, UV lamps are either suspended from the ceiling or mounted on the wall.⁴ Upper air UVGI units have two basic designs: a) a “pan” fixture with UVGI unshielded above the unit to direct the irradiation upward and b) a fixture with a series of parallel plates to columnize the irradiation outward while preventing the light from getting to the eyes of the room's occupants. The germicidal effect is dependent on air mixing via convection between the room's irradiated upper zone and the lower patient-care zones.^{233, 234}

Bacterial inactivation studies using BCG mycobacteria and *Serratia marcescens* have estimated the effect of UVGI as equivalent to 10 ACH–39 ACH.^{235, 236} Another study, however, suggests that UVGI may result in fewer equivalent ACH in the patient-care zone, especially if the mixing of air between zones is insufficient.²³⁴ The use of fans or HVAC systems to generate air movement may increase the effectiveness of UVGI if airborne microorganisms are exposed to the light energy for a sufficient length

of time.^{233, 235, 237–239} The optimal relationship between ventilation and UVGI is not known.

Because the clinical effectiveness of UV systems may vary, UVGI is not recommended for air management prior to air recirculation from airborne isolation rooms. It is also not recommended as a substitute for HEPA filtration, local exhaust of air to the outside, or negative pressure.⁴ The use of UV lamps and HEPA filtration in a single unit offers only minimal infection-control benefits over those provided by the use of a HEPA filter alone.²⁴⁰ Duct systems with UVGI are not recommended as a substitute for HEPA filters if the air from isolation rooms must be recirculated to other areas of the facility.⁴ Regular maintenance of UVGI systems is crucial and usually consists of keeping the bulbs free of dust and replacing old bulbs as necessary. Safety issues associated with the use of UVGI systems are described in other guidelines.⁴

d. Conditioned Air in Occupied Spaces

Temperature and humidity are two essential components of conditioned air. After outside air passes through a low- or medium-efficiency filter, the air undergoes conditioning for temperature and humidity control before it passes through high-efficiency or HEPA filtration.

i. Temperature

HVAC systems in health-care facilities are often single-duct or dual-duct systems.^{35, 241} A single-duct system distributes cooled air (55°F [12.8°C]) throughout the building and uses thermostatically controlled reheat boxes located in the terminal ductwork to warm the air for individual or multiple rooms. The dual-duct system consists of parallel ducts, one with a cold air stream and the other with a hot air stream. A mixing box in each room or group of rooms mixes the two air streams to achieve the desired temperature. Temperature standards are given as either a single temperature or a range, depending on the specific health-care zone. Cool temperature standards (68°F–73°F [20°C–23°C]) usually are associated with operating rooms, clean workrooms, and endoscopy suites.¹²⁰ A warmer temperature (75°F [24°C]) is needed in areas requiring greater degrees of patient comfort. Most other zones use a temperature range of 70°F–75°F (21°C–24°C).¹²⁰ Temperatures outside of these ranges may be needed occasionally in limited areas depending on individual circumstances during patient care (e.g., cooler temperatures in operating rooms during specialized operations).

ii. Humidity

Four measures of humidity are used to quantify different physical properties of the mixture of water vapor and air. The most common of these is relative humidity, which is the ratio of the amount of water vapor in the air to the amount of water vapor air can hold at that temperature.²⁴² The other measures of humidity are specific humidity, dew point, and vapor pressure.²⁴²

Relative humidity measures the percentage of saturation. At 100% relative humidity, the air is saturated. For most areas within health-care facilities, the designated comfort range is 30%–60% relative humidity.^{120, 214} Relative humidity levels >60%, in addition to being perceived as uncomfortable, promote fungal growth.²⁴³ Humidity levels can be manipulated by either of two mechanisms.²⁴⁴ In a water-wash unit, water is sprayed and drops are taken up by the filtered air; additional heating or cooling of this air sets the humidity levels. The second mechanism is by means of water vapor created from steam and added to filtered air in humidifying boxes. Reservoir-type humidifiers are not allowed in health-care facilities as per AIA guidelines and many state codes.¹²⁰ Cool-mist humidifiers should be avoided, because they can disseminate aerosols containing allergens and microorganisms.²⁴⁵ Additionally, the small, personal-use versions of this equipment can be difficult to clean.

iii. Ventilation

The control of air pollutants (e.g., microorganisms, dust, chemicals, and smoke) at the source is the most effective way to maintain clean air. The second most effective means of controlling indoor air pollution is through ventilation. Ventilation rates are voluntary unless a state or local government specifies a standard in health-care licensing or health department requirements. These standards typically apply to only the design of a facility, rather than its operation.^{220, 246} Health-care facilities without specific ventilation standards should follow the AIA guideline specific to the year in which the building was built or the ANSI/ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*.^{120, 214, 241}

Ventilation guidelines are defined in terms of air volume per minute per occupant and are based on the assumption that occupants and their activities are responsible for most of the contaminants in the conditioned space.²¹⁵ Most ventilation rates for health-care facilities are expressed as room ACH. Peak efficiency for particle removal in the air space occurs between 12 ACH–15 ACH.^{35, 247, 248} Ventilation rates vary among the different patient-care areas of a health-care facility (Appendix B).¹²⁰

Health-care facilities generally use recirculated air.^{35, 120, 241, 249, 250} Fans create sufficient positive pressure to force air through the building duct work and adequate negative pressure to evacuate air from the conditioned space into the return duct work and/or exhaust, thereby completing the circuit in a sealed system (Figure 1). However, because gaseous contaminants tend to accumulate as the air recirculates, a percentage of the recirculated air is exhausted to the outside and replaced by fresh outdoor air. In hospitals, the delivery of filtered air to an occupied space is an engineered system design issue, the full discussion of which is beyond the scope of this document.

Hospitals with areas not served by central HVAC systems often use through-the-wall or fan coil air conditioning units as the sole source of room ventilation. AIA guidelines for newly installed systems stipulate that through-the-wall fan-coil units be equipped with permanent (i.e., cleanable) or replaceable filters with a minimum efficiency of 68% weight arrestance.¹²⁰ These units may be used only as recirculating units; all outdoor air requirements must be met by a separate central air handling system with proper filtration, with a minimum of two outside air changes in general patient rooms (D. Erickson, ASHE, 2000).¹²⁰ If a patient room is equipped with an individual through-the-wall fan coil unit, the room should not be used as either AII or as PE.¹²⁰ These requirements, although directed to new HVAC installations also are appropriate for existing settings. Non-central air-handling systems are prone to problems associated with excess condensation accumulating in drip pans and improper filter maintenance; health-care facilities should clean or replace the filters in these units on a regular basis while the patient is out of the room.

Laminar airflow ventilation systems are designed to move air in a single pass, usually through a bank of HEPA filters either along a wall or in the ceiling, in a one-way direction through a clean zone with parallel streamlines. Laminar airflow can be directed vertically or horizontally; the unidirectional system optimizes airflow and minimizes air turbulence.^{63, 241} Delivery of air at a rate of 0.5 meters per second (90 ± 20 ft/min) helps to minimize opportunities for microorganism proliferation.^{63, 251, 252} Laminar airflow systems have been used in PE to help reduce the risk for health-care-associated airborne infections (e.g., aspergillosis) in high-risk patients.^{63, 93, 253, 254} However, data that demonstrate a survival benefit for patients in PE with laminar airflow are lacking. Given the high cost of installation and apparent lack of benefit, the value of laminar airflow in this setting is questionable.^{9, 37} Few data support the use of laminar airflow systems elsewhere in a hospital.²⁵⁵

iv. Pressurization

Positive and negative pressures refer to a pressure differential between two adjacent air spaces (e.g., rooms and hallways). Air flows away from areas or rooms with positive pressure (pressurized), while

air flows into areas with negative pressure (depressurized). All rooms are set at negative pressure to prevent airborne microorganisms in the room from entering hallways and corridors. PE rooms housing severely neutropenic patients are set at positive pressure to keep airborne pathogens in adjacent spaces or corridors from coming into and contaminating the airspace occupied by such high-risk patients. Self-closing doors are mandatory for both of these areas to help maintain the correct pressure differential.^{4,6,120} Older health-care facilities may have variable pressure rooms (i.e., rooms in which the ventilation can be manually switched between positive and negative pressure). These rooms are no longer permitted in the construction of new facilities or in renovated areas of the facility,¹²⁰ and their use in existing facilities has been discouraged because of difficulties in assuring the proper pressure differential, especially for the negative pressure setting, and because of the potential for error associated with switching the pressure differentials for the room. Continued use of existing variable pressure rooms depends on a partnership between engineering and infection control. Both positive- and negative-pressure rooms should be maintained according to specific engineering specifications (Table 6).

Table 6. Engineered specifications for positive- and negative pressure rooms*

	Positive pressure areas (e.g., protective environments [PE])	Negative pressure areas (e.g., airborne infection isolation [AII])
Pressure differentials	> +2.5 Pa§ (0.01" water gauge)	> -2.5 Pa (0.01" water gauge)
Air changes per hour (ACH)	>12	≥12 (for renovation or new construction)
Filtration efficiency	Supply: 99.97% @ 0.3 µm DOP¶ Return: none required**	Supply: 90% (dust spot test) Return: 99.97% @ 0.3 µm DOP¶ †
Room airflow direction	Out to the adjacent area	In to the room
Clean-to-dirty airflow in room	Away from the patient (high-risk patient, immunosuppressed patient)	Towards the patient (airborne disease patient)
Ideal pressure differential	> + 8 Pa	> - 2.5 Pa

* Material in this table was compiled from references 35 and 120. Table adapted from and used with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

§ Pa is the abbreviation for Pascal, a metric unit of measurement for pressure based on air velocity; 250 Pa equals 1.0 inch water gauge.

¶ DOP is the abbreviation for dioctylphthalate particles of 0.3 µm diameter.

** If the patient requires both PE and AII, return air should be HEPA-filtered or otherwise exhausted to the outside.

† HEPA filtration of exhaust air from AII rooms should not be required, providing that the exhaust is properly located to prevent re-entry into the building.

Health-care professionals (e.g., infection control, hospital epidemiologists) must perform a risk assessment to determine the appropriate number of AII rooms (negative pressure) and/or PE rooms (positive pressure) to serve the patient population. The AIA guidelines require a certain number of AII rooms as a minimum, and it is important to refer to the edition under which the building was built for appropriate guidance.¹²⁰

In large health-care facilities with central HVAC systems, sealed windows help to ensure the efficient operation of the system, especially with respect to creating and maintaining pressure differentials. Sealing the windows in PE areas helps minimize the risk of airborne contamination from the outside. One outbreak of aspergillosis among immunosuppressed patients in a hospital was attributed in part to an open window in the unit during a time when both construction and a fire happened nearby; sealing the window prevented further entry of fungal spores into the unit from the outside air.¹¹¹ Additionally, all emergency exits (e.g., fire escapes and emergency doors) in PE wards should be kept closed (except during emergencies) and equipped with alarms.

e. Infection Control Impact of HVAC System Maintenance and Repair

A failure or malfunction of any component of the HVAC system may subject patients and staff to discomfort and exposure to airborne contaminants. Only limited information is available from formal

studies on the infection-control implications of a complete air-handling system failure or shutdown for maintenance. Most experience has been derived from infectious disease outbreaks and adverse outcomes among high-risk patients when HVAC systems are poorly maintained. (See Table 7 for potential ventilation hazards, consequences, and correction measures.)

AIA guidelines prohibit U.S. hospitals and surgical centers from shutting down their HVAC systems for purposes other than required maintenance, filter changes, and construction.¹²⁰ Airflow can be reduced; however, sufficient supply, return, and exhaust must be provided to maintain required pressure relationships when the space is not occupied. Maintaining these relationships can be accomplished with special drives on the air-handling units (i.e., a variable air ventilation [VAV] system).

Microorganisms proliferate in environments wherever air, dust, and water are present, and air-handling systems can be ideal environments for microbial growth.³⁵ Properly engineered HVAC systems require routine maintenance and monitoring to provide acceptable indoor air quality efficiently and to minimize conditions that favor the proliferation of health-care-associated pathogens.^{35, 249} Performance monitoring of the system includes determining pressure differentials across filters, regular inspection of system filters, DOP testing of HEPA filters, testing of low- or medium efficiency filters, and manometer tests for positive- and negative-pressure areas in accordance with nationally recognized standards, guidelines, and manufacturers' recommendations. The use of hand-held, calibrated equipment that can provide a numerical reading on a daily basis is preferred for engineering purposes (A. Streifel, University of Minnesota, 2000).²⁵⁶ Several methods that provide a visual, qualitative measure of pressure differentials (i.e., airflow direction) include smoke-tube tests or placing flutter strips, ping-pong balls, or tissue in the air stream.

Preventive filter and duct maintenance (e.g., cleaning ductwork vents, replacing filters as needed, and properly disposing spent filters into plastic bags immediately upon removal) is important to prevent potential exposures of patients and staff during HVAC system shut-down. The frequency of filter inspection and the parameters of this inspection are established by each facility to meet their unique needs. Ductwork in older health-care facilities may have insulation on the interior surfaces that can trap contaminants. This insulation material tends to break down over time to be discharged from the HVAC system. Additionally, a malfunction of the air-intake system can overburden the filtering system and permit aerosolization of fungal pathogens. Keeping the intakes free from bird droppings, especially those from pigeons, helps to minimize the concentration of fungal spores entering from the outside.⁹⁸

Accumulation of dust and moisture within HVAC systems increases the risk for spread of health-care-associated environmental fungi and bacteria. Clusters of infections caused by *Aspergillus* spp., *P. aeruginosa*, *S. aureus*, and *Acinetobacter* spp. have been linked to poorly maintained and/or malfunctioning air conditioning systems.^{68, 161, 257, 258} Efforts to limit excess humidity and moisture in the infrastructure and on air-stream surfaces in the HVAC system can minimize the proliferation and dispersion of fungal spores and waterborne bacteria throughout indoor air.²⁵⁹⁻²⁶² Within the HVAC system, water is present in water-wash units, humidifying boxes, or cooling units. The dual-duct system may also create conditions of high humidity and excess moisture that favor fungal growth in drain pans as well as in fibrous insulation material that becomes damp as a result of the humid air passing over the hot stream and condensing.

If moisture is present in the HVAC system, periods of stagnation should be avoided. Bursts of organisms can be released upon system start-up, increasing the risk of airborne infection.²⁰⁶ Proper engineering of the HVAC system is critical to preventing dispersal of airborne organisms. In one hospital, endophthalmitis caused by *Acremonium kiliense* infection following cataract extraction in an ambulatory surgical center was traced to aerosols derived from the humidifier water in the ventilation system.²⁰⁶ The organism proliferated because the ventilation system was turned off routinely when the

center was not in operation; the air was filtered before humidification, but not afterwards.

Most health-care facilities have contingency plans in case of disruption of HVAC services. These plans include back-up power generators that maintain the ventilation system in high-risk areas (e.g., operating rooms, intensive-care units, negative- and positive-pressure rooms, transplantation units, and oncology units). Alternative generators are required to engage within 10 seconds of a loss of main power. If the ventilation system is out of service, rendering indoor air stagnant, sufficient time must be allowed to clean the air and re-establish the appropriate number of ACH once the HVAC system begins to function again. Air filters may also need to be changed, because reactivation of the system can dislodge substantial amounts of dust and create a transient burst of fungal spores.

Duct cleaning in health-care facilities has benefits in terms of system performance, but its usefulness for infection control has not been conclusively determined. Duct cleaning typically involves using specialized tools to dislodge dirt and a high-powered vacuum cleaner to clean out debris.²⁶³ Some duct-cleaning services also apply chemical biocides or sealants to the inside surfaces of ducts to minimize fungal growth and prevent the release of particulate matter. The U.S. Environmental Protection Agency (EPA), however, has concerns with the use of sanitizers and/or disinfectants to treat the surfaces of ductwork, because the label indications for most of these products may not specifically include the use of the product in HVAC systems.²⁶⁴ Further, EPA has not evaluated the potency of disinfectants in such applications, nor has the agency examined the potential attendant health and safety risks. The EPA recommends that companies use only those chemical biocides that are registered for use in HVAC systems.²⁶⁴ Although infrequent cleaning of the exhaust ducts in AII areas has been documented as a cause of diminishing negative pressure and a decrease in the air exchange rates,²¹⁴ no data indicate that duct cleaning, beyond what is recommended for optimal performance, improves indoor air quality or reduces the risk of infection. Exhaust return systems should be cleaned as part of routine system maintenance. Duct cleaning has not been shown to prevent any health problems,²⁶⁵ and EPA studies indicate that airborne particulate levels do not increase as a result of dirty air ducts, nor do they diminish after cleaning, presumably because much of the dirt inside air ducts adheres to duct surfaces and does not enter the conditioned space.²⁶⁵ Additional research is needed to determine if air-duct contamination can significantly increase the airborne infection risk in general areas of health-care facilities.

4. Construction, Renovation, Remediation, Repair, and Demolition

a. General Information

Environmental disturbances caused by construction and/or renovation and repair activities (e.g., disruption of the above-ceiling area, running cables through the ceiling, and structural repairs) in and near health-care facilities markedly increase the airborne *Aspergillus* spp. spore counts in the indoor air of such facilities, thereby increasing the risk for health-care-associated aspergillosis among high-risk patients. Although one case of health-care-associated aspergillosis is often difficult to link to a specific environmental exposure, the occurrence of temporarily clustered cases increase the likelihood that an environmental source within the facility may be identified and corrected.

Table 7. Ventilation hazards in health-care facilities that may be associated with increased potential of airborne disease transmission*

Problem§	Consequences	Possible solutions
Water-damaged building materials (18, 266)	Water leaks can soak wood, wall board, insulation, wall coverings, ceiling tiles, and carpeting. All of these materials can provide microbial habitat when wet. This is especially true for fungi growing on gypsum board.	<ol style="list-style-type: none"> 1. Replace water-damaged materials. 2. Incorporate fungistatic compounds into building materials in areas at risk for moisture problems. 3. Test for all moisture and dry in less than 72 hours. Replace if the material cannot dry within 72 hours.
Filter bypasses (17)	Rigorous air filtration requires air flow resistance. Air stream will elude filtration if openings are present because of filter damage or poor fit.	<ol style="list-style-type: none"> 1. Use pressure gauges to ensure that filters are performing at proper static pressure. 2. Make ease of installation and maintenance criteria for filter selection. 3. Properly train maintenance personnel in HVAC concerns. 4. Design system with filters downstream from fans. 5. Avoid water on filters or insulation.
Improper fan setting (267)	Air must be delivered at design volume to maintain pressure balances. Air flow in special vent rooms reverses.	<ol style="list-style-type: none"> 1. Routinely monitor air flow and pressure balances throughout critical parts of HVAC system. 2. Minimize or avoid using rooms that switch between positive and negative pressure.
Ductwork disconnections (268)	Dislodged or leaky supply duct runs can spill into and leaky returns may draw from hidden areas. Pressure balance will be interrupted, and infectious material may be disturbed and entrained into hospital air supply.	<ol style="list-style-type: none"> 1. Design a ductwork system that is easy to access, maintain, and repair. 2. Train maintenance personnel to regularly monitor air flow volumes and pressure balances throughout the system. 3. Test critical areas for appropriate air flow
Air flow impedance (213)	Debris, structural failure, or improperly adjusted dampers can block duct work and prevent designed air flow.	<ol style="list-style-type: none"> 1. Design and budget for a duct system that is easy to inspect, maintain, and repair. 2. Alert contractors to use caution when working around HVAC systems during the construction phase. 3. Regularly clean exhaust grilles. 4. Provide monitoring for special ventilation areas.
Open windows (96, 247)	Open windows can alter fan-induced pressure balance and allow dirty-to-clean air flow.	<ol style="list-style-type: none"> 1. Use sealed windows. 2. Design HVAC systems to deliver sufficient outdoor dilution ventilation. 3. Ensure that OSHA indoor air quality standards are met.
Dirty window air conditioners (96, 269)	Dirt, moisture, and bird droppings can contaminate window air conditioners, which can then introduce infectious material into hospital rooms.	<ol style="list-style-type: none"> 1. Eliminate such devices in plans for new construction. 2. Where they must be used, make sure that they are routinely cleaned and inspected.

Problem§	Consequences	Possible solutions
Inadequate filtration (270)	Infectious particles may pass through filters into vulnerable patient areas.	<ol style="list-style-type: none"> 1. Specify appropriate filters during new construction design phase. 2. Make sure that HVAC fans are sized to overcome pressure demands of filter system. 3. Inspect and test filters for proper installation.
Maintenance disruptions (271)	Fan shut-offs, dislodged filter cake material contaminates downstream air supply and drain pans. This may compromise air flow in special ventilation areas.	<ol style="list-style-type: none"> 1. Budget for a rigorous maintenance schedule when designing a facility. 2. Design system for easy maintenance. 3. Ensure communication between engineering and maintenance personnel. 4. Institute an ongoing training program for all involved staff members.
Excessive moisture in the HVAC system (120)	Chronically damp internal lining of the HVAC system, excessive condensate, and drip pans with stagnant water may result from this problem.	<ol style="list-style-type: none"> 1. Locate duct humidifiers upstream of the final filters. 2. Identify a means to remove water from the system. 3. Monitor humidity; all duct take-offs should be downstream of the humidifiers so that moisture is absorbed completely. 4. Use steam humidifiers in the HVAC system.
Duct contamination (18, 272)	Debris is released during maintenance or cleaning.	<ol style="list-style-type: none"> 1. Provide point-of-use filtration in the critical areas. 2. Design air-handling systems with insulation of the exterior of the ducts. 3. Do not use fibrous sound attenuators. 4. Decontaminate or encapsulate contamination.

* Reprinted with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

§ Numbers in parentheses are reference citations.

Construction, renovation, repair, and demolition activities in health-care facilities require substantial planning and coordination to minimize the risk for airborne infection both during projects and after their completion. Several organizations and experts have endorsed a multi-disciplinary team approach (Box 4) to coordinate the various stages of construction activities (e.g., project inception, project implementation, final walk-through, and completion).^{120, 249, 250, 273–276} Environmental services, employee health, engineering, and infection control must be represented in construction planning and design meetings should be convened with architects and design engineers. The number of members and disciplines represented is a function of the complexity of a project. Smaller, less complex projects and maintenance may require a minimal number of members beyond the core representation from engineering, infection control, environmental services, and the directors of the specialized departments.

Box 4. Suggested members and functions of a multi-disciplinary coordination team for construction, renovation, repair, and demolition projects

Members

Infection-control personnel, including hospital epidemiologists
Laboratory personnel
Facility administrators or their designated representatives, facility managers
Director of engineering
Risk-management personnel
Directors of specialized programs (e.g., transplantation, oncology and ICU* programs)
Employee safety personnel, industrial hygienists, and regulatory affairs personnel
Environmental services personnel
Information systems personnel
Construction administrators or their designated representatives
Architects, design engineers, project managers, and contractors

Functions and responsibilities

Coordinate members' input in developing a comprehensive project management plan.
Conduct a risk assessment of the project to determine potential hazards to susceptible patients.
Prevent unnecessary exposures of patients, visitors, and staff to infectious agents.
Oversee all infection-control aspects of construction activities.
Establish site-specific infection-control protocols for specialized areas.
Provide education about the infection-control impact of construction to staff and construction workers.
Ensure compliance with technical standards, contract provisions, and regulations.
Establish a mechanism to address and correct problems quickly.
Develop contingency plans for emergency response to power failures, water supply disruptions, and fires.
Provide a water-damage management plan (including drying protocols) for handling water intrusion from floods, leaks, and condensation.
Develop a plan for structural maintenance.

* ICU is intensive care unit.

Education of maintenance and construction workers, health-care staff caring for high-risk patients, and persons responsible for controlling indoor air quality heightens awareness that minimizing dust and moisture intrusion from construction sites into high-risk patient-care areas helps to maintain a safe environment.^{120, 250, 271, 275–278} Visual and printed educational materials should be provided in the language spoken by the workers. Staff and construction workers also need to be aware of the potentially catastrophic consequences of dust and moisture intrusion when an HVAC system or water system fails during construction or repair; action plans to deal quickly with these emergencies should be developed in advance and kept on file. Incorporation of specific standards into construction contracts may help to prevent departures from recommended practices as projects progress. Establishing specific lines of communication is important to address problems (e.g., dust control, indoor air quality, noise levels, and vibrations), resolve complaints, and keep projects moving toward completion. Health-care facility staff should develop a mechanism to monitor worker adherence to infection-control guidelines on a daily basis in and around the construction site for the duration of the project.

b. Preliminary Considerations

The three major topics to consider before initiating any construction or repair activity are as follows: a) design and function of the new structure or area, b) assessment of environmental risks for airborne disease and opportunities for prevention, and c) measures to contain dust and moisture during construction or repairs. A checklist of design and function considerations can help to ensure that a planned structure or area can be easily serviced and maintained for environmental infection control (Box 5).^{17, 250, 273, 275–277} Specifications for the construction, renovation, remodeling, and maintenance of health-care facilities are outlined in the AIA document, *Guidelines for Design and Construction of Hospitals and Health Care Facilities*.^{120, 275}

Box 5. Construction design and function considerations for environmental infection control

Location of sinks and dispensers for handwashing products and hand hygiene products
Types of faucets (e.g., aerated vs. non-aerated)
Air-handling systems engineered for optimal performance, easy maintenance, and repair
ACH and pressure differentials to accommodate special patient-care areas
Location of fixed sharps containers
Types of surface finishes (e.g., porous vs. non-porous)
Well-caulked walls with minimal seams
Location of adequate storage and supply areas
Appropriate location of medicine preparations areas (e.g., ≥ 3 ft. from a sink)
Appropriate location and type of ice machines (e.g., preferably ice dispensers rather than ice bins)
Appropriate materials for sinks and wall coverings
Appropriate traffic flow (e.g., no “dirty” movement through “clean” areas)
Isolation rooms with anterooms as appropriate
Appropriate flooring (e.g., seamless floors in dialysis units)
Sensible use carpeting (e.g., avoiding use of carpeting in special care areas or areas likely to become wet)*
Convenient location of soiled utility areas
Properly engineered areas for linen services and solid waste management
Location of main generator to minimize the risk of system failure from flooding or other emergency
Installation guidelines for sheetrock

* Use of carpet cleaning methods (e.g., “bonneting”) that disperse microorganisms into the air may increase the risk of airborne infection among at-risk patients, especially if they are in the vicinity of the cleaning activity.¹¹¹

Proactive strategies can help prevent environmentally mediated airborne infections in health-care facilities during demolition, construction, and renovation. The potential presence of dust and moisture and their contribution to health-care-associated infections must be critically evaluated early in the planning of any demolition, construction, renovation, and repairs.^{120, 250, 251, 273, 274, 276–279} Consideration must extend beyond dust generated by major projects to include dust that can become airborne if disturbed during routine maintenance and minor renovation activities (e.g., exposure of ceiling spaces for inspection; installation of conduits, cable, or sprinkler systems; rewiring; and structural repairs or replacement).^{273, 276, 277} Other projects that can compromise indoor air quality include construction and repair jobs that inadvertently allow substantial amounts of raw, unfiltered outdoor air to enter the facility (e.g., repair of elevators and elevator shafts) and activities that dampen any structure, area, or item made of porous materials or characterized by cracks and crevices (e.g., sink cabinets in need of repair, carpets, ceilings, floors, walls, vinyl wall coverings, upholstery, drapes, and countertops).^{18, 273, 277} Molds grow and proliferate on these surfaces when they become and remain wet.^{21, 120, 250, 266, 270, 272, 280} Scrubbable

materials are preferred for use in patient-care areas.

Containment measures for dust and/or moisture control are dictated by the location of the construction site. Outdoor demolition and construction require actions to keep dust and moisture out of the facility (e.g., sealing windows and vents and keeping doors closed or sealed). Containment of dust and moisture generated from construction inside a facility requires barrier structures (either pre-fabricated or constructed of more durable materials as needed) and engineering controls to clean the air in and around the construction or repair site.

c. Infection-Control Risk Assessment

An infection-control risk assessment (ICRA) conducted before initiating repairs, demolition, construction, or renovation activities can identify potential exposures of susceptible patients to dust and moisture and determine the need for dust and moisture containment measures. This assessment centers on the type and extent of the construction or repairs in the work area but may also need to include adjacent patient-care areas, supply storage, and areas on levels above and below the proposed project. An example of designing an ICRA as a matrix, the policy for performing an ICRA and implementing its results, and a sample permit form that streamlines the communication process are available.²⁸¹ Knowledge of the air flow patterns and pressure differentials helps minimize or eliminate the inadvertent dispersion of dust that could contaminate air space, patient-care items, and surfaces.^{57, 282, 283} A recent aspergillosis outbreak among oncology patients was attributed to depressurization of the building housing the HSCT unit while construction was underway in an adjacent building. Pressure readings in the affected building (including 12 of 25 HSCT-patient rooms) ranged from 0.1 Pa–5.8 Pa. Unfiltered outdoor air flowed into the building through doors and windows, exposing patients in the HSCT unit to fungal spores.²⁸³ During long-term projects, providing temporary essential services (e.g., toilet facilities) and conveniences (e.g., vending machines) to construction workers within the site will help to minimize traffic in and out of the area. The type of barrier systems necessary for the scope of the project must be defined.^{12, 120, 250, 279, 284}

Depending on the location and extent of the construction, patients may need to be relocated to other areas in the facility not affected by construction dust.^{51, 285} Such relocation might be especially prudent when construction takes place within units housing immunocompromised patients (e.g., severely neutropenic patients and patients on corticosteroid therapy). Advance assessment of high-risk locations and planning for the possible transport of patients to other departments can minimize delays and waiting time in hallways.⁵¹ Although hospitals have provided immunocompromised patients with some form of respiratory protection for use outside their rooms, the issue is complex and remains unresolved until more research can be done. Previous guidance on this issue has been inconsistent.⁹ Protective respirators (i.e., N95) were well tolerated by patients when used to prevent further cases of construction-related aspergillosis in a recent outbreak.²⁸³ The routine use of the N95 respirator by patients, however, has not been evaluated for preventing exposure to fungal spores during periods of non-construction. Although health-care workers who would be using the N95 respirator for personal respiratory protection must be fit-tested, there is no indication that either patients or visitors should undergo fit-testing.

Surveillance activities should augment preventive strategies during construction projects.^{3, 4, 20, 110, 286, 287} By determining baseline levels of health-care–acquired airborne and waterborne infections, infection-control staff can monitor changes in infection rates and patterns during and immediately after construction, renovations, or repairs.³

d. Air Sampling

Air sampling in health-care facilities may be conducted both during periods of construction and on a periodic basis to determine indoor air quality, efficacy of dust-control measures, or air-handling system performance via parametric monitoring. Parametric monitoring consists of measuring the physical

performance of the HVAC system in accordance with the system manufacturer's specifications. A periodic assessment of the system (e.g., air flow direction and pressure, ACH, and filter efficiency) can give assurance of proper ventilation, especially for special care areas and operating rooms.²⁸⁸

Air sampling is used to detect aerosols (i.e., particles or microorganisms). Particulate sampling (i.e., total numbers and size range of particulates) is a practical method for evaluating the infection-control performance of the HVAC system, with an emphasis on filter efficiency in removing respirable particles (<5 µm in diameter) or larger particles from the air. Particle size is reported in terms of the mass median aerodynamic diameter (MMAD), whereas count median aerodynamic diameter (CMAD) is useful with respect to particle concentrations.

Particle counts in a given air space within the health-care facility should be evaluated against counts obtained in a comparison area. Particle counts indoors are commonly compared with the particulate levels of the outdoor air. This approach determines the "rank order" air quality from "dirty" (i.e., the outdoor air) to "clean" (i.e., air filtered through high-efficiency filters [90%–95% filtration]) to "cleanest" (i.e., HEPA-filtered air).²⁸⁸ Comparisons from one indoor area to another may also provide useful information about the magnitude of an indoor air-quality problem. Making rank-order comparisons between clean, highly-filtered areas and dirty areas and/or outdoors is one way to interpret sampling results in the absence of air quality and action level standards.^{35, 289}

In addition to verifying filter performance, particle counts can help determine if barriers and efforts to control dust dispersion from construction are effective. This type of monitoring is helpful when performed at various times and barrier perimeter locations during the project. Gaps or breaks in the barriers' joints or seals can then be identified and repaired. The American Conference of Governmental Industrial Hygienists (ACGIH) has set a threshold limit value-time weighted average (TLV®-TWA) of 10 mg/m³ for nuisance dust that contains no asbestos and <1% crystalline silica.²⁹⁰ Alternatively, OSHA has set permissible exposure limits (PELs) for inert or nuisance dust as follows: respirable fraction at 5 mg/m³ and total dust at 15 mg/m³.²⁹¹ Although these standards are not measures of a bioaerosol, they are used for indoor air quality assessment in occupational settings and may be useful criteria in construction areas. Application of ACGIH guidance to health-care settings has not been standardized, but particulate counts in health-care facilities are likely to be well below this threshold value and approaching clean-room standards in certain care areas (e.g., operating rooms).¹⁰⁰

Particle counters and anemometers are used in particulate evaluation. The anemometer measures air flow velocity, which can be used to determine sample volumes. Particulate sampling usually does not require microbiology laboratory services for the reporting of results.

Microbiologic sampling of air in health-care facilities remains controversial because of currently unresolved technical limitations and the need for substantial laboratory support (Box 6). Infection-control professionals, laboratorians, and engineers should determine if microbiologic and/or particle sampling is warranted and assess proposed methods for sampling. The most significant technical limitation of air sampling for airborne fungal agents is the lack of standards linking fungal spore levels with infection rates. Despite this limitation, several health-care institutions have opted to use microbiologic sampling when construction projects are anticipated and/or underway in efforts to assess the safety of the environment for immunocompromised patients.^{35, 289} Microbiologic air sampling should be limited to assays for airborne fungi; of those, the thermotolerant fungi (i.e., those capable of growing at 95°F–98.6°F [35°C–37°C]) are of particular concern because of their pathogenicity in immunocompromised hosts.³⁵ Use of selective media (e.g., Sabouraud dextrose agar and inhibitory mold agar) helps with the initial identification of recovered organisms.

Microbiologic sampling for fungal spores performed as part of various airborne disease outbreak

investigations has also been problematic.^{18, 49, 106, 111, 112, 289} The precise source of a fungus is often difficult to trace with certainty, and sampling conducted after exposure may neither reflect the circumstances that were linked to infection nor distinguish between health-care–acquired and community-acquired infections. Because fungal strains may fluctuate rapidly in the environment, health-care–acquired *Aspergillus* spp. infection cannot be confirmed or excluded if the infecting strain is not found in the health-care setting.²⁸⁷ Sensitive molecular typing methods (e.g., randomly amplified polymorphic DNA (RAPD) techniques and a more recent DNA fingerprinting technique that detects restriction fragment length polymorphisms in fungal genomic DNA) to identify strain differences among *Aspergillus* spp., however, are becoming increasingly used in epidemiologic investigations of health-care–acquired fungal infection (A. Streifel, University of Minnesota, 2000).^{68, 110, 286, 287, 292–296} During case cluster evaluation, microbiologic sampling may provide an isolate from the environment for molecular typing and comparison with patient isolates. Therefore, it may be prudent for the clinical laboratory to save *Aspergillus* spp. isolated from colonizations and invasive disease cases among patients in PE, oncology, and transplant services for these purposes.

Box 6. Unresolved issues associated with microbiologic air sampling*

Lack of standards linking fungal spore levels with infection rates (i.e., no safe level of exposure)
Lack of standard protocols for testing (e.g., sampling intervals, number of samples, sampling locations)
Need for substantial laboratory support
Culture issues (e.g., false negatives, insensitivity, lag time between sampling and recording the results)
New, complex polymerase chain reaction (PCR) analytical methods
Unknown incubation period for *Aspergillus* spp. infection
Variability of sampler readings
Sensitivity of the sampler used (i.e., the volumes of air sampled)
Lack of details in the literature about describing sampling circumstances (e.g., unoccupied rooms vs. ongoing activities in rooms, expected fungal concentrations, and rate of outdoor air penetration)
Lack of correlation between fungal species and strains from the environment and clinical specimens
Confounding variables with high-risk patients (e.g., visitors and time spent outside of protective environment [PE] without respiratory protection)
Need for determination of ideal temperature for incubating fungal cultures (95°F [35°C] is the most commonly used temperature)

* Material in this box is compiled from references 35, 100, 222, 289, and 297.

Sedimentation methods using settle plates and volumetric sampling methods using solid impactors are commonly employed when sampling air for bacteria and fungi. Settle plates have been used by numerous investigators to detect airborne bacteria or to measure air quality during medical procedures (e.g., surgery).^{17, 60, 97, 151, 161, 287} Settle plates, because they rely on gravity during sampling, tend to select for larger particles and lack sensitivity for respirable particles (e.g., individual fungal spores), especially in highly-filtered environments. Therefore, they are considered impractical for general use.^{35, 289, 298–301} Settle plates, however, may detect fungi aerosolized during medical procedures (e.g., during wound dressing changes), as described in a recent outbreak of aspergillosis among liver transplant patients.³⁰²

The use of slit or sieve impactor samplers capable of collecting large volumes of air in short periods of time are needed to detect low numbers of fungal spores in highly filtered areas.^{35, 289} In some

outbreaks, aspergillosis cases have occurred when fungal spore concentrations in PE ambient air ranged as low as 0.9–2.2 colony-forming units per cubic meter (CFU/m³) of air.^{18, 94} On the basis of the expected spore counts in the ambient air and the performance parameters of various types of volumetric air samplers, investigators of a recent aspergillosis outbreak have suggested that an air volume of at least 1000 L (1 m³) should be considered when sampling highly filtered areas.²⁸³ Investigators have also suggested limits of 15 CFU/m³ for gross colony counts of fungal organisms and <0.1 CFU/m³ for *Aspergillus fumigatus* and other potentially opportunistic fungi in heavily filtered areas (≥12 ACH and filtration of ≥99.97% efficiency).¹²⁰ No correlation of these values with the incidence of health-care-associated fungal infection rates has been reported.

Air sampling in health-care facilities, whether used to monitor air quality during construction, to verify filter efficiency, or to commission new space prior to occupancy, requires careful notation of the circumstances of sampling. Most air sampling is performed under undisturbed conditions. However, when the air is sampled during or after human activity (e.g., walking and vacuuming), a higher number of airborne microorganisms likely is detected.²⁹⁷ The contribution of human activity to the significance of air sampling and its impact on health-care-associated infection rates remain to be defined. Comparing microbiologic sampling results from a target area (e.g., an area of construction) to those from an unaffected location in the facility can provide information about distribution and concentration of potential airborne pathogens. A comparison of microbial species densities in outdoor air versus indoor air has been used to help pinpoint fungal spore bursts. Fungal spore densities in outdoor air are variable, although the degree of variation with the seasons appears to be more dramatic in the United States than in Europe.^{92, 287, 303}

Particulate and microbiologic air sampling have been used when commissioning new HVAC system installations; however, such sampling is particularly important for newly constructed or renovated PE or operating rooms. Particulate sampling is used as part of a battery of tests to determine if a new HVAC system is performing to specifications for filtration and the proper number of ACH.^{268, 288, 304} Microbiologic air sampling, however, remains controversial in this application, because no standards for comparison purposes have been determined. If performed, sampling should be limited to determining the density of fungal spores per unit volume of air space. High numbers of spores may indicate contamination of air-handling system components prior to installation or a system deficiency when culture results are compared with known filter efficiencies and rates of air exchange.

e. External Demolition and Construction

External demolition, planned building implosions, and dirt excavation generate considerable dust and debris that can contain airborne microorganisms. In one study, peak concentrations in outdoor air at grade level and HVAC intakes during site excavation averaged 20,000 CFU/m³ for all fungi and 500 CFU/m³ for *Aspergillus fumigatus*, compared with 19 CFU/m³ and 4 CFU/m³, respectively, in the absence of construction.²⁸⁰ Many health-care institutions are located in large, urban areas; building implosions are becoming a more frequent concern. Infection-control risk assessment teams, particularly those in facilities located in urban renewal areas, would benefit by developing risk management strategies for external demolition and construction as a standing policy. In light of the events of 11 September 2001, it may be necessary for the team to identify those dust exclusion measures that can be implemented rapidly in response to emergency situations (Table 8). Issues to be reviewed prior to demolition include a) proximity of the air intake system to the work site, b) adequacy of window seals and door seals, c) proximity of areas frequented by immunocompromised patients, and d) location of the underground utilities (D. Erickson, ASHE, 2000).^{120, 250, 273, 276, 277, 280, 305}

Table 8. Strategies to reduce dust and moisture intrusion during external demolition and construction

<i>Item</i>	<i>Recommendation</i>
Demolition site	<ul style="list-style-type: none"> ● Shroud the site if possible to reduce environmental contamination.
Dust-generating equipment	<ul style="list-style-type: none"> ● Prior to placing dust-generating equipment, evaluate the location to ensure that dust produced by the equipment will not enter the building through open doorways or windows, or through ventilation air intakes.
Construction materials storage	<ul style="list-style-type: none"> ● Locate this storage away from the facility and ventilation air intakes.
Adjacent air intakes HVAC system	<ul style="list-style-type: none"> ● Seal off affected intakes, if possible, or move if funds permit. ● Consult with the facility engineer about pressure differentials and air recirculation options; keep facility air pressure positive to outside air.
Filters	<ul style="list-style-type: none"> ● Ensure that filters are properly installed; change roughing filters frequently to prevent dust build-up on high-efficiency filters.
Windows	<ul style="list-style-type: none"> ● Seal and caulk to prevent entry of airborne fungal spores.
Doors	<ul style="list-style-type: none"> ● Keep closed as much as possible; do not prop open; seal and caulk unused doors (i.e., those that are not designated as emergency exits); use mats with tacky surfaces at outside entrances.
Water utilities	<ul style="list-style-type: none"> ● Note location relative to construction area to prevent intrusion of dust into water systems.*
Medical gas piping	<ul style="list-style-type: none"> ● Ensure that these lines/pipes are insulated during periods of vibration.
Rooftops	<ul style="list-style-type: none"> ● Temporarily close off during active demolition/construction those rooftop areas that are normally open to the public (e.g., rooftop atrium).
Dust generation	<ul style="list-style-type: none"> ● Provide methods (e.g., misting the area with water) to minimize dust.
Immunocompromised patients	<ul style="list-style-type: none"> ● Use walk-ways protected from demolition/construction sites; avoid outside areas close to these sites; avoid rooftops.
Pedestrian traffic	<ul style="list-style-type: none"> ● Close off entry ways as needed to minimize dust intrusion.
Truck traffic	<ul style="list-style-type: none"> ● Reroute if possible, or arrange for frequent street cleaning.
Education and awareness+	<ul style="list-style-type: none"> ● Encourage reporting of hazardous or unsafe incidents associated with construction.

* Contamination of water pipes during demolition activities has been associated with health-care-associated transmission of *Legionella* spp.³⁰⁵

+ When health-care facilities have immunosuppressed patients in their census, telephoning the city building department each month to find out if buildings are scheduled for demolition is prudent.

Minimizing the entry of outside dust into the HVAC system is crucial in reducing the risk for airborne contaminants. Facility engineers should be consulted about the potential impact of shutting down the system or increasing the filtration. Selected air handlers, especially those located close to excavation sites, may have to be shut off temporarily to keep from overloading the system with dust and debris. Care is needed to avoid significant facility-wide reductions in pressure differentials that may cause the building to become negatively pressured relative to the outside. To prevent excessive particulate overload and subsequent reductions in effectiveness of intake air systems that cannot be shut off temporarily, air filters must be inspected frequently for proper installation and function. Excessive dust

penetration can be avoided if recirculated air is maximally utilized while outdoor air intakes are shut down. Scheduling demolition and excavation during the winter, when *Aspergillus* spp. spores may be present in lower numbers, can help, although seasonal variations in spore density differ around the world.^{92, 287, 303} Dust control can be managed by misting the dirt and debris during heavy dust-generating activities. To decrease the amount of aerosols from excavation and demolition projects, nearby windows, especially in areas housing immunocompromised patients, can be sealed and window and door frames caulked or weather-stripped to prevent dust intrusion.^{50, 301, 306} Monitoring for adherence to these control measures throughout demolition or excavation is crucial. Diverting pedestrian traffic away from the construction sites decreases the amount of dust tracked back into the health-care facility and minimizes exposure of high-risk patients to environmental pathogens. Additionally, closing entrances near construction or demolition sites might be beneficial; if this is not practical, creating an air lock (i.e., pressurizing the entry way) is another option.

f. Internal Demolition, Construction, Renovations, and Repairs

The focus of a properly implemented infection-control program during interior construction and repairs is containment of dust and moisture. This objective is achieved by a) educating construction workers about the importance of control measures; b) preparing the site; c) notifying and issuing advisories for staff, patients, and visitors; d) moving staff and patients and relocating patients as needed; e) issuing standards of practice and precautions during activities and maintenance; f) monitoring for adherence to control measures during construction and providing prompt feedback about lapses in control; g) monitoring HVAC performance; h) implementing daily clean-up, terminal cleaning and removal of debris upon completion; and i) ensuring the integrity of the water system during and after construction. These activities should be coordinated with engineering staff and infection-control professionals.

Physical barriers capable of containing smoke and dust will confine dispersed fungal spores to the construction zone.^{279, 284, 307, 308} The specific type of physical barrier required depends on the project's scope and duration and on local fire codes. Short-term projects that result in minimal dust dispersion (e.g., installation of new cables or wiring above ceiling tiles) require only portable plastic enclosures with negative pressure and HEPA filtration of the exhaust air from the enclosed work area. The placement of a portable industrial-grade HEPA filter device capable of filtration rate of 300–800 ft³/min. adjacent to the work area will help to remove fungal spores, but its efficacy is dependent on the supplied ACH and size of the area. If the project is extensive but short-term, dust-abatement, fire-resistant plastic curtains (e.g., Visqueen®) may be adequate. These should be completely airtight and sealed from ceiling to floor with overlapping curtains;^{276, 277, 309} holes, tears, or other perforations should be repaired promptly with tape. A portable, industrial-grade HEPA filter unit on continuous operation is needed within the contained area, with the filtered air exhausted to the outside of the work zone. Patients should not remain in the room when dust-generating activities are performed. Tools to assist the decision-making process regarding selection of barriers based on an ICRA approach are available.²⁸¹

More elaborate barriers are indicated for long-term projects that generate moderate to large amounts of dust. These barrier structures typically consist of rigid, noncombustible walls constructed from sheet rock, drywall, plywood, or plaster board and covered with sheet plastic (e.g., Visqueen®). Barrier requirements to prevent the intrusion of dust into patient-care areas include a) installing a plastic dust abatement curtain before construction of the rigid barrier; b) sealing and taping all joint edges including the top and bottom; c) extending the barrier from floor to floor, which takes into account the space [approximately 2–8 ft.] above the finished, lay-down ceiling; and d) fitting or sealing any temporary doors connecting the construction zone to the adjacent area. (See Box 7 for a list of the various construction and repair activities that require the use of some type of barrier.)

Box 7. Construction/repair projects that require barrier structures*

Demolition of walls, wallboard, plaster, ceramic tiles, ceiling tiles, and ceilings
Removal of flooring and carpeting, windows and doors, and casework
Working with sinks and plumbing that could result in aerosolization of water in high-risk areas
Exposure of ceiling spaces for demolition and for installation or rerouting of utility services (e.g., rewiring, electrical conduction installation, HVAC ductwork, and piping)
Crawling into ceiling spaces for inspection in a manner that may dislodge dust
Demolition, repair, or construction of elevator shafts
Repairing water damage

* Material for this box was compiled from references 120, 250, 273, 276, and 277.

Dust and moisture abatement and control rely primarily on the impermeable barrier containment approach; as construction continues, numerous opportunities can lead to dispersion of dust to other areas of the health-care facility. Infection-control measures that augment the use of barrier containment should be undertaken (Table 9).

Dust-control measures for clinical laboratories are an essential part of the infection-control strategy during hospital construction or renovation. Use of plastic or solid barriers may be needed if the ICRA determines that air flow from construction areas may introduce airborne contaminants into the laboratory space. In one facility, pseudofungemia clusters attributed to *Aspergillus* spp. and *Penicillium* spp. were linked to improper air flow patterns and construction projects adjacent to the laboratory; intrusion of dust and spores into a biological safety cabinet from construction activity immediately next to the cabinet resulted in a cluster of cultures contaminated with *Aspergillus niger*.^{310, 311} Reportedly, no barrier containment was used and the HEPA filtration system was overloaded with dust. In addition, an outbreak of pseudobacteremia caused by *Bacillus* spp. occurred in another hospital during construction above a storage area for blood culture bottles.²⁰⁷ Airborne spread of *Bacillus* spp. spores resulted in contamination of the bottles' plastic lids, which were not disinfected or handled with proper aseptic technique prior to collection of blood samples.

Table 9. Infection-control measures for internal construction and repair projects*+

Infection-control measure	Steps for implementation
Prepare for the project.	<ol style="list-style-type: none"> 1. Use a multi-disciplinary team approach to incorporate infection control into the project. 2. Conduct the risk assessment and a preliminary walk-through with project managers and staff.
Educate staff and construction workers.	<ol style="list-style-type: none"> 1. Educate staff and construction workers about the importance of adhering to infection-control measures during the project. 2. Provide educational materials in the language of the workers. 3. Include language in the construction contract requiring construction workers and subcontractors to participate in infection-control training.
Issue hazard and warning notices.	<ol style="list-style-type: none"> 1. Post signs to identify construction areas and potential hazards. 2. Mark detours requiring pedestrians to avoid the work area.
Relocate high-risk patients as needed, especially if the construction is in or adjacent to a PE area.	<ol style="list-style-type: none"> 1. Identify target patient populations for relocation based on the risk assessment. 2. Arrange for the transfer in advance to avoid delays. 3. At-risk patients should wear protective respiratory equipment (e.g., a high-efficiency mask) when outside their PE rooms.
Establish alternative traffic patterns for staff, patients, visitors, and construction workers.	<ol style="list-style-type: none"> 1. Determine appropriate alternate routes from the risk assessment. 2. Designate areas (e.g., hallways, elevators, and entrances/exits) for construction-worker use. 3. Do not transport patients on the same elevator with construction materials and debris.

Infection-control measure	Steps for implementation
Erect appropriate barrier containment.	<ol style="list-style-type: none"> 1. Use prefabricated plastic units or plastic sheeting for short-term projects that will generate minimal dust. 2. Use durable rigid barriers for ongoing, long-term projects.
Establish proper ventilation.	<ol style="list-style-type: none"> 1. Shut off return air vents in the construction zone, if possible, and seal around grilles. 2. Exhaust air and dust to the outside, if possible. 3. If recirculated air from the construction zone is unavoidable, use a pre-filter and a HEPA filter before the air returns to the HVAC system. 4. When vibration-related work is being done that may dislodge dust in the ventilation system or when modifications are made to ductwork serving occupied spaces, install filters on the supply air grilles temporarily. 5. Set pressure differentials so that the contained work area is under negative pressure. 6. Use air flow monitoring devices to verify the direction of the air pattern. 7. Exhaust air and dust to the outside, if possible. 8. Monitor temperature, air changes per hour (ACH), and humidity levels (humidity levels should be <65%). 9. Use portable, industrial grade HEPA filters in the adjacent area and/or the construction zone for additional ACH. 10. Keep windows closed, if possible.
Control solid debris.	<ol style="list-style-type: none"> 1. When replacing filters, place the old filter in a bag prior to transport and dispose as a routine solid waste. 2. Clean the construction zone daily or more often as needed. 3. Designate a removal route for small quantities of solid debris. 4. Mist debris and cover disposal carts before transport (i.e., leaving the construction zone). 5. Designate an elevator for construction crew use. 6. Use window chutes and negative pressure equipment for removal of larger pieces of debris while maintaining pressure differentials in the construction zone. 7. Schedule debris removal to periods when patient exposures to dust is minimal.
Control water damage.	<ol style="list-style-type: none"> 1. Make provisions for dry storage of building materials. 2. Do not install wet, porous building materials (i.e., sheet rock). 3. Replace water-damaged porous building materials if they cannot be completely dried out within 72 hours.
Control dust in air and on surfaces.	<ol style="list-style-type: none"> 1. Monitor the construction area daily for compliance with the infection-control plan. 2. Protective outer clothing for construction workers should be removed before entering clean areas. 3. Use mats with tacky surfaces within the construction zone at the entry; cover sufficient area so that both feet make contact with the mat while walking through the entry. 4. Construct an anteroom as needed where coveralls can be donned and removed. 5. Clean the construction zone and all areas used by construction workers with a wet mop. 6. If the area is carpeted, vacuum daily with a HEPA-filtered–equipped vacuum. 7. Provide temporary essential services (e.g., toilets) and worker conveniences (e.g. vending machines) in the construction zone as appropriate. 8. Damp-wipe tools if removed from the construction zone or left in the area. 9. Ensure that construction barriers remain well sealed; use particle sampling as needed. 10. Ensure that the clinical laboratory is free from dust contamination.

Infection-control measure	Steps for implementation
Complete the project.	<ol style="list-style-type: none"> 1. Flush the main water system to clear dust-contaminated lines. 2. Terminally clean the construction zone before the construction barriers are removed. 3. Check for visible mold and mildew and eliminate (i.e., decontaminate and remove), if present. 4. Verify appropriate ventilation parameters for the new area as needed. 5. Do not accept ventilation deficiencies, especially in special care areas. 6. Clean or replace HVAC filters using proper dust-containment procedures. 7. Remove the barriers and clean the area of any dust generated during this work. 8. Ensure that the designated air balances in the operating rooms (OR) and protective environments (PE) are achieved before occupancy. 9. Commission the space as indicated, especially in the OR and PE, ensuring that the room's required engineering specifications are met.

* Material in this table includes information from D. Erickson, ASHE, 2000.

+ Material in this table was compiled from references 19, 51, 67, 80, 106, 120, 250, 266, 273, 276–278, 280, 285, and 309, 312–315.

5. Environmental Infection-Control Measures for Special Health-Care Settings

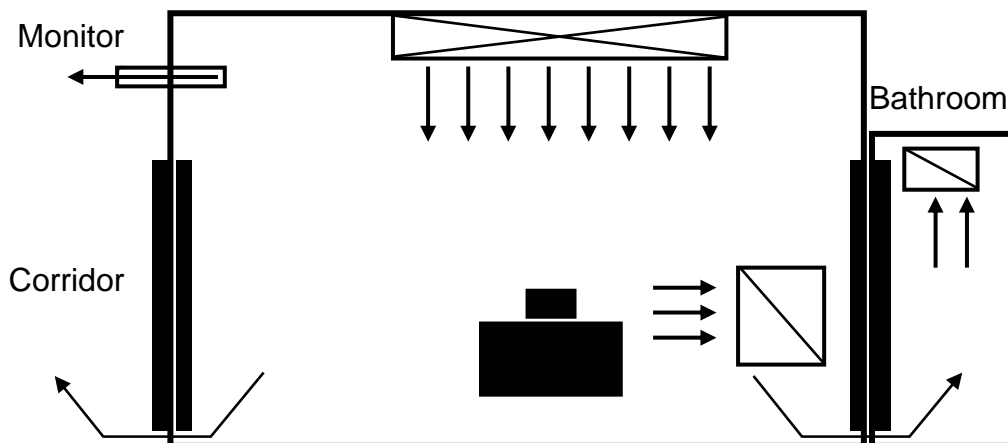
Areas in health-care facilities that require special ventilation include a) operating rooms; b) PE rooms used by high-risk, immunocompromised patients; and c) AII rooms for isolation of patients with airborne infections (e.g., those caused by *M. tuberculosis*, VZV, or measles virus). The number of rooms required for PE and AII are determined by a risk assessment of the health-care facility.⁶ Continuous, visual monitoring of air flow direction is required for new or renovated pressurized rooms.^{120, 256}

a. Protective Environments (PE)

Although the exact configuration and specifications of PEs might differ among hospitals, these care areas for high-risk, immunocompromised patients are designed to minimize fungal spore counts in air by maintaining a) filtration of incoming air by using central or point-of-use HEPA filters; b) directed room air flow [i.e., from supply on one side of the room, across the patient, and out through the exhaust on the opposite side of the room]; c) positive room air pressure of 2.5 Pa [0.01" water gauge] relative to the corridor; d) well-sealed rooms; and e) ≥ 12 ACH.^{44, 120, 251, 254, 316–319} Air flow rates must be adjusted accordingly to ensure sufficient ACH, and these rates vary depending on certain factors (e.g., room air leakage area). For example, to provide ≥ 12 ACH in a typical patient room with 0.5 sq. ft. air leakage, the air flow rate will be minimally 125 cubic feet/min (cfm).^{320, 321} Higher air flow rates may be needed. A general ventilation diagram for a positive-pressure room is given in Figure 2. Directed room air flow in PE rooms is not laminar; parallel air streams are not generated. Studies attempting to demonstrate patient benefit from laminar air flow in a PE setting are equivocal.^{316, 318, 319, 322–327}

Air flow direction at the entrances to these areas should be maintained and verified, preferably on a daily basis, using either a visual means of indication (e.g., smoke tubes and flutter strips) or manometers. Permanent installation of a visual monitoring device is indicated for new PE construction and renovation.¹²⁰ Facility service structures can interfere with the proper unidirectional air flow from the patients' rooms to the adjacent corridor. In one outbreak investigation, *Aspergillus* spp. infections in a critical care unit may have been associated with a pneumatic specimen transport system, a textile disposal duct system, and central vacuum lines for housekeeping, all of which disrupted proper air flow from the patients' rooms to the outside and allowed entry of fungal spores into the unit (M.McNeil, CDC, 2000).

Figure 2. Example of positive-pressure room control for protection from airborne environmental microbes (PE)* + §



* Stacked black boxes represent patient's bed. Long open box with cross-hatch represents supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate directions of air flow.

+ Possible uses include immunocompromised patient rooms (e.g., hematopoietic stem cell transplant or solid organ transplant procedure rooms) and orthopedic operating rooms.

§ Positive-pressure room engineering features include

- positive pressure (greater supply than exhaust air volume);
- pressure differential range of 2.5–8 Pa (0.01–0.03-in. water gauge), ideal at 8 Pa;
- air flow volume differential >125-cfm supply versus exhaust;
- sealed room, approximately 0.5-sq. ft. leakage;
- clean to dirty air flow;
- monitoring;
- ≥ 12 air changes per hour (ACH); and
- return air if refiltered.

¶ This diagram is a generic illustration of air flow in a typical installation. Alternative air flow arrangements are recognized. Adapted and used with permission from A. Streifel and the publisher of reference 328 (Penton Media, Inc.)

The use of surface fungicide treatments is becoming more common, especially for building materials.³²⁹ Copper-based compounds have demonstrated anti-fungal activity and are often applied to wood or paint. Copper-8-quinolinolate was used on environmental surfaces contaminated with *Aspergillus* spp. to control one reported outbreak of aspergillosis.³¹⁰ The compound was also incorporated into the fireproofing material of a newly constructed hospital to help decrease the environmental spore burden.³¹⁶

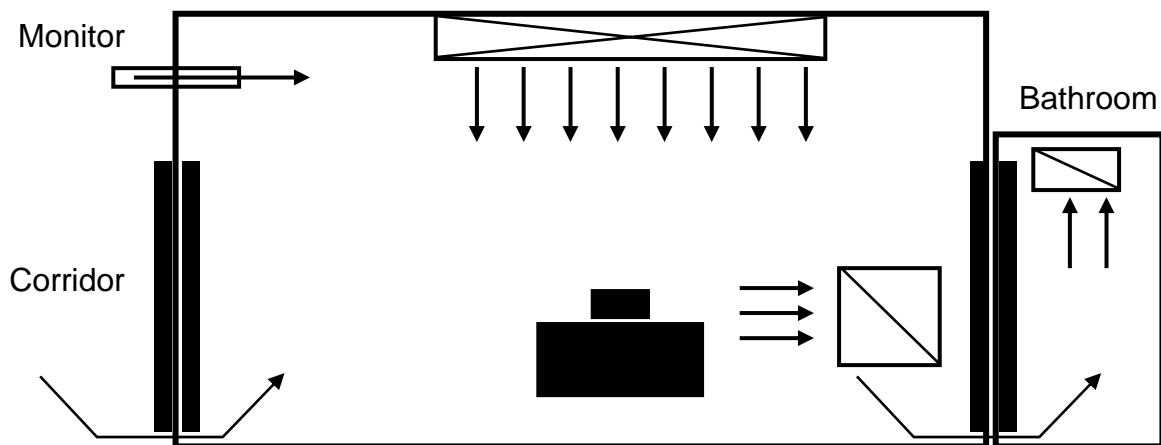
b. Airborne Infection Isolation (AII)

Acute-care inpatient facilities need at least one room equipped to house patients with airborne infectious disease. Every health-care facility, including ambulatory and long-term care facilities, should undertake an ICRA to identify the need for AII areas. Once the need is established, the appropriate ventilation equipment can be identified. Air handling systems for this purpose need not be restricted to central systems. Guidelines for the prevention of health-care-acquired TB have been published in response to multiple reports of health-care-associated transmission of multi-drug resistant strains.^{4, 330} In reports documenting health-care-acquired TB, investigators have noted a failure to comply fully with prevention measures in established guidelines.³³¹⁻³⁴⁵ These gaps highlight the importance of prompt recognition of the disease, isolation of patients, proper treatment, and engineering controls. AII rooms

are also appropriate for the care and management of smallpox patients.⁶ Environmental infection control with respect to smallpox is currently being revisited (see Appendix E).

Salient features of engineering controls for AII areas include a) use of negative pressure rooms with close monitoring of air flow direction using manometers or temporary or installed visual indicators [e.g., smoke tubes and flutter strips] placed in the room with the door closed; b) minimum 6 ACH for existing facilities, ≥ 12 ACH for areas under renovation or for new construction; and c) air from negative pressure rooms and treatment rooms exhausted directly to the outside if possible.^{4, 120, 248} As with PE, airflow rates need to be determined to ensure the proper numbers of ACH.^{320, 321} AII rooms can be constructed either with (Figure 3) or without (Figure 4) an anteroom. When the recirculation of air from AII rooms is unavoidable, HEPA filters should be installed in the exhaust duct leading from the room to the general ventilation system. In addition to UVGI fixtures in the room, UVGI can be placed in the ducts as an adjunct measure to HEPA filtration, but it can not replace the HEPA filter.^{4, 346} A UVGI fixture placed in the upper room, coupled with a minimum of 6 ACH, also provides adequate air cleaning.²⁴⁸

Figure 3. Example of negative-pressure room control for airborne infection isolation (AII)* + §¶



* Stacked black boxes represent patient's bed. Long open box with cross-hatch represents supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate direction of air flow.

+ Possible uses include treatment or procedure rooms, bronchoscopy rooms, and autopsy.

§ Negative-pressure room engineering features include

- negative pressure (greater exhaust than supply air volume);
- pressure differential of 2.5 Pa (0.01-in. water gauge);
- air flow volume differential >125 -cfm exhaust versus supply;
- sealed room, approximately 0.5-sq. ft. leakage;
- clean to dirty air flow;
- monitoring;
- ≥ 12 air changes per hour (ACH) new or renovation, 6 ACH existing; and
- exhaust to outside or HEPA-filtered if recirculated.

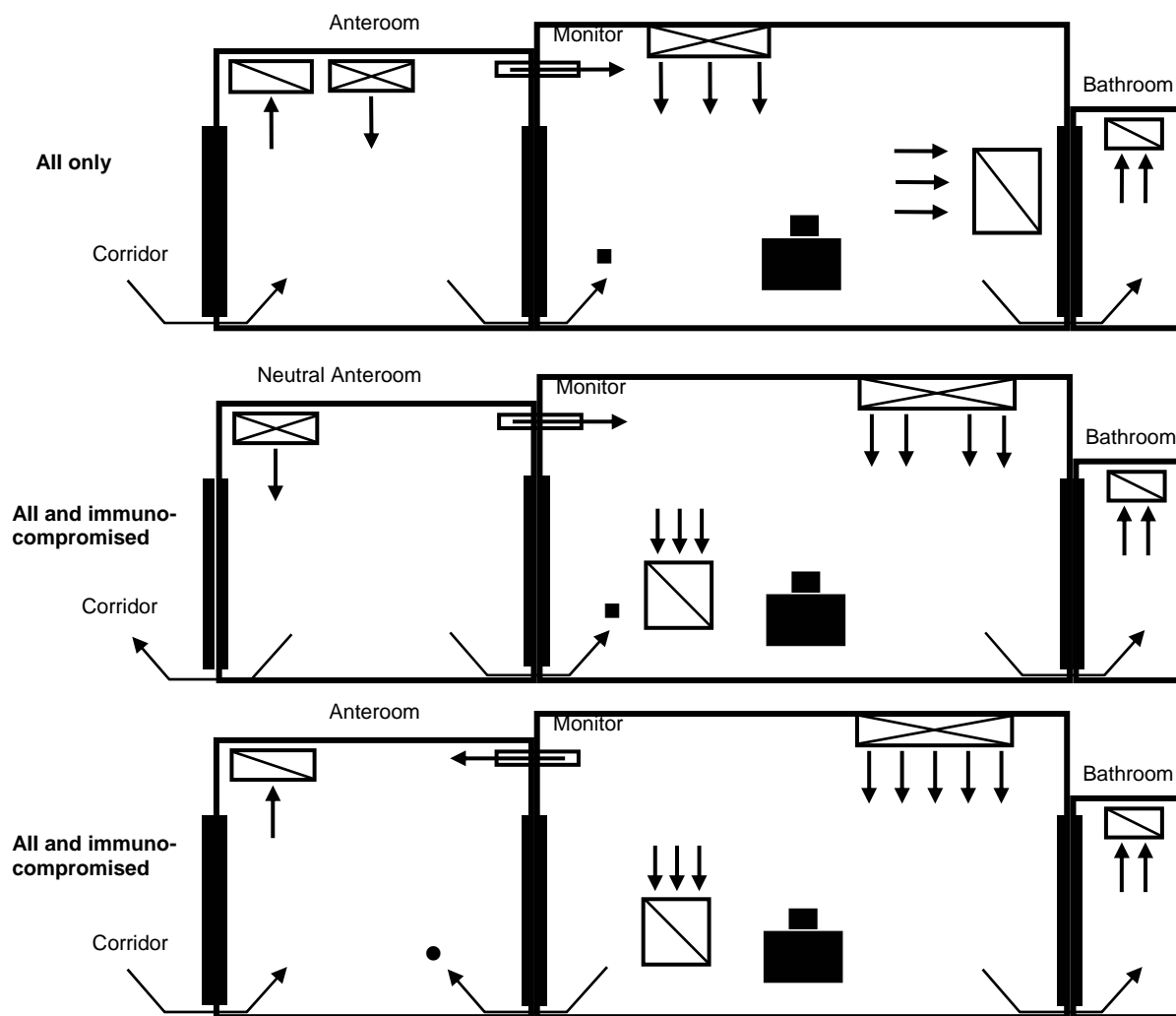
¶ This diagram is a generic illustration of air flow in a typical installation. Alternative air flow arrangements are recognized. Adapted and used with permission from A. Streifel and the publisher of reference 328 (Penton Media, Inc.)

One of the components of airborne infection isolation is respiratory protection for health-care workers and visitors when entering AII rooms.^{4, 6, 347} Recommendations of the type of respiratory protection are dependent on the patient's airborne infection (indicating the need for AII) and the risk of infection to

persons entering the AII room. A more in-depth discussion of respiratory protection in this instance is presented in the current isolation guideline;⁶ a revision of this guideline is in development. Cough-inducing procedures (e.g., endotracheal intubation and suctioning of known or suspected TB patients, diagnostic sputum induction, aerosol treatments, and bronchoscopy) require similar precautions.^{348–350}

Additional engineering measures are necessary for the management of patients requiring PE (i.e., allogeneic HSCT patients) who concurrently have airborne infection. For this type of patient treatment, an anteroom (Figure 4) is required in new construction and renovation as per AIA guidelines.¹²⁰

Figure 4. Example of airborne infection isolation (AII) room with anteroom and neutral anteroom* + §



* The top diagram indicates air flow patterns when patient with only airborne infectious disease occupies room. Middle and bottom diagrams indicate recommended air flow patterns when room is occupied by immunocompromised patient with airborne infectious disease. Stacked black boxes represent patient beds. Long open boxes with cross-hatches represent supply air. Open boxes with single, diagonal slashes represent air exhaust registers. Arrows indicate directions of air flow.

+ AII isolation room with anteroom engineering features include

- pressure differential of 2.5 Pa (0.01-in. water gauge) measured at the door between patient room and anteroom;
- air flow volume differential >125-cfm, depending on anteroom air flow direction (pressurized versus depressurized);

- sealed room with approximately 0.5-sq. ft. leakage;
- clean to dirty air flow
- monitoring;
- ≥ 12 air changes per hour (ACH) new or renovation, 6 ACH existing; and
- anteroom air flow patterns. The small ■ in panels 1 and 2 indicate the anteroom is pressurized (supply versus exhaust), while the small ● in panel 3 indicates the anteroom is depressurized (exhaust versus supply).

§ Used with permission of A. Streifel, University of Minnesota

The pressure differential of an anteroom can be positive or negative relative to the patient in the room.¹²⁰ An anteroom can act as an airlock (Figure 4). If the anteroom is positive relative to the air space in the patient's room, staff members do not have to mask prior to entry into the anteroom if air is directly exhausted to the outside and a minimum of 10 ACH (Figure 4, top diagram).¹²⁰ When an anteroom is negative relative to both the AII room and the corridor, health-care workers must mask prior to entering the anteroom (Figure 4, bottom diagram). If an AII room with an anteroom is not available, use of a portable, industrial-grade HEPA filter unit may help to increase the number of ACHs while facilitating the removal of fungal spores; however, a fresh air source must be present to achieve the proper air exchange rate. Incoming ambient air should receive HEPA filtration.

c. Operating Rooms

Operating room air may contain microorganisms, dust, aerosol, lint, skin squamous epithelial cells, and respiratory droplets. The microbial level in operating room air is directly proportional to the number of people moving in the room.³⁵¹ One study documented lower infection rates with coagulase-negative staphylococci among patients when operating room traffic during the surgical procedure was limited.³⁵² Therefore, efforts should be made to minimize personnel traffic during operations. Outbreaks of SSIs caused by group A beta-hemolytic streptococci have been traced to airborne transmission from colonized operating-room personnel to patients.^{150–154} Several potential health-care-associated pathogens (e.g., *Staphylococcus aureus* and *Staphylococcus epidermidis*) and drug-resistant organisms have also been recovered from areas adjacent to the surgical field,³⁵³ but the extent to which the presence of bacteria near the surgical field influences the development of postoperative SSIs is not clear.³⁵⁴

Proper ventilation, humidity (<68%), and temperature control in the operating room is important for the comfort of surgical personnel and patients, but also in preventing environmental conditions that encourage growth and transmission of microorganisms.³⁵⁵ Operating rooms should be maintained at positive pressure with respect to corridors and adjacent areas.³⁵⁶ Operating rooms typically do not have a variable air handling system. Variable air handling systems are permitted for use in operating rooms only if they continue to provide a positive pressure with respect to the corridors and adjacent areas and the proper ACHs are maintained when the room is occupied. Conventional operating-room ventilation systems produce a minimum of about 15 ACH of filtered air for thermal control, three (20%) of which must be fresh air.^{120, 357, 358} Air should be introduced at the ceiling and exhausted near the floor.^{357, 359}

Laminar airflow and UVGI have been suggested as adjunct measures to reduce SSI risk for certain operations. Laminar airflow is designed to move particle-free air over the aseptic operating field at a uniform velocity (0.3–0.5 m/sec), sweeping away particles in its path. This air flow can be directed vertically or horizontally, and recirculated air is passed through a HEPA filter.^{360–363} Neither laminar airflow nor UV light, however, has been conclusively shown to decrease overall SSI risk.^{356, 364–370}

Elective surgery on infectious TB patients should be postponed until such patients have received adequate drug therapy. The use of general anesthesia in TB patients poses infection-control challenges because intubation can induce coughing, and the anesthesia breathing circuit apparatus potentially can become contaminated.³⁷¹ Although operating room suites at 15 ACH exceed the air exchanges required

for TB isolation, the positive air flow relative to the corridor could result in health-care-associated transmission of TB to operating-room personnel. If feasible, intubation and extubation of the TB surgical patient should be performed in AII. AIA currently does not recommend changing pressure from positive to negative or setting it to neutral; most facilities lack the capability to do so.¹²⁰ When emergency surgery is indicated for a suspected/diagnosed infectious TB patient, taking specific infection-control measures is prudent (Box 8).

Box 8. Strategy for managing TB patients and preventing airborne transmission in operating rooms*

1. If emergency surgery is indicated for a patient with active TB, schedule the TB patient as the last surgical case to provide maximum time for adequate ACH.
2. Operating room personnel should use NIOSH-approved N95 respirators without exhalation valves.³⁴⁷
3. Keep the operating room door closed after the patient is intubated, and allow adequate time for sufficient ACH to remove 99% of airborne particles (Appendix B, Table B.1.):
 - a) after the patient is intubated and particularly if intubation produces coughing;
 - b) if the door to the operating suite must be opened, and intubation induces coughing in the patient; or
 - c) after the patient is extubated and suctioned [unless a closed suctioning system is present].
4. Extubate the patient in the operating room or allow the patient to recover in AII rather than in the regular open recovery facilities.
5. Temporary use of a portable, industrial grade HEPA filter may expedite removal of airborne contaminants (fresh-air exchange requirements for proper ventilation must still be met).+
6. Breathing circuit filters with 0.1–0.2 μm pore size can be used as an adjunct infection-control measure.^{373, 374}

* Material in this table was compiled from references 4, 347, and 372–374.

+ The placement of portable HEPA filter units in the operating room must be carefully evaluated for potential disruptions in normal air flow. The portable unit should be turned off while the surgical procedure is underway and turned on following extubation. Portable HEPA filter units previously placed in construction areas may be used in subsequent patient care, provided that all internal and external surfaces are cleaned and the filter's performance is verified with appropriate particle testing and is changed, if needed.

Table 10. Summary of ventilation specifications in selected areas of health-care facilities*

Specifications	AII room+	PE room	Critical care room§	Isolation anteroom	Operating room
Air pressure¶	Negative	Positive	Positive, negative, or neutral	Positive or negative	Positive
Room air changes	≥ 6 ACH (for existing rooms); ≥ 12 ACH (for renovation or new construction)	≥ 12 ACH	≥ 6 ACH	≥ 10 ACH	≥ 15 ACH
Sealed**	Yes	Yes	No	Yes	Yes
Filtration supply	90% (dust-spot ASHRAE 52.1 1992)	99.97%++	$\geq 90\%$	$\geq 90\%$	90%
Recirculation	No§§	Yes	Yes	No	Yes

* Material in this table is compiled from references 35 and 120.

+ Includes bronchoscopy suites.

§ Positive pressure and HEPA filters may be preferred in some rooms in intensive care units (ICUs) caring for large numbers of immunocompromised patients.

¶ Clean-to-dirty: negative to an infectious patient, positive away from an immunocompromised patient.

** Minimized infiltration for ventilation control; pertains to windows, closed doors, and surface joints.

++ Fungal spore filter at point of use (HEPA at 99.97% of 0.3 μm particles).

§§ Recirculated air may be used if the exhaust air is first processed through a HEPA filter.

¶¶ Table used with permission of the publisher of reference 35 (Lippincott Williams and Wilkins).

6. Other Aerosol Hazards in Health-Care Facilities

In addition to infectious bioaerosols, several crucial non-infectious, indoor air-quality issues must be addressed by health-care facilities. The presence of sensitizing and allergenic agents and irritants in the workplace (e.g., ethylene oxide, glutaraldehyde, formaldehyde, hexachlorophene, and latex allergens³⁷⁵) is increasing. Asthma and dermatologic and systemic reactions often result with exposure to these chemicals. Anesthetic gases and aerosolized medications (e.g., ribavirin, pentamidine, and aminoglycosides) represent some of the emerging potentially hazardous exposures to health-care workers. Containment of the aerosol at the source is the first level of engineering control, but personal protective equipment (e.g., masks, respirators, and glove liners) that distances the worker from the hazard also may be needed.

Laser plumes and surgical smoke represent another potential risk for health-care workers.^{376–378} Lasers transfer electromagnetic energy into tissues, resulting in the release of a heated plume that includes particles, gases, tissue debris, and offensive smells. One concern is that aerosolized infectious material in the laser plume might reach the nasal mucosa of surgeons and adjacent personnel. Although some viruses (i.e., varicella-zoster virus, pseudorabies virus, and herpes simplex virus) do not aerosolize efficiently,^{379, 380} other viruses and bacteria (e.g., human papilloma virus [HPV], HIV, coagulase-negative *Staphylococcus*, *Corynebacterium* spp., and *Neisseria* spp.) have been detected in laser plumes.^{381–387} The presence of an infectious agent in a laser plume may not, however, be sufficient to cause disease from airborne exposure, especially if the normal mode of transmission for the agent is not airborne. No evidence indicated that HIV or hepatitis B virus (HBV) has been transmitted via aerosolization and inhalation.³⁸⁸

Although continuing studies are needed to fully evaluate the risk of laser plumes to surgical personnel, the prevention measures in these other guidelines should be followed: a) NIOSH recommendations,³⁷⁸ b) the *Recommended Practices for Laser Safety in Practice Settings* developed by the Association of periOperative Registered Nurses [AORN],³⁸⁹ c) the assessments of ECRI,^{390–392} and d) the ANSI standard.³⁹³ These guidelines recommend the use of a) respirators (N95 or N100) or full face shields and masks,²⁶⁰ b) central wall-suction units with in-line filters to collect particulate matter from minimal plumes, and c) dedicated mechanical smoke exhaust systems with a high-efficiency filter to remove large amounts of laser plume. Although transmission of TB has occurred as a result of abscess management practices that lacked airborne particulate control measures and respiratory protection, use of a smoke evacuator or needle aspirator and a high degree of clinical awareness can help protect health-care workers when excising and draining an extrapulmonary TB abscess.¹³⁷

D. Water

1. Modes of Transmission of Waterborne Diseases

Moist environments and aqueous solutions in health-care settings have the potential to serve as reservoirs for waterborne microorganisms. Under favorable environmental circumstances (e.g., warm temperature and the presence of a source of nutrition), many bacterial and some protozoal microorganisms can either proliferate in active growth or remain for long periods in highly stable, environmentally resistant (yet infectious) forms. Modes of transmission for waterborne infections

include a) direct contact [e.g., that required for hydrotherapy]; b) ingestion of water [e.g., through consuming contaminated ice]; c) indirect-contact transmission [e.g., from an improperly reprocessed medical device];⁶ d) inhalation of aerosols dispersed from water sources;³ and e) aspiration of contaminated water. The first three modes of transmission are commonly associated with infections caused by gram-negative bacteria and nontuberculous mycobacteria (NTM). Aerosols generated from water sources contaminated with *Legionella* spp. often serve as the vehicle for introducing legionellae to the respiratory tract.³⁹⁴

2. Waterborne Infectious Diseases in Health-Care Facilities

a. Legionellosis

Legionellosis is a collective term describing infection produced by *Legionella* spp., whereas Legionnaires disease is a multi-system illness with pneumonia.³⁹⁵ The clinical and epidemiologic aspects of these diseases (Table 11) are discussed extensively in another guideline.³ Although Legionnaires disease is a respiratory infection, infection-control measures intended to prevent health-care-associated cases center on the quality of water—the principal reservoir for *Legionella* spp.

Table 11. Clinical and epidemiologic characteristics of legionellosis/Legionnaires disease

		References
Causative agent	<i>Legionella pneumophila</i> (90% of infections); <i>L. micdadei</i> , <i>L. bozemanii</i> , <i>L. dumoffii</i> , <i>L. longbeachii</i> , (14 additional species can cause infection in humans)	395–399
Mode of transmission	Aspiration of water, direct inhalation or water aerosols	3, 394–398, 400
Source of exposure	Exposure to environmental sources of <i>Legionella</i> spp. (i.e., water or water aerosols)	31, 33, 401–414
Clinical syndromes and diseases	Two distinct illnesses: a) Pontiac fever [a milder, influenza-like illness]; and b) progressive pneumonia that may be accompanied by cardiac, renal, and gastrointestinal involvement	3, 397–399, 415–422
Populations at greatest risk	Immunosuppressed patients (e.g., transplant patients, cancer patients, and patients receiving corticosteroid therapy); immunocompromised patients (e.g., surgical patients, patients with underlying chronic lung disease, and dialysis patients); elderly persons; and patients who smoke	395–397, 423–433
Occurrence	Proportion of community-acquired pneumonia caused by <i>Legionella</i> spp. ranges from 1%–5%; estimated annual incidence among the general population is 8,000–18,000 cases in the United States; the incidence of health-care-associated pneumonia (0%–14%) may be underestimated if appropriate laboratory diagnostic methods are unavailable.	396, 397, 434–444
Mortality rate	Mortality declined markedly during 1980–1998, from 34% to 12% for all cases; the mortality rate is higher among persons with health-care-associated pneumonia compared with the rate among community-acquired pneumonia patients (14% for health-care-associated pneumonia versus 10% for community-acquired pneumonia [1998 data]).	395–397, 445

Legionella spp. are commonly found in various natural and man-made aquatic environments^{446, 447} and can enter health-care facility water systems in low or undetectable numbers.^{448, 449} Cooling towers, evaporative condensers, heated potable water distribution systems, and locally-produced distilled water can provide environments for multiplication of legionellae.^{450–454} In several hospital outbreaks, patients have been infected through exposure to contaminated aerosols generated by cooling towers, showers, faucets, respiratory therapy equipment, and room-air humidifiers.^{401–410, 455} Factors that enhance

colonization and amplification of legionellae in man-made water environments include a) temperatures of 77°F–107.6°F [25°C–42°C],^{456–460} b) stagnation,⁴⁶¹ c) scale and sediment,⁴⁶² and d) presence of certain free-living aquatic amoebae that can support intracellular growth of legionellae.^{462, 463} The bacteria multiply within single-cell protozoa in the environment and within alveolar macrophages in humans.

b. Other Gram-Negative Bacterial Infections

Other gram-negative bacteria present in potable water also can cause health-care–associated infections. Clinically important, opportunistic organisms in tap water include *Pseudomonas aeruginosa*, *Pseudomonas* spp., *Burkholderia cepacia*, *Ralstonia pickettii*, *Stenotrophomonas maltophilia*, and *Sphingomonas* spp. (Tables 12 and 13). Immunocompromised patients are at greatest risk of developing infection. Medical conditions associated with these bacterial agents range from colonization of the respiratory and urinary tracts to deep, disseminated infections that can result in pneumonia and bloodstream bacteremia. Colonization by any of these organisms often precedes the development of infection. The use of tap water in medical care (e.g., in direct patient care, as a diluent for solutions, as a water source for medical instruments and equipment, and during the final stages of instrument disinfection) therefore presents a potential risk for exposure. Colonized patients also can serve as a source of contamination, particularly for moist environments of medical equipment (e.g., ventilators).

In addition to *Legionella* spp., *Pseudomonas aeruginosa* and *Pseudomonas* spp. are among the most clinically relevant, gram-negative, health-care–associated pathogens identified from water. These and other gram-negative, non-fermentative bacteria have minimal nutritional requirements (i.e., these organisms can grow in distilled water) and can tolerate a variety of physical conditions. These attributes are critical to the success of these organisms as health-care–associated pathogens. Measures to prevent the spread of these organisms and other waterborne, gram-negative bacteria include hand hygiene, glove use, barrier precautions, and eliminating potentially contaminated environmental reservoirs.^{464, 465}

Table 12. *Pseudomonas aeruginosa* infections in health-care facilities

		References
Clinical syndromes and diseases	Septicemia, pneumonia (particularly ventilator-associated), chronic respiratory infections among cystic fibrosis patients, urinary tract infections, skin and soft-tissue infections (e.g., tissue necrosis and hemorrhage), burn-wound infections, folliculitis, endocarditis, central nervous system infections (e.g., meningitis and abscess), eye infections, and bone and joint infections	466–503
Modes of transmission	Direct contact with water, aerosols; aspiration of water and inhalation of water aerosols; and indirect transfer from moist environmental surfaces via hands of health-care workers	28, 502–506
Environmental sources of pseudomonads in health-care settings	Potable (tap) water, distilled water, antiseptic solutions contaminated with tap water, sinks, hydrotherapy pools, whirlpools and whirlpool spas, water baths, lithotripsy therapy tanks, dialysis water, eyewash stations, flower vases, and endoscopes with residual moisture in the channels	28, 29, 466, 468, 507–520
Environmental sources of pseudomonads in the community	Fomites (e.g., drug injection equipment stored in contaminated water)	494, 495
Populations at greatest risk	Intensive care unit (ICU) patients (including neonatal ICU), transplant patients (organ and hematopoietic stem cell), neutropenic patients, burn therapy and hydrotherapy patients, patients with malignancies, cystic fibrosis patients, patients with underlying medical conditions, and dialysis patients	28, 466, 467, 472, 477, 493, 506–508, 511, 512, 521–526

Table 13. Other gram-negative bacteria associated with water and moist environments

Implicated contaminated environmental vehicle	References
<i>Burkholderia cepacia</i>	
Distilled water	527
Contaminated solutions and disinfectants	528, 529
Dialysis machines	527
Nebulizers	530–532
Water baths	533
Intrinsically-contaminated mouthwash*	534
Ventilator temperature probes	535
<i>Stenotrophomonas maltophilia, Sphingomonas spp.</i>	
Distilled water	536, 537
Contaminated solutions and disinfectants	529
Dialysis machines	527
Nebulizers	530–532
Water	538
Ventilator temperature probes	539
<i>Ralstonia pickettii</i>	
Fentanyl solutions	540
Chlorhexidine	541
Distilled water	541
Contaminated respiratory therapy solution	541, 542
<i>Serratia marcescens</i>	
Potable water	543
Contaminated antiseptics (i.e., benzalkonium chloride and chlorhexidine)	544–546
Contaminated disinfectants (i.e., quaternary ammonium compounds and glutaraldehyde)	547, 548
<i>Acinetobacter spp.</i>	
Medical equipment that collects moisture (e.g., mechanical ventilators, cool mist humidifiers, vaporizers, and mist tents)	549–556
Room humidifiers	553, 555
Environmental surfaces	557–564
<i>Enterobacter spp.</i>	
Humidifier water	565
Intravenous fluids	566–578
Unsterilized cotton swabs	573
Ventilators	565, 569
Rubber piping on a suctioning machine	565, 569
Blood gas analyzers	570

* This report describes intrinsic contamination (i.e., occurring during manufacture) prior to use by the health-care facility staff. All other entries reflect extrinsic sources of contamination.

Two additional gram-negative bacterial pathogens that can proliferate in moist environments are *Acinetobacter spp.* and *Enterobacter spp.*^{571, 572} Members of both genera are responsible for health-care-associated episodes of colonization, bloodstream infections, pneumonia, and urinary tract infections among medically compromised patients, especially those in ICUs and burn therapy units.^{566, 572–583} Infections caused by *Acinetobacter spp.* represent a significant clinical problem. Average infection rates are higher from July through October compared with rates from November through June.⁵⁸⁴ Mortality rates associated with *Acinetobacter* bacteremia are 17%–52%, and rates as high as 71% have been reported for pneumonia caused by infection with either *Acinetobacter spp.* or

Pseudomonas spp.^{574–576} Multi-drug resistance, especially in third generation cephalosporins for *Enterobacter* spp., contributes to increased morbidity and mortality.^{569, 572}

Patients and health-care workers contribute significantly to the environmental contamination of surfaces and equipment with *Acinetobacter* spp. and *Enterobacter* spp., especially in intensive care areas, because of the nature of the medical equipment (e.g., ventilators) and the moisture associated with this equipment.^{549, 571, 572, 585} Hand carriage and hand transfer are commonly associated with health-care-associated transmission of these organisms and for *S. marcescens*.⁵⁸⁶ *Enterobacter* spp. are primarily spread in this manner among patients by the hands of health-care workers.^{567, 587} *Acinetobacter* spp. have been isolated from the hands of 4%–33% of health-care workers in some studies,^{585–590} and transfer of an epidemic strain of *Acinetobacter* from patients' skin to health-care workers' hands has been demonstrated experimentally.⁵⁹¹ *Acinetobacter* infections and outbreaks have also been attributed to medical equipment and materials (e.g., ventilators, cool mist humidifiers, vaporizers, and mist tents) that may have contact with water of uncertain quality (e.g., rinsing a ventilator circuit in tap water).^{549–556} Strict adherence to hand hygiene helps prevent the spread of both *Acinetobacter* spp. and *Enterobacter* spp.^{577, 592}

Acinetobacter spp. have also been detected on dry environmental surfaces (e.g., bed rails, counters, sinks, bed cupboards, bedding, floors, telephones, and medical charts) in the vicinity of colonized or infected patients; such contamination is especially problematic for surfaces that are frequently touched.^{557–564} In two studies, the survival periods of *Acinetobacter baumannii* and *Acinetobacter calcoaceticus* on dry surfaces approximated that for *S. aureus* (e.g., 26–27 days).^{593, 594} Because *Acinetobacter* spp. may come from numerous sources at any given time, laboratory investigation of health-care-associated *Acinetobacter* infections should involve techniques to determine biotype, antibiotype, plasmid profile, and genomic fingerprinting (i.e., macrorestriction analysis) to accurately identify sources and modes of transmission of the organism(s).⁵⁹⁵

c. Infections and Pseudo-Infections Due to Nontuberculous Mycobacteria

NTM are acid-fast bacilli (AFB) commonly found in potable water. NTM include both saprophytic and opportunistic organisms. Many NTM are of low pathogenicity, and some measure of host impairment is necessary to enhance clinical disease.⁵⁹⁶ The four most common forms of human disease associated with NTM are a) pulmonary disease in adults; b) cervical lymph node disease in children; c) skin, soft tissue, and bone infections; and d) disseminated disease in immunocompromised patients.^{596, 597} Person-to-person acquisition of NTM infection, especially among immunocompetent persons, does not appear to occur, and close contacts of patients are not readily infected, despite the high numbers of organisms harbored by such patients.^{596, 598–600} NTM are spread via all modes of transmission associated with water. In addition to health-care-associated outbreaks of clinical disease, NTM can colonize patients in health-care facilities through consumption of contaminated water or ice or through inhalation of aerosols.^{601–605} Colonization following NTM exposure, particularly of the respiratory tract, occurs when a patient's local defense mechanisms are impaired; overt clinical disease does not develop.⁶⁰⁶ Patients may have positive sputum cultures in the absence of clinical disease.

Using tap water during patient procedures and specimen collection and in the final steps of instrument reprocessing can result in pseudo-outbreaks of NTM contamination.^{607–609} NTM pseudo-outbreaks of *Mycobacterium chelonae*, *M. gordonae*, and *M. xenopi* have been associated with both bronchoscopy and gastrointestinal endoscopy when a) tap water is used to provide irrigation to the site or to rinse off the viewing tip *in situ* or b) the instruments are inappropriately reprocessed with tap water in the final steps.^{610–612}

Table 14. Nontuberculous mycobacteria—environmental vehicles

Vehicles associated with infections or colonizations	References
<i>Mycobacterium abscessus</i>	
Inadequately sterilized medical instruments	613
<i>Mycobacterium avium</i> complex (MAC)	
Potable water	614–616
<i>Mycobacterium chelonae</i>	
Dialysis, reprocessed dialyzers	31, 32
Inadequately-sterilized medical instruments, jet injectors	617, 618
Contaminated solutions	619, 620
Hydrotherapy tanks	621
<i>Mycobacterium fortuitum</i>	
Aerosols from showers or other water sources	605, 606
Ice	602
Inadequately sterilized medical instruments	603
Hydrotherapy tanks	622
<i>Mycobacterium marinum</i>	
Hydrotherapy tanks	623
<i>Mycobacterium ulcerans</i>	
Potable water	624
Vehicles associated with pseudo-outbreaks	References
<i>Mycobacterium chelonae</i>	
Potable water used during bronchoscopy and instrument reprocessing	610
<i>Mycobacterium fortuitum</i>	
Ice	607
<i>Mycobacterium gordonae</i>	
Deionized water	611
Ice	603
Laboratory solution (intrinsically contaminated)	625
Potable water ingestion prior to sputum specimen collection	626
<i>Mycobacterium kansasii</i>	
Potable water	627
<i>Mycobacterium terrae</i>	
Potable water	608
<i>Mycobacterium xenopi</i>	
Potable water	609, 612, 627

NTM can be isolated from both natural and man-made environments. Numerous studies have identified various NTM in municipal water systems and in hospital water systems and storage tanks.^{615, 616, 624, 627–632} Some NTM species (e.g., *Mycobacterium xenopi*) can survive in water at 113°F (45°C), and can be isolated from hot water taps, which can pose a problem for hospitals that lower the temperature of their hot water systems.⁶²⁷ Other NTM (e.g., *Mycobacterium kansasii*, *M. gordonae*, *M. fortuitum*, and *M. chelonae*) cannot tolerate high temperatures and are associated more often with cold water lines and taps.⁶²⁹

NTM have a high resistance to chlorine; they can tolerate free chlorine concentrations of 0.05–0.2 mg/L (0.05–0.2 ppm) found at the tap.^{598, 633, 634} They are 20–100 times more resistant to chlorine compared with coliforms; slow-growing strains of NTM (e.g., *Mycobacterium avium* and *M. kansasii*) appear to be

more resistant to chlorine inactivation compared to fast-growing NTM.⁶³⁵ Slow-growing NTM species have also demonstrated some resistance to formaldehyde and glutaraldehyde, which has posed problems for reuse of hemodialyzers.³¹ The ability of NTM to form biofilms at fluid-surface interfaces (e.g., interior surfaces of water pipes) contributes to the organisms' resistance to chemical inactivation and provides a microenvironment for growth and proliferation.^{636, 637}

d. Cryptosporidiosis

Cryptosporidium parvum is a protozoan parasite that causes self-limiting gastroenteritis in normal hosts but can cause severe, life-threatening disease in immunocompromised patients. First recognized as a human pathogen in 1976, *C. parvum* can be present in natural and finished waters after fecal contamination from either human or animal sources.^{638–641}

The health risks associated with drinking potable water contaminated with minimal numbers of *C. parvum* oocysts are unknown.⁶⁴² It remains to be determined if immunosuppressed persons are more susceptible to lower doses of oocysts than are immunocompetent persons. One study demonstrated that a median 50% infectious dose (ID₅₀) of 132 oocysts of calf origin was sufficient to cause infection among healthy volunteers.⁶⁴³ In a second study, the same researchers found that oocysts obtained from infected foals (newborn horses) were infectious for human volunteers at median ID₅₀ of 10 oocysts, indicating that different strains or species of *Cryptosporidium* may vary in their infectivity for humans.⁶⁴⁴ In a small study population of 17 healthy adults with pre-existing antibody to *C. parvum*, the ID₅₀ was determined to be 1,880 oocysts, more than 20-fold higher than in seronegative persons.⁶⁴⁵ These data suggest that pre-existing immunity derived from previous exposures to *Cryptosporidium* offers some protection from infection and illness that ordinarily would result from exposure to low numbers of oocysts.^{645, 646}

Oocysts, particularly those with thick walls, are environmentally resistant, but their survival under natural water conditions is poorly understood. Under laboratory conditions, some oocysts remain viable and infectious in cold (41°F [5°C]) for months.⁶⁴¹ The prevalence of *Cryptosporidium* in the U.S. drinking water supply is notable. Two surveys of approximately 300 surface water supplies revealed that 55%–77% of the water samples contained *Cryptosporidium* oocysts.^{647, 648} Because the oocysts are highly resistant to common disinfectants (e.g., chlorine) used to treat drinking water, filtration of the water is important in reducing the risk of waterborne transmission. Coagulation-flocculation and sedimentation, when used with filtration, can collectively achieve approximately a 2.5 log₁₀ reduction in the number of oocysts.⁶⁴⁹ However, outbreaks have been associated with both filtered and unfiltered drinking water systems (e.g., the 1993 outbreak in Milwaukee, Wisconsin that affected 400,000 people).^{641, 650–652} The presence of oocysts in the water is not an absolute indicator that infection will occur when the water is consumed, nor does the absence of detectable oocysts guarantee that infection will not occur. Health-care-associated outbreaks of cryptosporidiosis primarily have been described among groups of elderly patients and immunocompromised persons.⁶⁵³

3. Water Systems in Health-Care Facilities

a. Basic Components and Point-of-Use Fixtures

Treated municipal water enters a health-care facility via the water mains and is distributed throughout the building(s) by a network of pipes constructed of galvanized iron, copper, and polyvinylchloride (PVC). The pipe runs should be as short as is practical. Where recirculation is employed, the pipe runs should be insulated and long dead legs avoided in efforts to minimize the potential for water stagnation, which favors the proliferation of *Legionella* spp. and NTM. In high-risk applications (e.g., PE areas for severely immunosuppressed patients), insulated recirculation loops should be incorporated as a design

feature. Recirculation loops prevent stagnation and insulation maintains return water temperature with minimal loss.

Each water service main, branch main, riser, and branch (to a group of fixtures) has a valve and a means to reach the valves via an access panel.¹²⁰ Each fixture has a stop valve. Valves permit the isolation of a portion of the water system within a facility during repairs or maintenance. Vacuum breakers and other similar devices in the lines prevent water from back-flowing into the system. All systems that supply water should be evaluated to determine risk for potential back siphonage and cross connections.

Health-care facilities generate hot water from municipal water using a boiler system. Hot water heaters and storage vessels for such systems should have a drainage facility at the lowest point, and the heating element should be located as close as possible to the bottom of the vessel to facilitate mixing and to prevent water temperature stratification. Those hot or cold water systems that incorporate an elevated holding tank should be inspected and cleaned annually. Lids should fit securely to exclude foreign materials.

The most common point-of-use fixtures for water in patient-care areas are sinks, faucets, aerators, showers, and toilets; eye-wash stations are found primarily in laboratories. The potential for these fixtures to serve as a reservoir for pathogenic microorganisms has long been recognized (Table 15).^{509, 654–656} Wet surfaces and the production of aerosols facilitate the multiplication and dispersion of microbes. The level of risk associated with aerosol production from point-of-use fixtures varies. Aerosols from shower heads and aerators have been linked to a limited number of clusters of gram-negative bacterial colonizations and infections, including Legionnaires disease, especially in areas where immunocompromised patients are present (e.g., surgical ICUs, transplant units, and oncology units).^{412, 415, 656–659} In one report, clinical infection was not evident among immunocompetent persons (e.g., hospital staff) who used hospital showers when *Legionella pneumophila* was present in the water system.⁶⁶⁰ Given the infrequency of reported outbreaks associated with faucet aerators, consensus has not been reached regarding the disinfection of or removal of these devices from general use. If additional clusters of infections or colonizations occur in high-risk patient-care areas, it may be prudent to clean and decontaminate the aerators or to remove them.^{658, 659} ASHRAE recommends cleaning and monthly disinfection of aerators in high-risk patient-care areas as part of *Legionella* control measures.⁶⁶¹ Although aerosols are produced with toilet flushing,^{662, 663} no epidemiologic evidence suggests that these aerosols pose a direct infection hazard.

Although not considered a standard point-of-use fixture, decorative fountains are being installed in increasing numbers in health-care facilities and other public buildings. Aerosols from a decorative fountain have been associated with transmission of *Legionella pneumophila* serogroup 1 infection to a small cluster of older adults.⁶⁶⁴ This hotel lobby fountain had been irregularly maintained, and water in the fountain may have been heated by submersed lighting, all of which favored the proliferation of *Legionella* in the system.⁶⁶⁴ Because of the potential for generations of infectious aerosols, a prudent prevention measure is to avoid locating these fixtures in or near high-risk patient-care areas and to adhere to written policies for routine fountain maintenance.¹²⁰

Table 15. Water and point-of-use fixtures as sources and reservoirs of waterborne pathogens*

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Potable water	<i>Pseudomonas</i> , gram-negative bacteria, NTM	Contact	Moderate	Follow public health guidelines.	(See Tables 12–14)

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Potable water	<i>Legionella</i>	Aerosol inhalation	Moderate	Provide supplemental treatment for water.	(See Table 11)
Holy water	Gram-negative bacteria	Contact	Low	Avoid contact with severe burn injuries. Minimize use among immunocompromised patients.	665
Dialysis water	Gram-negative bacteria	Contact	Moderate	Dialysate should be $\leq 2,000$ cfu/mL; water should be ≤ 200 cfu/mL.	2, 527, 666–668
Automated endoscope reprocessors and rinse water	Gram-negative bacteria	Contact	Moderate	Use and maintain equipment according to instructions; eliminate residual moisture by drying the channels (e.g., through alcohol rinse and forced air drying).	669–675
Water baths	<i>Pseudomonas</i> , <i>Burkholderia</i> , <i>Acinetobacter</i>	Contact	Moderate	Add germicide to the water; wrap transfusion products in protective plastic wrap if using the bath to modulate the temperature of these products.	29, 533, 676, 677
Tub immersion	<i>Pseudomonas</i> , <i>Enterobacter</i> , <i>Acinetobacter</i>	Contact	Moderate	Drain and disinfect tub after each use; consider adding germicide to the water; water in large hydrotherapy pools should be properly disinfected and filtered.	678–683
Ice and ice machines	NTM, <i>Enterobacter</i> , <i>Pseudomonas</i> , <i>Cryptosporidium</i>	Ingestion, contact	Moderate	Clean periodically; use automatic dispenser (avoid open chest storage compartments in patient areas).	601, 684–687
Faucet aerators	<i>Legionella</i>	Aerosol inhalation	Low		
Faucet aerators	<i>Legionella</i>	Aerosol inhalation	Moderate	Clean and disinfect monthly in high-risk patient areas; consider removing if additional infections occur.	415, 661
Faucet aerators	<i>Pseudomonas</i> , <i>Acinetobacter</i> , <i>Stenotrophomonas</i> , <i>Chryseobacterium</i>	Contact, droplet	Low	No precautions are necessary at present in immunocompetent patient-care areas.	658, 659, 688, 689
Sinks	<i>Pseudomonas</i>	Contact, droplet	Moderate	Use separate sinks for handwashing and disposal of contaminated fluids.	509, 653, 685–693
Showers	<i>Legionella</i>	Aerosol inhalation	Low	Provide sponge baths for hematopoietic stem cell transplant patients; avoid shower use for immunocompromised patients when <i>Legionella</i> is detected in facility water.	656

Reservoir	Associated pathogens	Transmission	Strength of evidence+	Prevention and control	References
Dental unit water lines	<i>Pseudomonas</i> , <i>Legionella</i> , <i>Sphingomonas</i> , <i>Acinetobacter</i>	Contact	Low	Clean water systems according to system manufacturer's instructions.	636, 694–696
Ice baths for thermodilution catheters	<i>Ewingella</i> , <i>Staphylococcus</i>	Contact	Low	Use sterile water.	697, 698
Decorative fountains	<i>Legionella</i>	Aerosol inhalation	Low	Perform regular maintenance, including water disinfection; avoid use in or near high-risk patient-care areas.	664
Eyewash stations	<i>Pseudomonas</i> , amoebae, <i>Legionella</i>	Contact	Low Minimum	Flush eyewash stations weekly; have sterile water available for eye flushes.	518, 699, 700
Toilets	Gram-negative bacteria	–	Minimum	Clean regularly; use good hand hygiene.	662
Flowers	Gram-negative bacteria, <i>Aspergillus</i>	–	Minimum	Avoid use in intensive care units and in immunocompromised patient-care settings.	515, 701, 702

* Modified from reference 654 and used with permission of the publisher (Slack, Inc.)

+ **Moderate:** occasional well-described outbreaks. **Low:** few well-described outbreaks. **Minimal:** actual infections not demonstrated.

b. Water Temperature and Pressure

Hot water temperature is usually measured at the point of use or at the point at which the water line enters equipment requiring hot water for proper operation.¹²⁰ Generally, the hot water temperature in hospital patient-care areas is no greater than a temperature within the range of 105°F–120°F (40.6°C–49°C), depending on the AIA guidance issued at the year in which the facility was built.¹²⁰ Hot water temperature in patient-care areas of skilled nursing-care facilities is set within a slightly lower range of 95°F–110°F (35°C–43.3°C) depending on the AIA guidance at the time of facility construction.¹²⁰ Many states have adopted a temperature setting in these ranges into their health-care regulations and building codes. ASHRAE, however, has recommended higher settings.⁶⁶¹ Steam jets or booster heaters are usually needed to meet the hot water temperature requirements in certain service areas of the hospital (e.g., the kitchen [120°F (49°C)] or the laundry [160°F (71°C)]).¹²⁰ Additionally, water lines may need to be heated to a particular temperature specified by manufacturers of specific hospital equipment. Hot-water distribution systems serving patient-care areas are generally operated under constant recirculation to provide continuous hot water at each hot-water outlet.¹²⁰ If a facility is or has a hemodialysis unit, then continuously circulated, cold treated water is provided to that unit.¹²⁰

To minimize the growth and persistence of gram-negative waterborne bacteria (e.g., thermophilic NTM and *Legionella* spp.),^{627, 703–709} cold water in health-care facilities should be stored and distributed at temperatures below 68°F (20°C); hot water should be stored above 140°F (60°C) and circulated with a minimum return temperature of 124°F (51°C),⁶⁶¹ or the highest temperature specified in state regulations and building codes. If the return temperature setting of 124°F (51°C) is permitted, then installation of preset thermostatic mixing valves near the point-of-use can help to prevent scalding. Valve maintenance is especially important in preventing valve failure, which can result in scalding. New shower systems in large buildings, hospitals, and nursing homes should be designed to permit mixing of hot and cold water near the shower head. The warm water section of pipe between the control valve and shower head should be self-draining. Where buildings can not be retrofitted, other

approaches to minimize the growth of *Legionella* spp. include a) periodically increasing the temperature to at least 150°F [66°C] at the point of use [i.e., faucets] and b) adding additional chlorine and flushing the water.^{661, 710, 711} Systems should be inspected annually to ensure that thermostats are functioning properly.

Adequate water pressure ensures sufficient water supplies for a) direct patient care; b) operation of water-cooled instruments and equipment [e.g., lasers, computer systems, telecommunications systems, and automated endoscope reprocessors⁷¹²]; c) proper function of vacuum suctioning systems; d) indoor climate control; and e) fire-protection systems. Maintaining adequate pressure also helps to ensure the integrity of the piping system.

c. Infection-Control Impact of Water System Maintenance and Repair

Corrective measures for water-system failures have not been studied in well-designed experiments; these measures are instead based on empiric engineering and infection-control principles. Health-care facilities can occasionally sustain both intentional cut-offs by the municipal water authority to permit new construction project tie-ins and unintentional disruptions in service when a water main breaks as a result of aging infrastructure or a construction accident. Vacuum breakers or other similar devices can prevent backflow of water in the facility's distribution system during water-disruption emergencies.¹¹ To be prepared for such an emergency, all health-care facilities need contingency plans that identify a) the total demand for potable water, b) the quantity of replacement water [e.g., bottled water] required for a minimum of 24 hours when the water system is down, c) mechanisms for emergency water distribution, and 4) procedures for correcting drops in water pressure that affect operation of essential devices and equipment that are driven or cooled by a water system [Table 16].⁷¹³

Table 16. Water demand in health-care facilities during water disruption emergencies

	Potable water	Bottled, sterile water
Water use needs	Drinking water Handwashing Cafeteria services Ice Manual flushing of toilets Patient baths, hygiene Hemodialysis Hydrotherapy Fire prevention (e.g., sprinkler systems) Surgery and critical care areas Laboratory services Laundry and central sterile services* Cooling towers+ Steam generation	Surgical scrub Emergency surgical procedures Pharmaceutical preparations Patient-care equipment (e.g., ventilators)§

* Arrange to have a contingency provision of these services from another resource, if possible (e.g., another health-care facility or contractor).

+ Some cooling towers may use a potable water source, but most units use non-potable water.

§ This item is included in the table under the assumption that electrical power is available during the water emergency.

Detailed, up-to-date plans for hot and cold water piping systems should be readily available for maintenance and repair purposes in case of system problems. Opening potable water systems for repair or construction and subjecting systems to water-pressure changes can result in water discoloration and dramatic increases in the concentrations of *Legionella* spp. and other gram-negative bacteria. The maintenance of a chlorine residual at all points within the piping system also offers some protection from entry of contamination to the pipes in the event of inadvertent cross-connection between potable and non-potable water lines. As a minimum preventive measure, ASHRAE recommends a thorough flushing of the system.⁶⁶¹ High-temperature flushing or hyperchlorination may also be appropriate

strategies to decrease potentially high concentrations of waterborne organisms. The decision to pursue either of these remediation strategies, however, should be made on a case-by-case basis. If only a portion of the system is involved, high temperature flushing or chlorination can be used on only that portion of the system.⁶⁶¹

When shock decontamination of hot water systems is necessary (e.g., after disruption caused by construction and after cross-connections), the hot water temperature should be raised to 160°F–170°F (71°C–77°C) and maintained at that level while each outlet around the system is progressively flushed. A minimum flush time of 5 minutes has been recommended;³ the optimal flush time is not known, however, and longer flush times may be necessary.⁷¹⁴ The number of outlets that can be flushed simultaneously depends on the capacity of the water heater and the flow capability of the system. Appropriate safety procedures to prevent scalding are essential. When possible, flushing should be performed when the fewest building occupants are present (e.g., during nights and weekends).

When thermal shock treatment is not possible, shock chlorination may serve as an alternative method.⁶⁶¹ Experience with this method of decontamination is limited, however, and high levels of free chlorine can corrode metals. Chlorine should be added, preferably overnight, to achieve a free chlorine residual of at least 2 mg/L (2 ppm) throughout the system.⁶⁶¹ This may require chlorination of the water heater or tank to levels of 20–50 mg/L (20–50 ppm). The pH of the water should be maintained at 7.0–8.0.⁶⁶¹ After completion of the decontamination, recolonization of the hot water system is likely to occur unless proper temperatures are maintained or a procedure such as continuous supplemental chlorination is continued.

Interruptions of the water supply and sewage spills are situations that require immediate recovery and remediation measures to ensure the health and safety of patients and staff.⁷¹⁵ When delivery of potable water through the municipal distribution system has been disrupted, the public water supplier must issue a “boil water” advisory if microbial contamination presents an immediate public health risk to customers. The hospital engineer should oversee the restoration of the water system in the facility and clear it for use when appropriate. Hospitals must maintain a high level of surveillance for waterborne disease among patients and staff after the advisory is lifted.⁶⁴²

Flooding from either external (e.g., from a hurricane) or internal sources (e.g., a water system break) usually results in property damage and a temporary loss of water and sanitation.^{716–718} JCAHO requires all hospitals to have plans that address facility response for recovery from both internal and external disasters.^{713, 719} The plans are required to discuss a) general emergency preparedness, b) staffing, c) regional planning among area hospitals, d) emergency supply of potable water, e) infection control and medical services needs, f) climate control, and g) remediation. The basic principles of structural recovery from flooding are similar to those for recovery from sewage contamination (Box 9 and 10). Following a major event (e.g., flooding), facilities may elect to conduct microbial sampling of water after the system is restored to verify that water quality has been returned to safe levels (<500 CFU/mL, heterotrophic plate count). This approach may help identify point-of-use fixtures that may harbor contamination as a result of design or engineering features.⁷²⁰ Medical records should be allowed to dry and then either photocopied or placed in plastic covers before returning them to the record.

Moisture meters can be used to assess water-damaged structural materials. If porous structural materials for walls have a moisture content of >20% after 72 hours, the affected material should be removed.^{266, 278, 313} The management of water-damaged structural materials is not strictly limited to major water catastrophes (e.g., flooding and sewage intrusions); the same principles are used to evaluate the damage from leaking roofs, point-of-use fixtures, and equipment. Additional sources of moisture include condensate on walls from boilers and poorly engineered humidification in HVAC systems.

Box 9. Recovery and remediation measures for water-related emergencies*

Potable water disruptions

Contingency plan items

- Ensure access to plumbing network so that repairs can be easily made.
- Provide sufficient potable water, either from bottled sources or truck delivery.
- Post advisory notices against consuming tap water, ice, or beverages made with water.
- Rope off or bag drinking fountains to designate these as being “out of service” until further notice.
- Rinse raw foods as needed in disinfected water.
- Disconnect ice machines whenever possible.+
- Postpone laundry services until after the water system is restored.

Water treatment

- Heat water to a rolling boil for ≥ 1 minute.

Remediation of the water system after the “boil water” advisory is rescinded

- Flush fixtures (e.g., faucets and drinking fountains) and equipment for several minutes and restart.
 - Run water softeners through a regeneration cycle.
 - Drain, disinfect, and refill water storage tanks, if needed.
 - Change pretreatment filters and disinfect the dialysis water system.
-

Sewage spills/malfunction

Overall strategy

- Move patients and clean/sterile supplies out of the area.
- Redirect traffic away from the area.
- Close the doors or use plastic sheeting to isolate the area prior to clean-up.
- Restore sewage system function first, then the potable water system (if both are malfunctioning).
- Remove sewage solids, drain the area, and let dry.

Remediation of the structure

- Hard surfaces: clean with detergent/disinfectant after the area has been drained.
- Carpeting, loose tiles, buckled flooring: remove and allow the support surface to dry; replace the items; wet down carpeting with a low-level disinfectant or sanitizer prior to removal to minimize dust dispersion to the air.
- Wallboard and other porous structural materials: remove and replace if they cannot be cleaned and dried within 72 hours.§

Furniture

- Hard surface furniture (e.g., metal or plastic furniture): clean and allow to dry.
- Wood furniture: let dry, sand the wood surface, and reapply varnish.
- Cloth furniture: replace.

Electrical equipment

- Replace if the item cannot be easily dismantled, cleaned, and reassembled.
-

* Material in this box is compiled from references 266, 278, 315, 713, 716–719, 721–729.

+ Ice machines should always be disconnected from the water source in advance of planned water disruptions.

§ Moisture meter readings should be <20% moisture content.

An exception to these recommendations is made for hemodialysis units where water is further treated either by portable water treatment or large-scale water treatment systems usually involving reverse osmosis (RO). In the United States, >97% of dialysis facilities use RO treatment for their water.⁷²¹ However, changing pre-treatment filters and disinfecting the system to prevent colonization of the RO membrane and microbial contamination down-stream of the pre-treatment filter are prudent measures.

Box 10. Contingency planning for flooding

General emergency preparedness

- Ensure that emergency electrical generators are not located in flood-prone areas of the facility.
- Develop alternative strategies for moving patients, water containers, medical records, equipment, and supplies in the event that the elevators are inoperable.
- Establish in advance a centralized base of operations with batteries, flashlights, and cellular phones.
- Ensure sufficient supplies of sandbags to place at the entrances and the area around boilers, incinerators, and generators.
- Establish alternative strategies for bringing core employees to the facility if high water prevents travel.

Staffing Patterns

- Temporarily reassign licensed staff as needed to critical care areas to provide manual ventilation and to perform vital assessments on patients.
- Designate a core group of employees to remain on site to keep all services operational if the facility remains open.
- Train all employees in emergency preparedness procedures.

Regional planning among are facilities for disaster management

- Incorporate community support and involvement (e.g., media alerts, news, and transportation).
- Develop in advance strategies for transferring patients, as needed.
- Develop strategies for sharing supplies and providing essential services among participating facilities (e.g., central sterile department services, and laundry services).
- Identify sources for emergency provisions (e.g., blood, emergency vehicles, and bottled water).

Medical services and infection control

- Use alcohol-based hand rubs in general patient-care areas.
- Postpone elective surgeries until full services are restored, or transfer these patients to other facilities.
- Consider using portable dialysis machines.+
- Provide an adequate supply of tetanus and hepatitis A immunizations for patients and staff.

Climate control

- Provide adequate water for cooling towers.§
-

* Material in this box was compiled from references 713, 716–719.

+ Portable dialysis machines require less water compared to the larger units situated in dialysis settings.

§ Water for cooling towers may need to be trucked in, especially if the tower uses a potable water source.

4. Strategies for Controlling Waterborne Microbial Contamination

a. Supplemental Treatment of Water with Heat and/or Chemicals

In addition to using supplemental treatment methods as remediation measures after inadvertent contamination of water systems, health-care facilities sometimes use special measures to control waterborne microorganisms on a sustained basis. This decision is most often associated with outbreaks of legionellosis and subsequent efforts to control legionellae,⁷²² although some facilities have tried supplemental measures to better control thermophilic NTM.⁶²⁷

The primary disinfectant for both cold and hot water systems is chlorine. However, chlorine residuals are expected to be low, and possibly nonexistent, in hot water tanks because of extended retention time in the tank and elevated water temperature. Flushing, especially that which removes sludge from the bottom of the tank, probably provides the most effective treatment of water systems. Unlike the situation for disinfecting cooling towers, no equivalent recommendations have been made for potable water systems, although specific intervention strategies have been published.^{403, 723} The principal approaches to disinfection of potable systems are heat flushing using temperatures 160°F–170°F (71°C–77°C), hyperchlorination, and physical cleaning of hot-water tanks.^{3, 403, 661} Potable systems are easily recolonized and may require continuous intervention (e.g., raising of hot water temperatures or continuous chlorination).^{403, 711} Chlorine solutions lose potency over time, thereby rendering the stocking of large quantities of chlorine impractical.

Some hospitals with hot water systems identified as the source of *Legionella* spp. have performed emergency decontamination of their systems by pulse (i.e., one-time) thermal disinfection/superheating or hyperchlorination.^{711, 714, 724, 725} After either of these procedures, hospitals either maintain their heated water with a minimum return temperature of 124°F (51°C) and cold water at <68°F (<20°C) or chlorinate their hot water to achieve 1–2 mg/L (1–2 ppm) of free residual chlorine at the tap.^{26, 437, 709–711, 726, 727} Additional measures (e.g., physical cleaning or replacement of hot-water storage tanks, water heaters, faucets, and shower heads) may be required to help eliminate accumulations of scale and sediment that protect organisms from the biocidal effects of heat and chlorine.^{457, 711} Alternative methods for controlling and eradicating legionellae in water systems (e.g., treating water with chlorine dioxide, heavy metal ions [i.e., copper/silver ions], ozone, and UV light) have limited the growth of legionellae under laboratory and operating conditions.^{728–742} Further studies on the long-term efficacy of these treatments are needed before these methods can be considered standard applications.

Renewed interest in the use of chloramines stems from concerns about adverse health effects associated with disinfectants and disinfection by-products.⁷⁴³ Monochloramine usage minimizes the formation of disinfection by-products, including trihalomethanes and haloacetic acids. Monochloramine can also reach distal points in a water system and can penetrate into bacterial biofilms more effectively than free chlorine.⁷⁴⁴ However, monochloramine use is limited to municipal water treatment plants and is currently not available to health-care facilities as a supplemental water-treatment approach. A recent study indicated that 90% of Legionnaires disease outbreaks associated with drinking water could have been prevented if monochloramine rather than free chlorine has been used for residual disinfection.⁷⁴⁵ In a retrospective comparison of health-care-associated Legionnaires disease incidence in central Texas hospitals, the same research group documented an absence of cases in facilities located in communities with monochloramine-treated municipal water.⁷⁴⁶ Additional data are needed regarding the effectiveness of using monochloramine before its routine use as a disinfectant in water systems can be recommended. No data have been published regarding the effectiveness of monochloramine installed at the level of the health-care facility.

Additional filtration of potable water systems is not routinely necessary. Filters are used in water lines in dialysis units, however, and may be inserted into the lines for specific equipment (e.g., endoscope washers and disinfectors) for the purpose of providing bacteria-free water for instrument reprocessing. Additionally, an RO unit is usually added to the distribution system leading to PE areas.

b. Primary Prevention of Legionnaires Disease (No Cases Identified)

The primary and secondary environmental infection-control strategies described in this section on the guideline pertain to health-care facilities without transplant units. Infection-control measures specific to PE or transplant units (i.e., patient-care areas housing patients at the highest risk for morbidity and mortality from *Legionella* spp. infection) are described in the subsection titled *Preventing Legionnaires Disease in Protective Environments*.

Health-care facilities use at least two general strategies to prevent health-care-associated legionellosis when no cases or only sporadic cases have been detected. The first is an environmental surveillance approach involving periodic culturing of water samples from the hospital's potable water system to monitor for *Legionella* spp.^{747–750} If any sample is culture-positive, diagnostic testing is recommended for all patients with health-care-associated pneumonia.^{748, 749} In-house testing is recommended for facilities with transplant programs as part of a comprehensive treatment/management program. If $\geq 30\%$ of the samples are culture-positive for *Legionella* spp., decontamination of the facility's potable water system is warranted.⁷⁴⁸ The premise for this approach is that no cases of health-care-associated legionellosis can occur if *Legionella* spp. are not present in the potable water system, and, conversely, cases of health-care-associated legionellosis could potentially occur if *Legionella* spp. are cultured from the water.^{26, 751} Physicians who are informed that the hospital's potable water system is culture-positive

for *Legionella* spp. are more likely to order diagnostic tests for legionellosis.

A potential advantage of the environmental surveillance approach is that periodic culturing of water is less costly than routine laboratory diagnostic testing for all patients who have health-care-associated pneumonia. The primary argument against this approach is that, in the absence of cases, the relationship between water-culture results and legionellosis risk remains undefined.³ *Legionella* spp. can be present in the water systems of buildings⁷⁵² without being associated with known cases of disease.^{437, 707, 753} In a study of 84 hospitals in Québec, 68% of the water systems were found to be colonized with *Legionella* spp., and 26% were colonized at >30% of sites sampled; cases of Legionnaires disease, however, were infrequently reported from these hospitals.⁷⁰⁷

Other factors also argue against environmental surveillance. Interpretation of results from periodic water culturing might be confounded by differing results among the sites sampled in a single water system and by fluctuations in the concentration of *Legionella* spp. at the same site.^{709, 754} In addition, the risk for illness after exposure to a given source might be influenced by several factors other than the presence or concentration of organisms, including a) the degree to which contaminated water is aerosolized into respirable droplets, b) the proximity of the infectious aerosol to the potential host, c) the susceptibility of the host, and d) the virulence properties of the contaminating strain.^{755–757} Thus, data are insufficient to assign a level of disease risk even on the basis of the number of colony-forming units detected in samples from areas for immunocompetent patients. Conducting environmental surveillance would obligate hospital administrators to initiate water-decontamination programs if *Legionella* spp. are identified. Therefore, periodic monitoring of water from the hospital's potable water system and from aerosol-producing devices is not widely recommended in facilities that have not experienced cases of health-care-associated legionellosis.^{661, 758}

The second strategy to prevent and control health-care-associated legionellosis is a clinical approach, in which providers maintain a high index of suspicion for legionellosis and order appropriate diagnostic tests (i.e., culture, urine antigen, and direct fluorescent antibody [DFA] serology) for patients with health-care-associated pneumonia who are at high risk for legionellosis and its complications.^{437, 759, 760} The testing of autopsy specimens can be included in this strategy should a death resulting from health-care-associated pneumonia occur. Identification of one case of definite or two cases of possible health-care-associated Legionnaires disease should prompt an epidemiologic investigation for a hospital source of *Legionella* spp., which may involve culturing the facility's water for *Legionella*. Routine maintenance of cooling towers, and use of sterile water for the filling and terminal rinsing of nebulization devices and ventilation equipment can help to minimize potential sources of contamination. Circulating potable water temperatures should match those outlined in the subsection titled *Water Temperature and Pressure*, as permitted by state code.

c. Secondary prevention of Legionnaires Disease (With Identified Cases)

The indications for a full-scale environmental investigation to search for and subsequently decontaminate identified sources of *Legionella* spp. in health-care facilities without transplant units have not been clarified; these indications would likely differ depending on the facility. Case categories for health-care-associated Legionnaires disease in facilities without transplant units include definite cases (i.e., laboratory-confirmed cases of legionellosis that occur in patients who have been hospitalized continuously for ≥ 10 days before the onset of illness) and possible cases (i.e., laboratory-confirmed infections that occur 2–9 days after hospital admission).³ In settings in which as few as one to three health-care-associated cases are recognized over several months, intensified surveillance for Legionnaires disease has frequently identified numerous additional cases.^{405, 408, 432, 453, 739, 759, 760} This finding suggests the need for a low threshold for initiating an investigation after laboratory confirmation of cases of health-care-associated legionellosis. When developing a strategy for responding to such a finding, however, infection-control personnel should consider the level of risk for health-care-

associated acquisition of, and mortality from, *Legionella* spp. infection at their particular facility.

An epidemiologic investigation conducted to determine the source of *Legionella* spp. involves several important steps (Box 11). Laboratory assessment is crucial in supporting epidemiologic evidence of a link between human illness and a specific environmental source.⁷⁶¹ Strain determination from subtype analysis is most frequently used in these investigations.^{410, 762–764} Once the environmental source is established and confirmed with laboratory support, supplemental water treatment strategies can be initiated as appropriate.

Box 11. Steps in an epidemiologic investigation for legionellosis

Review medical and microbiologic records.

Initiate active surveillance to identify all recent or ongoing cases.

Develop a line listing of cases by time, place, and person.

Determine the type of epidemiologic investigation needed for assessing risk factors:

- Case-control study,
- Cohort study.

Gather and analyze epidemiologic information:

- Evaluate risk factors associated with potential environmental exposures (e.g., showers, cooling towers, and respiratory-therapy equipment).

Collect water samples:

- Sample environmental sources implicated by epidemiologic investigation,
- Sample other potential source of water aerosols.

Subtype strains of *Legionella* spp. cultured from both patients and environmental sources.

Review autopsy records and include autopsy specimens in diagnostic testing.

The decision to search for hospital environmental sources of *Legionella* spp. and the choice of procedures to eradicate such contamination are based on several considerations, as follows: a) the hospital's patient population; b) the cost of an environmental investigation and institution of control measures to eradicate *Legionella* spp. from the water supply,^{765–768} and c) the differential risk, based on host factors, for acquiring health-care-associated legionellosis and developing severe and fatal infection.

d. Preventing Legionnaires Disease in Protective Environments

This subsection outlines infection-control measures applicable to those health-care facilities providing care to severely immunocompromised patients. Indigenous microorganisms in the tap water of these facilities may pose problems for such patients. These measures are designed to prevent the generation of potentially infectious aerosols from water and the subsequent exposure of PE patients or other immunocompromised patients (e.g., transplant patients) (Table 17). Infection-control measures that address the use of water with medical equipment (e.g., ventilators, nebulizers, and equipment humidifiers) are described in other guidelines and publications.^{3, 455}

If one case of laboratory-confirmed, health-care-associated Legionnaires disease is identified in a patient in a solid-organ transplant program or in PE (i.e., an inpatient in PE for all or part of the 2–10 days prior to onset of illness) or if two or more laboratory-confirmed cases occur among patients who had visited an outpatient PE setting, the hospital should report the cases to the local and state health departments. The hospital should then initiate a thorough epidemiologic and environmental investigation to determine the likely environmental sources of *Legionella* spp.⁹ The source of *Legionella* should be decontaminated or removed. Isolated cases may be difficult to investigate. Because transplant recipients are at substantially higher risk for disease and death from legionellosis

compared with other hospitalized patients, periodic culturing for *Legionella* spp. in water samples from the potable water in the solid-organ transplant and/or PE unit can be performed as part of an overall strategy to prevent Legionnaires disease in PE units.^{9, 431, 710, 769} The optimal methodology (i.e., frequency and number of sites) for environmental surveillance cultures in PE units has not been determined, and the cost-effectiveness of this strategy has not been evaluated. Because transplant recipients are at high risk for Legionnaires disease and because no data are available to determine a safe concentration of legionellae organisms in potable water, the goal of environmental surveillance for *Legionella* spp. should be to maintain water systems with no detectable organisms.^{9, 431} Culturing for legionellae may be used to assess the effectiveness of water treatment or decontamination methods, a practice that provides benefits to both patients and health-care workers.^{767, 770}

Table 17. Additional infection-control measures to prevent exposure of high-risk patients to waterborne pathogens

Measures	References
<ul style="list-style-type: none"> • Restrict patients from taking showers if the water is contaminated with <i>Legionella</i> spp. • Use water that is not contaminated with <i>Legionella</i> spp. for patients' sponge baths. • Provide sterile water for drinking, tooth brushing, or for flushing nasogastric tubes. • Perform supplemental treatment of the water for the unit. • Consider periodic monitoring (i.e., culturing) of the unit water supply for <i>Legionella</i> spp. • Remove shower heads and faucet aerators monthly for cleaning.* • Use a 500–600 ppm (1:100 v/v dilution) solution of sodium hypochlorite to disinfect shower heads and faucet aerators.* • Do not use large-volume room air humidifiers that create aerosols unless these are subjected to cleaning and high-level disinfection daily and filled with distilled water. • Eliminate water-containing bath toys.+ 	<ul style="list-style-type: none"> • 407, 412, 654, 655, 658 • 9 • 9, 412 • 732 • 9, 431 • 661 • 661 • 3 • 30

* These measures can be considered in settings where legionellosis cases have occurred. These measures are not generally recommended in routine patient-care setting..

+ These items have been associated with outbreaks of *Pseudomonas*.

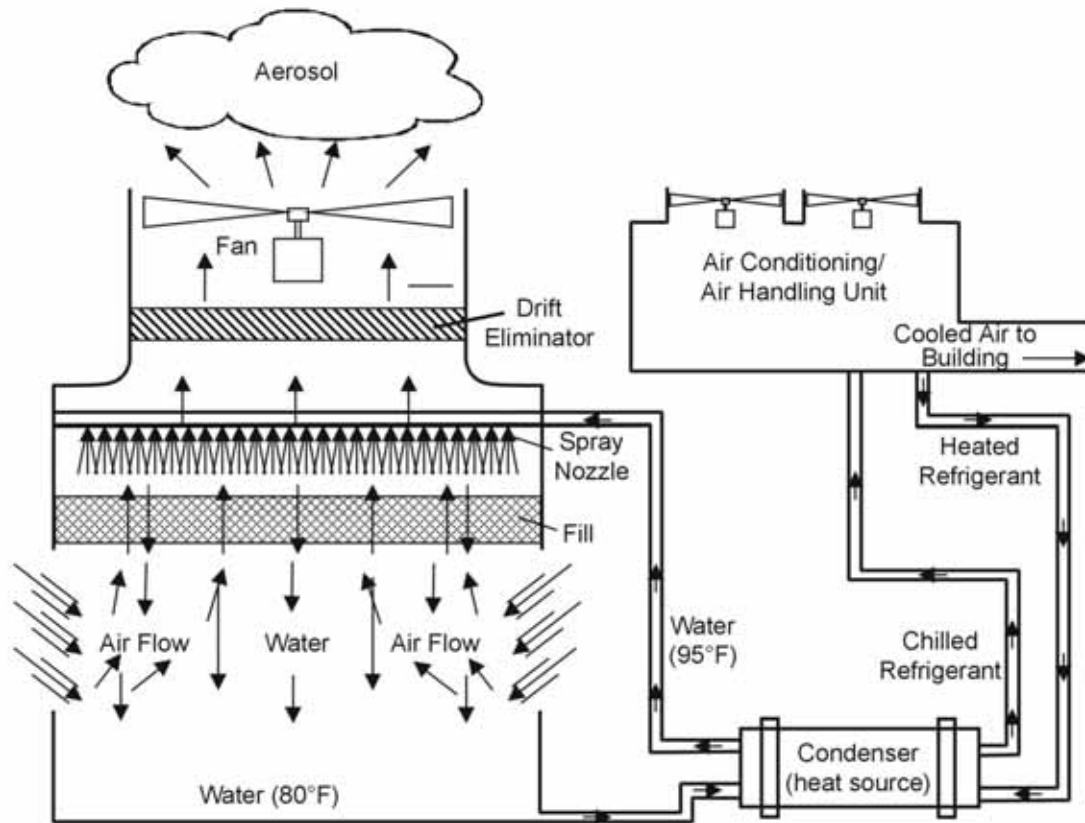
Protecting patient-care devices and instruments from inadvertent tap water contamination during room cleaning procedures is also important in any immunocompromised patient-care area. In a recent outbreak of gram-negative bacteremias among open-heart-surgery patients, pressure-monitoring equipment that was assembled and left uncovered overnight prior to the next day's surgeries was inadvertently contaminated with mists and splashing water from a hose-disinfectant system used for cleaning.⁷⁷¹

5. Cooling Towers and Evaporative Condensers

Modern health-care facilities maintain indoor climate control during warm weather by use of cooling towers (large facilities) or evaporative condensers (smaller buildings). A cooling tower is a wet-type, evaporative heat transfer device used to discharge to the atmosphere waste heat from a building's air conditioning condensers (Figure 5).^{772, 773} Warm water from air-conditioning condensers is piped to the cooling tower where it is sprayed downward into a counter- or cross-current air flow. To accelerate heat transfer to the air, the water passes over the fill, which either breaks water into droplets or causes it to spread into a thin film.^{772, 773} Most systems use fans to move air through the tower, although some large industrial cooling towers rely on natural draft circulation of air. The cooled water from the tower is piped back to the condenser, where it again picks up heat generated during the process of chilling the system's refrigerant. The water is cycled back to the cooling tower to be cooled. Closed-circuit cooling towers and evaporative condensers are also evaporative heat-transfer devices. In these systems, the

process fluid (e.g., water, ethylene glycol/water mixture, oil, or a condensing refrigerant) does not directly contact the cooling air, but is contained inside a coil assembly.⁶⁶¹

Figure 5. Diagram of a typical air conditioning (induced draft) cooling tower*



Water temperatures are approximate and may differ substantially according to system use and design. Warm water from the condenser (or chiller) is sprayed downward into a counter- or cross-current air flow. Water passes over the fill (a component of the system designed to increase the surface area of the water exposed to air), and heat from the water is transferred to the air. Some of the water becomes aerosolized during this process, although the volume of aerosol discharged to the air can be reduced by the placement of a drift eliminator. Water cooled in the tower returns to the heat source to cool refrigerant from the air conditioning unit.

* This figure is reprinted with permission of the publisher of reference 773 (Plenum Medical).

Cooling towers and evaporative condensers incorporate inertial stripping devices called drift eliminators to remove water droplets generated within the unit. Although the effectiveness of these eliminators varies substantially depending on design and condition, some water droplets in the size range of $<5 \mu\text{m}$ will likely leave the unit, and some larger droplets leaving the unit may be reduced to $\leq 5 \mu\text{m}$ by evaporation. Thus, even with proper operation, a cooling tower or evaporative condenser can generate and expel respirable water aerosols. If either the water in the unit's basin or the make-up water (added to replace water lost to evaporation) contains *Legionella* spp. or other waterborne microorganisms, these organisms can be aerosolized and dispersed from the unit.⁷⁷⁴ Clusters of both Legionnaires disease and Pontiac fever have been traced to exposure to infectious water aerosols originating from cooling towers and evaporative condensers contaminated with *Legionella* spp. Although most of these outbreaks have been community-acquired episodes of pneumonia,⁷⁷⁵⁻⁷⁸² health-care-associated Legionnaires disease

has been linked to cooling tower aerosol exposure.^{404, 405} Contaminated aerosols from cooling towers on hospital premises gained entry to the buildings either through open windows or via air handling system intakes located near the tower equipment.

Cooling towers and evaporative condensers provide ideal ecological niches for *Legionella* spp. The typical temperature of the water in cooling towers ranges from 85°F–95°F (29°C–35°C), although temperatures can be above 120°F (49°C) and below 70°F (21°C) depending on system heat load, ambient temperature, and operating strategy.⁶⁶¹ An Australian study of cooling towers found that legionellae colonized or multiplied in towers with basin temperatures above 60.8°F (16°C), and multiplication became explosive at temperatures above 73.4°F (23°C).⁷⁸³ Water temperature in closed-circuit cooling towers and evaporative condensers is similar to that in cooling towers. Considerable variation in the piping arrangement occurs. In addition, stagnant areas or dead legs may be difficult to clean or penetrate with biocides.

Several documents address the routine maintenance of cooling towers, evaporative condensers, and whirlpool spas.^{661, 784–787} They suggest following manufacturer's recommendations for cleaning and biocide treatment of these devices; all health-care facilities should ensure proper maintenance for their cooling towers and evaporative condensers, even in the absence of *Legionella* spp (Appendix C). Because cooling towers and evaporative condensers can be shut down during periods when air conditioning is not needed, this maintenance cleaning and treatment should be performed before starting up the system for the first time in the warm season.⁷⁸² Emergency decontamination protocols describing cleaning procedures and hyperchlorination for cooling towers have been developed for towers implicated in the transmission of legionellosis.^{786, 787}

6. Dialysis Water Quality and Dialysate

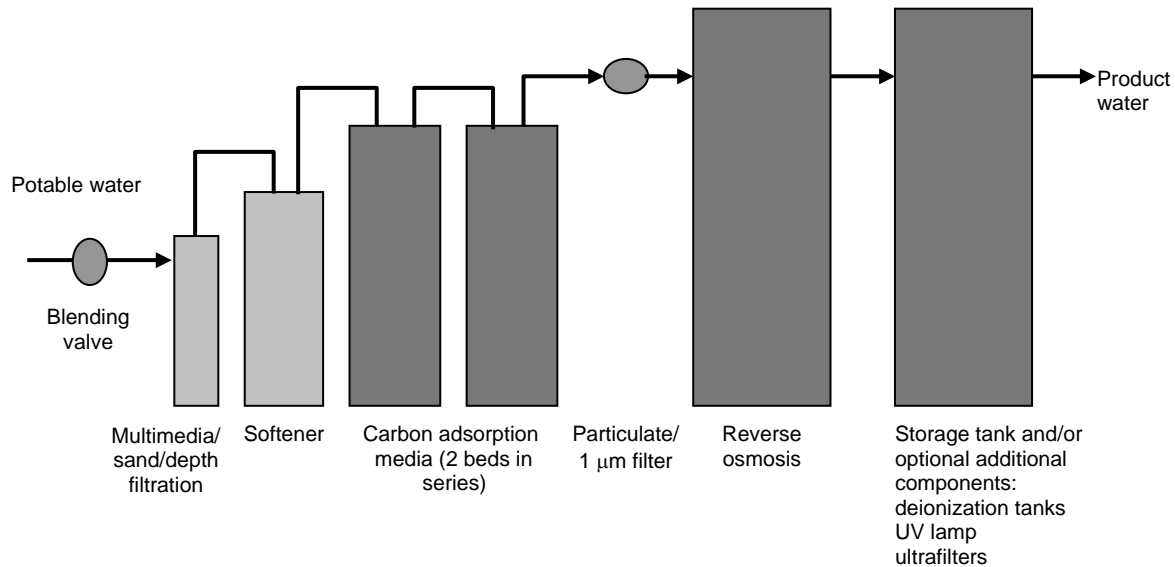
a. Rationale for Water Treatment in Hemodialysis

Hemodialysis, hemofiltration, and hemodiafiltration require special water-treatment processes to prevent adverse patient outcomes of dialysis therapy resulting from improper formulation of dialysate with water containing high levels of certain chemical or biological contaminants. The Association for the Advancement of Medical Instrumentation (AAMI) has established chemical and microbiologic standards for the water used to prepare dialysate, substitution fluid, or to reprocess hemodialyzers for renal replacement therapy.^{788–792} The AAMI standards address: a) equipment and processes used to purify water for the preparation of concentrates and dialysate and the reprocessing of dialyzers for multiple use and b) the devices used to store and distribute this water. Future revisions to these standards may include hemofiltration and hemodiafiltration.

Water treatment systems used in hemodialysis employ several physical and/or chemical processes either singly or in combination (Figure 6). These systems may be portable units or large systems that feed several rooms. In the United States, >97% of maintenance hemodialysis facilities use RO alone or in combination with deionization.⁷⁹³ Many acute-care facilities use portable hemodialysis machines with attached portable water treatment systems that use either deionization or RO. These machines were exempted from earlier versions of AAMI recommendations, but given current knowledge about toxic exposures to and inflammatory processes in patients new to dialysis, these machines should now come into compliance with current AAMI recommendations for hemodialysis water and dialysate quality.^{788, 789} Previous recommendations were based on the assumption that acute-care patients did not experience the same degree of adverse effects from short-term, cumulative exposures to either chemicals or microbiologic agents present in hemodialysis fluids compared with the effects encountered by patients during chronic, maintenance dialysis.^{788, 789} Additionally, JCAHO is reviewing inpatient

practices and record-keeping for dialysis (acute and maintenance) for adherence to AAMI standards and recommended practices.

Figure 6. Dialysis water treatment system*



* See text for description of the placement and function of these components.

Neither the water used to prepare dialysate nor the dialysate itself needs to be sterile, but tap water can not be used without additional treatment. Infections caused by rapid-growing NTM (e.g., *Mycobacterium chelonae* and *M. abscessus*) present a potential risk to hemodialysis patients (especially those in hemodialyzer reuse programs) if disinfection procedures to inactivate mycobacteria in the water (low-level disinfection) and the hemodialyzers (high-level disinfection) are inadequate.^{31, 32, 633} Other factors associated with microbial contamination in dialysis systems could involve the water treatment system, the water and dialysate distribution systems, and the type of hemodialyzer.^{666, 667, 794-799} Understanding the various factors and their influence on contamination levels is the key to preventing high levels of microbial contamination in dialysis therapy.

In several studies, pyrogenic reactions were demonstrated to have been caused by lipopolysaccharide or endotoxin associated with gram-negative bacteria.^{794, 800-803} Early studies demonstrated that parenteral exposure to endotoxin at a concentration of 1 ng/kg body weight/hour was the threshold dose for producing pyrogenic reactions in humans, and that the relative potencies of endotoxin differ by bacterial species.^{804, 805} Gram-negative water bacteria (e.g., *Pseudomonas* spp.) have been shown to multiply rapidly in a variety of hospital-associated fluids that can be used as supply water for hemodialysis (e.g., distilled water, deionized water, RO water, and softened water) and in dialysate (a balanced salt solution made with this water).⁸⁰⁶ Several studies have demonstrated that the attack rates of pyrogenic reactions are directly associated with the number of bacteria in dialysate.^{666, 667, 807} These studies provided the rationale for setting the heterotrophic bacteria standards in the first AAMI hemodialysis guideline at $\leq 2,000$ CFU/mL in dialysate and one log lower (≤ 200 CFU/mL) for the water used to prepare dialysate.^{668, 788} If the level of bacterial contamination exceeded 200 CFU/mL in water, this level could be amplified in the system and effectively constitute a high inoculum for dialysate at the start of a

dialysis treatment.^{807, 808} Pyrogenic reactions did not appear to occur when the level of contamination was below 2,000 CFU/mL in dialysate unless the source of the endotoxin was exogenous to the dialysis system (i.e., present in the community water supply). Endotoxins in a community water supply have been linked to the development of pyrogenic reactions among dialysis patients.⁷⁹⁴

Whether endotoxin actually crosses the dialyzer membrane is controversial. Several investigators have shown that bacteria growing in dialysate-generated products that could cross the dialyzer membrane.^{809,}

⁸¹⁰ Gram-negative bacteria growing in dialysate have produced endotoxins that in turn stimulated the production of anti-endotoxin antibodies in hemodialysis patients;^{801, 811} these data suggest that bacterial endotoxins, although large molecules, cross dialyzer membranes either intact or as fragments. The use of the very permeable membranes known as high-flux membranes (which allow large molecules [e.g., β_2 microglobulin] to traverse the membrane) increases the potential for passage of endotoxins into the blood path. Several studies support this contention. In one such study, an increase in plasma endotoxin concentrations during dialysis was observed when patients were dialyzed against dialysate containing 10^3 – 10^4 CFU/mL *Pseudomonas* spp.⁸¹² *In vitro* studies using both radiolabeled lipopolysaccharide and biologic assays have demonstrated that biologically active substances derived from bacteria found in dialysate can cross a variety of dialyzer membranes.^{802, 813–816} Patients treated with high-flux membranes have had higher levels of anti-endotoxin antibodies than subjects or patients treated with conventional membranes.⁸¹⁷ Finally, since 1989, 19%–22% of dialysis centers have reported pyrogenic reactions in the absence of septicemia.^{818, 819}

Investigations of adverse outcomes among patients using reprocessed dialyzers have demonstrated a greater risk for developing pyrogenic reactions when the water used to reprocess these devices contained >6 ng/mL endotoxin and $>10^4$ CFU/mL bacteria.⁸²⁰ In addition to the variability in endotoxin assays, host factors also are involved in determining whether a patient will mount a response to endotoxin.⁸⁰³ Outbreak investigations of pyrogenic reactions and bacteremias associated with hemodialyzer reuse have demonstrated that pyrogenic reactions are prevented once the endotoxin level in the water used to reprocess the dialyzers is returned to below the AAMI standard level.⁸²¹

Reuse of dialyzers and use of bicarbonate dialysate, high-flux dialyzer membranes, or high-flux dialysis may increase the potential for pyrogenic reactions if the water in the dialysis setting does not meet standards.^{796–798} Although investigators have been unable to demonstrate endotoxin transfer across dialyzer membranes,^{803, 822, 823} the preponderance of reports now supports the ability of endotoxin to transfer across at least some high-flux membranes under some operating conditions. In addition to the acute risk of pyrogenic reactions, indirect evidence is increasingly demonstrating that chronic exposure to low amounts of endotoxin may play a role in some of the long-term complications of hemodialysis therapy. Patients treated with ultrafiltered dialysate for 5–6 months have demonstrated a decrease in serum β_2 microglobulin concentrations and a decrease in markers of an inflammatory response.^{824–826} In studies of longer duration, use of microbiologically ultrapure dialysate has been associated with a decreased incidence of β_2 microglobulin-associated amyloidosis.^{827, 828}

Although patient benefit likely is associated with the use of ultrapure dialysate, no consensus has been reached regarding the potential adoption of this as standard in the United States. Debate continues regarding the bacterial and endotoxin limits for dialysate. As advances in water treatment and hemodialysis processes occur, efforts are underway to move improved technology from the manufacturer out into the user community. Cost-benefit studies, however, have not been done, and substantially increased costs to implement newer water treatment modalities are anticipated.

To reconcile AAMI documents with current International Organization for Standardization (ISO) format, AAMI has determined that its hemodialysis standards will be discussed in the following four installments: RD 5 for hemodialysis equipment, RD 62 for product water quality, RD 47 for dialyzer

reprocessing, and RD 52 for dialysate quality. The Renal Diseases and Dialysis Committee of AAMI is expected to finalize and promulgated the dialysate standard pertinent to the user community (RD 52), adopting by reference the bacterial and endotoxin limits in product water as currently outlined in the AAMI standard that applies to systems manufacturers (RD 62). At present, the user community should continue to observe water quality and dialysate standards as outlined in AAMI RD 5 (Hemodialysis Systems, 1992) and AAMI RD 47 (Reuse of Hemodialyzers, 1993) until the new RD 52 standard becomes available (Table 18).^{789, 791}

Table 18. Microbiologic limits for hemodialysis fluids*

Hemodialysis fluid	Maximum total heterotrophs (CFU/mL)+	Maximum endotoxin level (EU/mL)§
<i>Present standard</i>		
Product water¶		
Used to prepare dialysate	200	No standard
Used to reprocess dialyzers	200	5
Dialysate	2,000	No standard
<i>Proposed standard**</i>		
Product water	200	2
Dialysate	200	2

* The material in this table was compiled from references 789 and 791 (ANSI/AAMI standards RD 5-1992 and ANSI/AAMI RD 47-1993).

+ Colony forming units per milliliter.

§ Endotoxin units per milliliter.

¶ Product water presently includes water used to prepare dialysate and water used to reprocess dialyzers.

** Dialysate for hemodialysis, RD 52, under development, American National Standards Institute, Association for the Advancement of Medical Instrumentation (AAMI).

The current AAMI standard directed at systems manufacturers (RD 62 [Water Treatment Equipment for Hemodialysis Applications, 2001]) now specifies that all product water used to prepare dialysate or to reprocess dialyzers for multiple use should contain <2 endotoxin units per milliliter (EU/mL).⁷⁹² A level of 2 EU/mL was chosen as the upper limit for endotoxin because this level is easily achieved with contemporary water treatment systems using RO and/or ultrafiltration. CDC has advocated monthly endotoxin testing along with microbiologic assays of water, because endotoxin activity may not correspond to the total heterotrophic plate counts.⁸²⁹ Additionally, the current AAMI standard RD 62 for manufacturers includes action levels for product water. Because 48 hours can elapse between the time of sampling water for microbial contamination and the time when results are received, and because bacterial proliferation can be rapid, action levels for microbial counts and endotoxin concentrations are reported as 50 CFU/mL and 1 EU/mL, respectively, in this revision of the standard.⁷⁹² These recommendations will allow users to initiate corrective action before levels exceed the maximum levels established by the standard.

In hemodialysis, the net movement of water is from the blood to the dialysate, although within the dialyzer, local movement of water from the dialysate to the blood through the phenomenon of back-filtration may occur, particularly in dialyzers with highly permeable membranes.⁸³⁰ In contrast, hemofiltration and hemodiafiltration feature infusion of large volumes of electrolyte solution (20–70 L) into the blood. Increasingly, this electrolyte solution is being prepared on-line from water and concentrate. Because of the large volumes of fluid infused, AAMI considered the necessity of setting more stringent requirements for water to be used in this application, but this organization has not yet established these because of lack of expert consensus and insufficient experience with on-line therapies in the United States. On-line hemofiltration and hemodiafiltration systems use sequential ultrafiltration as the final step in the preparation of infusion fluid. Several experts from AAMI concur that these

point-of-use ultrafiltration systems should be capable of further reducing the bacteria and endotoxin burden of solutions prepared from water meeting the requirements of the AAMI standard to a safe level for infusion.

b. Microbial Control Strategies

The strategy for controlling massive accumulations of gram-negative water bacteria and NTM in dialysis systems primarily involves preventing their growth through proper disinfection of water-treatment systems and hemodialysis machines. Gram-negative water bacteria, their associated lipopolysaccharides (bacterial endotoxins), and NTM ultimately come from the community water supply, and levels of these bacteria can be amplified depending on the water treatment system, dialysate distribution system, type of dialysis machine, and method of disinfection (Table 19).^{634, 794, 831} Control strategies are designed to reduce levels of microbial contamination in water and dialysis fluid to relatively low levels but not to completely eradicate it.

Table 19. Factors influencing microbial contamination in hemodialysis systems

Factors	Comments
<u>Water supply</u> Source of community water Ground water Surface water	Contains endotoxin and bacteria Contains high levels of endotoxin and bacteria
<u>Water treatment at the dialysis center</u> None Filtration Prefilter Absolute filter (depth or membrane filter) Activated carbon filter	Not recommended Particulate filter to protect equipment; does not remove microorganisms Removes bacteria, however, unless the filter is changed frequently or disinfected, bacteria will accumulate and grow through the filter; acts as a significant reservoir of bacteria and endotoxin Removes organics and available chlorine or chloramines; acts as a significant reservoir of bacteria and endotoxin
<u>Water treatment devices</u> Deionization/ion-exchange softener Reverse osmosis (RO) Ultraviolet light Ultrafilter	Both softeners and deionizers are significant reservoirs of bacteria and do not remove endotoxin. Removes bacteria and endotoxin, but must be disinfected; operates at high water pressure Kills some bacteria, but there is no residual; ultraviolet-resistant bacteria can develop if the unit is not properly maintained Removes bacteria and endotoxin; operates on normal line pressure; can be positioned distal to deionizer; must be disinfected
<u>Water and dialysate distribution system</u> Distribution pipes Size Construction Elevation Storage tanks	Oversized diameter and length decrease fluid flow and increase bacterial reservoir for both treated water and centrally-prepared dialysate. Rough joints, dead ends, unused branches, and polyvinyl chloride (PVC) piping can act as bacterial reservoirs. Outlet taps should be located at the highest elevation to prevent loss of disinfectant; keep a recirculation loop in the system; flush unused ports routinely. Tanks are undesirable because they act as a reservoir for water bacteria; if tanks are present, they must be routinely scrubbed and disinfected.
<u>Dialysis machines</u> Single-pass Recirculating single-pass or recirculating (batch)	Disinfectant should have contact with all parts of the machine that are exposed to water or dialysis fluid. Recirculating pumps and machine design allow for massive contamination levels if not properly disinfected; overnight chemical germicide treatment is recommended.

Two components of hemodialysis water distribution systems – pipes (particularly those made of polyvinyl chloride [PVC]) and storage tanks – can serve as reservoirs of microbial contamination. Hemodialysis systems frequently use pipes that are wider and longer than are needed to handle the required flow, which slows the fluid velocity and increases both the total fluid volume and the wetted surface area of the system. Gram-negative bacteria in fluids remaining in pipes overnight multiply rapidly and colonize the wet surfaces, producing bacterial populations and endotoxin quantities in proportion to the volume and surface area. Such colonization results in formation of protective biofilm that is difficult to remove and protects the bacteria from disinfection.⁸³² Routine (i.e., monthly), low-level disinfection of the pipes can help to control bacterial contamination of the distribution system. Additional measures to protect pipes from contaminations include a) situating all outlet taps at equal elevation and at the highest point of the system so that the disinfectant cannot drain from pipes by gravity before adequate contact time has elapsed and b) eliminating rough joints, dead-end pipes, and unused branches and taps that can trap fluid and serve as reservoirs of bacteria capable of continuously inoculating the entire volume of the system.⁸⁰⁰ Maintain a flow velocity of 3–5 ft/sec.

A storage tank in the distribution system greatly increases the volume of fluid and surface area available and can serve as a niche for water bacteria. Storage tanks are therefore not recommended for use in dialysis systems unless they are frequently drained and adequately disinfected, including scrubbing the sides of the tank to remove bacterial biofilm. An ultrafilter should be used distal to the storage tank.^{808, 833}

Microbiologic sampling of dialysis fluids is recommended because gram-negative bacteria can proliferate rapidly in water and dialysate in hemodialysis systems; high levels of these organisms place patients at risk for pyrogenic reactions or health-care–associated infection.^{667, 668, 808}

Health-care facilities are advised to sample dialysis fluids at least monthly using standard microbiologic assay methods for waterborne microorganisms.^{788, 793, 799, 834–836} Product water used to prepare dialysate and to reprocess hemodialyzers for reuse on the same patient should also be tested for bacterial endotoxin on a monthly basis.^{792, 829, 837} (See Appendix C for information about water sampling methods for dialysis.)

Cross-contamination of dialysis machines and inadequate disinfection measures can facilitate the spread of waterborne organisms to patients. Steps should be taken to ensure that dialysis equipment is performing correctly and that all connectors, lines, and other components are specific for the equipment, in good repair, and properly in place. A recent outbreak of gram-negative bacteremias among dialysis patients was attributed to faulty valves in a drain port of the machine that allowed backflow of saline used to flush the dialyzer before patient use.^{838, 839} This backflow contaminated the drain priming connectors, which contaminated the blood lines and exposed the patients to high concentrations of gram-negative bacteria. Environmental infection control in dialysis settings also includes low-level disinfection of housekeeping surfaces and spot decontamination of spills of blood (see Environmental Services in Part I of this guideline for further information).

c. Infection-Control Issues in Peritoneal Dialysis

Peritoneal dialysis (PD), most commonly administered as continuous ambulatory peritoneal dialysis (CAPD) and continual cycling peritoneal dialysis (CCPD), is the third most common treatment for end-stage renal disease (ESRD) in the United States, accounting for 12% of all dialysis patients.⁸⁴⁰ Peritonitis is the primary complication of CAPD, with coagulase-negative staphylococci the most clinically significant causative organisms.⁸⁴¹ Other organisms that have been found to produce peritonitis include *Staphylococcus aureus*, *Mycobacterium fortuitum*, *M. mucogenicum*, *Stenotrophomonas maltophilia*, *Burkholderia cepacia*, *Corynebacterium jeikeium*, *Candida* spp., and

other fungi.⁸⁴²⁻⁸⁵⁰ Substantial morbidity is associated with peritoneal dialysis infections. Removal of peritoneal dialysis catheters usually is required for treatment of peritonitis caused by fungi, NTM, or other bacteria that are not cleared within the first several days of effective antimicrobial treatment. Furthermore, recurrent episodes of peritonitis may lead to fibrosis and loss of the dialysis membrane.

Many reported episodes of peritonitis are associated with exit-site or tunneled catheter infections. Risk factors for the development of peritonitis in PD patients include a) under dialysis, b) immune suppression, c) prolonged antimicrobial treatment, d) patient age [more infections occur in younger patients and older hospitalized patients], e) length of hospital stay, and f) hypoalbuminemia.^{844, 851, 852} Concern has been raised about infection risk associated with the use of automated cyclers in both inpatient and outpatient settings; however, studies suggest that PD patients who use automated cyclers have much lower infection rates.⁸⁵³ One study noted that a closed-drainage system reduced the incidence of system-related peritonitis among intermittent peritoneal dialysis (IPD) patients from 3.6 to 1.5 cases/100 patient days.⁸⁵⁴ The association of peritonitis with management of spent dialysate fluids requires additional study. Therefore, ensuring that the tip of the waste line is not submerged beneath the water level in a toilet or in a drain is prudent.

7. Ice Machines and Ice

Microorganisms may be present in ice, ice-storage chests, and ice-making machines. The two main sources of microorganisms in ice are the potable water from which it is made and a transferral of organisms from hands (Table 20). Ice from contaminated ice machines has been associated with patient colonization, blood stream infections, pulmonary and gastrointestinal illnesses, and pseudoinfections.^{602, 603, 683, 684, 854, 855} Microorganisms in ice can secondarily contaminate clinical specimens and medical solutions that require cold temperatures for either transport or holding.^{601, 620} An outbreak of surgical-site infections was interrupted when sterile ice was used in place of tap water ice to cool cardioplegia solutions.⁶⁰¹

Table 20. Microorganisms and their sources in ice and ice machines

Sources of microorganisms	References
From potable water	
<i>Legionella</i> spp.	684, 685, 857, 858
Nontuberculous mycobacteria (NTM)	602, 603, 859
<i>Pseudomonas aeruginosa</i>	859
<i>Burkholderia cepacia</i>	859, 860
<i>Stenotrophomonas maltophilia</i>	860
<i>Flavobacterium</i> spp.	860
From fecally-contaminated water	
Norwalk virus	861-863
<i>Giardia lamblia</i>	864
<i>Cryptosporidium parvum</i>	685
From hand-transfer of organisms	
<i>Acinetobacter</i> spp.	859
Coagulase-negative staphylococci	859
<i>Salmonella enteritidis</i>	865
<i>Cryptosporidium parvum</i>	685

In a study comparing the microbial populations of hospital ice machines with organisms recovered from ice samples gathered from the community, samples from 27 hospital ice machines yielded low numbers (<10 CFU/mL) of several potentially opportunistic microorganisms, mainly gram-negative bacilli.⁸⁵⁹ During the survey period, no health-care-associated infections were attributed to the use of ice. Ice from community sources had higher levels of microbial contamination (75%–95% of 194 samples had total heterotrophic plate counts <500 CFU/mL, with the proportion of positive cultures dependent on the incubation temperature) and showed evidence of fecal contamination from the source water.⁸⁵⁹ Thus, ice machines in health-care settings are no more heavily contaminated compared with ice machines in the community. If the source water for ice in a health-care facility is not fecally contaminated, then ice from clean ice machines and chests should pose no increased hazard for immunocompetent patients. Some waterborne bacteria found in ice could potentially be a risk to immunocompromised patients if they consume ice or drink beverages with ice. For example, *Burkholderia cepacia* in ice could present an infection risk for cystic fibrosis patients.^{859, 860} Therefore, protecting immunosuppressed and otherwise medically at-risk patients from exposure to tap water and ice potentially contaminated with opportunistic pathogens is prudent.⁹

No microbiologic standards for ice, ice-making machines, or ice storage equipment have been established, although several investigators have suggested the need for such standards.^{859, 866} Culturing of ice machines is not routinely recommended, but it may be useful as part of an epidemiologic investigation.^{867–869} Sampling might also help determine the best schedule for cleaning open ice-storage chests. Recommendations for a regular program of maintenance and disinfection have been published.^{866–869} Health-care facilities are advised to clean ice-storage chests on a regular basis. Open ice chests may require a more frequent cleaning schedule compared with chests that have covers. Portable ice chests and containers require cleaning and low-level disinfection before the addition of ice intended for consumption. Ice-making machines may require less frequent cleaning, but their maintenance is important to proper performance. The manufacturer's instructions for both the proper method of cleaning and/or maintenance should be followed. These instructions may also recommend an EPA-registered disinfectant to ensure chemical potency, materials compatibility, and safety. In the event that instructions and suitable EPA-registered disinfectants are not available for this process, then a generic approach to cleaning, disinfecting, and maintaining ice machines and dispensers can be used (Box 12).

Ice and ice-making machines also may be contaminated via improper storage or handling of ice by patients and/or staff.^{684–686, 855–858, 870} Suggested steps to avoid this means of contamination include a) minimizing or avoiding direct hand contact with ice intended for consumption, b) using a hard-surface scoop to dispense ice, and c) installing machines that dispense ice directly into portable containers at the touch of a control.^{687, 869}

Box 12. General steps for cleaning and maintaining ice machines, dispensers, and storage chests*+

-
- 1. Disconnect unit from power supply.**
 - 2. Remove and discard ice from bin or storage chest.**
 - 3. Allow unit to warm to room temperature.**
 - 4. Disassemble removable parts of machine that make contact with water to make ice.**
 - 5. Thoroughly clean machine and parts with water and detergent.**
 - 6. Dry external surfaces of removable parts before reassembling.**
 - 7. Check for any needed repair.**
 - 8. Replace feeder lines, as appropriate (e.g., when damaged, old, or difficult to clean).**
 - 9. Ensure presence of an air space in tubing leading from water inlet into water distribution system of machine.**

(Box 12. continued)

10. Inspect for rodent or insect infestations under the unit and treat, as needed.
11. Check door gaskets (open compartment models) for evidence of leakage or dripping into the storage chest.
12. Clean the ice-storage chest or bin with fresh water and detergent; rinse with fresh tap water.
13. Sanitize machine by circulating a 50–100 parts per million (ppm) solution of sodium hypochlorite (i.e., 4–8 mL sodium hypochlorite/gallon of water) through the ice-making and storage systems for 2 hours (100 ppm solution), or 4 hours (50 ppm solution).
14. Drain sodium hypochlorite solutions and flush with fresh tap water.
15. Allow all surfaces of equipment to dry before returning to service.

* Material in this box is adapted from reference 869.

+ These general guidelines should be used only where manufacturer-recommended methods and EPA-registered disinfectants are not available.

8. Hydrotherapy Tanks and Pools

a. General Information

Hydrotherapy equipment (e.g., pools, whirlpools, whirlpool spas, hot tubs, and physiotherapy tanks) traditionally has been used to treat patients with certain medical conditions (e.g., burns,^{871, 872} septic ulcers, lesions, amputations,⁸⁷³ orthopedic impairments and injuries, arthritis,⁸⁷⁴ and kidney lithotripsy).⁶⁵⁴ Wound-care medicine is increasingly moving away from hydrotherapy, however, in favor of bedside pulsed-lavage therapy using sterile solutions for cleaning and irrigation.^{492, 875–878}

Several episodes of health-care-associated infections have been linked to use of hydrotherapy equipment (Table 21). Potential routes of infection include incidental ingestion of the water, sprays and aerosols, and direct contact with wounds and intact skin (folliculitis). Risk factors for infection include a) age and sex of the patient, b) underlying medical conditions, c) length of time spent in the hydrotherapy water, and d) portals of entry.⁸⁷⁹

Table 21. Infections associated with use of hydrotherapy equipment

Microorganisms	Medical conditions	References
<i>Acinetobacter baumannii</i>	Sepsis	572
<i>Citrobacter freundii</i>	Cellulitis	880
<i>Enterobacter cloacae</i>	Sepsis	881
<i>Legionella</i> spp.	Legionellosis	882
<i>Mycobacterium abscessus</i> , <i>Mycobacterium fortuitum</i> , <i>Mycobacterium marinum</i>	Skin ulcers and soft tissue infections	621–623, 883
<i>Pseudomonas aeruginosa</i>	Sepsis, soft tissue infections, folliculitis, and wound infections	492, 493, 506, 679, 884–888
Adenovirus, adeno-associated virus	Conjunctivitis	889

Infection control for hydrotherapy tanks, pools, or birthing tanks presents unique challenges because indigenous microorganisms are always present in the water during treatments. In addition, some studies have found free living amoebae (i.e., *Naegleria lovaniensis*), which are commonly found in association with *Naegleria fowleri*, in hospital hydrotherapy pools.⁸⁹⁰ Although hydrotherapy is at times appropriate for patients with wounds, burns, or other types of non-intact skin conditions (determined on a case-by-case basis), this equipment should not be considered “semi-critical” in accordance with the Spaulding classification.⁸⁹¹ Microbial data to evaluate the risk of infection to patients using hydrotherapy pools and birthing tanks are insufficient. Nevertheless, health-care facilities should maintain stringent cleaning and disinfection practices in accordance with the manufacturer’s instructions

and with relevant scientific literature until data supporting more rigorous infection-control measures become available. Factors that should be considered in therapy decisions in this situation would include a) availability of alternative aseptic techniques for wound management and b) a risk-benefit analysis of using traditional hydrotherapy.

b. Hydrotherapy Tanks

Hydrotherapy tanks (e.g., whirlpools, Hubbard tanks and whirlpool bath tubs) are shallow tanks constructed of stainless steel, plexiglass, or tile. They are closed-cycle water systems with hydrojets to circulate, aerate, and agitate the water. The maximum water temperature range is 50°F–104°F (10°C–40°C). The warm water temperature, constant agitation and aeration, and design of the hydrotherapy tanks provide ideal conditions for bacterial proliferation if the equipment is not properly maintained, cleaned, and disinfected. The design of the hydrotherapy equipment should be evaluated for potential infection-control problems that can be associated with inaccessible surfaces that can be difficult to clean and/or remain wet in between uses (i.e., recessed drain plates with fixed grill plates).⁸⁸⁷ Associated equipment (e.g., parallel bars, plinths, Hoyer lifts, and wheelchairs) can also be potential reservoirs of microorganisms, depending on the materials used in these items (i.e., porous vs. non-porous materials) and the surfaces that may become wet during use. Patients with active skin colonizations and wound infections can serve as sources of contamination for the equipment and the water. Contamination from spilled tub water can extend to drains, floors, and walls.^{680–683} Health-care-associated colonization or infection can result from exposure to endogenous sources of microorganisms (autoinoculation) or exogenous sources (via cross-contamination from other patients previously receiving treatment in the unit).

Although some facilities have used tub liners to minimize environmental contamination of the tanks, the use of a tub liner does not eliminate the need for cleaning and disinfection. Draining these small pools and tanks after each patient use, thoroughly cleaning with a detergent, and disinfecting according to manufacturers' instructions have reduced bacterial contamination levels in the water from 10⁴ CFU/mL to <10 CFU/mL.⁸⁹² A chlorine residual of 15 ppm in the water should be obtained prior to the patient's therapy session (e.g., by adding 15 grams of calcium hypochlorite 70% [e.g., HTH®] per 100 gallons of water).⁸⁹² A study of commercial and residential whirlpools found that superchlorination or draining, cleaning, disinfection, and refilling of whirlpools markedly reduced densities of *Pseudomonas aeruginosa* in whirlpool water.⁸⁹³ The bacterial populations were rapidly replenished, however, when disinfectant concentrations dropped below recommended levels for recreational use (i.e., chlorine at 3.0 ppm or bromine at 6.0 ppm). When using chlorine, however, knowing whether the community drinking-water system is disinfected with chloramine is important, because municipal utilities adjust the pH of the water to the basic side to enhance chloramine formation. Because chlorine is not very effective at pH levels above 8, it may be necessary to re-adjust the pH of the water to a more acidic level.⁸⁹⁴

A few reports describe the addition of antiseptic chemicals to hydrotherapy tank water, especially for burn patient therapy.^{895–897} One study involving a minimal number of participants demonstrated a reduction in the number of *Pseudomonas* spp. and other gram-negative bacteria from both patients and equipment surfaces when chloramine-T ("chlorazene") was added to the water.⁸⁹⁸ Chloramine-T has not, however, been approved for water treatment in the United States.

c. Hydrotherapy Pools

Hydrotherapy pools typically serve large numbers of patients and are usually heated to 91.4°F–98.6°F (31°C–37°C). The temperature range is more narrow (94°F–96.8°F [35°C–36°C]) for pediatric and geriatric patient use.⁸⁹⁹ Because the size of hydrotherapy pools precludes draining after patient use, proper management is required to maintain the proper balance of water conditioning (i.e., alkalinity, hardness, and temperature) and disinfection. The most widely used chemicals for disinfection of pools

are chlorine and chlorine compounds – calcium hypochlorite, sodium hypochlorite, lithium hypochlorite, chloroisocyanurates, and chlorine gas. Solid and liquid formulations of chlorine chemicals are the easiest and safest to use.⁹⁰⁰ Other halogenated compounds have also been used for pool-water disinfection, albeit on a limited scale. Bromine, which forms bactericidal bromamines in the presence of ammonia, has limited use because of its association with contact dermatitis.⁹⁰¹ Iodine does not bleach hair, swim suits, or cause eye irritation, but when introduced at proper concentrations, it gives water a greenish-yellowish cast.⁸⁹²

In practical terms, maintenance of large hydrotherapy pools (e.g., those used for exercise) is similar to that for indoor public pools (i.e., continuous filtration, chlorine residuals no less than 0.4 ppm, and pH of 7.2–7.6).^{902,903} Supply pipes and pumps also need to be maintained to eliminate the possibility of this equipment serving as a reservoir for waterborne organisms.⁹⁰⁴ Specific standards for chlorine residual and pH of the water are addressed in local and state regulations. Patients who are fecally incontinent or who have draining wounds should refrain from using these pools until their condition improves.

d. Birthing Tanks and Other Equipment

The use of birthing tanks, whirlpool spas, and whirlpools is a recent addition to obstetrical practice.⁹⁰⁵ Few studies on the potential risks associated with these pieces of equipment have been conducted. In one study of 32 women, a newborn contracted a *Pseudomonas* infection after being birthed in such a tank, the strain of which was identical to the organism isolated from the tank water.⁹⁰⁶ Another report documented identical strains of *P. aeruginosa* isolates from a newborn with sepsis and on the environmental surfaces of a tub that the mother used for relaxation while in labor.⁹⁰⁷ Other studies have shown no significant increases in the rates of post-immersion infections among mothers and infants.^{908,909}

Because the water and the tub surfaces routinely become contaminated with the mother's skin flora and blood during labor and delivery, birthing tanks and other tub equipment must be drained after each patient use and the surfaces thoroughly cleaned and disinfected. Health-care facilities are advised to follow the manufacturer's instructions for selection of disinfection method and chemical germicide. The range of chlorine residuals for public whirlpools and whirlpool spas is 2–5 ppm.⁹¹⁰ Use of an inflatable tub is an alternative solution, but this item must be cleaned and disinfected between patients if it is not considered a single-use unit.

Recreational tanks and whirlpool spas are increasingly being used as hydrotherapy equipment. Although such home equipment appears to be suitable for hydrotherapy, they are neither designed nor constructed to function in this capacity. Additionally, manufacturers generally are not obligated to provide the health-care facility with cleaning and disinfecting instructions appropriate for medical equipment use, and the U.S. Food and Drug Administration (FDA) does not evaluate recreational equipment. Health-care facilities should therefore carefully evaluate this “off-label” use of home equipment before proceeding with a purchase.

9. Miscellaneous Medical/Dental Equipment Connected to Main Water Systems

a. Automated Endoscope Reprocessors

The automated endoscopic reprocessor (AER) is classified by the FDA as an accessory for the flexible endoscope.⁶⁵⁴ A properly operating AER can provide a more consistent, reliable method of decontaminating and terminal reprocessing for endoscopes between patient procedures than manual reprocessing methods alone.⁹¹¹ An endoscope is generally subjected to high-level disinfection using a

liquid chemical sterilant or a high-level disinfectant. Because the instrument is a semi-critical device, the optimal rinse fluid for a disinfected endoscope would be sterile water.³ Sterile water, however, is expensive and difficult to produce in sufficient quantities and with adequate quality assurance for instrument rinsing in an AER.^{912, 913} Therefore, one option to be used for AERs is rinse water that has been passed through filters with a pore size of 0.1–0.2 μm to render the water “bacteria-free.” These filters usually are located in the water line at or near the port where the mains water enters the equipment. The product water (i.e., tap water passing through these filters) in these applications is not considered equivalent in microbial quality to that for membrane-filtered water as produced by pharmaceutical firms. Membrane filtration in pharmaceutical applications is intended to ensure the microbial quality of polished product water.

Water has been linked to the contamination of flexible fiberoptic endoscopes in the following two scenarios: a) rinsing a disinfected endoscope with unfiltered tap water, followed by storage of the instrument without drying out the internal channels and b) contamination of AERs from tap water inadvertently introduced into the equipment. In the latter instance, the machine’s water reservoirs and fluid circuitry become contaminated with waterborne, heterotrophic bacteria (e.g., *Pseudomonas aeruginosa* and NTM), which can survive and persist in biofilms attached to these components.^{914–917} Colonization of the reservoirs and water lines of the AER becomes problematic if the required cleaning, disinfection, and maintenance are not performed on the equipment as recommended by the manufacturer.^{669, 916, 917} Use of the 0.1–0.2- μm filter in the water line helps to keep bacterial contamination to a minimum,^{670, 911, 917} but filters may fail and allow bacteria to pass through to the equipment and then to the instrument undergoing reprocessing.^{671–674, 913, 918} Filters also require maintenance for proper performance.^{670, 911, 912, 918, 919} Heightened awareness of the proper disinfection of the connectors that hook the instrument to the AER may help to further reduce the potential for contaminating endoscopes during reprocessing.⁹²⁰ An emerging issue in the field of endoscopy is that of the possible role of rinse water monitoring and its potential to help reduce endoscopy/bronchoscopy-associated infections.⁹¹⁸

Studies have linked deficiencies in endoscope cleaning and/or disinfecting processes to the incidence of post-endoscopic adverse outcomes.^{921–924} Several clusters have been traced to AERs of older designs and these were associated with water quality.^{675, 914–916} Regardless of whether manual or automated terminal reprocessing is used for endoscopes, the internal channels of the instrument should be dried before storage.⁹²⁵ The presence of residual moisture in the internal channels encourages the proliferation of waterborne microorganisms, some of which may be pathogenic. One of the most frequently used methods employs 70% isopropyl alcohol to flush the internal channels, followed by forced air drying of these channels and hanging the endoscope vertically in a protected cabinet; this method ensures internal drying of the endoscope, lessens the potential for proliferation of waterborne microorganisms,^{669, 913, 917, 922, 926, 927} and is consistent with professional organization guidance for endoscope reprocessing.⁹²⁸

An additional problem with waterborne microbial contamination of AERs centers on increased microbial resistance to alkaline glutaraldehyde, a widely used liquid chemical sterilant/high-level disinfectant.^{669, 929} Opportunistic waterborne microorganisms (e.g., *Mycobacterium chelonae*, *Methylobacterium* spp.) have been associated with pseudo-outbreaks and colonization; infection caused by these organisms has been associated with procedures conducted in clinical settings (e.g., bronchoscopy).^{669, 913, 929–931} Increasing microbial resistance to glutaraldehyde has been attributed to improper use of the disinfectant in the equipment, allowing the dilution of glutaraldehyde to fall below the manufacturer’s recommended minimal use concentration.⁹²⁹

b. Dental Unit Water Lines

Dental unit water lines (DUWLs) consist of small-bore plastic tubing that delivers water used for general, non-surgical irrigation and as a coolant to dental handpieces, sonic and ultrasonic scalers, and air-water syringes; municipal tap water is the source water for these lines. The presence of biofilms of waterborne bacteria and fungi (e.g., *Legionella* spp., *Pseudomonas aeruginosa*, and NTM) in DUWLs has been established.^{636, 637, 694, 695, 932–954} Biofilms continually release planktonic microorganisms into the water, the titers of which can exceed 1×10^6 CFU/mL.⁶⁹⁴ However, scientific evidence indicates that immunocompetent persons are only at minimal risk for substantial adverse health effects after contact with water from a dental unit. Nonetheless, exposing patients or dental personnel to water of uncertain microbiological quality is not consistent with universally accepted infection-control principles.⁹³⁵

In 1993, CDC issued guidelines relative to water quality in a dental setting. These guidelines recommend that all dental instruments that use water (including high-speed handpieces) should be run to discharge water for 20–30 seconds after each patient and for several minutes before the start of each clinic day.⁹³⁶ This practice can help to flush out any patient materials that may have entered the turbine, air, or waterlines.^{937, 938} The 1993 guidance also indicated that waterlines be flushed at the beginning of the clinic day. Although these guidelines are designed to help reduce the number of microorganisms present in treatment water, they do not address the issue of reducing or preventing biofilm formation in the waterlines. Research published subsequent to the 1993 dental infection control guideline suggests that flushing the lines at the beginning of the day has only minimal effect on the status of the biofilm in the lines and does not reliably improve the quality of water during dental treatment.^{939–941} Updated recommendations on infection-control practices for water line use in dentistry will be available in late 2003.⁹⁴²

The numbers of microorganisms in water used as coolant or irrigant for non-surgical dental treatment should be as low as reasonably achievable and, at a minimum, should meet nationally recognized standards for safe drinking water.^{935, 943} Only minimal evidence suggests that water meeting drinking water standards poses a health hazard for immunocompetent persons. The EPA, the American Public Health Association (APHA), and the American Water Works Association (AWWA) have set a maximum limit of 500 CFU/mL for aerobic, heterotrophic, mesophilic bacteria in drinking water in municipal distribution systems.^{944, 945} This standard is achievable, given improvements in water-line technology. Dentists should consult with the manufacturer of their dental unit to determine the best equipment and method for maintaining and monitoring good water quality.^{935, 946}

E. Environmental Services

1. Principles of Cleaning and Disinfecting Environmental Surfaces

Although microbiologically contaminated surfaces can serve as reservoirs of potential pathogens, these surfaces generally are not directly associated with transmission of infections to either staff or patients. The transferral of microorganisms from environmental surfaces to patients is largely via hand contact with the surface.^{947, 948} Although hand hygiene is important to minimize the impact of this transfer, cleaning and disinfecting environmental surfaces as appropriate is fundamental in reducing their potential contribution to the incidence of healthcare-associated infections.

The principles of cleaning and disinfecting environmental surfaces take into account the intended use of the surface or item in patient care. CDC retains the Spaulding classification for medical and surgical instruments, which outlines three categories based on the potential for the instrument to transmit infection if the instrument is microbiologically contaminated before use.^{949, 950} These categories are

“critical,” “semicritical,” and “noncritical.” In 1991, CDC proposed an additional category designated “environmental surfaces” to Spaulding’s original classification⁹⁵¹ to represent surfaces that generally do not come into direct contact with patients during care. Environmental surfaces carry the least risk of disease transmission and can be safely decontaminated using less rigorous methods than those used on medical instruments and devices. Environmental surfaces can be further divided into medical equipment surfaces (e.g., knobs or handles on hemodialysis machines, x-ray machines, instrument carts, and dental units) and housekeeping surfaces (e.g., floors, walls, and tabletops).⁹⁵¹

The following factors influence the choice of disinfection procedure for environmental surfaces: a) the nature of the item to be disinfected, b) the number of microorganisms present, c) the innate resistance of those microorganisms to the inactivating effects of the germicide, d) the amount of organic soil present, e) the type and concentration of germicide used, f) duration and temperature of germicide contact, and g) if using a proprietary product, other specific indications and directions for use.^{952, 953}

Cleaning is the necessary first step of any sterilization or disinfection process. Cleaning is a form of decontamination that renders the environmental surface safe to handle or use by removing organic matter, salts, and visible soils, all of which interfere with microbial inactivation.⁹⁵⁴⁻⁹⁶⁰ The physical action of scrubbing with detergents and surfactants and rinsing with water removes large numbers of microorganisms from surfaces.⁹⁵⁷ If the surface is not cleaned before the terminal reprocessing procedures are started, the success of the sterilization or disinfection process is compromised.

Spaulding proposed three levels of disinfection for the treatment of devices and surfaces that do not require sterility for safe use. These disinfection levels are “high-level,” “intermediate-level,” and “low-level.”^{949, 950} The basis for these levels is that microorganisms can usually be grouped according to their innate resistance to a spectrum of physical or chemical germicidal agents (Table 22). This information, coupled with the instrument/surface classification, determines the appropriate level of terminal disinfection for an instrument or surface.

Table 22. Levels of disinfection by type of microorganism*

Disinfection level	Bacteria			Fungi+	Viruses	
	Vegetative	Tubercle bacillus	Spores		Lipid and medium size	Nonlipid and small size
High	+ §	+	+ ¶	+	+	+
Intermediate	+	+	—**	+	+	± ⁺⁺
Low	+	—	—	±	+	±

* Material in this table compiled from references 2 and 951.

+ This class of microorganisms includes asexual spores but not necessarily chlamydo spores or sexual spores.

§ The “plus” sign indicates that a killing effect can be expected when the normal use-concentrations of chemical disinfectants or pasteurization are properly employed; a “negative” sign indicates little or no killing effect.

¶ Only with extended exposure times are high-level disinfectant chemicals capable of killing high numbers of bacterial spores in laboratory tests; they are, however, capable of sporicidal activity.

** Some intermediate-level disinfectants (e.g., hypochlorites) can exhibit some sporicidal activity; others (e.g., alcohols and phenolics) have no demonstrable sporicidal activity.

++ Some intermediate-level disinfectants, although they are tuberculocidal, may have limited virucidal activity.

The process of high-level disinfection, an appropriate standard of treatment for heat-sensitive, semi-critical medical instruments (e.g., flexible, fiberoptic endoscopes), inactivates all vegetative bacteria, mycobacteria, viruses, fungi, and some bacterial spores. High-level disinfection is accomplished with powerful, sporicidal chemicals (e.g., glutaraldehyde, peracetic acid, and hydrogen peroxide) that are not appropriate for use on housekeeping surfaces. These liquid chemical sterilants/high-level disinfectants

are highly toxic.^{961–963} Use of these chemicals for applications other than those indicated in their label instructions (i.e., as immersion chemicals for treating heat-sensitive medical instruments) is not appropriate.⁹⁶⁴ Intermediate-level disinfection does not necessarily kill bacterial spores, but it does inactivate *Mycobacterium tuberculosis* var. *bovis*, which is substantially more resistant to chemical germicides than ordinary vegetative bacteria, fungi, and medium to small viruses (with or without lipid envelopes). Chemical germicides with sufficient potency to achieve intermediate-level disinfection include chlorine-containing compounds (e.g., sodium hypochlorite), alcohols, some phenolics, and some iodophors. Low-level disinfection inactivates vegetative bacteria, fungi, enveloped viruses (e.g., human immunodeficiency virus [HIV], and influenza viruses), and some non-enveloped viruses (e.g., adenoviruses). Low-level disinfectants include quaternary ammonium compounds, some phenolics, and some iodophors. Sanitizers are agents that reduce the numbers of bacterial contaminants to safe levels as judged by public health requirements, and are used in cleaning operations, particularly in food service and dairy applications. Germicidal chemicals that have been approved by FDA as skin antiseptics are not appropriate for use as environmental surface disinfectants.⁹⁵¹

The selection and use of chemical germicides are largely matters of judgment, guided by product label instructions, information, and regulations. Liquid sterilant chemicals and high-level disinfectants intended for use on critical and semi-critical medical/dental devices and instruments are regulated exclusively by the FDA as a result of recent memoranda of understanding between FDA and the EPA that delineates agency authority for chemical germicide regulation.^{965, 966} Environmental surface germicides (i.e., primarily intermediate- and low-level disinfectants) are regulated by the EPA and labeled with EPA registration numbers. The labels and technical data or product literature of these germicides specify indications for product use and provide claims for the range of antimicrobial activity. The EPA requires certain pre-registration laboratory potency tests for these products to support product label claims. EPA verifies (through laboratory testing) manufacturers' claims to inactivate microorganisms for selected products and organisms. Germicides labeled as "hospital disinfectant" have passed the potency tests for activity against three representative microorganisms – *Pseudomonas aeruginosa*, *Staphylococcus aureus*, and *Salmonella cholerae suis*. Low-level disinfectants are often labeled "hospital disinfectant" without a tuberculocidal claim, because they lack the potency to inactivate mycobacteria. Hospital disinfectants with demonstrated potency against mycobacteria (i.e., intermediate-level disinfectants) may list "tuberculocidal" on the label as well. Other claims (e.g., "fungicidal," "pseudomonocidal," and "virucidal") may appear on labels of environmental surface germicides, but the designations of "tuberculocidal hospital disinfectant" and "hospital disinfectant" correlate directly to Spaulding's assessment of intermediate-level disinfectants and low-level disinfectants, respectively.⁹⁵¹

A common misconception in the use of surface disinfectants in health-care settings relates to the underlying purpose for use of proprietary products labeled as a "tuberculocidal" germicide. Such products will not interrupt and prevent the transmission of TB in health-care settings because TB is not acquired from environmental surfaces. The tuberculocidal claim is used as a benchmark by which to measure germicidal potency. Because mycobacteria have the highest intrinsic level of resistance among the vegetative bacteria, viruses, and fungi, any germicide with a tuberculocidal claim on the label (i.e., an intermediate-level disinfectant) is considered capable of inactivating a broad spectrum of pathogens, including much less resistant organisms such as the bloodborne pathogens (e.g., hepatitis B virus [HBV], hepatitis C virus [HCV], and HIV). It is this broad spectrum capability, rather than the product's specific potency against mycobacteria, that is the basis for protocols and OSHA regulations indicating the appropriateness of using tuberculocidal chemicals for surface disinfection.⁹⁶⁷

2. General Cleaning Strategies for Patient-Care Areas

The number and types of microorganisms present on environmental surfaces are influenced by the following factors: a) number of people in the environment, b) amount of activity, c) amount of moisture, d) presence of material capable of supporting microbial growth, e) rate at which organisms suspended in the air are removed, and f) type of surface and orientation [i.e., horizontal or vertical].⁹⁶⁸ Strategies for cleaning and disinfecting surfaces in patient-care areas take into account a) potential for direct patient contact, b) degree and frequency of hand contact, and c) potential contamination of the surface with body substances or environmental sources of microorganisms (e.g., soil, dust, and water).

a. Cleaning of Medical Equipment

Manufacturers of medical equipment should provide care and maintenance instructions specific to their equipment. These instructions should include information about a) the equipments' compatibility with chemical germicides, b) whether the equipment is water-resistant or can be safely immersed for cleaning, and c) how the equipment should be decontaminated if servicing is required.⁹⁶⁷ In the absence of manufacturers' instructions, non-critical medical equipment (e.g., stethoscopes, blood pressure cuffs, dialysis machines, and equipment knobs and controls) usually only require cleansing followed by low- to intermediate-level disinfection, depending on the nature and degree of contamination. Ethyl alcohol or isopropyl alcohol in concentrations of 60%–90% (v/v) is often used to disinfect small surfaces (e.g., rubber stoppers of multiple-dose medication vials, and thermometers)^{952, 969} and occasionally external surfaces of equipment (e.g., stethoscopes and ventilators). However, alcohol evaporates rapidly, which makes extended contact times difficult to achieve unless items are immersed, a factor that precludes its practical use as a large-surface disinfectant.⁹⁵¹ Alcohol may cause discoloration, swelling, hardening, and cracking of rubber and certain plastics after prolonged and repeated use and may damage the shellac mounting of lenses in medical equipment.⁹⁷⁰

Barrier protection of surfaces and equipment is useful, especially if these surfaces are a) touched frequently by gloved hands during the delivery of patient care, b) likely to become contaminated with body substances, or c) difficult to clean. Impervious-backed paper, aluminum foil, and plastic or fluid-resistant covers are suitable for use as barrier protection. An example of this approach is the use of plastic wrapping to cover the handle of the operatory light in dental-care settings.^{936, 942} Coverings should be removed and discarded while the health-care worker is still gloved.^{936, 942} The health-care worker, after ungloving and performing hand hygiene, must cover these surfaces with clean materials before the next patient encounter.

b. Cleaning Housekeeping Surfaces

Housekeeping surfaces require regular cleaning and removal of soil and dust. Dry conditions favor the persistence of gram-positive cocci (e.g., coagulase-negative *Staphylococcus* spp.) in dust and on surfaces, whereas moist, soiled environments favor the growth and persistence of gram-negative bacilli.^{948, 971, 972} Fungi are also present on dust and proliferate in moist, fibrous material.

Most, if not all, housekeeping surfaces need to be cleaned only with soap and water or a detergent/disinfectant, depending on the nature of the surface and the type and degree of contamination. Cleaning and disinfection schedules and methods vary according to the area of the health-care facility, type of surface to be cleaned, and the amount and type of soil present. Disinfectant/detergent formulations registered by EPA are used for environmental surface cleaning, but the actual physical removal of microorganisms and soil by wiping or scrubbing is probably as important, if not more so, than any antimicrobial effect of the cleaning agent used.⁹⁷³ Therefore, cost, safety, product-surface compatibility, and acceptability by housekeepers can be the main criteria for selecting a registered agent. If using a proprietary detergent/disinfectant, the manufacturers' instructions for appropriate use

of the product should be followed.⁹⁷⁴ Consult the products' material safety data sheets (MSDS) to determine appropriate precautions to prevent hazardous conditions during product application. Personal protective equipment (PPE) used during cleaning and housekeeping procedures should be appropriate to the task.

Housekeeping surfaces can be divided into two groups – those with minimal hand-contact (e.g., floors, and ceilings) and those with frequent hand-contact (“high touch surfaces”). The methods, thoroughness, and frequency of cleaning and the products used are determined by health-care facility policy.⁶ However, high-touch housekeeping surfaces in patient-care areas (e.g., doorknobs, bedrails, light switches, wall areas around the toilet in the patient's room, and the edges of privacy curtains) should be cleaned and/or disinfected more frequently than surfaces with minimal hand contact. Infection-control practitioners typically use a risk-assessment approach to identify high-touch surfaces and then coordinate an appropriate cleaning and disinfecting strategy and schedule with the housekeeping staff.

Horizontal surfaces with infrequent hand contact (e.g., window sills and hard-surface flooring) in routine patient-care areas require cleaning on a regular basis, when soiling or spills occur, and when a patient is discharged from the facility.⁶ Regular cleaning of surfaces and decontamination, as needed, is also advocated to protect potentially exposed workers.⁹⁶⁷ Cleaning of walls, blinds, and window curtains is recommended when they are visibly soiled.^{972, 973, 975} Disinfectant fogging is not recommended for general infection control in routine patient-care areas.^{2, 976} Further, paraformaldehyde, which was once used in this application, is no longer registered by EPA for this purpose. Use of paraformaldehyde in these circumstances requires either registration or an exemption issued by EPA under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA). Infection control, industrial hygienists, and environmental services supervisors should assess the cleaning procedures, chemicals used, and the safety issues to determine if a temporary relocation of the patient is needed when cleaning in the room.

Extraordinary cleaning and decontamination of floors in health-care settings is unwarranted. Studies have demonstrated that disinfection of floors offers no advantage over regular detergent/water cleaning and has minimal or no impact on the occurrence of health-care-associated infections.^{947, 948, 977–980} Additionally, newly cleaned floors become rapidly recontaminated from airborne microorganisms and those transferred from shoes, equipment wheels, and body substances.^{971, 975, 981} Nevertheless, health-care institutions or contracted cleaning companies may choose to use an EPA-registered detergent/disinfectant for cleaning low-touch surfaces (e.g., floors) in patient-care areas because of the difficulty that personnel may have in determining if a spill contains blood or body fluids (requiring a detergent/disinfectant for clean-up) or when a multi-drug resistant organism is likely to be in the environment. Methods for cleaning non-porous floors include wet mopping and wet vacuuming, dry dusting with electrostatic materials, and spray buffing.^{973, 982–984} Methods that produce minimal mists and aerosols or dispersion of dust in patient-care areas are preferred.^{9, 20, 109, 272}

Part of the cleaning strategy is to minimize contamination of cleaning solutions and cleaning tools. Bucket solutions become contaminated almost immediately during cleaning, and continued use of the solution transfers increasing numbers of microorganisms to each subsequent surface to be cleaned.^{971, 981, 985} Cleaning solutions should be replaced frequently. A variety of “bucket” methods have been devised to address the frequency with which cleaning solutions are replaced.^{986, 987} Another source of contamination in the cleaning process is the cleaning cloth or mop head, especially if left soaking in dirty cleaning solutions.^{971, 988–990} Laundering of cloths and mop heads after use and allowing them to dry before re-use can help to minimize the degree of contamination.⁹⁹⁰ A simplified approach to cleaning involves replacing soiled cloths and mop heads with clean items each time a bucket of detergent/disinfectant is emptied and replaced with fresh, clean solution (B. Stover, Kosair Children's Hospital, 2000). Disposable cleaning cloths and mop heads are an alternative option, if costs permit.

Another reservoir for microorganisms in the cleaning process may be dilute solutions of the detergents or disinfectants, especially if the working solution is prepared in a dirty container, stored for long periods of time, or prepared incorrectly.⁵⁴⁷ Gram-negative bacilli (e.g., *Pseudomonas* spp. and *Serratia marcescens*) have been detected in solutions of some disinfectants (e.g., phenolics and quaternary ammonium compounds).^{547, 991} Contemporary EPA registration regulations have helped to minimize this problem by asking manufacturers to provide potency data to support label claims for detergent/disinfectant properties under real-use conditions (e.g., diluting the product with tap water instead of distilled water). Application of contaminated cleaning solutions, particularly from small-quantity aerosol spray bottles or with equipment that might generate aerosols during operation, should be avoided, especially in high-risk patient areas.^{992, 993} Making sufficient fresh cleaning solution for daily cleaning, discarding any remaining solution, and drying out the container will help to minimize the degree of bacterial contamination. Containers that dispense liquid as opposed to spray-nozzle dispensers (e.g., quart-sized dishwashing liquid bottles) can be used to apply detergent/disinfectants to surfaces and then to cleaning cloths with minimal aerosol generation. A pre-mixed, “ready-to-use” detergent/disinfectant solution may be used if available.

c. Cleaning Special Care Areas

Guidelines have been published regarding cleaning strategies for isolation areas and operating rooms.^{6, 7} The basic strategies for areas housing immunosuppressed patients include a) wet dusting horizontal surfaces daily with cleaning cloths pre-moistened with detergent or an EPA-registered hospital disinfectant or disinfectant wipes;^{94, 98463} b) using care when wet dusting equipment and surfaces above the patient to avoid patient contact with the detergent/disinfectant; c) avoiding the use of cleaning equipment that produces mists or aerosols; d) equipping vacuums with HEPA filters, especially for the exhaust, when used in any patient-care area housing immunosuppressed patients,^{9, 94, 986} and e) regular cleaning and maintenance of equipment to ensure efficient particle removal. When preparing the cleaning cloths for wet-dusting, freshly prepared solutions of detergents or disinfectants should be used rather than cloths that have soaked in such solutions for long periods of time. Dispersal of microorganisms in the air from dust or aerosols is more problematic in these settings than elsewhere in health-care facilities. Vacuum cleaners can serve as dust disseminators if they are not operating properly.⁹⁹⁴ Doors to immunosuppressed patients’ rooms should be closed when nearby areas are being vacuumed.⁹ Bacterial and fungal contamination of filters in cleaning equipment is inevitable, and these filters should be cleaned regularly or replaced as per equipment manufacturer instructions.

Mats with tacky surfaces placed in operating rooms and other patient-care areas only slightly minimize the overall degree of contamination of floors and have little impact on the incidence rate of health-care-associated infection in general.^{351, 971, 983} An exception, however, is the use of tacky mats inside the entry ways of cordoned-off construction areas inside the health-care facility; these mats help to minimize the intrusion of dust into patient-care areas.

Special precautions for cleaning incubators, mattresses, and other nursery surfaces have been recommended to address reports of hyperbilirubinemia in newborns linked to inadequately diluted solutions of phenolics and poor ventilation.⁹⁹⁵⁻⁹⁹⁷ These medical conditions have not, however, been associated with the use of properly prepared solutions of phenolics. Non-porous housekeeping surfaces in neonatal units can be disinfected with properly diluted or pre-mixed phenolics, followed by rinsing with clean water.⁹⁹⁷ However, phenolics are not recommended for cleaning infant bassinets and incubators during the stay of the infant. Infants who remain in the nursery for an extended period should be moved periodically to freshly cleaned and disinfected bassinets and incubators.⁹⁹⁷ If phenolics are used for cleaning bassinets and incubators after they have been vacated, the surfaces should be rinsed thoroughly with water and dried before either piece of equipment is reused. Cleaning

and disinfecting protocols should allow for the full contact time specified for the product used. Bassinet mattresses should be replaced, however, if the mattress cover surface is broken.⁹⁹⁷

3. Cleaning Strategies for Spills of Blood and Body Substances

Neither HBV, HCV, nor HIV has ever been transmitted from a housekeeping surface (i.e., floors, walls, or countertops). Nonetheless, prompt removal and surface disinfection of an area contaminated by either blood or body substances are sound infection-control practices and OSHA requirements.⁹⁶⁷

Studies have demonstrated that HIV is inactivated rapidly after being exposed to commonly used chemical germicides at concentrations that are much lower than those used in practice.^{998–1003} HBV is readily inactivated with a variety of germicides, including quaternary ammonium compounds.¹⁰⁰⁴ Embalming fluids (e.g., formaldehyde) are also capable of completely inactivating HIV and HBV.^{1005, 1006} OSHA has revised its regulation for disinfecting spills of blood or other potentially infectious material to include proprietary products whose label includes inactivation claims for HBV and HIV, provided that such surfaces have not become contaminated with agent(s) or volumes of or concentrations of agent(s) for which a higher level of disinfection is recommended.¹⁰⁰⁷ These registered products are listed in EPA's List D – *Registered Antimicrobials Effective Against Hepatitis B Virus and Human HIV-1*, which may include products tested against duck hepatitis B virus (DHBV) as a surrogate for HBV.^{1008, 1009} Additional lists of interest include EPA's List C – *Registered Antimicrobials Effective Against Human HIV-1* and EPA's List E – *Registered Antimicrobials Effective Against Mycobacterium spp., Hepatitis B Virus, and Human HIV-1*.

Sodium hypochlorite solutions are inexpensive and effective broad-spectrum germicidal solutions.^{1010, 1011} Generic sources of sodium hypochlorite include household chlorine bleach or reagent grade chemical. Concentrations of sodium hypochlorite solutions with a range of 5,000–6,150 ppm (1:10 v/v dilution of household bleaches marketed in the United States) to 500–615 ppm (1:100 v/v dilution) free chlorine are effective depending on the amount of organic material (e.g., blood, mucus, and urine) present on the surface to be cleaned and disinfected.^{1010, 1011} EPA-registered chemical germicides may be more compatible with certain materials that could be corroded by repeated exposure to sodium hypochlorite, especially the 1:10 dilution. Appropriate personal protective equipment (e.g., gloves and goggles) should be worn when preparing and using hypochlorite solutions or other chemical germicides.⁹⁶⁷

Despite laboratory evidence demonstrating adequate potency against bloodborne pathogens (e.g., HIV and HBV), many chlorine bleach products available in grocery and chemical-supply stores are not registered by the EPA for use as surface disinfectants. Use of these chlorine products as surface disinfectants is considered by the EPA to be an “unregistered use.” EPA encourages the use of registered products because the agency reviews them for safety and performance when the product is used according to label instructions. When unregistered products are used for surface disinfection, users do so at their own risk.

Strategies for decontaminating spills of blood and other body fluids differ based on the setting in which they occur and the volume of the spill.¹⁰¹⁰ In patient-care areas, workers can manage small spills with cleaning and then disinfecting using an intermediate-level germicide or an EPA-registered germicide from the EPA List D or E.^{967, 1007} For spills containing large amounts of blood or other body substances, workers should first remove visible organic matter with absorbent material (e.g., disposable paper towels discarded into leak-proof, properly labeled containment) and then clean and decontaminate the area.^{1002, 1003, 1012} If the surface is nonporous and a generic form of a sodium hypochlorite solution is used (e.g., household bleach), a 1:100 dilution is appropriate for decontamination assuming that a) the

worker assigned to clean the spill is wearing gloves and other personal protective equipment appropriate to the task, b) most of the organic matter of the spill has been removed with absorbent material, and c) the surface has been cleaned to remove residual organic matter. A recent study demonstrated that even strong chlorine solutions (i.e., 1:10 dilution of chlorine bleach) may fail to totally inactivate high titers of virus in large quantities of blood, but in the absence of blood these disinfectants can achieve complete viral inactivation.¹⁰¹¹ This evidence supports the need to remove most organic matter from a large spill before final disinfection of the surface. Additionally, EPA-registered proprietary disinfectant label claims are based on use on a pre-cleaned surface.^{951, 954}

Managing spills of blood, body fluids, or other infectious materials in clinical, public health, and research laboratories requires more stringent measures because of a) the higher potential risk of disease transmission associated with large volumes of blood and body fluids and b) high numbers of microorganisms associated with diagnostic cultures. The use of an intermediate-level germicide for routine decontamination in the laboratory is prudent.⁹⁵⁴ Recommended practices for managing large spills of concentrated infectious agents in the laboratory include a) confining the contaminated area, b) flooding the area with a liquid chemical germicide before cleaning, and c) decontaminating with fresh germicidal chemical of at least intermediate-level disinfectant potency.¹⁰¹⁰ A suggested technique when flooding the spill with germicide is to lay absorbent material down on the spill and apply sufficient germicide to thoroughly wet both the spill and the absorbent material.¹⁰¹³ If using a solution of household chlorine bleach, a 1:10 dilution is recommended for this purpose. EPA-registered germicides should be used according to the manufacturers' instructions for use dilution and contact time. Gloves should be worn during the cleaning and decontamination procedures in both clinical and laboratory settings. PPE in such a situation may include the use of respiratory protection (e.g., an N95 respirator) if clean-up procedures are expected to generate infectious aerosols. Protocols for cleaning spills should be developed and made available on record as part of good laboratory practice.¹⁰¹³ Workers in laboratories and in patient-care areas of the facility should receive periodic training in environmental-surface infection-control strategies and procedures as part of an overall infection-control and safety curriculum.

4. Carpeting and Cloth Furnishings

a. Carpeting

Carpeting has been used for more than 30 years in both public and patient-care areas of health-care facilities. Advantages of carpeting in patient-care areas include a) its noise-limiting characteristics; b) the "humanizing" effect on health care; and c) its contribution to reductions in falls and resultant injuries, particularly for the elderly.¹⁰¹⁴⁻¹⁰¹⁶ Compared to hard-surface flooring, however, carpeting is harder to keep clean, especially after spills of blood and body substances. It is also harder to push equipment with wheels (e.g., wheelchairs, carts, and gurneys) on carpeting.

Several studies have documented the presence of diverse microbial populations, primarily bacteria and fungi, in carpeting;^{111, 1017-1024} the variety and number of microorganisms tend to stabilize over time. New carpeting quickly becomes colonized, with bacterial growth plateauing after about 4 weeks.¹⁰¹⁹ Vacuuming and cleaning the carpeting can temporarily reduce the numbers of bacteria, but these populations soon rebound and return to pre-cleaning levels.^{1019, 1020, 1023} Bacterial contamination tends to increase with higher levels of activity.^{1018-1020, 1025} Soiled carpeting that is or remains damp or wet provides an ideal setting for the proliferation and persistence of gram-negative bacteria and fungi.¹⁰²⁶ Carpeting that remains damp should be removed, ideally within 72 hours.

Despite the evidence of bacterial growth and persistence in carpeting, only limited epidemiologic evidence demonstrates that carpets influence health-care-associated infection rates in areas housing

immunocompetent patients.^{1023, 1025, 1027} This guideline, therefore, includes no recommendations against the use of carpeting in these areas. Nonetheless, avoiding the use of carpeting is prudent in areas where spills are likely to occur (e.g., laboratories, areas around sinks, and janitor closets) and where patients may be at greater risk of infection from airborne environmental pathogens (e.g., HSCT units, burn units, ICUs, and ORs).^{111, 1028} An outbreak of aspergillosis in an HSCT unit was recently attributed to carpet contamination and a particular method of carpet cleaning.¹¹¹ A window in the unit had been opened repeatedly during the time of a nearby building fire, which allowed fungal spore intrusion into the unit. After the window was sealed, the carpeting was cleaned using a “bonnet buffing” machine, which dispersed *Aspergillus* spores into the air.¹¹¹ Wet vacuuming was instituted, replacing the dry cleaning method used previously; no additional cases of invasive aspergillosis were identified.

The care setting and the method of carpet cleaning are important factors to consider when attempting to minimize or prevent production of aerosols and dispersal of carpet microorganisms into the air.^{94, 111} Both vacuuming and shampooing or wet cleaning with equipment can disperse microorganisms to the air.^{111, 994} Vacuum cleaners should be maintained to minimize dust dispersal in general, and be equipped with HEPA filters, especially for use in high-risk patient-care areas.^{9, 94, 986} Some formulations of carpet-cleaning chemicals, if applied or used improperly, can be dispersed into the air as a fine dust capable of causing respiratory irritation in patients and staff.¹⁰²⁹ Cleaning equipment, especially those that engage in wet cleaning and extraction, can become contaminated with waterborne organisms (e.g., *Pseudomonas aeruginosa*) and serve as a reservoir for these organisms if this equipment is not properly maintained. Substantial numbers of bacteria can then be transferred to carpeting during the cleaning process.¹⁰³⁰ Therefore, keeping the carpet cleaning equipment in good repair and allowing such equipment to dry between uses is prudent.

Carpet cleaning should be performed on a regular basis determined by internal policy. Although spills of blood and body substances on non-porous surfaces require prompt spot cleaning using standard cleaning procedures and application of chemical germicides,⁹⁶⁷ similar decontamination approaches to blood and body substance spills on carpeting can be problematic from a regulatory perspective.¹⁰³¹ Most, if not all, modern carpet brands suitable for public facilities can tolerate the activity of a variety of liquid chemical germicides. However, according to OSHA, carpeting contaminated with blood or other potentially infectious materials can not be fully decontaminated.¹⁰³² Therefore, facilities electing to use carpeting for high-activity patient-care areas may choose carpet tiles in areas at high risk for spills.^{967, 1032} In the event of contamination with blood or other body substances, carpet tiles can be removed, discarded, and replaced. OSHA also acknowledges that only minimal direct skin contact occurs with carpeting, and therefore, employers are expected to make reasonable efforts to clean and sanitize carpeting using carpet detergent/cleaner products.¹⁰³²

Over the last few years, some carpet manufacturers have treated their products with fungicidal and/or bactericidal chemicals. Although these chemicals may help to reduce the overall numbers of bacteria or fungi present in carpet, their use does not preclude the routine care and maintenance of the carpeting. Limited evidence suggests that chemically treated carpet may have helped to keep health-care–associated aspergillosis rates low in one HSCT unit,¹¹¹ but overall, treated carpeting has not been shown to prevent the incidence of health-care–associated infections in care areas for immunocompetent patients.

b. Cloth Furnishings

Upholstered furniture and furnishings are becoming increasingly common in patient-care areas. These furnishings range from simple cloth chairs in patients’ rooms to a complete decorating scheme that gives the interior of the facility more the look of an elegant hotel.¹⁰³³ Even though pathogenic microorganisms have been isolated from the surfaces of cloth chairs, no epidemiologic evidence suggests that general patient-care areas with cloth furniture pose increased risks of health-care–

associated infection compared with areas that contain hard-surfaced furniture.^{1034, 1035} Allergens (e.g., dog and cat dander) have been detected in or on cloth furniture in clinics and elsewhere in hospitals in concentrations higher than those found on bed linens.^{1034, 1035} These allergens presumably are transferred from the clothing of visitors. Researchers have therefore suggested that cloth chairs should be vacuumed regularly to keep the dust and allergen levels to a minimum. This recommendation, however, has generated concerns that aerosols created from vacuuming could place immunocompromised patients or patients with preexisting lung disease (e.g., asthma) at risk for development of health-care-associated, environmental airborne disease.^{9, 20, 109, 988} Recovering worn, upholstered furniture (especially the seat cushion) with covers that are easily cleaned (e.g., vinyl), or replacing the item is prudent; minimizing the use of upholstered furniture and furnishings in any patient-care areas where immunosuppressed patients are located (e.g., HSCT units) reduces the likelihood of disease.⁹

5. Flowers and Plants in Patient-Care Areas

Fresh flowers, dried flowers, and potted plants are common items in health-care facilities. In 1974, clinicians isolated an *Erwinia* sp. post mortem from a neonate diagnosed with fulminant septicemia, meningitis, and respiratory distress syndrome.¹⁰³⁸ Because *Erwinia* spp. are plant pathogens, plants brought into the delivery room were suspected to be the source of the bacteria, although the case report did not definitively establish a direct link. Several subsequent studies evaluated the numbers and diversity of microorganisms in the vase water of cut flowers. These studies revealed that high concentrations of bacteria, ranging from 10^4 – 10^{10} CFU/mL, were often present, especially if the water was changed infrequently.^{515, 702, 1039} The major group of microorganisms in flower vase water was gram-negative bacteria, with *Pseudomonas aeruginosa* the most frequently isolated organism.^{515, 702, 1039, 1040} *P. aeruginosa* was also the primary organism directly isolated from chrysanthemums and other potted plants.^{1041, 1042} However, flowers in hospitals were not significantly more contaminated with bacteria compared with flowers in restaurants or in the home.⁷⁰² Additionally, no differences in the diversity and degree of antibiotic resistance of bacteria have been observed in samples isolated from hospital flowers versus those obtained from flowers elsewhere.⁷⁰²

Despite the diversity and large numbers of bacteria associated with flower-vase water and potted plants, minimal or no evidence indicates that the presence of plants in immunocompetent patient-care areas poses an increased risk of health-care-associated infection.⁵¹⁵ In one study involving a limited number of surgical patients, no correlation was observed between bacterial isolates from flowers in the area and the incidence and etiology of postoperative infections among the patients.¹⁰⁴⁰ Similar conclusions were reached in a study that examined the bacteria found in potted plants.¹⁰⁴² Nonetheless, some precautions for general patient-care settings should be implemented, including a) limiting flower and plant care to staff with no direct patient contact, b) advising health-care staff to wear gloves when handling plants, c) washing hands after handling plants, d) changing vase water every 2 days and discharging the water into a sink outside the immediate patient environment, and e) cleaning and disinfecting vases after use.⁷⁰²

Some researchers have examined the possibility of adding a chemical germicide to vase water to control bacterial populations. Certain chemicals (e.g., hydrogen peroxide and chlorhexidine) are well tolerated by plants.^{1040, 1043, 1044} Use of these chemicals, however, was not evaluated in studies to assess impact on health-care-associated infection rates. Modern florists now have a variety of products available to add to vase water to extend the life of cut flowers and to minimize bacterial clouding of the water.

Flowers (fresh and dried) and ornamental plants, however, may serve as a reservoir of *Aspergillus* spp., and dispersal of conidiospores into the air from this source can occur.¹⁰⁹ Health-care-associated outbreaks of invasive aspergillosis reinforce the importance of maintaining an environment as free of

Aspergillus spp. spores as possible for patients with severe, prolonged neutropenia. Potted plants, fresh-cut flowers, and dried flower arrangements may provide a reservoir for these fungi as well as other fungal species (e.g., *Fusarium* spp.).^{109, 1045, 1046} Researchers in one study of bacteria and flowers suggested that flowers and vase water should be avoided in areas providing care to medically at-risk patients (e.g., oncology patients and transplant patients), although this study did not attempt to correlate the observations of bacterial populations in the vase water with the incidence of health-care–associated infections.⁵¹⁵ Another study using molecular epidemiology techniques demonstrated identical *Aspergillus terreus* types among environmental and clinical specimens isolated from infected patients with hematological malignancies.¹⁰⁴⁶ Therefore, attempts should be made to exclude flowers and plants from areas where immunosuppressed patients are located (e.g., HSCT units).^{9, 1046}

6. Pest Control

Cockroaches, flies and maggots, ants, mosquitoes, spiders, mites, midges, and mice are among the typical arthropod and vertebrate pest populations found in health-care facilities. Insects can serve as agents for the mechanical transmission of microorganisms, or as active participants in the disease transmission process by serving as a vector.^{1047–1049} Arthropods recovered from health-care facilities have been shown to carry a wide variety of pathogenic microorganisms.^{1050–1056} Studies have suggested that the diversity of microorganisms associated with insects reflects the microbial populations present in the indoor health-care environment; some pathogens encountered in insects from hospitals were either absent from or present to a lesser degree in insects trapped from residential settings.^{1057–1060} Some of the microbial populations associated with insects in hospitals have demonstrated resistance to antibiotics.^{1048, 1059, 1061–1063}

Insect habitats are characterized by warmth, moisture, and availability of food.¹⁰⁶⁴ Insects forage in and feed on substrates, including but not limited to food scraps from kitchens/cafeteria, foods in vending machines, discharges on dressings either in use or discarded, other forms of human detritus, medical wastes, human wastes, and routine solid waste.^{1057–1061} Cockroaches, in particular, have been known to feed on fixed sputum smears in laboratories.^{1065, 1066} Both cockroaches and ants are frequently found in the laundry, central sterile supply departments, and anywhere in the facility where water or moisture is present (e.g., sink traps, drains and janitor closets). Ants will often find their way into sterile packs of items as they forage in a warm, moist environment.¹⁰⁵⁷ Cockroaches and other insects frequent loading docks and other areas with direct access to the outdoors.

Although insects carry a wide variety of pathogenic microorganisms on their surfaces and in their gut, the direct association of insects with disease transmission (apart from vector transmission) is limited, especially in health-care settings; the presence of insects in itself likely does not contribute substantially to health-care–associated disease transmission in developed countries. However, outbreaks of infection attributed to microorganisms carried by insects may occur because of infestation coupled with breaks in standard infection-control practices.¹⁰⁶³ Studies have been conducted to examine the role of houseflies as possible vectors for shigellosis and other forms of diarrheal disease in non-health-care settings.^{1046, 1067} When control measures aimed at reducing the fly population density were implemented, a concomitant reduction in the incidence of diarrheal infections, carriage of *Shigella* organisms, and mortality caused by diarrhea among infants and young children was observed.

Myiasis is defined as a parasitosis in which the larvae of any of a variety of flies use living or necrotic tissue or body substances of the host as a nutritional source.¹⁰⁶⁸ Larvae from health-care–acquired myiasis have been observed in nares, wounds, eyes, ears, sinuses, and the external urogenital structures.^{1069–1071} Patients with this rare condition are typically older adults with underlying medical conditions (e.g., diabetes, chronic wounds, and alcoholism) who have a decreased capacity to ward off

the flies. Persons with underlying conditions who live or travel to tropical regions of the world are especially at risk.^{1070, 1071} Cases occur in the summer and early fall months in temperate climates when flies are most active.¹⁰⁷¹ An environmental assessment and review of the patient's history are necessary to verify that the source of the myiasis is health-care-acquired and to identify corrective measures.^{1069, 1072} Simple prevention measures (e.g., installing screens on windows) are important in reducing the incidence of myiasis.¹⁰⁷²

From a public health and hygiene perspective, arthropod and vertebrate pests should be eradicated from all indoor environments, including health-care facilities.^{1073, 1074} Modern approaches to institutional pest management usually focus on a) eliminating food sources, indoor habitats, and other conditions that attract pests; b) excluding pests from the indoor environments; and c) applying pesticides as needed.¹⁰⁷⁵ Sealing windows in modern health-care facilities helps to minimize insect intrusion. When windows need to be opened for ventilation, ensuring that screens are in good repair and closing doors to the outside can help with pest control. Insects should be kept out of all areas of the health-care facility, especially ORs and any area where immunosuppressed patients are located. A pest-control specialist with appropriate credentials can provide a regular insect-control program that is tailored to the needs of the facility and uses approved chemicals and/or physical methods. Industrial hygienists can provide information on possible adverse reactions of patients and staff to pesticides and suggest alternative methods for pest control, as needed.

7. Special Pathogen Concerns

a. Antibiotic-Resistant Gram-Positive Cocci

Vancomycin-resistant enterococci (VRE), methicillin-resistant *Staphylococcus aureus* (MRSA), and *S. aureus* with intermediate levels of resistance to glycopeptide antibiotics (vancomycin intermediate resistant *S. aureus* [VISA] or glycopeptide intermediate resistant *S. aureus* [GISA]) represent crucial and growing concerns for infection control. Although the term GISA is technically a more accurate description of the strains isolated to date (most of which are classified as having intermediate resistance to both vancomycin and teicoplanin), the term “glycopeptide” may not be recognized by many clinicians. Thus, the label of VISA, which emphasizes a change in minimum inhibitory concentration (MICs) to vancomycin, is similar to that of VRE and is more meaningful to clinicians.¹⁰⁷⁶ According to National Nosocomial Infection Surveillance (NNIS) statistics for infections acquired among ICU patients in the United States in 1999, 52.3% of infections resulting from *S. aureus* were identified as MRSA infections, and 25.2% of enterococcal infections were attributed to VRE. These figures reflect a 37% and a 43% increase, respectively, since 1994–1998.¹⁰⁷⁷

People represent the primary reservoir of *S. aureus*.¹⁰⁷⁸ Although *S. aureus* has been isolated from a variety of environmental surfaces (e.g., stethoscopes, floors, charts, furniture, dry mops, and hydrotherapy tanks), the role of environmental contamination in transmission of this organism in health care appears to be minimal.^{1079–1082} *S. aureus* contamination of surfaces and tanks within burn therapy units, however, may be a major factor in the transmission of infection among burn patients.¹⁰⁸³

Colonized patients are the principal reservoir of VRE, and patients who are immunosuppressed (e.g., transplant patients) or otherwise medically at-risk (e.g., ICU patients, cardio-thoracic surgical patients, patients previously hospitalized for extended periods, and those having received multi-antimicrobial or vancomycin therapy) are at greatest risk for VRE colonization.^{1084–1087} The mechanisms by which cross-colonization take place are not well defined, although recent studies have indicated that both MRSA and VRE may be transmitted either a) directly from patient to patient, b) indirectly by transient carriage on the hands of health-care workers,^{1088–1091} or c) by hand transfer of these gram-positive organisms from contaminated environmental surfaces and patient-care equipment.^{1084, 1087, 1092–1097} In

one survey, hand carriage of VRE in workers in a long-term care facility ranged from 13%–41%.¹⁰⁹⁸ Many of the environmental surfaces found to be contaminated with VRE in outbreak investigations have been those that are touched frequently by the patient or the health-care worker.¹⁰⁹⁹ Such high-touch surfaces include bedrails, doorknobs, bed linens, gowns, overbed tables, blood pressure cuffs, computer table, bedside tables, and various medical equipment.^{22, 1087, 1094, 1095, 1100–1102} Contamination of environmental surfaces with VRE generally occurs in clinical laboratories and areas where colonized patients are present,^{1087, 1092, 1094, 1095, 1103} but the potential for contamination increases when such patients have diarrhea¹⁰⁸⁷ or have multiple body-site colonization.¹¹⁰⁴ Additional factors that can be important in the dispersion of these pathogens to environmental surfaces are misuse of glove techniques by health-care workers (especially when cleaning fecal contamination from surfaces) and patient, family, and visitor hygiene.

Interest in the importance of environmental reservoirs of VRE increased when laboratory studies demonstrated that enterococci can persist in a viable state on dry environmental surfaces for extended periods of time (7 days to 4 months)^{1099, 1105} and multiple strains can be identified during extensive periods of surveillance.¹¹⁰⁴ VRE can be recovered from inoculated hands of health-care workers (with or without gloves) for up to 60 minutes.²² The presence of either MRSA, VISA, or VRE on environmental surfaces, however, does not mean that patients in the contaminated areas will become colonized. Strict adherence to hand hygiene/handwashing and the proper use of barrier precautions help to minimize the potential for spread of these pathogens. Published recommendations for preventing the spread of vancomycin resistance address isolation measures, including patient cohorting and management of patient-care items.⁵ Direct patient-care items (e.g., blood pressure cuffs) should be disposable whenever possible when used in contact isolation settings for patients with multiply resistant microorganisms.¹¹⁰²

Careful cleaning of patient rooms and medical equipment contributes substantially to the overall control of MRSA, VISA, or VRE transmission. The major focus of a control program for either VRE or MRSA should be the prevention of hand transfer of these organisms. Routine cleaning and disinfection of the housekeeping surfaces (e.g., floors and walls) and patient-care surfaces (e.g., bedrails) should be adequate for inactivation of these organisms. Both MRSA and VRE are susceptible to several EPA-registered low- and intermediate-level disinfectants (e.g., alcohols, sodium hypochlorite, quaternary ammonium compounds, phenolics, and iodophors) at recommended use dilutions for environmental surface disinfection.^{1103, 1106–1109} Additionally, both VRE and vancomycin-sensitive enterococci are equally sensitive to inactivation by chemical germicides,^{1106, 1107, 1109} and similar observations have been made when comparing the germicidal resistance of MRSA to that of either methicillin-sensitive *S. aureus* (MSSA) or VISA.¹¹¹⁰ The use of stronger solutions of disinfectants for inactivation of either VRE, MRSA, or VISA is not recommended based on the organisms' resistance to antibiotics.^{1110–1112} VRE from clinical specimens have exhibited some measure of increased tolerance to heat inactivation in temperature ranges <212°F (<100°C),^{1106, 1113} however, the clinical significance of these observations is unclear because the role of cleaning the surface or item prior to heat treatment was not evaluated. Although routine environmental sampling is not recommended, laboratory surveillance of environmental surfaces during episodes when VRE contamination is suspected can help determine the effectiveness of the cleaning and disinfecting procedures. Environmental culturing should be approved and supervised by the infection-control program in collaboration with the clinical laboratory.^{1084, 1087, 1088, 1092, 1096}

Two cases of wound infections associated with vancomycin-resistant *Staphylococcus aureus* (VRSA) determined to be resistant by NCCLS standards for sensitivity/resistance testing were identified in Michigan and Pennsylvania in 2002.^{1114, 1115} These represented isolated cases, and neither the family members nor the health-care providers of these case-patients had evidence of colonization or infection with VRSA. Conventional environmental infection-control measures (i.e., cleaning and then

disinfecting surfaces using EPA-registered disinfectants with label claims for *S. aureus*) were used during the environmental investigation of these two cases;^{1110–1112} however, studies have yet to evaluate the potential intrinsic resistance of these VRSA strains to surface disinfectants.

Standard procedures during terminal cleaning and disinfection of surfaces, if performed incorrectly, may be inadequate for the elimination of VRE from patient rooms.^{1113, 1116–1118} Given the sensitivity of VRE to hospital disinfectants, current disinfecting protocols should be effective if they are diligently carried out and properly performed. Health-care facilities should be sure that housekeeping staff use correct procedures for cleaning and disinfecting surfaces in VRE-contaminated areas, which include using sufficient amounts of germicide at proper use dilution and allowing adequate contact time.¹¹¹⁸

b. Clostridium difficile

Clostridium difficile is the most frequent etiologic agent for health-care–associated diarrhea.^{1119, 1120} In one hospital, 30% of adults who developed health-care–associated diarrhea were positive for *C. difficile*.¹¹²¹ One recent study employing PCR-ribotyping techniques demonstrated that cases of *C. difficile*-acquired diarrhea occurring in the hospital included patients whose infections were attributed to endogenous *C. difficile* strains and patients whose illnesses were considered to be health-care–associated infections.¹¹²² Most patients remain asymptomatic after infection, but the organism continues to be shed in their stools. Risk factors for acquiring *C. difficile*-associated infection include a) exposure to antibiotic therapy, particularly with beta-lactam agents;¹¹²³ b) gastrointestinal procedures and surgery;¹¹²⁴ c) advanced age; and d) indiscriminate use of antibiotics.^{1125–1128} Of all the measures that have been used to prevent the spread of *C. difficile*-associated diarrhea, the most successful has been the restriction of the use of antimicrobial agents.^{1129, 1130}

C. difficile is an anaerobic, gram-positive bacterium. Normally fastidious in its vegetative state, it is capable of sporulating when environmental conditions no longer support its continued growth. The capacity to form spores enables the organism to persist in the environment (e.g., in soil and on dry surfaces) for extended periods of time. Environmental contamination by this microorganism is well known, especially in places where fecal contamination may occur.¹¹³¹ The environment (especially housekeeping surfaces) rarely serves as a direct source of infection for patients.^{1024, 1132–1136} However, direct exposure to contaminated patient-care items (e.g., rectal thermometers) and high-touch surfaces in patients' bathrooms (e.g., light switches) have been implicated as sources of infection.^{1130, 1135, 1136, 1138}

Transfer of the pathogen to the patient via the hands of health-care workers is thought to be the most likely mechanism of exposure.^{24, 1133, 1139} Standard isolation techniques intended to minimize enteric contamination of patients, health-care–workers' hands, patient-care items, and environmental surfaces have been published.¹¹⁴⁰ Handwashing remains the most effective means of reducing hand contamination. Proper use of gloves is an ancillary measure that helps to further minimize transfer of these pathogens from one surface to another.

The degree to which the environment becomes contaminated with *C. difficile* spores is proportional to the number of patients with *C. difficile*-associated diarrhea,^{24, 1132, 1135} although asymptomatic, colonized patients may also serve as a source of contamination. Few studies have examined the use of specific chemical germicides for the inactivation of *C. difficile* spores, and no well-controlled trials have been conducted to determine efficacy of surface disinfection and its impact on health-care–associated diarrhea. Some investigators have evaluated the use of chlorine-containing chemicals (e.g., 1,000 ppm hypochlorite at recommended use-dilution, 5,000 ppm sodium hypochlorite [1:10 v/v dilution], 1:100 v/v dilutions of unbuffered hypochlorite, and phosphate-buffered hypochlorite [1,600 ppm]). One of the studies demonstrated that the number of contaminated environmental sites was reduced by half,¹¹³⁵ whereas another two studies demonstrated declines in health-care–associated *C. difficile* infections in a HSCT unit¹¹⁴¹ and in two geriatric medical units¹¹⁴² during a period of hypochlorite use. The presence

of confounding factors, however, was acknowledged in one of these studies.¹¹⁴² The recommended approach to environmental infection control with respect to *C. difficile* is meticulous cleaning followed by disinfection using hypochlorite-based germicides as appropriate.^{952, 1130, 1143} However, because no EPA-registered surface disinfectants with label claims for inactivation of *C. difficile* spores are available, the recommendation is based on the best available evidence from the scientific literature.

c. Respiratory and Enteric Viruses in Pediatric-Care Settings

Although the viruses mentioned in this guideline are not unique to the pediatric-care setting in health-care facilities, their prevalence in these areas, especially during the winter months, is substantial. Children (particularly neonates) are more likely to develop infection and substantial clinical disease from these agents compared with adults and therefore are more likely to require supportive care during their illness.

Common respiratory viruses in pediatric-care areas include rhinoviruses, respiratory syncytial virus (RSV), adenoviruses, influenza viruses, and parainfluenza viruses. Transmission of these viruses occurs primarily via direct contact with small-particle aerosols or via hand contamination with respiratory secretions that are then transferred to the nose or eyes. Because transmission primarily requires close personal contact, contact precautions are appropriate to interrupt transmission.⁶ Hand contamination can occur from direct contact with secretions or indirectly from touching high-touch environmental surfaces that have become contaminated with virus from large droplets. The indirect transfer of virus from one person to other via hand contact with frequently-touched fomites was demonstrated in a study using a bacteriophage whose environmental stability approximated that of human viral pathogens (e.g., poliovirus and parvovirus).¹¹⁴⁴ The impact of this mode of transmission with respect to human respiratory- and enteric viruses is dependent on the ability of these agents to survive on environmental surfaces. Infectious RSV has been recovered from skin, porous surfaces, and non-porous surfaces after 30 minutes, 1 hour, and 7 hours, respectively.¹¹⁴⁵ Parainfluenza viruses are known to persist for up to 4 hours on porous surfaces and up to 10 hours on non-porous surfaces.¹¹⁴⁶ Rhinoviruses can persist on porous surfaces and non-porous surfaces for approximately 1 and 3 hours respectively; study participants in a controlled environment became infected with rhinoviruses after first touching a surface with dried secretions and then touching their nasal or conjunctival mucosa.¹¹⁴⁷ Although the efficiency of direct transmission of these viruses from surfaces in uncontrolled settings remains to be defined, these data underscore the basis for maintaining regular protocols for cleaning and disinfecting of high-touch surfaces.

The clinically important enteric viruses encountered in pediatric care settings include enteric adenovirus, astroviruses, caliciviruses, and rotavirus. Group A rotavirus is the most common cause of infectious diarrhea in infants and children. Transmission of this virus is primarily fecal-oral, however, the role of fecally contaminated surfaces and fomites in rotavirus transmission is unclear. During one epidemiologic investigation of enteric disease among children attending day care, rotavirus contamination was detected on 19% of inanimate objects in the center.^{1148, 1149} In an outbreak in a pediatric unit, secondary cases of rotavirus infection clustered in areas where children with rotaviral diarrhea were located.¹¹⁵⁰ Astroviruses cause gastroenteritis and diarrhea in newborns and young children and can persist on fecally contaminated surfaces for several months during periods of relatively low humidity.^{1151, 1152} Outbreaks of small round-structured viruses (i.e., caliciviruses [Norwalk virus and Norwalk-like viruses]) can affect both patients and staff, with attack rates of $\geq 50\%$.¹¹⁵³ Routes of person-to-person transmission include fecal-oral spread and aerosols generated from vomiting.^{1154–1156} Fecal contamination of surfaces in care settings can spread large amounts of virus to the environment. Studies that have attempted to use low- and intermediate-level disinfectants to inactivate rotavirus suspended in feces have demonstrated a protective effect of high concentrations of organic matter.^{1157, 1158} Intermediate-level disinfectants (e.g., alcoholic quaternary ammonium compounds, and chlorine solutions) can be effective in inactivating enteric viruses provided that a cleaning step to remove most of

the organic matter precedes terminal disinfection.¹¹⁵⁸ These findings underscore the need for proper cleaning and disinfecting procedures where contamination of environmental surfaces with body substances is likely. EPA-registered surface disinfectants with label claims for these viral agents should be used in these settings. Using disposable, protective barrier coverings may help to minimize the degree of surface contamination.⁹³⁶

d. Severe Acute Respiratory Syndrome (SARS) Virus

In November 2002 an atypical pneumonia of unknown etiology emerged in Asia and subsequently developed into an international outbreak of respiratory illness among persons in 29 countries during the first six months of 2003. “Severe acute respiratory syndrome” (SARS) is a viral upper respiratory infection associated with a newly described coronavirus (SARS-associated Co-V [SARS-CoV]). SARS-CoV is an enveloped RNA virus. It is present in high titers in respiratory secretions, stool, and blood of infected persons. The modes of transmission determined from epidemiologic investigations were primarily forms of direct contact (i.e., large droplet aerosolization and person-to-person contact). Respiratory secretions were presumed to be the major source of virus in these situations; airborne transmission of virus has not been completely ruled out. Little is known about the impact of fecal-oral transmission and SARS.

The epidemiology of SARS-CoV infection is not completely understood, and therefore recommended infection control and prevention measures to contain the spread of SARS will evolve as new information becomes available.¹¹⁵⁹ At present there is no indication that established strategies for cleaning (i.e., to remove the majority of bioburden) and disinfecting equipment and environmental surfaces need to be changed for the environmental infection control of SARS. In-patient rooms housing SARS patients should be cleaned and disinfected at least daily and at the time of patient transfer or discharge. More frequent cleaning and disinfection may be indicated for high-touch surfaces and following aerosol-producing procedures (e.g., intubation, bronchoscopy, and sputum production). While there are presently no disinfectant products registered by EPA specifically for inactivation of SARS-CoV, EPA-registered hospital disinfectants that are equivalent to low- and intermediate-level germicides may be used on pre-cleaned, hard, non-porous surfaces in accordance with manufacturer’s instructions for environmental surface disinfection. Monitoring adherence to guidelines established for cleaning and disinfection is an important component of environmental infection control to contain the spread of SARS.

e. Creutzfeldt-Jakob Disease (CJD) in Patient-Care Areas

Creutzfeldt-Jakob disease (CJD) is a rare, invariably fatal, transmissible spongiform encephalopathy (TSE) that occurs worldwide with an average annual incidence of 1 case per million population.¹¹⁶⁰⁻¹¹⁶² CJD is one of several TSEs affecting humans; other diseases in this group include kuru, fatal familial insomnia, and Gerstmann-Sträussler-Scheinker syndrome. A TSE that affects a younger population (compared to the age range of CJD cases) has been described primarily in the United Kingdom since 1996.¹¹⁶³ This variant form of CJD (vCJD) is clinically and neuropathologically distinguishable from classic CJD; epidemiologic and laboratory evidence suggests a causal association for bovine spongiform encephalopathy (BSE [Mad Cow disease]) and vCJD.¹¹⁶³⁻¹¹⁶⁶

The agent associated with CJD is a prion, which is an abnormal isoform of a normal protein constituent of the central nervous system.¹¹⁶⁷⁻¹¹⁶⁹ The mechanism by which the normal form of the protein is converted to the abnormal, disease-causing prion is unknown. The tertiary conformation of the abnormal prion protein appears to confer a heightened degree of resistance to conventional methods of sterilization and disinfection.^{1170, 1171}

Although about 90% of CJD cases occur sporadically, a limited number of cases are the result of a direct exposure to prion-containing material (usually central nervous system tissue or pituitary

hormones) acquired as a result of health care (iatrogenic cases). These cases have been linked to a) pituitary hormone therapy [from human sources as opposed to hormones prepared through the use of recombinant technology],^{1170–1174} b) transplants of either dura mater or corneas,^{1175–1181} and c) neurosurgical instruments and depth electrodes.^{1182–1185} In the cases involving instruments and depth electrodes, conventional cleaning and terminal reprocessing methods of the day failed to fully inactivate the contaminating prions and are considered inadequate by today's standards.

Prion inactivation studies involving whole tissues and tissue homogenates have been conducted to determine the parameters of physical and chemical methods of sterilization or disinfection necessary for complete inactivation,^{1170, 1186–1191} however, the application of these findings to environmental infection control in health-care settings is problematic. No studies have evaluated the effectiveness of medical instrument reprocessing in inactivating prions. Despite a consensus that abnormal prions display some extreme measure of resistance to inactivation by either physical or chemical methods, scientists disagree about the exact conditions needed for sterilization. Inactivation studies utilizing whole tissues present extraordinary challenges to any sterilizing method.¹¹⁹² Additionally, the experimental designs of these studies preclude the evaluation of surface cleaning as a part of the total approach to pathogen inactivation.^{951, 1192}

Some researchers have recommended the use of either a 1:2 v/v dilution of sodium hypochlorite (approximately 20,000 ppm), full-strength sodium hypochlorite (50,000–60,000 ppm), or 1–2 N sodium hydroxide (NaOH) for the inactivation of prions on certain surfaces (e.g., those found in the pathology laboratory).^{1170, 1188} Although these chemicals may be appropriate for the decontamination of laboratory, operating-room, or autopsy-room surfaces that come into contact with central nervous system tissue from a known or suspected patient, this approach is not indicated for routine or terminal cleaning of a room previously occupied by a CJD patient. Both chemicals pose hazards for the health-care worker doing the decontamination. NaOH is caustic and should not make contact with the skin. Sodium hypochlorite solutions (i.e., chlorine bleach) can corrode metals (e.g., aluminum). MSDS information should be consulted when attempting to work with concentrated solutions of either chemical. Currently, no EPA-registered products have label claims for prion inactivation; therefore, this guidance is based on the best available evidence from the scientific literature.

Environmental infection-control strategies must be based on the principles of the “chain of infection,” regardless of the disease of concern.¹³ Although CJD is transmissible, it is not highly contagious. All iatrogenic cases of CJD have been linked to a direct exposure to prion-contaminated central nervous system tissue or pituitary hormones. The six documented iatrogenic cases associated with instruments and devices involved neurosurgical instruments and devices that introduced residual contamination directly to the recipient's brain. No evidence suggests that vCJD has been transmitted iatrogenically or that either CJD or vCJD has been transmitted from environmental surfaces (e.g., housekeeping surfaces). Therefore, routine procedures are adequate for terminal cleaning and disinfection of a CJD patient's room. Additionally, in epidemiologic studies involving highly transfused patients, blood was not identified as a source for prion transmission.^{1193–1198} Routine procedures for containing, decontaminating, and disinfecting surfaces with blood spills should be adequate for proper infection control in these situations.^{951, 1199}

Guidance for environmental infection control in ORs and autopsy areas has been published.^{1197, 1199} Hospitals should develop risk-assessment procedures to identify patients with known or suspected CJD in efforts to implement prion-specific infection-control measures for the OR and for instrument reprocessing.¹²⁰⁰ This assessment also should be conducted for older patients undergoing non-lesionous neurosurgery when such procedures are being done for diagnosis. Disposable, impermeable coverings should be used during these autopsies and neurosurgeries to minimize surface contamination. Surfaces that have become contaminated with central nervous system tissue or cerebral spinal fluid should be

cleaned and decontaminated by a) removing most of the tissue or body substance with absorbent materials, b) wetting the surface with a sodium hypochlorite solution containing $\geq 5,000$ ppm or a 1 N NaOH solution, and c) rinsing thoroughly.^{951, 1197–1199, 1201} The optimum duration of contact exposure in these instances is unclear. Some researchers recommend a 1-hour contact time on the basis of tissue-inactivation studies,^{1197, 1198, 1201} whereas other reviewers of the subject draw no conclusions from this research.¹¹⁹⁹ Factors to consider before cleaning a potentially contaminated surface are a) the degree to which gross tissue/body substance contamination can be effectively removed and b) the ease with which the surface can be cleaned.

F. Environmental Sampling

This portion of Part I addresses the basic principles and methods of sampling environmental surfaces and other environmental sources for microorganisms. The applied strategies of sampling with respect to environmental infection control have been discussed in the appropriate preceding subsections.

1. General Principles: Microbiologic Sampling of the Environment

Before 1970, U.S. hospitals conducted regularly scheduled culturing of the air and environmental surfaces (e.g., floors, walls, and table tops).¹²⁰² By 1970, CDC and the American Hospital Association (AHA) were advocating the discontinuation of routine environmental culturing because rates of health-care–associated infection had not been associated with levels of general microbial contamination of air or environmental surfaces, and because meaningful standards for permissible levels of microbial contamination of environmental surfaces or air did not exist.^{1203–1205} During 1970–1975, 25% of U.S. hospitals reduced the extent of such routine environmental culturing — a trend that has continued.^{1206, 1207}

Random, undirected sampling (referred to as “routine” in previous guidelines) differs from the current practice of targeted sampling for defined purposes.^{2, 1204} Previous recommendations against routine sampling were not intended to discourage the use of sampling in which sample collection, culture, and interpretation are conducted in accordance with defined protocols.² In this guideline, targeted microbiologic sampling connotes a monitoring process that includes a) a written, defined, multidisciplinary protocol for sample collection and culturing; b) analysis and interpretation of results using scientifically determined or anticipatory baseline values for comparison; and c) expected actions based on the results obtained. Infection control, in conjunction with laboratorians, should assess the health-care facility’s capability to conduct sampling and determine when expert consultation and/or services are needed.

Microbiologic sampling of air, water, and inanimate surfaces (i.e., environmental sampling) is an expensive and time-consuming process that is complicated by many variables in protocol, analysis, and interpretation. It is therefore indicated for only four situations.¹²⁰⁸ The first is to support an investigation of an outbreak of disease or infections when environmental reservoirs or fomites are implicated epidemiologically in disease transmission.^{161, 1209, 1210} It is important that such culturing be supported by epidemiologic data. Environmental sampling, as with all laboratory testing, should not be conducted if there is no plan for interpreting and acting on the results obtained.^{11, 1211, 1212} Linking microorganisms from environmental samples with clinical isolates by molecular epidemiology is crucial whenever it is possible to do so.

The second situation for which environmental sampling may be warranted is in research. Well-designed and controlled experimental methods and approaches can provide new information about the spread of health-care–associated diseases.^{126, 129} A classic example is the study of environmental microbial

contamination that compared health-care–associated infection rates in an old hospital and a new facility before and shortly after occupancy.⁹⁴⁷

The third indication for sampling is to monitor a potentially hazardous environmental condition, confirm the presence of a hazardous chemical or biological agent, and validate the successful abatement of the hazard. This type of sampling can be used to: a) detect bioaerosols released from the operation of health-care equipment (e.g., an ultrasonic cleaner) and determine the success of repairs in containing the hazard,¹²¹³ b) detect the release of an agent of bioterrorism in an indoor environmental setting and determine its successful removal or inactivation, and c) sample for industrial hygiene or safety purposes (e.g., monitoring a “sick building”).

The fourth indication is for quality assurance to evaluate the effects of a change in infection-control practice or to ensure that equipment or systems perform according to specifications and expected outcomes. Any sampling for quality-assurance purposes must follow sound sampling protocols and address confounding factors through the use of properly selected controls. Results from a single environmental sample are difficult to interpret in the absence of a frame of reference or perspective. Evaluations of a change in infection-control practice are based on the assumption that the effect will be measured over a finite period, usually of short duration. Conducting quality-assurance sampling on an extended basis, especially in the absence of an adverse outcome, is usually unjustified. A possible exception might be the use of air sampling during major construction periods to qualitatively detect breaks in environmental infection-control measures. In one study, which began as part of an investigation of an outbreak of health-care–associated aspergillosis, airborne concentrations of *Aspergillus* spores were measured in efforts to evaluate the effectiveness of sealing hospital doors and windows during a period of construction of a nearby building.⁵⁰ Other examples of sampling for quality-assurance purposes may include commissioning newly constructed space in special care areas (i.e., ORs and units for immunosuppressed patients) or assessing a change in housekeeping practice. However, the only types of routine environmental microbiologic sampling recommended as part of a quality-assurance program are a) the biological monitoring of sterilization processes by using bacterial spores¹²¹⁴ and b) the monthly culturing of water used in hemodialysis applications and for the final dialysate use dilution. Some experts also advocate periodic environmental sampling to evaluate the microbial/particulate quality for regular maintenance of the air handling system (e.g., filters) and to verify that the components of the system meet manufacturer’s specifications (A. Streifel, University of Minnesota, 2000). Certain equipment in health-care settings (e.g., biological safety cabinets) may also be monitored with air flow and particulate sampling to determine performance or as part of adherence to a certification program; results can then be compared with a predetermined standard of performance. These measurements, however, usually do not require microbiologic testing.

2. Air Sampling

Biological contaminants occur in the air as aerosols and may include bacteria, fungi, viruses, and pollens.^{1215, 1216} Aerosols are characterized as solid or liquid particles suspended in air. Talking for 5 minutes and coughing each can produce 3,000 droplet nuclei; sneezing can generate approximately 40,000 droplets which then evaporate to particles in the size range of 0.5–12 μm .^{137, 1217} Particles in a biological aerosol usually vary in size from $<1 \mu\text{m}$ to $\geq 50 \mu\text{m}$. These particles may consist of a single, unattached organism or may occur in the form of clumps composed of a number of bacteria. Clumps can also include dust and dried organic or inorganic material. Vegetative forms of bacterial cells and viruses may be present in the air in a lesser number than bacterial spores or fungal spores. Factors that determine the survival of microorganisms within a bioaerosol include a) the suspending medium, b) temperature, c) relative humidity, d) oxygen sensitivity, and e) exposure to UV or electromagnetic radiation.¹²¹⁵ Many vegetative cells will not survive for lengthy periods of time in the air unless the

relative humidity and other factors are favorable for survival and the organism is enclosed within some protective cover (e.g., dried organic or inorganic matter).¹²¹⁶ Pathogens that resist drying (e.g., *Staphylococcus* spp., *Streptococcus* spp., and fungal spores) can survive for long periods and can be carried considerable distances via air and still remain viable. They may also settle on surfaces and become airborne again as secondary aerosols during certain activities (e.g., sweeping and bed making).^{1216, 1218}

Microbiologic air sampling is used as needed to determine the numbers and types of microorganisms, or particulates, in indoor air.²⁸⁹ Air sampling for quality control is, however, problematic because of lack of uniform air-quality standards. Although airborne spores of *Aspergillus* spp. can pose a risk for neutropenic patients, the critical number (i.e., action level) of these spores above which outbreaks of aspergillosis would be expected to occur has not been defined. Health-care professionals considering the use of air sampling should keep in mind that the results represent indoor air quality at singular points in time, and these may be affected by a variety of factors, including a) indoor traffic, b) visitors entering the facility, c) temperature, d) time of day or year, e) relative humidity, f) relative concentration of particles or organisms, and g) the performance of the air-handling system components. To be meaningful, air-sampling results must be compared with those obtained from other defined areas, conditions, or time periods.

Several preliminary concerns must be addressed when designing a microbiologic air sampling strategy (Box 13). Because the amount of particulate material and bacteria retained in the respiratory system is largely dependent on the size of the inhaled particles, particle size should be determined when studying airborne microorganisms and their relation to respiratory infections. Particles $>5\ \mu\text{m}$ are efficiently trapped in the upper respiratory tract and are removed primarily by ciliary action.¹²¹⁹ Particles $\leq 5\ \mu\text{m}$ in diameter reach the lung, but the greatest retention in the alveoli is of particles 1–2 μm in diameter.^{1220–1222}

Box 13. Preliminary concerns for conducting air sampling

-
- Consider the possible characteristics and conditions of the aerosol, including size range of particles, relative amount of inert material, concentration of microorganisms, and environmental factors.
 - Determine the type of sampling instruments, sampling time, and duration of the sampling program.
 - Determine the number of samples to be taken.
 - Ensure that adequate equipment and supplies are available.
 - Determine the method of assay that will ensure optimal recovery of microorganisms.
 - Select a laboratory that will provide proper microbiologic support.
 - Ensure that samples can be refrigerated if they cannot be assayed in the laboratory promptly.
-

Bacteria, fungi, and particulates in air can be identified and quantified with the same methods and equipment (Table 23). The basic methods include a) impingement in liquids, b) impaction on solid surfaces, c) sedimentation, d) filtration, e) centrifugation, f) electrostatic precipitation, and g) thermal precipitation.¹²¹⁸ Of these, impingement in liquids, impaction on solid surfaces, and sedimentation (on settle plates) have been used for various air-sampling purposes in health-care settings.²⁸⁹

Several instruments are available for sampling airborne bacteria and fungi (Box 14). Some of the samplers are self-contained units requiring only a power supply and the appropriate collecting medium, but most require additional auxiliary equipment (e.g., a vacuum pump and an airflow measuring device [i.e., a flowmeter or anemometer]). Sedimentation or depositional methods use settle plates and

therefore need no special instruments or equipment. Selection of an instrument for air sampling requires a clear understanding of the type of information desired and the particular determinations that must be made (Box 14). Information may be needed regarding a) one particular organism or all organisms that may be present in the air, b) the concentration of viable particles or of viable organisms, c) the change in concentration with time, and d) the size distribution of the collected particles. Before sampling begins, decisions should be made regarding whether the results are to be qualitative or quantitative. Comparing quantities of airborne microorganisms to those of outdoor air is also standard operating procedure. Infection-control professionals, hospital epidemiologists, industrial hygienists, and laboratory supervisors, as part of a multidisciplinary team, should discuss the potential need for microbial air sampling to determine if the capacity and expertise to conduct such sampling exists within the facility and when it is appropriate to enlist the services of an environmental microbiologist consultant.

Table 23. Air sampling methods and examples of equipment*

Method	Principle	Suitable for measuring:	Collection media or surface	Rate of collection (L/min.)	Auxilliary equipment needed+	Points to consider	Prototype samplers§
Impingement in liquids	Air drawn through a small jet and directed against a liquid surface	Viable organisms, and concentration over time. Example use: sampling water aerosols to <i>Legionella</i> spp.	Buffered gelatin, tryptose saline, peptone, nutrient broth	12.5	Yes	Antifoaming agent may be needed. Ambient temperature and humidity will influence length of collection time	Chemical Corps. All Glass Impinger (AGI)
Impaction on solid surfaces	Air drawn into the sampler; particles deposited on a dry surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to organisms that resist drying and spores); size measurement, and concentration over time. Example use: sampling air for <i>Aspergillus</i> spp., fungal spores	Dry surface, coated surfaces, and agar	28 (sieve) 30–800 (slit)	Yes	Available as sieve impactors or slit impactors. Sieve impactors can be set up to measure particle size. Slit impactors have a rotating support stage for agar plates to allow for measurement of concentration over time.	Andersen Air Sampler (sieve impactor); TDL, Cassella MK-2 (slit impactors)
Sedimentation	Particles and micro-organisms settle onto surfaces via gravity	Viable particles. Example uses: sampling air for bacteria in the vicinity of and during a medical procedure; general measurements of microbial air quality.	Nutrient media (agars) on plates or slides	—	No	Simple and inexpensive; best suited for qualitative sampling; significant airborne fungal spores are too buoyant to settle efficiently for collection using this method.	Settle plates

Method	Principle	Suitable for measuring:	Collection media or surface	Rate of collection (L/min.)	Auxilliary equipment needed+	Points to consider	Prototype samplers§
Filtration	Air drawn through a filter unit; particles trapped; 0.2 µm pore size	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., fungal spores, and dust	Paper, cellulose, glass wool, gelatin foam, and membrane filters	1–50	Yes	Filter must be agitated first in rinse fluid to remove and disperse trapped micro-organisms; rinse fluid is assayed; used more for sampling dust and chemicals.	—
Centrifugation	Aerosols subjected to centrifugal force; particles impacted onto a solid surface	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time. Example use: air sampling for <i>Aspergillus</i> spp., and fungal spores	Coated glass or plastic slides, and agar surfaces	40–50	Yes	Calibration is difficult and is done only by the factory; relative comparison of airborne contamination is its general use.	Biotest RCS Plus
Electrostatic precipitation	Air drawn over an electrostatically charged surface; particles become charged	Viable particles; viable organisms (on non-nutrient surfaces, limited to spores and organisms that resist drying); concentration over time	Solid collecting surfaces (glass, and agar)	85	Yes	High volume sampling rate, but equipment is complex and must be handled carefully; not practical for use in health-care settings.	—
Thermal precipitation	Air drawn over a thermal gradient; particles repelled from hot surfaces, settle on colder surfaces	Size measurements	Glass coverslip, and electron microscope grid	0.003–0.4	Yes	Determine particle size by direct observation; not frequently used because of complex adjustments and low sampling rates.	—

* Material in this table is compiled from references 289, 1218, 1223, and 1224.

+ Most samplers require a flow meter or anemometer and a vacuum source as auxiliary equipment.

§ Trade names listed are for identification purposes only and are not intended as endorsements by the U.S. Public Health Service.

Box 14. Selecting an air sampling device*

The following factors must be considered when choosing an air sampling instrument:

- Viability and type of the organism to be sampled
- Compatibility with the selected method of analysis
- Sensitivity of particles to sampling
- Assumed concentrations and particle size
- Whether airborne clumps must be broken (i.e., total viable organism count vs. particle count)
- Volume of air to be sampled and length of time sampler is to be continuously operated
- Background contamination
- Ambient conditions
- Sampler collection efficiency
- Effort and skill required to operate sampler
- Availability and cost of sampler, plus back-up samplers in case of equipment malfunction
- Availability of auxiliary equipment and utilities (e.g., vacuum pumps, electricity, and water)

* Material in this box is compiled from reference 1218.

Liquid impinger and solid impactor samplers are the most practical for sampling bacteria, particles, and fungal spores, because they can sample large volumes of air in relatively short periods of time.²⁸⁹ Solid impactor units are available as either “slit” or “sieve” designs. Slit impactors use a rotating disc as support for the collecting surface, which allows determinations of concentration over time. Sieve impactors commonly use stages with calibrated holes of different diameters. Some impactor-type samplers use centrifugal force to impact particles onto agar surfaces. The interior of either device must be made sterile to avoid inadvertent contamination from the sampler. Results obtained from either sampling device can be expressed as organisms or particles per unit volume of air (CFU/m³).

Sampling for bacteria requires special attention, because bacteria may be present as individual organisms, as clumps, or mixed with or adhering to dust or covered with a protective coating of dried organic or inorganic substances. Reports of bacterial concentrations determined by air sampling therefore must indicate whether the results represent individual organisms or particles bearing multiple cells. Certain types of samplers (e.g., liquid impingers) will completely or partially disintegrate clumps and large particles; the sampling result will therefore reflect the total number of individual organisms present in the air.

The task of sizing a bioaerosol is simplified through the use of sieves or slit impactors because these samplers will separate the particles and microorganisms into size ranges as the sample is collected. These samplers must, however, be calibrated first by sampling aerosols under similar use conditions.¹²²⁵

The use of settle plates (i.e., the sedimentation or depositional method) is not recommended when sampling air for fungal spores, because single spores can remain suspended in air indefinitely.²⁸⁹ Settle plates have been used mainly to sample for particulates and bacteria either in research studies or during epidemiologic investigations.^{161, 1226–1229} Results of sedimentation sampling are typically expressed as numbers of viable particles or viable bacteria per unit area per the duration of sampling time (i.e., CFU/area/time); this method can not quantify the volume of air sampled. Because the survival of microorganisms during air sampling is inversely proportional to the velocity at which the air is taken into the sampler,¹²¹⁵ one advantage of using a settle plate is its reliance on gravity to bring organisms and particles into contact with its surface, thus enhancing the potential for optimal survival of collected organisms. This process, however, takes several hours to complete and may be impractical for some situations.

Air samplers are designed to meet differing measurement requirements. Some samplers are better suited for one form of measurement than others. No one type of sampler and assay procedure can be used to collect and enumerate 100% of airborne organisms. The sampler and/or sampling method chosen should, however, have an adequate sampling rate to collect a sufficient number of particles in a reasonable time period so that a representative sample of air is obtained for biological analysis. Newer analytical techniques for assaying air samples include PCR methods and enzyme-linked immunosorbent assays (ELISAs).

3. Water Sampling

A detailed discussion of the principles and practices of water sampling has been published.⁹⁴⁵ Water sampling in health-care settings is used to detect waterborne pathogens of clinical significance or to determine the quality of finished water in a facility's distribution system. Routine testing of the water in a health-care facility is usually not indicated, but sampling in support of outbreak investigations can help determine appropriate infection-control measures. Water-quality assessments in dialysis settings have been discussed in this guideline (see Water, Dialysis Water Quality and Dialysate, and Appendix C).

Health-care facilities that conduct water sampling should have their samples assayed in a laboratory that uses established methods and quality-assurance protocols. Water specimens are not "static specimens" at ambient temperature; potential changes in both numbers and types of microbial populations can occur during transport. Consequently, water samples should be sent to the testing laboratory cold (i.e., at approximately 39.2°F [4°C]) and testing should be done as soon as practical after collection (preferably within 24 hours).

Because most water sampling in health-care facilities involves the testing of finished water from the facility's distribution system, a reducing agent (i.e., sodium thiosulfate [Na₂S₂O₃]) needs to be added to neutralize residual chlorine or other halogen in the collected sample. If the water contains elevated levels of heavy metals, then a chelating agent should be added to the specimen. The minimum volume of water to be collected should be sufficient to complete any and all assays indicated; 100 mL is considered a suitable minimum volume. Sterile collection equipment should always be used.

Sampling from a tap requires flushing of the water line before sample collection. If the tap is a mixing faucet, attachments (e.g., screens and aerators) must be removed, and hot and then cold water must be run through the tap before collecting the sample.⁹⁴⁵ If the cleanliness of the tap is questionable, disinfection with 500–600 ppm sodium hypochlorite (1:100 v/v dilution of chlorine bleach) and flushing the tap should precede sample collection.

Microorganisms in finished or treated water often are physically damaged ("stressed") to the point that growth is limited when assayed under standard conditions. Such situations lead to false-negative readings and misleading assessments of water quality. Appropriate neutralization of halogens and chelation of heavy metals are crucial to the recovery of these organisms. The choice of recovery media and incubation conditions will also affect the assay. Incubation temperatures should be closer to the ambient temperature of the water rather than at 98.6°F (37°C), and recovery media should be formulated to provide appropriate concentrations of nutrients to support organisms exhibiting less than rigorous growth.⁹⁴⁵ High-nutrient content media (e.g., blood agar and tryptic soy agar [TSA]) may actually inhibit the growth of these damaged organisms. Reduced nutrient media (e.g., diluted peptone and R2A) are preferable for recovery of these organisms.⁹⁴⁵

Use of aerobic, heterotrophic plate counts allows both a qualitative and quantitative measurement for water quality. If bacterial counts in water are expected to be high in number (e.g., during waterborne outbreak investigations), assaying small quantities using pour plates or spread plates is appropriate.⁹⁴⁵ Membrane filtration is used when low-count specimens are expected and larger sampling volumes are required (≥ 100 mL). The sample is filtered through the membrane, and the filter is applied directly face-up onto the surface of the agar plate and incubated.

Unlike the testing of potable water supplies for coliforms (which uses standardized test and specimen collection parameters and conditions), water sampling to support epidemiologic investigations of disease outbreaks may be subjected to modifications dictated by the circumstances present in the facility. Assay methods for waterborne pathogens may also not be standardized. Therefore, control or comparison samples should be included in the experimental design. Any departure from a standard method should be fully documented and should be considered when interpreting results and developing strategies. Assay methods specific for clinically significant waterborne pathogens (e.g., *Legionella* spp., *Aeromonas* spp, *Pseudomonas* spp., and *Acinetobacter* spp.) are more complicated and costly compared with both methods used to detect coliforms and other standard indicators of water quality.

4. Environmental Surface Sampling

Routine environmental-surface sampling (e.g., surveillance cultures) in health-care settings is neither cost-effective nor warranted.^{951, 1225} When indicated, surface sampling should be conducted with multidisciplinary approval in adherence to carefully considered plans of action and policy (Box 15).

Box 15. Undertaking environmental-surface sampling*

The following factors should be considered before engaging in environmental-surface sampling:

- **Background information from the literature and present activities (i.e., preliminary results from an epidemiologic investigation)**
 - **Location of surfaces to be sampled**
 - **Method of sample collection and the appropriate equipment for this task**
 - **Number of replicate samples needed and which control or comparison samples are required**
 - **Parameters of the sample assay method and whether the sampling will be qualitative, quantitative, or both**
 - **An estimate of the maximum allowable microbial numbers or types on the surface(s) sampled (refer to the Spaulding classification for devices and surfaces)**
 - **Some anticipation of a corrective action plan**
-

* The material in this box is compiled from reference 1214.

Surface sampling is used currently for research, as part of an epidemiologic investigation, or as part of a comprehensive approach for specific quality assurance purposes. As a research tool, surface sampling has been used to determine a) potential environmental reservoirs of pathogens,^{564, 1230–1232} b) survival of microorganisms on surfaces,^{1232, 1233} and c) the sources of the environmental contamination.¹⁰²³ Some or all of these approaches can also be used during outbreak investigations.¹²³² Discussion of surface sampling of medical devices and instruments is beyond the scope of this document and is deferred to future guidelines on sterilization and disinfection issues.

Meaningful results depend on the selection of appropriate sampling and assay techniques.¹²¹⁴ The media, reagents, and equipment required for surface sampling are available from any well-equipped

microbiology laboratory and laboratory supplier. For quantitative assessment of surface organisms, non-selective, nutrient-rich agar media and broth (e.g., TSA and brain-heart infusion broth [BHI] with or without 5% sheep or rabbit blood supplement) are used for the recovery of aerobic bacteria. Broth media are used with membrane-filtration techniques. Further sample work-up may require the use of selective media for the isolation and enumeration of specific groups of microorganisms. Examples of selective media are MacConkey agar (MAC [selects for gram-negative bacteria]), Cetrimide agar (selects for *Pseudomonas aeruginosa*), or Sabouraud dextrose- and malt extract agars and broths (select for fungi). Qualitative determinations of organisms from surfaces require only the use of selective or non-selective broth media.

Effective sampling of surfaces requires moisture, either already present on the surface to be sampled or via moistened swabs, sponges, wipes, agar surfaces, or membrane filters.^{1214, 1234–1236} Dilution fluids and rinse fluids include various buffers or general purpose broth media (Table 24). If disinfectant residuals are expected on surfaces being sampled, specific neutralizer chemicals should be used in both the growth media and the dilution or rinse fluids. Lists of the neutralizers, the target disinfectant active ingredients, and the use concentrations have been published.^{1214, 1237} Alternatively, instead of adding neutralizing chemicals to existing culture media (or if the chemical nature of the disinfectant residuals is unknown), the use of either a) commercially available media including a variety of specific and non-specific neutralizers or b) double-strength broth media will facilitate optimal recovery of microorganisms. The inclusion of appropriate control specimens should be included to rule out both residual antimicrobial activity from surface disinfectants and potential toxicity caused by the presence of neutralizer chemicals carried over into the assay system.¹²¹⁴

Table 24. Examples of eluents and diluents for environmental-surface sampling* +

Solutions	Concentration in water
Ringer	¼ strength
Peptone water	0.1%–1.0%
Buffered peptone water	0.067 M phosphate, 0.43% NaCl, 0.1% peptone
Phosphate-buffered saline	0.02 M phosphate, 0.9% NaCl
Sodium chloride (NaCl)	0.25%–0.9%
Calgon Ringer§	¼ strength
Thiosulfate Ringer¶	¼ strength
Water	–
Tryptic soy broth (TSB)	–
Brain-heart infusion broth (BHI) supplemented with 0.5% beef extract	–

* Material in this table is compiled from references 1214 and 1238.

+ A surfactant (e.g., polysorbate [i.e., Tween® 80]) may be added to eluents and diluents. A concentration ranging from 0.01%–0.1% is generally used, depending on the specific application. Foaming may occur during use.

§ This solution is used for dissolution of calcium alginate swabs.

¶ This solution is used for neutralization of residual chlorine.

Several methods can be used for collecting environmental surface samples (Table 25). Specific step-by-step discussions of each of the methods have been published.^{1214, 1239} For best results, all methods should incorporate aseptic techniques, sterile equipment, and sterile recovery media.

Table 25. Methods of environmental-surface sampling

Method	Suitable for appropriate surface(s)	Assay technique	Procedural notes	Points of interpretation	Available standards	References
Sample/rinse Moistened swab/rinse	Non-absorbent surfaces, corners, crevices, devices, and instruments	Dilutions; qualitative or quantitative assays	Assay multiple measures areas or devices with separate swabs	Report results per measured areas or if assaying an object, per the entire sample site	YES – food industry; NO – health care	1214, 1239–1242
Moistened sponge/rinse	Large areas and housekeeping surfaces (e.g., floors or walls)	Dilutions; qualitative or quantitative assays	Vigorously rub a sterile sponge over the surface	Report results per measured area	YES – food industry; NO – health care	1214, 1239–1242
Moistened wipe/rinse	Large areas and housekeeping surfaces (e.g., countertops)	Dilutions; qualitative or quantitative assays	Use a sterile wipe	Report results per measured area	YES – food industry; NO – health care	1214, 1239–1242
Direct immersion	Small items capable of being immersed	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinse volume is large and anticipated microbiological concentration is low	Report results per item	NO	1214
Containment	Interior surfaces of containers, tubes, or bottles	Dilutions; qualitative or quantitative assays	Use membrane filtration if rinse volume is large	Evaluate both the types and numbers of microorganisms	YES – food and industrial applications for containers prior to fill	1214
RODAC*	Previously cleaned and sanitized flat, non-absorbent surfaces; not suitable for irregular surfaces	Direct assay	Overgrowth occurs if used on heavily contaminated surfaces; use neutralizers in the agar if surface disinfectant residuals are present	Provides direct, quantitative results; use a minimum of 15 plates per an average hospital room	NO	1214, 1237, 1239, 1243, 1244

* RODAC stands for “replicate organism direct agar contact.”

Sample/rinse methods are frequently chosen because of their versatility. However, these sampling methods are the most prone to errors caused by manipulation of the swab, gauze pad, or sponge.¹²³⁸ Additionally, no microbiocidal or microbiostatic agents should be present in any of these items when used for sampling.¹²³⁸ Each of the rinse methods requires effective elution of microorganisms from the item used to sample the surface. Thorough mixing of the rinse fluids after elution (e.g., via manual or mechanical mixing using a vortex mixer, shaking with or without glass beads, and ultrasonic bath) will help to remove and suspend material from the sampling device and break up clumps of organisms for a more accurate count.¹²³⁸ In some instances, the item used to sample the surface (e.g., gauze pad and sponge) may be immersed in the rinse fluids in a sterile bag and subjected to stomaching.¹²³⁸ This technique, however, is suitable only for soft or absorbent items that will not puncture the bag during the elution process.

If sampling is conducted as part of an epidemiologic investigation of a disease outbreak, identification of isolates to species level is mandatory, and characterization beyond the species level is preferred.¹²¹⁴ When interpreting the results of the sampling, the expected degree of microbial contamination

associated with the various categories of surfaces in the Spaulding classification must be considered. Environmental surfaces should be visibly clean; recognized pathogens in numbers sufficient to result in secondary transfer to other animate or inanimate surfaces should be absent from the surface being sampled.¹²¹⁴ Although the interpretation of a sample with positive microbial growth is self-evident, an environmental surface sample, especially that obtained from housekeeping surfaces, that shows no growth does not represent a “sterile” surface. Sensitivities of the sampling and assay methods (i.e., level of detection) must be taken into account when no-growth samples are encountered. Properly collected control samples will help rule out extraneous contamination of the surface sample.

G. Laundry and Bedding

1. General Information

Laundry in a health-care facility may include bed sheets and blankets, towels, personal clothing, patient apparel, uniforms, scrub suits, gowns, and drapes for surgical procedures.¹²⁴⁵ Although contaminated textiles and fabrics in health-care facilities can be a source of substantial numbers of pathogenic microorganisms, reports of health-care–associated diseases linked to contaminated fabrics are so few in number that the overall risk of disease transmission during the laundry process likely is negligible. When the incidence of such events are evaluated in the context of the volume of items laundered in health-care settings (estimated to be 5 billion pounds annually in the United States),¹²⁴⁶ existing control measures (e.g., standard precautions) are effective in reducing the risk of disease transmission to patients and staff. Therefore, use of current control measures should be continued to minimize the contribution of contaminated laundry to the incidence of health-care–associated infections. The control measures described in this section of the guideline are based on principles of hygiene, common sense, and consensus guidance; they pertain to laundry services utilized by health-care facilities, either in-house or contract, rather than to laundry done in the home.

2. Epidemiology and General Aspects of Infection Control

Contaminated textiles and fabrics often contain high numbers of microorganisms from body substances, including blood, skin, stool, urine, vomitus, and other body tissues and fluids. When textiles are heavily contaminated with potentially infective body substances, they can contain bacterial loads of 10^6 – 10^8 CFU/100 cm² of fabric.¹²⁴⁷ Disease transmission attributed to health-care laundry has involved contaminated fabrics that were handled inappropriately (i.e., the shaking of soiled linens). Bacteria (*Salmonella* spp., *Bacillus cereus*), viruses (hepatitis B virus [HBV]), fungi (*Microsporium canis*), and ectoparasites (scabies) presumably have been transmitted from contaminated textiles and fabrics to workers via a) direct contact or b) aerosols of contaminated lint generated from sorting and handling contaminated textiles.^{1248–1252} In these events, however, investigations could not rule out the possibility that some of these reported infections were acquired from community sources. Through a combination of soil removal, pathogen removal, and pathogen inactivation, contaminated laundry can be rendered hygienically clean. Hygienically clean laundry carries negligible risk to health-care workers and patients, provided that the clean textiles, fabric, and clothing are not inadvertently contaminated before use.

OSHA defines contaminated laundry as “laundry which has been soiled with blood or other potentially infectious materials or may contain sharps.”⁹⁶⁷ The purpose of the laundry portion of the standard is to protect the worker from exposure to potentially infectious materials during collection, handling, and sorting of contaminated textiles through the use of personal protective equipment, proper work practices, containment, labeling, hazard communication, and ergonomics.

Experts are divided regarding the practice of transporting clothes worn at the workplace to the health-care worker's home for laundering. Although OSHA regulations prohibit home laundering of items that are considered personal protective apparel or equipment (e.g., laboratory coats),⁹⁶⁷ experts disagree about whether this regulation extends to uniforms and scrub suits that are not contaminated with blood or other potentially infectious material. Health-care facility policies on this matter vary and may be inconsistent with recommendations of professional organizations.^{1253, 1254} Uniforms without blood or body substance contamination presumably do not differ appreciably from street clothes in the degree and microbial nature of soilage. Home laundering would be expected to remove this level of soil adequately. However, if health-care facilities require the use of uniforms, they should either make provisions to launder them or provide information to the employee regarding infection control and cleaning guidelines for the item based on the tasks being performed at the facility. Health-care facilities should address the need to provide this service and should determine the frequency for laundering these items. In a recent study examining the microbial contamination of medical students' white coats, the students perceived the coats as "clean" as long as the garments were not visibly contaminated with body substances, even after wearing the coats for several weeks.¹²⁵⁵ The heaviest bacterial load was found on the sleeves and the pockets of these garments; the organisms most frequently isolated were *Staphylococcus aureus*, diphtheroids, and *Acinetobacter* spp.¹²⁵⁵ Presumably, the sleeves of the coat may make contact with a patient and potentially serve to transfer environmentally stable microorganisms among patients. In this study, however, surveillance was not conducted among patients to detect new infections or colonizations. The students did, however, report that they would likely replace their coats more frequently and regularly if clean coats were provided.¹²⁵⁵ Apart from this study, which documents the presence of pathogenic bacteria on health-care facility clothing, reports of infections attributed to either the contact with such apparel or with home laundering have been rare.^{1256, 1257}

Laundry services for health-care facilities are provided either in-house (i.e., on-premise laundry [OPL]), co-operatives (i.e., those entities owned and operated by a group of facilities), or by off-site commercial laundries. In the latter, the textiles may be owned by the health-care facility, in which case the processor is paid for laundering only. Alternatively, the textiles may be owned by the processor who is paid for every piece laundered on a "rental" fee. The laundry facility in a health-care setting should be designed for efficiency in providing hygienically clean textiles, fabrics, and apparel for patients and staff. Guidelines for laundry construction and operation for health-care facilities, including nursing facilities, have been published.^{120, 1258} The design and engineering standards for existing facilities are those cited in the AIA edition in effect during the time of the facility's construction.¹²⁰ A laundry facility is usually partitioned into two separate areas - a "dirty" area for receiving and handling the soiled laundry and a "clean" area for processing the washed items.¹²⁵⁹ To minimize the potential for recontaminating cleaned laundry with aerosolized contaminated lint, areas receiving contaminated textiles should be at negative air pressure relative to the clean areas.¹²⁶⁰⁻¹²⁶² Laundry areas should have handwashing facilities readily available to workers. Laundry workers should wear appropriate personal protective equipment (e.g., gloves and protective garments) while sorting soiled fabrics and textiles.⁹⁶⁷ Laundry equipment should be used and maintained according to the manufacturer's instructions to prevent microbial contamination of the system.^{1250, 1263} Damp textiles should not be left in machines overnight.¹²⁵⁰

3. Collecting, Transporting, and Sorting Contaminated Textiles and Fabrics

The laundry process starts with the removal of used or contaminated textiles, fabrics, and/or clothing from the areas where such contamination occurred, including but not limited to patients' rooms, surgical/operating areas, and laboratories. Handling contaminated laundry with a minimum of agitation

can help prevent the generation of potentially contaminated lint aerosols in patient-care areas.^{967, 1259} Sorting or rinsing contaminated laundry at the location where contamination occurred is prohibited by OSHA.⁹⁶⁷ Contaminated textiles and fabrics are placed into bags or other appropriate containment in this location; these bags are then securely tied or otherwise closed to prevent leakage.⁹⁶⁷ Single bags of sufficient tensile strength are adequate for containing laundry, but leak-resistant containment is needed if the laundry is wet and capable of soaking through a cloth bag.¹²⁶⁴ Bags containing contaminated laundry must be clearly identified with labels, color-coding, or other methods so that health-care workers handle these items safely, regardless of whether the laundry is transported within the facility or destined for transport to an off-site laundry service.⁹⁶⁷

Typically, contaminated laundry originating in isolation areas of the hospital is segregated and handled with special practices; however, few, if any, cases of health-care–associated infection have been linked to this source.¹²⁶⁵ Single-blinded studies have demonstrated that laundry from isolation areas is no more heavily contaminated with microorganisms than laundry from elsewhere in the hospital.¹²⁶⁶ Therefore, adherence to standard precautions when handling contaminated laundry in isolation areas and minimizing agitation of the contaminated items are considered sufficient to prevent the dispersal of potentially infectious aerosols.⁶

Contaminated textiles and fabrics in bags can be transported by cart or chute.^{1258, 1262} Laundry chutes require proper design, maintenance, and use, because the piston-like action of a laundry bag traveling in the chute can propel airborne microbial contaminants throughout the facility.^{1267–1269} Laundry chutes should be maintained under negative air pressure to prevent the spread of microorganisms from floor to floor. Loose, contaminated pieces of laundry should not be tossed into chutes, and laundry bags should be closed or otherwise secured to prevent the contents from falling out into the chute.¹²⁷⁰ Health-care facilities should determine the point in the laundry process at which textiles and fabrics should be sorted. Sorting after washing minimizes the exposure of laundry workers to infective material in soiled fabrics, reduces airborne microbial contamination in the laundry area, and helps to prevent potential percutaneous injuries to personnel.¹²⁷¹ Sorting laundry before washing protects both the machinery and fabrics from hard objects (e.g., needles, syringes, and patients' property) and reduces the potential for recontamination of clean textiles.¹²⁷² Sorting laundry before washing also allows for customization of laundry formulas based on the mix of products in the system and types of soils encountered. Additionally, if work flow allows, increasing the amount of segregation by specific product types will usually yield the greatest amount of work efficiency during inspection, folding, and pack-making operations.¹²⁵³ Protective apparel for the workers and appropriate ventilation can minimize these exposures.^{967, 1258–1260} Gloves used for the task of sorting laundry should be of sufficient thickness to minimize sharps injuries.⁹⁶⁷ Employee safety personnel and industrial hygienists can help to determine the appropriate glove choice.

4. Parameters of the Laundry Process

Fabrics, textiles, and clothing used in health-care settings are disinfected during laundering and generally rendered free of vegetative pathogens (i.e., hygienically clean), but they are not sterile.¹²⁷³ Laundering cycles consist of flush, main wash, bleaching, rinsing, and souring.¹²⁷⁴ Cleaned wet textiles, fabrics, and clothing are then dried, pressed as needed, and prepared (e.g., folded and packaged) for distribution back to the facility. Clean linens provided by an off-site laundry must be packaged prior to transport to prevent inadvertent contamination from dust and dirt during loading, delivery, and unloading. Functional packaging of laundry can be achieved in several ways, including a) placing clean linen in a hamper lined with a previously unused liner, which is then closed or covered; b) placing clean linen in a properly cleaned cart and covering the cart with disposable material or a properly cleaned reusable textile material that can be secured to the cart; and c) wrapping individual bundles of clean

textiles in plastic or other suitable material and sealing or taping the bundles.

The antimicrobial action of the laundering process results from a combination of mechanical, thermal, and chemical factors.^{1271, 1275, 1276} Dilution and agitation in water remove substantial quantities of microorganisms. Soaps and detergents function to suspend soils and also exhibit some microbiocidal properties. Hot water provides an effective means of destroying microorganisms.¹²⁷⁷ A temperature of at least 160°F (71°C) for a minimum of 25 minutes is commonly recommended for hot-water washing.² Water of this temperature can be provided by steam jet or separate booster heater.¹²⁰ The use of chlorine bleach assures an extra margin of safety.^{1278, 1279} A total available chlorine residual of 50–150 ppm is usually achieved during the bleach cycle.¹²⁷⁷ Chlorine bleach becomes activated at water temperatures of 135°F–145°F (57.2°C–62.7°C). The last of the series of rinse cycles is the addition of a mild acid (i.e., sour) to neutralize any alkalinity in the water supply, soap, or detergent. The rapid shift in pH from approximately 12 to 5 is an effective means to inactivate some microorganisms.¹²⁴⁷ Effective removal of residual alkali from fabrics is an important measure in reducing the risk for skin reactions among patients.

Chlorine bleach is an economical, broad-spectrum chemical germicide that enhances the effectiveness of the laundering process. Chlorine bleach is not, however, an appropriate laundry additive for all fabrics. Traditionally, bleach was not recommended for laundering flame-retardant fabrics, linens, and clothing because its use diminished the flame-retardant properties of the treated fabric.¹²⁷³ However, some modern-day flame retardant fabrics can now tolerate chlorine bleach. Flame-retardant fabrics, whether topically treated or inherently flame retardant, should be thoroughly rinsed during the rinse cycles, because detergent residues are capable of supporting combustion. Chlorine alternatives (e.g., activated oxygen-based laundry detergents) provide added benefits for fabric and color safety in addition to antimicrobial activity. Studies comparing the antimicrobial potencies of chlorine bleach and oxygen-based bleach are needed. Oxygen-based bleach and detergents used in health-care settings should be registered by EPA to ensure adequate disinfection of laundry. Health-care workers should note the cleaning instructions of textiles, fabrics, drapes, and clothing to identify special laundering requirements and appropriate hygienic cleaning options.¹²⁷⁸

Although hot-water washing is an effective laundry disinfection method, the cost can be substantial. Laundries are typically the largest users of hot water in hospitals. They consume 50%–75% of the total hot water,¹²⁸⁰ representing an average of 10%–15% of the energy used by a hospital. Several studies have demonstrated that lower water temperatures of 71°F–77°F (22°C–25°C) can reduce microbial contamination when the cycling of the washer, the wash detergent, and the amount of laundry additive are carefully monitored and controlled.^{1247, 1281–1285} Low-temperature laundry cycles rely heavily on the presence of chlorine- or oxygen-activated bleach to reduce the levels of microbial contamination. The selection of hot- or cold-water laundry cycles may be dictated by state health-care facility licensing standards or by other regulation. Regardless of whether hot or cold water is used for washing, the temperatures reached in drying and especially during ironing provide additional significant microbiocidal action.¹²⁴⁷ Dryer temperatures and cycle times are dictated by the materials in the fabrics. Man-made fibers (i.e., polyester and polyester blends) require shorter times and lower temperatures.

After washing, cleaned and dried textiles, fabrics, and clothing are pressed, folded, and packaged for transport, distribution, and storage by methods that ensure their cleanliness until use.² State regulations and/or accrediting standards may dictate the procedures for this activity. Clean/sterile and contaminated textiles should be transported from the laundry to the health-care facility in vehicles (e.g., trucks, vans, and carts) that allow for separation of clean/sterile and contaminated items. Clean/sterile textiles and contaminated textiles may be transported in the same vehicle, provided that the use of physical barriers and/or space separation can be verified to be effective in protecting the clean/sterile items from

contamination. Clean, uncovered/unwrapped textiles stored in a clean location for short periods of time (e.g., uncovered and used within a few hours) have not been demonstrated to contribute to increased levels of health-care–acquired infection. Such textiles can be stored in convenient places for use during the provision of care, provided that the textiles can be maintained dry and free from soil and body-substance contamination.

In the absence of microbiologic standards for laundered textiles, no rationale exists for routine microbiologic sampling of cleaned health-care textiles and fabrics.¹²⁸⁶ Sampling may be used as part of an outbreak investigation if epidemiologic evidence suggests that textiles, fabrics, or clothing are a suspected vehicle for disease transmission. Sampling techniques include aseptically macerating the fabric into pieces and adding these to broth media or using contact plates (RODAC plates) for direct surface sampling.^{1271, 1286} When evaluating the disinfecting properties of the laundering process specifically, placing pieces of fabric between two membrane filters may help to minimize the contribution of the physical removal of microorganisms.¹²⁸⁷

Washing machines and dryers in residential-care settings are more likely to be consumer items rather than the commercial, heavy-duty, large volume units typically found in hospitals and other institutional health-care settings. Although all washing machines and dryers in health-care settings must be properly maintained for performance according to the manufacturer's instructions, questions have been raised about the need to disinfect washers and dryers in residential-care settings. Disinfection of the tubs and tumblers of these machines is unnecessary when proper laundry procedures are followed; these procedures involve a) the physical removal of bulk solids (e.g., feces) before the wash/dry cycle and b) proper use of temperature, detergent, and laundry additives. Infection has not been linked to laundry procedures in residential-care facilities, even when consumer versions of detergents and laundry additives are used.

5. Special Laundry Situations

Some textile items (e.g., surgical drapes and reusable gowns) must be sterilized before use and therefore require steam autoclaving after laundering.⁷ Although the American Academy of Pediatrics in previous guidelines recommended autoclaving for linens in neonatal intensive care units (NICUs), studies on the microbial quality of routinely cleaned NICU linen have not identified any increased risk for infection among the neonates receiving care.¹²⁸⁸ Consequently, hygienically clean linens are suitable for use in this setting.⁹⁹⁷ The use of sterile linens in burn therapy units remains unresolved.

Coated or laminated fabrics are often used in the manufacture of PPE. When these items become contaminated with blood or other body substances, the manufacturer's instructions for decontamination and cleaning take into account the compatibility of the rubber backing with the chemical germicides or detergents used in the process. The directions for decontaminating these items should be followed as indicated; the item should be discarded when the backing develops surface cracks.

Dry cleaning, a cleaning process that utilizes organic solvents (e.g., perchloroethylene) for soil removal, is an alternative means of cleaning fabrics that might be damaged in conventional laundering and detergent washing. Several studies, however, have shown that dry cleaning alone is relatively ineffective in reducing the numbers of bacteria and viruses on contaminated linens;^{1289, 1290} microbial populations are significantly reduced only when dry-cleaned articles are heat pressed. Dry cleaning should therefore not be considered a routine option for health-care facility laundry and should be reserved for those circumstances in which fabrics can not be safely cleaned with water and detergent.¹²⁹¹

6. Surgical Gowns, Drapes, and Disposable Fabrics

An issue of recent concern involves the use of disposable (i.e., single use) versus reusable (i.e., multiple use) surgical attire and fabrics in health-care settings.¹²⁹² Regardless of the material used to manufacture gowns and drapes, these items must be resistant to liquid and microbial penetration.^{7, 1293–1297} Surgical gowns and drapes must be registered with FDA to demonstrate their safety and effectiveness. Repellency and pore size of the fabric contribute to gown performance, but performance capability can be influenced by the item's design and construction.^{1298, 1299} Reinforced gowns (i.e., gowns with double-layered fabric) generally are more resistant to liquid strike-through.^{1300, 1301} Reinforced gowns may, however, be less comfortable. Guidelines for selection and use of barrier materials for surgical gowns and drapes have been published.¹³⁰² When selecting a barrier product, repellency level and type of barrier should be compatible for the exposure expected.⁹⁶⁷ However, data are limited regarding the association between gown or drape characteristics and risk for surgical site infections.^{7, 1303} Health-care facilities must ensure optimal protection of patients and health-care workers. Not all fabric items in health care lend themselves to single-use. Facilities exploring options for gowns and drapes should consider the expense of disposable items and the impact on the facility's waste-management costs once these items are discarded. Costs associated with the use of durable goods involve the fabric or textile items; staff expenses to collect, sort, clean, and package the laundry; and energy costs to operate the laundry if on-site or the costs to contract with an outside service.^{1304, 1305}

7. Antimicrobial-Impregnated Articles and Consumer Items Bearing Antimicrobial Labeling

Manufacturers are increasingly incorporating antibacterial or antimicrobial chemicals into consumer and health-care items. Some consumer products bearing labels that indicate treatment with antimicrobial chemicals have included pens, cutting boards, toys, household cleaners, hand lotions, cat litter, soaps, cotton swabs, toothbrushes, and cosmetics. The “antibacterial” label on household cleaning products, in particular, gives consumers the impression that the products perform “better” than comparable products without this labeling, when in fact all household cleaners have antibacterial properties.

In the health-care setting, treated items may include children's pajamas, mattresses, and bed linens with label claims of antimicrobial properties. These claims require careful evaluation to determine whether they pertain to the use of antimicrobial chemicals as preservatives for the fabric or other components or whether they imply a health claim.^{1306, 1307} No evidence is available to suggest that use of these products will make consumers and patients healthier or prevent disease. No data support the use of these items as part of a sound infection-control strategy, and therefore, the additional expense of replacing a facility's bedding and sheets with these treated products is unwarranted.

EPA has reaffirmed its position that manufacturers who make public health claims for articles containing antimicrobial chemicals must provide evidence to support those claims as part of the registration process.¹³⁰⁸ Current EPA regulations outlined in the Treated Articles Exemption of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) require manufacturers to register both the antimicrobial chemical used in or on the product and the finished product itself if a public health claim is maintained for the item. The exemption applies to the use of antimicrobial chemicals for the purpose of preserving the integrity of the product's raw material(s). The U.S. Federal Trade Commission (FTC) is evaluating manufacturer advertising of products with antimicrobial claims.¹³⁰⁹

8. Standard Mattresses, Pillows, and Air-Fluidized Beds

Standard mattresses and pillows can become contaminated with body substances during patient care if the integrity of the covers of these items is compromised. The practice of sticking needles into the mattress should be avoided. A mattress cover is generally a fitted, protective material, the purpose of which is to prevent the mattress from becoming contaminated with body fluids and substances. A linen sheet placed on the mattress is not considered a mattress cover. Patches for tears and holes in mattress covers do not provide an impermeable surface over the mattress. Mattress covers should be replaced when torn; the mattress should be replaced if it is visibly stained. Wet mattresses, in particular, can be a substantial environmental source of microorganisms. Infections and colonizations caused by *Acinetobacter* spp., MRSA, and *Pseudomonas aeruginosa* have been described, especially among burn patients.^{1310–1315} In these reports, the removal of wet mattresses was an effective infection-control measure. Efforts were made to ensure that pads and covers were cleaned and disinfected between patients using disinfectant products compatible with mattress-cover materials to ensure that these covers remained impermeable to fluids.^{1310–1314} Pillows and their covers should be easily cleanable, preferably in a hot water laundry cycle.¹³¹⁵ These should be laundered between patients or if contaminated with body substances.

Air-fluidized beds are used for the care of patients immobilized for extended periods of time because of therapy or injury (e.g., pain, decubitus ulcers, and burns).¹³¹⁶ These specialized beds consist of a base unit filled with microsphere beads fluidized by warm, dry air flowing upward from a diffuser located at the bottom of the unit. A porous, polyester filter sheet separates the patient from direct contact with the beads but allows body fluids to pass through to the beads. Moist beads aggregate into clumps which settle to the bottom where they are removed as part of routine bed maintenance.

Because the beads become contaminated with the patient's body substances, concerns have been raised about the potential for these beds to serve as an environmental source of pathogens. Certain pathogens (e.g., *Enterococcus* spp., *Serratia marcescens*, *Staphylococcus aureus*, and *Streptococcus fecalis*) have been recovered either from the microsphere beads or the polyester sheet after cleaning.^{1317, 1318} Reports of cross-contamination of patients, however, are few.¹³¹⁸ Nevertheless, routine maintenance and between-patient decontamination procedures can minimize potential risks to patients. Regular removal of bead clumps, coupled with the warm, dry air of the bed, can help to minimize bacterial growth in the unit.^{1319–1321} Beads are decontaminated between patients by high heat (113°F–194°F [45°C–90°C], depending on the manufacturer's specifications) for at least 1 hour; this procedure is particularly important for the inactivation of *Enterococcus* spp. which are relatively resistant to heat.^{1322, 1323} The polyester filter sheet requires regular changing and thorough cleaning and disinfection, especially between patients.^{1317, 1318, 1322, 1323}

Microbial contamination of the air space in the immediate vicinity of a properly maintained air-fluidized bed is similar to that found in air around conventional bedding, despite the air flow out of the base unit and around the patient.^{1320, 1324, 1325} An operational air-fluidized bed can, however, interfere with proper pressure differentials, especially in negative-pressure rooms;¹³²⁶ the effect varies with the location of the bed relative to the room's configuration and supply and exhaust vent locations. Use of an air-fluidized bed in a negative-pressure room requires consultation with a facility engineer to determine appropriate placement of the bed.

H. Animals in Health-Care Facilities

1. General Information

Animals in health-care facilities traditionally have been limited to laboratories and research areas. However, their presence in patient-care areas is now more frequent, both in acute-care and long-term care settings, prompting consideration for the potential transmission of zoonotic pathogens from animals to humans in these settings. Although dogs and cats may be commonly encountered in health-care settings, other animals (e.g., fish, birds, non-human primates, rabbits, rodents, and reptiles) also can be present as research, resident, or service animals. These animals can serve as sources of zoonotic pathogens that could potentially infect patients and health-care workers (Table 26).¹³²⁷⁻¹³⁴⁰ Animals potentially can serve as reservoirs for antibiotic-resistant microorganisms, which can be introduced to the health-care setting while the animal is present. VRE have been isolated from both farm animals and pets,¹³⁴¹ and a cat in a geriatric care center was found to be colonized with MRSA.¹³⁴²

Table 26. Examples of diseases associated with zoonotic transmission*+

Infectious disease	Cats	Dogs	Fish	Birds	Rabbits	Reptiles§	Primates	Rodents§
Virus								
Lymphocytic choriomeningitis								+¶
Rabies	+	+						
Bacteria								
Campylobacteriosis	+	+				+	+	+
<i>Capnocytophaga canimorsus</i> infection	+	+						
Cat scratch disease (<i>Bartonella henselae</i>)	+							
Leptospirosis	+						+	+
Mycobacteriosis			+	+				
Pasteurellosis	+	+			+			
Plague	+			+			+	+
Psittacosis				+				
Q fever (<i>Coxiella burnetti</i>)	+							
Rat bite fever (<i>Spirillum minus</i> , <i>Streptobacillus moniliformis</i>)								+
Salmonellosis	+	+		+	+	+	+	+
Tularemia	+				+			+
Yersiniosis					+	+	+	+
Parasites								
Ancylostomiasis	+	+					+	
Cryptosporidiosis	+							
Giardiasis	+	+					+	
Toxocariasis	+	+					+	
Toxoplasmosis	+	+					+	
Fungi								
Blastomycosis		+						
Dermatophytosis		+			+		+	+

* Material in this table is adapted from reference 1331 and used with permission of the publisher (Lippincott Williams and Wilkins).

+ This table does not include vectorborne diseases.

§ Reptiles include lizards, snakes, and turtles. Rodents include hamsters, mice, and rats.

¶ The + symbol indicates that the pathogen associated with the infection has been isolated from animals and is considered to pose potential risk to humans.

Zoonoses can be transmitted from animals to humans either directly or indirectly via bites, scratches, aerosols, ectoparasites, accidental ingestion, or contact with contaminated soil, food, water, or unpasteurized milk.^{1331, 1332, 1343–1345} Colonization and hand transferral of pathogens acquired from pets in health-care workers' homes represent potential sources and modes of transmission of zoonotic pathogens in health-care settings. An outbreak of infections caused by a yeast (*Malassezia pachydermatis*) among newborns was traced to transfer of the yeast from the hands of health-care workers with pet dogs at home.¹³⁴⁶ In addition, an outbreak of ringworm in a NICU caused by *Microsporum canis* was associated with a nurse and her cat,¹³⁴⁷ and an outbreak of *Rhodococcus (Gordona) bronchialis* sternal SSIs after coronary-artery bypass surgery was traced to a colonized nurse whose dogs were culture-positive for the organism.¹³⁴⁸ In the latter outbreak, whether the dogs were the sole source of the organism and whether other environmental reservoirs contributed to the outbreak are unknown. Nonetheless, limited data indicate that outbreaks of infectious disease have occurred as a result of contact with animals in areas housing immunocompetent patients. However, the low frequency of outbreaks may result from a) the relatively limited presence of the animals in health-care facilities and b) the immunocompetency of the patients involved in the encounters. Formal scientific studies to evaluate potential risks of transmission of zoonoses in health-care settings outside of the laboratory are lacking.

2. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animals

Animal-Assisted Activities (AAA) are those programs that enhance the patients' quality of life. These programs allow patients to visit animals in either a common, central location in the facility or in individual patient rooms. A group session with the animals enhances opportunities for ambulatory patients and facility residents to interact with caregivers, family members, and volunteers.^{1349–1351} Alternatively, allowing the animals access to individual rooms provides the same opportunity to non-ambulatory patients and patients for whom privacy or dignity issues are a consideration. The decision to allow this access to patients' rooms should be made on a case-by-case basis, with the consultation and consent of the attending physician and nursing staff.

Animal-Assisted Therapy (AAT) is a goal-directed intervention that incorporates an animal into the treatment process provided by a credentialed therapist.^{1330, 1331} The concept for AAT arose from the observation that some patients with pets at home recover from surgical and medical procedures more rapidly than patients without pets.^{1352, 1353} Contact with animals is considered beneficial for enhancing wellness in certain patient populations (e.g., children, the elderly, and extended-care hospitalized patients).^{1349, 1354–1357} However, evidence supporting this benefit is largely derived from anecdotal reports and observations of patient/animal interactions.^{1357–1359} Guidelines for establishing AAT programs are available for facilities considering this option.¹³⁶⁰

The incorporation of non-human primates into an AAA or AAT program is not encouraged because of concerns regarding potential disease transmission from and unpredictable behavior of these animals.^{1361, 1362} Animals participating in either AAA or AAT sessions should be in good health and up-to-date with recommended immunizations and prophylactic medications (e.g., heartworm prevention) as determined by a licensed veterinarian based on local needs and recommendations. Regular re-evaluation of the animal's health and behavior status is essential.¹³⁶⁰ Animals should be routinely screened for enteric parasites and/or have evidence of a recently completed antihelminthic regimen.¹³⁶³ They should also be free of ectoparasites (e.g., fleas and ticks) and should have no sutures, open wounds, or obvious dermatologic lesions that could be associated with bacterial, fungal, or viral infections or parasitic infestations. Incorporating young animals (i.e., those aged <1 year) into these programs is not encouraged because of issues regarding unpredictable behavior and elimination control. Additionally,

the immune systems of very young puppies and kittens is not completely developed, thereby placing the health of these animals at risk. Animals should be clean and well-groomed. The visits must be supervised by persons who know the animals and their behavior. Animal handlers should be trained in these activities and receive site-specific orientation to ensure that they work efficiently with the staff in the specific health-care environment.¹³⁶⁰ Additionally, animal handlers should be in good health.¹³⁶⁰

The most important infection-control measure to prevent potential disease transmission is strict enforcement of hand-hygiene measures (e.g., using either soap and water or an alcohol-based hand rub) for all patients, staff, and residents after handling the animals.^{1355, 1364} Care should also be taken to avoid direct contact with animal urine or feces. Clean-up of these substances from environmental surfaces requires gloves and the use of leak-resistant plastic bags to discard absorbent material used in the process.² The area must be cleaned after visits according to standard cleaning procedures.

The American Academy of Allergy, Asthma, and Immunology estimates that dog or cat allergies occur in approximately 15% of the population.¹³⁶⁵ Minimizing contact with animal saliva, dander, and/or urine helps to mitigate allergic responses.^{1365–1367} Some facilities may not allow animal visitation for patients with a) underlying asthma, b) known allergies to cat or dog hair, c) respiratory allergies of unknown etiology, and d) immunosuppressive disorders. Hair shedding can be minimized by processes that remove dead hair (e.g., grooming) and that prevent the shedding of dead hair (e.g., therapy capes for dogs). Allergens can be minimized by bathing therapy animals within 24 hours of a visit.^{1333, 1368}

Animal therapists and handlers must take precautions to prevent animal bites. Common pathogens associated with animal bites include *Capnocytophaga canimorsus*, *Pasteurella* spp., *Staphylococcus* spp., and *Streptococcus* spp. Selecting well-behaved and well-trained animals for these programs greatly decreases the incidence of bites. Rodents, exotic species, wild/domestic animals (i.e., wolf-dog hybrids), and wild animals whose behavior is unpredictable should be excluded from AAA or AAT programs. A well-trained animal handler should be able to recognize stress in the animal and to determine when to terminate a session to minimize risk. When an animal bites a person during AAA or AAT, the animal is to be permanently removed from the program. If a bite does occur, the wound must be cleansed immediately and monitored for subsequent infection. Most infections can be treated with antibiotics, and antibiotics often are prescribed prophylactically in these situations.

The health-care facility's infection-control staff should participate actively in planning for and coordinating AAA and AAT sessions. Many facilities do not offer AAA or AAT programs for severely immunocompromised patients (e.g., HSCT patients and patients on corticosteroid therapy).¹³³⁹ The question of whether family pets or companion animals can visit terminally-ill HSCT patients or other severely immunosuppressed patients is best handled on a case-by-case basis, although animals should not be brought into the HSCT unit or any other unit housing severely immunosuppressed patients. An in-depth discussion of this issue is presented elsewhere.¹³⁶⁶

Immunocompromised patients who have been discharged from a health-care facility may be at higher risk for acquiring some pet-related zoonoses. Although guidelines have been developed to minimize the risk of disease transmission to HIV-infected patients,⁸ these recommendations may be applicable for patients with other immunosuppressive disorders. In addition to handwashing or hand hygiene, these recommendations include avoiding contact with a) animal feces and soiled litter box materials, b) animals with diarrhea, c) very young animals (i.e., dogs <6 months of age and cats <1 year of age), and d) exotic animals and reptiles.⁸ Pets or companion animals with diarrhea should receive veterinary care to resolve their condition.

Many health-care facilities are adopting more home-like environments for residential-care or extended-stay patients in acute-care settings, and resident animals are one element of this approach.¹³⁶⁹ One

concept, the “Eden Alternative,” incorporates children, plants, and animals (e.g., dogs, cats, fish, birds, rabbits, and rodents) into the daily care setting.^{1370, 1371} The concept of working with resident animals has not been scientifically evaluated. Several issues beyond the benefits of therapy must be considered before embarking on such a program, including a) whether the animals will come into direct contact with patients and/or be allowed to roam freely in the facility; b) how the staff will provide care for the animals; c) the management of patients’ or residents’ allergies, asthma, and phobias; d) precautionary measures to prevent bites and scratches; and e) measures to properly manage the disposal of animal feces and urine, thereby preventing environmental contamination by zoonotic microorganisms (e.g., *Toxoplasma* spp., *Toxocara* spp., and *Ancylostoma* spp.).^{1372, 1373} Few data document a link between health-care–acquired infection rates and frequency of cleaning fish tanks or rodent cages. Skin infections caused by *Mycobacterium marinum* have been described among persons who have fish aquariums at home.^{1374, 1375} Nevertheless, immunocompromised patients should avoid direct contact with fish tanks and cages and the aerosols that these items produce. Further, fish tanks should be kept clean on a regular basis as determined by facility policy, and this task should be performed by gloved staff members who are not responsible for patient care. The use of the infection-control risk assessment can help determine whether a fish tank poses a risk for patient or resident safety and health in these situations. No evidence, however, links the incidence of health-care–acquired infections among immunocompetent patients or residents with the presence of a properly cleaned and maintained fish tank, even in dining areas. As a general preventive measure, resident animal programs are advised to restrict animals from a) food preparation kitchens, b) laundries, c) central sterile supply and any storage areas for clean supplies, and d) medication preparation areas. Resident-animal programs in acute-care facilities should not allow the animals into the isolation areas, protective environments, ORs, or any area where immunocompromised patients are housed. Patients and staff routinely should wash their hands or use waterless, alcohol-based hand-hygiene products after contact with animals.

3. Service Animals

Although this section provides an overview about service animals in health-care settings, it cannot address every situation or question that may arise (see Appendix E - Information Resources). A service animal is any animal individually trained to do work or perform tasks for the benefit of a person with a disability.^{1366, 1376} A service animal is not considered a pet but rather an animal trained to provide assistance to a person because of a disability. Title III of the “Americans with Disabilities Act” (ADA) of 1990 mandates that persons with disabilities accompanied by service animals be allowed access with their service animals into places of public accommodation, including restaurants, public transportation, schools, and health-care facilities.^{1366, 1376} In health-care facilities, a person with a disability requiring a service animal may be an employee, a visitor, or a patient.

An overview of the subject of service animals and their presence in health-care facilities has been published.¹³⁶⁶ No evidence suggests that animals pose a more significant risk of transmitting infection than people; therefore, service animals should not be excluded from such areas, unless an individual patient’s situation or a particular animal poses greater risk that cannot be mitigated through reasonable measures. If health-care personnel, visitors, and patients are permitted to enter care areas (e.g., in-patient rooms, some ICUs, and public areas) without taking additional precautions to prevent transmission of infectious agents (e.g., donning gloves, gowns, or masks), a clean, healthy, well-behaved service animal should be allowed access with its handler.¹³⁶⁶ Similarly, if immunocompromised patients are able to receive visitors without using protective garments or equipment, an exclusion of service animals from this area would not be justified.¹³⁶⁶

Because health-care facilities are covered by the ADA or the Rehabilitation Act, a person with a disability may be accompanied by a service animal within the facility unless the animal’s presence or

behavior creates a fundamental alteration in the nature of a facility's services in a particular area or a direct threat to other persons in a particular area.¹³⁶⁶ A "direct threat" is defined as a significant risk to the health or safety of others that cannot be mitigated or eliminated by modifying policies, practices, or procedures.¹³⁷⁶ The determination that a service animal poses a direct threat in any particular health-care setting must be based on an individualized assessment of the service animal, the patient, and the health-care situation. When evaluating risk in such situations, health-care personnel should consider the nature of the risk (including duration and severity); the probability that injury will occur; and whether reasonable modifications of policies, practices, or procedures will mitigate the risk (J. Wodatch, U.S. Department of Justice, 2000). The person with a disability should contribute to the risk-assessment process as part of a pre-procedure health-care provider/patient conference.

Excluding a service animal from an OR or similar special care areas (e.g., burn units, some ICUs, PE units, and any other area containing equipment critical for life support) is appropriate if these areas are considered to have "restricted access" with regards to the general public. General infection-control measures that dictate such limited access include a) the area is required to meet environmental criteria to minimize the risk of disease transmission, b) strict attention to hand hygiene and absence of dermatologic conditions, and c) barrier protective measures [e.g., using gloves, wearing gowns and masks] are indicated for persons in the affected space. No infection-control measures regarding the use of barrier precautions could be reasonably imposed on the service animal. Excluding a service animal that becomes threatening because of a perceived danger to its handler during treatment also is appropriate; however, exclusion of such an animal must be based on the actual behavior of the particular animal, not on speculation about how the animal might behave.

Another issue regarding service animals is whether to permit persons with disabilities to be accompanied by their service animals during all phases of their stay in the health-care facility. Health-care personnel should discuss all aspects of anticipatory care with the patient who uses a service animal. Health-care personnel may not exclude a service animal because health-care staff may be able to perform the same services that the service animal does (e.g., retrieving dropped items and guiding an otherwise ambulatory person to the restroom). Similarly, health-care personnel can not exclude service animals because the health-care staff perceive a lack of need for the service animal during the person's stay in the health-care facility. A person with a disability is entitled to independent access (i.e., to be accompanied by a service animal unless the animal poses a direct threat or a fundamental alteration in the nature of services); "need" for the animal is not a valid factor in either analysis. For some forms of care (e.g., ambulation as physical therapy following total hip replacement or knee replacement), the service animal should not be used in place of a credentialed health-care worker who directly provides therapy. However, service animals need not be restricted from being in the presence of its handler during this time; in addition, rehabilitation and discharge planning should incorporate the patient's future use of the animal. The health-care personnel and the patient with a disability should discuss both the possible need for the service animal to be separated from its handler for a period of time during non-emergency care and an alternate plan of care for the service animal in the event the patient is unable or unwilling to provide that care. This plan might include family members taking the animal out of the facility several times a day for exercise and elimination, the animal staying with relatives, or boarding off-site. Care of the service animal, however, remains the obligation of the person with the disability, not the health-care staff.

Although animals potentially carry zoonotic pathogens transmissible to man, the risk is minimal with a healthy, clean, vaccinated, well-behaved, and well-trained service animal, the most common of which are dogs and cats. No reports have been published regarding infectious disease that affects humans originating in service dogs. Standard cleaning procedures are sufficient following occupation of an area by a service animal.¹³⁶⁶ Clean-up of spills of animal urine, feces, or other body substances can be accomplished with blood/body substance procedures outlined in the Environmental Services section of

this guideline. No special bathing procedures are required prior to a service animal accompanying its handler into a health-care facility.

Providing access to exotic animals (e.g., reptiles and non-human primates) that are used as service animals is problematic. Concerns about these animals are discussed in two published reviews.^{1331, 1366} Because some of these animals exhibit high-risk behaviors that may increase the potential for zoonotic disease transmission (e.g., herpes B infection), providing health-care facility access to nonhuman primates used as service animals is discouraged, especially if these animals might come into contact with the general public.^{1361, 1362} Health-care administrators should consult the Americans with Disabilities Act for guidance when developing policies about service animals in their facilities.^{1366, 1376}

Requiring documentation for access of a service animal to an area generally accessible to the public would impose a burden on a person with a disability. When health-care workers are not certain that an animal is a service animal, they may ask the person who has the animal if it is a service animal required because of a disability; however, no certification or other documentation of service animal status can be required.¹³⁷⁷

4. Animals as Patients in Human Health-Care Facilities

The potential for direct and indirect transmission of zoonoses must be considered when rooms and equipment in human health-care facilities are used for the medical or surgical treatment or diagnosis of animals.¹³⁷⁸ Inquiries should be made to veterinary medical professionals to determine an appropriate facility and equipment to care for an animal.

The central issue associated with providing medical or surgical care to animals in human health-care facilities is whether cross-contamination occurs between the animal patient and the human health-care workers and/or human patients. The fundamental principles of infection control and aseptic practice should differ only minimally, if at all, between veterinary medicine and human medicine. Health-care-associated infections can and have occurred in both patients and workers in veterinary medical facilities when lapses in infection-control procedures are evident.^{1379–1384} Further, veterinary patients can be at risk for acquiring infection from veterinary health-care workers if proper precautions are not taken.¹³⁸⁵

The issue of providing care to veterinary patients in human health-care facilities can be divided into the following three areas of infection-control concerns: a) whether the room/area used for animal care can be made safe for human patients, b) whether the medical/surgical instruments used on animals can be subsequently used on human patients, and c) which disinfecting or sterilizing procedures need to be done for these purposes. Studies addressing these concerns are lacking. However, with respect to disinfection or sterilization in veterinary settings, only minimal evidence suggests that zoonotic microbial pathogens are unusually resistant to inactivation by chemical or physical agents (with the exception of prions). Ample evidence supports the contrary observation (i.e., that pathogens from human- and animal sources are similar in their relative intrinsic resistance to inactivation).^{1386–1391} Further, no evidence suggests that zoonotic pathogens behave differently from human pathogens with respect to ventilation. Despite this knowledge, an aesthetic and sociologic perception that animal care must remain separate from human care persists. Health-care facilities, however, are increasingly faced with requests from the veterinary medical community for access to human health-care facilities for reasons that are largely economical (e.g., costs of acquiring sophisticated diagnostic technology and complex medical instruments). If hospital guidelines allow treatment of animals, alternate veterinary resources (including veterinary hospitals, clinics, and universities) should be exhausted before using human health-care settings. Additionally, the hospital's public/media relations should be notified of the situation. The goal is to develop policies and procedures to proactively and positively discuss and

disclose this activity to the general public.

An infection-control risk assessment (ICRA) must be undertaken to evaluate the circumstances specific to providing care to animals in a human health-care facility. Individual hospital policies and guidelines should be reviewed before any animal treatment is considered in such facilities. Animals treated in human health-care facilities should be under the direct care and supervision of a licensed veterinarian; they also should be free of known infectious diseases, ectoparasites, and other external contaminants (e.g., soil, urine, and feces). Measures should be taken to avoid treating animals with a known or suspected zoonotic disease in a human health-care setting (e.g., lambs being treated for Q fever).

If human health-care facilities must be used for animal treatment or diagnostics, the following general infection-control actions are suggested: a) whenever possible, the use of ORs or other rooms used for invasive procedures should be avoided [e.g., cardiac catheterization labs and invasive nuclear medicine areas]; b) when all other space options are exhausted and use of the aforementioned rooms is unavoidable, the procedure should be scheduled late in the day as the last procedure for that particular area such that patients are not present in the department/unit/area; c) environmental surfaces should be thoroughly cleaned and disinfected using procedures discussed in the Environmental Services portion of this guideline after the animal is removed from the care area; d) sufficient time should be allowed for ACH to help prevent allergic reactions by human patients [Table B.1. in Appendix B]; e) only disposable equipment or equipment that can be thoroughly and easily cleaned, disinfected, or sterilized should be used; f) when medical or surgical instruments, especially those invasive instruments that are difficult to clean [e.g., endoscopes], are used on animals, these instruments should be reserved for future use only on animals; and g) standard precautions should be followed.

5. Research Animals in Health-Care Facilities

The risk of acquiring a zoonotic infection from research animals has decreased in recent years because many small laboratory animals (e.g., mice, rats, and rabbits) come from quality stock and have defined microbiologic profiles.¹³⁹² Larger animals (e.g., nonhuman primates) are still obtained frequently from the wild and may harbor pathogens transmissible to humans. Primates, in particular, benefit from vaccinations to protect their health during the research period provided the vaccination does not interfere with the study of the particular agent. Animals serving as models for human disease studies pose some risk for transmission of infection to laboratory or health-care workers from percutaneous or mucosal exposure. Exposures can occur either through a) direct contact with an infected animal or its body substances and secretions or b) indirect contact with infectious material on equipment, instruments, surfaces, or supplies.¹³⁹² Uncontained aerosols generated during laboratory procedures can also transmit infection.

Infection-control measures to prevent transmission of zoonotic infections from research animals are largely derived from the following basic laboratory safety principles: a) purchasing pathogen-free animals, b) quarantining incoming animals to detect any zoonotic pathogens, c) treating infected animals or removing them from the facility, d) vaccinating animal carriers and high-risk contacts if possible, e) using specialized containment caging or facilities, and f) using protective clothing and equipment [e.g., gloves, face shields, gowns, and masks].¹³⁹² An excellent resource for detailed discussion of these safety measures has been published.¹⁰¹³

The animal research unit within a health-care facility should be engineered to provide a) adequate containment of animals and pathogens; b) daily decontamination and transport of equipment and waste; c) proper ventilation and air filtration, which prevents recirculation of the air in the unit to other areas of the facility; and d) negative air pressure in the animal rooms relative to the corridors. To ensure

adequate security and containment, no through traffic to other areas of the health-care facility should flow through this unit; access should be restricted to animal-care staff, researchers, environmental services, maintenance, and security personnel.

Occupational health programs for animal-care staff, researchers, and maintenance staff should take into consideration the animals' natural pathogens and research pathogens. Components of such programs include a) prophylactic vaccines, b) TB skin testing when primates are used, c) baseline serums, and d) hearing and respiratory testing. Work practices, PPE, and engineering controls specific for each of the four animal biosafety levels have been published.^{1013, 1393} The facility's occupational or employee health clinic should be aware of the appropriate post-exposure procedures involving zoonoses and have available the appropriate post-exposure biologicals and medications.

Animal-research-area staff should also develop standard operating procedures for a) daily animal husbandry [e.g., protection of the employee while facilitating animal welfare]; b) pathogen containment and decontamination; c) management, cleaning, disinfecting and/or sterilizing equipment and instruments; and d) employee training for laboratory safety and safety procedures specific to animal research worksites.¹⁰¹³ The federal Animal Welfare Act of 1966 and its amendments serve as the regulatory basis for ensuring animal welfare in research.^{1394, 1395}

I. Regulated Medical Waste

1. Epidemiology

No epidemiologic evidence suggests that most of the solid- or liquid wastes from hospitals, other health-care facilities, or clinical/research laboratories is any more infective than residential waste. Several studies have compared the microbial load and the diversity of microorganisms in residential wastes and wastes obtained from a variety of health-care settings.¹³⁹⁹⁻¹⁴⁰² Although hospital wastes had a greater number of different bacterial species compared with residential waste, wastes from residences were more heavily contaminated.^{1397, 1398} Moreover, no epidemiologic evidence suggests that traditional waste-disposal practices of health-care facilities (whereby clinical and microbiological wastes were decontaminated on site before leaving the facility) have caused disease in either the health-care setting or the general community.^{1400, 1401} This statement excludes, however, sharps injuries sustained during or immediately after the delivery of patient care before the sharp is "discarded." Therefore, identifying wastes for which handling and disposal precautions are indicated is largely a matter of judgment about the relative risk of disease transmission, because no reasonable standards on which to base these determinations have been developed. Aesthetic and emotional considerations (originating during the early years of the HIV epidemic) have, however, figured into the development of treatment and disposal policies, particularly for pathology and anatomy wastes and sharps.¹⁴⁰²⁻¹⁴⁰⁵ Public concerns have resulted in the promulgation of federal, state, and local rules and regulations regarding medical waste management and disposal.¹⁴⁰⁶⁻¹⁴¹⁴

2. Categories of Medical Waste

Precisely defining medical waste on the basis of quantity and type of etiologic agents present is virtually impossible. The most practical approach to medical waste management is to identify wastes that represent a sufficient potential risk of causing infection during handling and disposal and for which some precautions likely are prudent.² Health-care facility medical wastes targeted for handling and disposal precautions include microbiology laboratory waste (e.g., microbiologic cultures and stocks of microorganisms), pathology and anatomy waste, blood specimens from clinics and laboratories, blood

products, and other body-fluid specimens.² Moreover, the risk of either injury or infection from certain sharp items (e.g., needles and scalpel blades) contaminated with blood also must be considered. Although any item that has had contact with blood, exudates, or secretions may be potentially infective, treating all such waste as infective is neither practical nor necessary. Federal, state, and local guidelines and regulations specify the categories of medical waste that are subject to regulation and outline the requirements associated with treatment and disposal. The categorization of these wastes has generated the term “regulated medical waste.” This term emphasizes the role of regulation in defining the actual material and as an alternative to “infectious waste,” given the lack of evidence of this type of waste’s infectivity. State regulations also address the degree or amount of contamination (e.g., blood-soaked gauze) that defines the discarded item as a regulated medical waste. The EPA’s *Manual for Infectious Waste Management* identifies and categorizes other specific types of waste generated in health-care facilities with research laboratories that also require handling precautions.¹⁴⁰⁶

3. Management of Regulated Medical Waste in Health-Care Facilities

Medical wastes require careful disposal and containment before collection and consolidation for treatment. OSHA has dictated initial measures for discarding regulated medical-waste items. These measures are designed to protect the workers who generate medical wastes and who manage the wastes from point of generation to disposal.⁹⁶⁷ A single, leak-resistant biohazard bag is usually adequate for containment of regulated medical wastes, provided the bag is sturdy and the waste can be discarded without contaminating the bag’s exterior. The contamination or puncturing of the bag requires placement into a second biohazard bag. All bags should be securely closed for disposal. Puncture-resistant containers located at the point of use (e.g., sharps containers) are used as containment for discarded slides or tubes with small amounts of blood, scalpel blades, needles and syringes, and unused sterile sharps.⁹⁶⁷ To prevent needlestick injuries, needles and other contaminated sharps should not be recapped, purposefully bent, or broken by hand. CDC has published general guidelines for handling sharps.^{6, 1415} Health-care facilities may need additional precautions to prevent the production of aerosols during the handling of blood-contaminated items for certain rare diseases or conditions (e.g., Lassa fever and Ebola virus infection).²⁰³

Transporting and storing regulated medical wastes within the health-care facility prior to terminal treatment is often necessary. Both federal and state regulations address the safe transport and storage of on- and off-site regulated medical wastes.^{1406–1408} Health-care facilities are instructed to dispose medical wastes regularly to avoid accumulation. Medical wastes requiring storage should be kept in labeled, leak-proof, puncture-resistant containers under conditions that minimize or prevent foul odors. The storage area should be well ventilated and be inaccessible to pests. Any facility that generates regulated medical wastes should have a regulated medical waste management plan to ensure health and environmental safety as per federal, state, and local regulations.

4. Treatment of Regulated Medical Waste

Regulated medical wastes are treated or decontaminated to reduce the microbial load in or on the waste and to render the by-products safe for further handling and disposal. From a microbiologic standpoint, waste need not be rendered “sterile” because the treated waste will not be deposited in a sterile site. In addition, waste need not be subjected to the same reprocessing standards as are surgical instruments. Historically, treatment methods involved steam-sterilization (i.e., autoclaving), incineration, or interment (for anatomy wastes). Alternative treatment methods developed in recent years include chemical disinfection, grinding/shredding/disinfection methods, energy-based technologies (e.g., microwave or radiowave treatments), and disinfection/encapsulation methods.¹⁴⁰⁹ State medical waste regulations specify appropriate treatment methods for each category of regulated medical waste.

Of all the categories comprising regulated medical waste, microbiologic wastes (e.g., untreated cultures, stocks, and amplified microbial populations) pose the greatest potential for infectious disease transmission, and sharps pose the greatest risk for injuries. Untreated stocks and cultures of microorganisms are subsets of the clinical laboratory or microbiologic waste stream. If the microorganism must be grown and amplified in culture to high concentration to permit work with the specimen, this item should be considered for on-site decontamination, preferably within the laboratory unit. Historically, this was accomplished effectively by either autoclaving (steam sterilization) or incineration. If steam sterilization in the health-care facility is used for waste treatment, exposure of the waste for up to 90 minutes at 250°F (121°C) in an autoclave (depending on the size of the load and type container) may be necessary to ensure an adequate decontamination cycle.^{1416–1418} After steam sterilization, the residue can be safely handled and discarded with all other nonhazardous solid waste in accordance with state solid-waste disposal regulations. On-site incineration is another treatment option for microbiologic, pathologic, and anatomic waste, provided the incinerator is engineered to burn these wastes completely and stay within EPA emissions standards.¹⁴¹⁰ Improper incineration of waste with high moisture and low energy content (e.g., pathology waste) can lead to emission problems. State medical-waste regulatory programs identify acceptable methods for inactivating amplified stocks and cultures of microorganisms, some of which may employ technology rather than steam sterilization or incineration.

Concerns have been raised about the ability of modern health-care facilities to inactivate microbiologic wastes on-site, given that many of these institutions have decommissioned their laboratory autoclaves. Current laboratory guidelines for working with infectious microorganisms at biosafety level (BSL) 3 recommend that all laboratory waste be decontaminated before disposal by an approved method, preferably within the laboratory.¹⁰¹³ These same guidelines recommend that all materials removed from a BSL 4 laboratory (unless they are biological materials that are to remain viable) are to be decontaminated before they leave the laboratory.¹⁰¹³ Recent federal regulations for laboratories that handle certain biological agents known as “select agents” (i.e., those that have the potential to pose a severe threat to public health and safety) require these agents (and those obtained from a clinical specimen intended for diagnostic, reference, or verification purposes) to be destroyed on-site before disposal.¹⁴¹² Although recommendations for laboratory waste disposal from BSL 1 or 2 laboratories (e.g., most health-care clinical and diagnostic laboratories) allow for these materials to be decontaminated off-site before disposal, on-site decontamination by a known effective method is preferred to reduce the potential of exposure during the handling of infectious material.

A recent outbreak of TB among workers in a regional medical-waste treatment facility in the United States demonstrated the hazards associated with aerosolized microbiologic wastes.^{1419, 1420} The facility received diagnostic cultures of *Mycobacterium tuberculosis* from several different health-care facilities before these cultures were chemically disinfected; this facility treated this waste with a grinding/shredding process that generated aerosols from the material.^{1419, 1420} Several operational deficiencies facilitated the release of aerosols and exposed workers to airborne *M. tuberculosis*. Among the suggested control measures was that health-care facilities perform on-site decontamination of laboratory waste containing live cultures of microorganisms before release of the waste to a waste management company.^{1419, 1420} This measure is supported by recommendations found in the CDC/NIH guideline for laboratory workers.¹⁰¹³ This outbreak demonstrates the need to avoid the use of any medical-waste treatment method or technology that can aerosolize pathogens from live cultures and stocks (especially those of airborne microorganisms) unless aerosols can be effectively contained and workers can be equipped with proper PPE.^{1419–1421} Safe laboratory practices, including those addressing waste management, have been published.^{1013, 1422}

In an era when local, state, and federal health-care facilities and laboratories are developing bioterrorism

response strategies and capabilities, the need to reinstate in-laboratory capacity to destroy cultures and stocks of microorganisms becomes a relevant issue.¹⁴²³ Recent federal regulations require health-care facility laboratories to maintain the capability of destroying discarded cultures and stocks on-site if these laboratories isolate from a clinical specimen any microorganism or toxin identified as a “select agent” from a clinical specimen (Table 27).^{1412, 1413} As an alternative, isolated cultures of select agents can be transferred to a facility registered to accept these agents in accordance with federal regulations.¹⁴¹² State medical waste regulations can, however, complicate or completely prevent this transfer if these cultures are determined to be medical waste, because most states regulate the inter-facility transfer of untreated medical wastes.

Table 27. Microorganisms and biologicals identified as select agents*+

<i>HHS Non-overlap select agents and toxins (42 CFR Part 73 §73.4)</i>	
Viruses	Crimean-Congo hemorrhagic fever virus; Ebola viruses; Cercopithecine herpesvirus 1 (herpes B virus); Lassa fever virus; Marburg virus; monkeypox virus; South American hemorrhagic fever viruses (Junin, Machupo, Sabia, Flexal, Guanarito); tick-borne encephalitis complex (flavi) viruses (Central European tick-borne encephalitis, Far Eastern tick-borne encephalitis [Russian spring and summer encephalitis, Kyasnaur Forest disease, Omsk hemorrhagic fever]); variola major virus (smallpox virus); and variola minor virus (alastrim)
Exclusions¶	Vaccine strain of Junin virus (Candid. #1)
Bacteria	<i>Rickettsia prowazekii</i> , <i>R. rickettsii</i> , <i>Yersinia pestis</i>
Fungi	<i>Coccidioides posadasii</i>
Toxins	Abrin; conotoxins; diacetoxyscirpenol; ricin; saxitoxin; Shiga-like ribosome inactivating proteins; tetrodotoxin
Exclusions¶	The following toxins (in purified form or in combinations of pure and impure forms) if the aggregate amount under the control of a principal investigator does not, at any time, exceed the amount specified: 100 mg of abrin; 100 mg of conotoxins; 1,000 mg of diacetoxyscirpenol; 100 mg of ricin; 100 mg of saxitoxin; 100 mg of Shiga-like ribosome inactivating proteins; or 100 mg of tetrodotoxin
Genetic elements, recombinant nucleic acids, and recombinant organisms¶	<ul style="list-style-type: none"> • Select agent viral nucleic acids (synthetic or naturally-derived, contiguous or fragmented, in host chromosomes or in expression vectors) that can encode infectious and/or replication competent forms of any of the select agent viruses; • Nucleic acids (synthetic or naturally-derived) that encode for the functional form(s) of any of the toxins listed in this table if the nucleic acids: a) are in a vector or host chromosome; b) can be expressed <i>in vivo</i> or <i>in vitro</i>; or c) are in a vector or host chromosome and can be expressed <i>in vivo</i> or <i>in vitro</i>; • Viruses, bacteria, fungi, and toxins listed in this table that have been genetically modified.
<i>High consequence livestock pathogens and toxins/select agents (overlap agents) (42 CFR Part 73 §73.5 and USDA regulation 9 CFR Part 121)</i>	
Viruses	Eastern equine encephalitis virus; Nipah and Hendra complex viruses; Rift Valley fever virus; Venezuelan equine encephalitis virus
Exclusions¶	MP-12 vaccine strain of Rift Valley fever virus; TC-83 vaccine strain of Venezuelan equine encephalitis virus
Bacteria	<i>Bacillus anthracis</i> ; <i>Brucella abortus</i> , <i>B. melitensis</i> , <i>B. suis</i> ; <i>Burkholderia mallei</i> (formerly <i>Pseudomonas mallei</i>), <i>B. pseudomallei</i> (formerly <i>P. pseudomallei</i>); botulinum neurotoxin-producing species of <i>Clostridium</i> ; <i>Coxiella burnetii</i> ; <i>Francisella tularensis</i>
Fungi	<i>Coccidioides immitis</i>
Toxins	Botulinum neurotoxins; <i>Clostridium perfringens</i> epsilon toxin; Shigatoxin; staphylococcal enterotoxins; T-2 toxin
Exclusions¶	The following toxins (in purified form or in combinations of pure and impure forms) if the aggregate amount under the control of a principal investigator does not, at any time, exceed the amount specified: 0.5 mg of botulinum neurotoxins; 100 mg of <i>Clostridium perfringens</i> epsilon toxin; 100 mg of Shigatoxin; 5 mg of staphylococcal enterotoxins; or 1,000 mg of T-2 toxin

High consequence livestock pathogens and toxins/select agents (overlap agents) (42 CFR Part 73 §73.5 and USDA regulation 9 CFR Part 121) (continued)	
Genetic elements, recombinant nucleic acids, and recombinant organisms¶	<ul style="list-style-type: none"> • Select agent viral nuclei acids (synthetic or naturally derived, contiguous or fragmented, in host chromosomes or in expression vectors) that can encode infectious and/or replication competent forms of any of the select agent viruses; • Nucleic acids (synthetic or naturally derived) that encode for the functional form(s) of any of the toxins listed in this table if the nucleic acids: a) are in a vector or host chromosome; b) can be expressed <i>in vivo</i> or <i>in vitro</i>; or c) are in a vector or host chromosome and can be expressed <i>in vivo</i> or <i>in vitro</i>; • Viruses, bacteria, fungi, and toxins listed in this table that have been genetically modified

* Material in this table is compiled from references 1412, 1413, and 1424. Reference 1424 also contains lists of select agents that include plant pathogens and pathogens affecting livestock.

+ 42 CFR 73 §§73.4 and 73.5 do not include any select agent or toxin that is in its naturally-occurring environment, provided it has not been intentionally introduced, cultivated, collected, or otherwise extracted from its natural source. These sections also do not include non-viable select agent organisms or nonfunctional toxins. This list of select agents is current as of 3 October 2003 and is subject to change pending the final adoption of 42 CFR Part 73.

¶ These table entries are listed in reference 1412 and 1413, but were not included in reference 1424.

5. Discharging Blood, Fluids to Sanitary Sewers or Septic Tanks

The contents of all vessels that contain more than a few milliliters of blood remaining after laboratory procedures, suction fluids, or bulk blood can either be inactivated in accordance with state-approved treatment technologies or carefully poured down a utility sink drain or toilet.¹⁴¹⁴ State regulations may dictate the maximum volume allowable for discharge of blood/body fluids to the sanitary sewer. No evidence indicates that bloodborne diseases have been transmitted from contact with raw or treated sewage. Many bloodborne pathogens, particularly bloodborne viruses, are not stable in the environment for long periods of time;^{1425, 1426} therefore, the discharge of small quantities of blood and other body fluids to the sanitary sewer is considered a safe method of disposing of these waste materials.¹⁴¹⁴ The following factors increase the likelihood that bloodborne pathogens will be inactivated in the disposal process: a) dilution of the discharged materials with water; b) inactivation of pathogens resulting from exposure to cleaning chemicals, disinfectants, and other chemicals in raw sewage; and c) effectiveness of sewage treatment in inactivating any residual bloodborne pathogens that reach the treatment facility. Small amounts of blood and other body fluids should not affect the functioning of a municipal sewer system. However, large quantities of these fluids, with their high protein content, might interfere with the biological oxygen demand (BOD) of the system. Local municipal sewage treatment restrictions may dictate that an alternative method of bulk fluid disposal be selected. State regulations may dictate what quantity constitutes a small amount of blood or body fluids.

Although concerns have been raised about the discharge of blood and other body fluids to a septic tank system, no evidence suggests that septic tanks have transmitted bloodborne infections. A properly functioning septic system is adequate for inactivating bloodborne pathogens. System manufacturers' instructions specify what materials may be discharged to the septic tank without jeopardizing its proper operation.

6. Medical Waste and CJD

Concerns also have been raised about the need for special handling and treatment procedures for wastes generated during the care of patients with CJD or other transmissible spongiform encephalopathies (TSEs). Prions, the agents that cause TSEs, have significant resistance to inactivation by a variety of physical, chemical, or gaseous methods.¹⁴²⁷ No epidemiologic evidence, however, links acquisition of CJD with medical-waste disposal practices. Although handling neurologic tissue for pathologic examination and autopsy materials with care, using barrier precautions, and following specific

procedures for the autopsy are prudent measures,¹¹⁹⁷ employing extraordinary measures once the materials are discarded is unnecessary. Regulated medical wastes generated during the care of the CJD patient can be managed using the same strategies as wastes generated during the care of other patients. After decontamination, these wastes may then be disposed in a sanitary landfill or discharged to the sanitary sewer, as appropriate.

Part II. Recommendations for Environmental Infection Control in Health-Care Facilities

A. Rationale for Recommendations

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretic rationale, applicability, and possible economic benefit. The recommendations are evidence-based wherever possible. However, certain recommendations are derived from empiric infection-control or engineering principles, theoretic rationale, or from experience gained from events that cannot be readily studied (e.g., floods).

The HICPAC system for categorizing recommendations has been modified to include a category for engineering standards and actions required by state or federal regulations. Guidelines and standards published by the American Institute of Architects (AIA), American Society of Heating, Refrigeration, and Air-Conditioning Engineers (ASHRAE), and the Association for the Advancement in Medical Instrumentation (AAMI) form the basis of certain recommendations. These standards reflect a consensus of expert opinions and extensive consultation with agencies of the U.S. Department of Health and Human Services. Compliance with these standards is usually voluntary. However, state and federal governments often adopt these standards as regulations. For example, the standards from AIA regarding construction and design of new or renovated health-care facilities, have been adopted by reference by >40 states. Certain recommendations have two category ratings (e.g., Categories IA and IC or Categories IB and IC), indicating the recommendation is evidence-based as well as a standard or regulation.

B. Rating Categories

Recommendations are rated according to the following categories:

- **Category IA.** Strongly recommended for implementation and strongly supported by well-designed experimental, clinical, or epidemiologic studies.
- **Category IB.** Strongly recommended for implementation and supported by certain experimental, clinical, or epidemiologic studies and a strong theoretical rationale.
- **Category IC.** Required by state or federal regulation, or representing an established association standard. (Note: Abbreviations for governing agencies and regulatory citations are listed, where appropriate. Recommendations from regulations adopted at state levels are also noted. Recommendations from AIA guidelines cite the appropriate sections of the standard).
- **Category II.** Suggested for implementation and supported by suggestive clinical or epidemiologic studies, or a theoretical rationale.
- **Unresolved Issue.** No recommendation is offered. No consensus or insufficient evidence exists regarding efficacy.

C. Recommendations—Air

I. Air-Handling Systems in Health-Care Facilities

- A. Use AIA guidelines as minimum standards where state or local regulations are not in place for design and construction of ventilation systems in new or renovated health-care facilities. Ensure that existing structures continue to meet the specifications in effect at the time of construction.¹²⁰ **Category IC** (AIA: 1.1.A, 5.4)
- B. Monitor ventilation systems in accordance with engineers' and manufacturers' recommendations to ensure preventive engineering, optimal performance for removal of particulates, and elimination of excess moisture.^{18, 35, 106, 120, 220, 222, 333, 336} **Category IB, IC** (AIA: 7.2, 7.31.D, 8.31.D, 9.31.D, 10.31.D, 11.31.D, EPA guidance)
 1. Ensure that heating, ventilation, air conditioning (HVAC) filters are properly installed and maintained to prevent air leakages and dust overloads.^{17, 18, 106, 222} **Category IB**
 2. Monitor areas with special ventilation requirements (e.g., AII or PE) for ACH, filtration, and pressure differentials.^{21, 120, 249, 250, 273–275, 277, 333–344} **Category IB, IC** (AIA: 7.2.C7, 7.2.D6)
 - a. Develop and implement a maintenance schedule for ACH, pressure differentials, and filtration efficiencies using facility-specific data as part of the multidisciplinary risk assessment. Take into account the age and reliability of the system.
 - b. Document these parameters, especially the pressure differentials.
 3. Engineer humidity controls into the HVAC system and monitor the controls to ensure proper moisture removal.¹²⁰ **Category IC** (AIA: 7.31.D9)
 - a. Locate duct humidifiers upstream from the final filters.
 - b. Incorporate a water-removal mechanism into the system.
 - c. Locate all duct takeoffs sufficiently down-stream from the humidifier so that moisture is completely absorbed.
 4. Incorporate steam humidifiers, if possible, to reduce potential for microbial proliferation within the system, and avoid use of cool mist humidifiers. **Category II**
 5. Ensure that air intakes and exhaust outlets are located properly in construction of new facilities and renovation of existing facilities.^{3, 120} **Category IC** (AIA: 7.31.D3, 8.31.D3, 9.31.D3, 10.31.D3, 11.31.D3)
 - a. Locate exhaust outlets >25 ft. from air-intake systems.
 - b. Locate outdoor air intakes ≥6 ft. above ground or ≥3 ft. above roof level.
 - c. Locate exhaust outlets from contaminated areas above roof level to minimize recirculation of exhausted air.
 6. Maintain air intakes and inspect filters periodically to ensure proper operation.^{3, 120, 249, 250, 273–275, 277} **Category IC** (AIA: 7.31.D8)
 7. Bag dust-filled filters immediately upon removal to prevent dispersion of dust and fungal spores during transport within the facility.^{106, 221} **Category IB**
 - a. Seal or close the bag containing the discarded filter.
 - b. Discard spent filters as regular solid waste, regardless of the area from which they were removed.²²¹
 8. Remove bird roosts and nests near air intakes to prevent mites and fungal spores from entering the ventilation system.^{3, 98, 119} **Category IB**
 9. Prevent dust accumulation by cleaning air-duct grilles in accordance with facility-specific procedures and schedules when rooms are not occupied by patients.^{21, 120, 249, 250, 273–275, 277} **Category IC, II** (AIA: 7.31.D10)

10. Periodically measure output to monitor system function; clean ventilation ducts as part of routine HVAC maintenance to ensure optimum performance.^{120, 263, 264}
Category II (AIA: 7.31.D10)
- C. Use portable, industrial-grade HEPA filter units capable of filtration rates in the range of 300–800 ft³/min. to augment removal of respirable particles as needed.²¹⁹ **Category II**
 1. Select portable HEPA filters that can recirculate all or nearly all of the room air and provide the equivalent of ≥ 12 ACH.⁴ **Category II**
 2. Portable HEPA filter units previously placed in construction zones can be used later in patient-care areas, provided all internal and external surfaces are cleaned, and the filter's performance verified by appropriate particle testing. **Category II**
 3. Situate portable HEPA units with the advice of facility engineers to ensure that all room air is filtered.⁴ **Category II**
 4. Ensure that fresh-air requirements for the area are met.^{214, 219} **Category II**
- D. Follow appropriate procedures for use of areas with through-the-wall ventilation units.¹²⁰
Category IC (AIA: 8.31.D1, 8.31.D8, 9.31.D23, 10.31.D18, 11.31.D15)
 1. Do not use such areas as PE rooms.¹²⁰ **Category IC** (AIA: 7.2.D3)
 2. Do not use a room with a through-the-wall ventilation unit as an AII room unless it can be demonstrated that all required AII engineering controls required are met.^{4, 120}
Category IC (AIA: 7.2.C3)
- E. Conduct an infection-control risk assessment (ICRA) and provide an adequate number of AII and PE rooms (if required) or other areas to meet the needs of the patient population.^{4, 6, 9, 18, 19, 69, 94, 120, 142, 331–334, 336–338} **Category IA, IC** (AIA: 7.2.C, 7.2.D)
- F. When UVGI is used as a supplemental engineering control, install fixtures 1) on the wall near the ceiling or suspended from the ceiling as an upper air unit; 2) in the air-return duct of an AII room; or 3) in designated enclosed areas or booths for sputum induction.⁴
Category II
- G. Seal windows in buildings with centralized HVAC systems and especially with PE areas.^{35, 111, 120} **Category IB, IC** (AIA: 7.2.D3)
- H. Keep emergency doors and exits from PE rooms closed except during an emergency; equip emergency doors and exits with alarms. **Category II**
- I. Develop a contingency plan for backup capacity in the event of a general power failure.⁷¹³
Category IC (Joint Commission on Accreditation of Healthcare Organizations [JCAHO]: Environment of Care [EC] 1.4)
 1. Emphasize restoration of proper air quality and ventilation conditions in AII rooms, PE rooms, operating rooms, emergency departments, and intensive care units.^{120, 713}
Category IC (AIA: 1.5.A1; JCAHO: EC 1.4)
 2. Deploy infection-control procedures to protect occupants until power and systems functions are restored.^{6, 120, 713} **Category IC** (AIA: 5.1, 5.2; JCAHO: EC 1.4)
- J. Do not shut down HVAC systems in patient-care areas except for maintenance, repair, testing of emergency backup capacity, or new construction.^{120, 206} **Category IB, IC** (AIA: 5.1, 5.2.B, C)
 1. Coordinate HVAC system maintenance with infection-control staff to allow for relocation of immunocompromised patients if necessary.¹²⁰ **Category IC** (AIA: 5.1, 5.2)
 2. Provide backup emergency power and air-handling and pressurization systems to maintain filtration, constant ACH, and pressure differentials in PE rooms, AII rooms, operating rooms, and other critical-care areas.^{9, 120, 278} **Category IC** (AIA: 1.5, 5.1, 5.2)
 3. For areas not served by installed emergency ventilation and backup systems, use portable units and monitor ventilation parameters and patients in those areas.²¹⁹
Category II
 4. Coordinate system startups with infection-control staff to protect patients in PE rooms from bursts of fungal spores.^{9, 35, 120, 278} **Category IC** (AIA: 5.1, 5.2)

5. Allow sufficient time for ACH to clean the air once the system is operational (Appendix B, Table B.1).^{4, 120} **Category IC** (AIA: 5.1, 5.2)
- K. HVAC systems serving offices and administration areas may be shut down for energy conservation purposes, but the shutdown must not alter or adversely affect pressure differentials maintained in laboratories or critical-care areas with specific ventilation requirements (i.e., PE rooms, AII rooms, operating rooms). **Category II**
- L. Whenever possible, avoid inactivating or shutting down the entire HVAC system at one time, especially in acute-care facilities. **Category II**
- M. Whenever feasible, design and install fixed backup ventilation systems for new or renovated construction for PE rooms, AII rooms, operating rooms, and other critical care areas identified by ICRA.¹²⁰ **Category IC** (AIA: 1.5.A1)

II. Construction, Renovation, Remediation, Repair, and Demolition

- A. Establish a multidisciplinary team that includes infection-control staff to coordinate demolition, construction, and renovation projects and consider proactive preventive measures at the inception; produce and maintain summary statements of the team's activities.^{17, 19, 20, 97, 109, 120, 249, 250, 273–277} **Category IB, IC** (AIA: 5.1)
- B. Educate both the construction team and the health-care staff in immunocompromised patient-care areas regarding the airborne infection risks associated with construction projects, dispersal of fungal spores during such activities, and methods to control the dissemination of fungal spores.^{3, 249, 250, 273–277, 1428–1432} **Category IB**
- C. Incorporate mandatory adherence agreements for infection control into construction contracts, with penalties for noncompliance and mechanisms to ensure timely correction of problems.^{3, 120, 249, 273–277} **Category IC** (AIA: 5.1)
- D. Establish and maintain surveillance for airborne environmental disease (e.g., aspergillosis) as appropriate during construction, renovation, repair, and demolition activities to ensure the health and safety of immunocompromised patients.^{3, 64, 65, 79} **Category IB**
 1. Using active surveillance, monitor for airborne fungal infections in immunocompromised patients.^{3, 9, 64, 65} **Category IB**
 2. Periodically review the facility's microbiologic, histopathologic, and postmortem data to identify additional cases.^{3, 9, 64, 65} **Category IB**
 3. If cases of aspergillosis or other health-care-associated airborne fungal infections occur, aggressively pursue the diagnosis with tissue biopsies and cultures as feasible.^{3, 64, 65, 79, 249, 273–277} **Category IB**
- E. Implement infection-control measures relevant to construction, renovation, maintenance, demolition, and repair.^{96, 97, 120, 276, 277} **Category IB, IC** (AIA: 5.1, 5.2)
 1. Before the project gets underway, perform an ICRA to define the scope of the project and the need for barrier measures.^{96, 97, 120, 249, 273–277} **Category IB, IC** (AIA: 5.1)
 - a. Determine if immunocompromised patients may be at risk for exposure to fungal spores from dust generated during the project.^{20, 109, 273–275, 277}
 - b. Develop a contingency plan to prevent such exposures.^{20, 109, 273–275, 277}
 2. Implement infection-control measures for external demolition and construction activities.^{50, 249, 273–277, 283} **Category IB**
 - a. Determine if the facility can operate temporarily on recirculated air; if feasible, seal off adjacent air intakes.
 - b. If this is not possible or practical, check the low-efficiency (roughing) filter banks frequently and replace as needed to avoid buildup of particulates.
 - c. Seal windows and reduce wherever possible other sources of outside air intrusion (e.g., open doors in stairwells and corridors), especially in PE areas.
 3. Avoid damaging the underground water distribution system (i.e., buried pipes) to prevent soil and dust contamination of the water.^{120, 305} **Category IB, IC** (AIA: 5.1)

4. Implement infection-control measures for internal construction activities.^{20, 49, 97, 120, 249, 273–277} **Category IB, IC** (AIA: 5.1, 5.2)
 - a. Construct barriers to prevent dust from construction areas from entering patient-care areas; ensure that barriers are impermeable to fungal spores and in compliance with local fire codes.^{20, 49, 97, 120, 284, 312, 713, 1431}
 - b. Block and seal off return air vents if rigid barriers are used for containment.^{120, 276, 277}
 - c. Implement dust control measures on surfaces and by diverting pedestrian traffic away from work zones.^{20, 49, 97, 120}
 - d. Relocate patients whose rooms are adjacent to work zones, depending upon their immune status, the scope of the project, the potential for generation of dust or water aerosols, and the methods used to control these aerosols.^{49, 120, 281}
5. Perform those engineering and work-site related infection-control measures as needed for internal construction, repairs, and renovations.^{20, 49, 97, 109, 120, 312} **Category IB, IC** (AIA: 5.1, 5.2)
 - a. Ensure proper operation of the air-handling system in the affected area after erection of barriers and before the room or area is set to negative pressure.^{49, 69, 276, 278} **Category IB**
 - b. Create and maintain negative air pressure in work zones adjacent to patient-care areas and ensure that required engineering controls are maintained.^{20, 49, 97, 109, 120, 312}
 - c. Monitor negative air flow inside rigid barriers.^{120, 281}
 - d. Monitor barriers and ensure the integrity of the construction barriers; repair gaps or breaks in barrier joints.^{120, 284, 307, 312}
 - e. Seal windows in work zones if practical; use window chutes for disposal of large pieces of debris as needed, but ensure that the negative pressure differential for the area is maintained.^{20, 120, 273}
 - f. Direct pedestrian traffic from construction zones away from patient-care areas to minimize the dispersion of dust.^{20, 49, 97, 109, 111, 120, 273–277}
 - g. Provide construction crews with 1) designated entrances, corridors, and elevators whenever practical; 2) essential services [e.g., toilet facilities], and convenience services [e.g., vending machines]; 3) protective clothing [e.g., coveralls, footwear, and headgear] for travel to patient-care areas; and 4) a space or anteroom for changing clothing and storing equipment.^{120, 249, 273–277}
 - h. Clean work zones and their entrances daily by 1) wet-wiping tools and tool carts before their removal from the work zone; 2) placing mats with tacky surfaces inside the entrance; and 3) covering debris and securing this covering before removing debris from the work zone.^{120, 249, 273–277}
 - i. In patient-care areas, for major repairs that include removal of ceiling tiles and disruption of the space above the false ceiling, use plastic sheets or prefabricated plastic units to contain dust; use a negative pressure system within this enclosure to remove dust; and either pass air through an industrial grade, portable HEPA filter capable of filtration rates ranging from 300–800 ft³/min., or exhaust air directly to the outside.^{49, 276, 277, 281, 309}
 - j. Upon completion of the project, clean the work zone according to facility procedures, and install barrier curtains to contain dust and debris before removal of rigid barriers.^{20, 97, 120, 249, 273–277}
 - k. Flush the water system to clear sediment from pipes to minimize waterborne microorganism proliferation.^{120, 305}
 - l. Restore appropriate ACH, humidity, and pressure differential; clean or replace air filters; dispose of spent filters.^{35, 106, 221, 278}

- F. Use airborne-particle sampling as a tool to evaluate barrier integrity.^{35, 100} **Category II**
- G. Commission the HVAC system for newly constructed health-care facilities and renovated spaces before occupancy and use, with emphasis on ensuring proper ventilation for operating rooms, AII rooms, and PE areas.^{100, 120, 288, 304} **Category IC** (AIA: 5.1; ASHRAE: 1-1996)
- H. **No recommendation is offered** on routine microbiologic air sampling before, during, or after construction or before or during occupancy of areas housing immunocompromised patients.^{17, 20, 49, 97, 109, 272, 1433} **Unresolved issue**
- I. If a case of health-care-acquired aspergillosis or other opportunistic environmental airborne fungal disease occurs during or immediately after construction, implement appropriate follow-up measures.^{20, 55, 62, 77, 94, 95} **Category IB**
1. Review pressure differential monitoring documentation to verify that pressure differentials in the construction zone and in PE rooms were appropriate for their settings.^{94, 95, 120} **Category IB, IC** (AIA: 5.1)
 2. Implement corrective engineering measures to restore proper pressure differentials as needed.^{94, 95, 120} **Category IB, IC** (AIA: 5.1)
 3. Conduct a prospective search for additional cases and intensify retrospective epidemiologic review of the hospital's medical and laboratory records.^{3, 20, 62, 63, 104} **Category IB**
 4. If there is no evidence of ongoing transmission, continue routine maintenance in the area to prevent health-care-acquired fungal disease.^{3, 55} **Category IB**
- J. If there is epidemiologic evidence of ongoing transmission of fungal disease, conduct an environmental assessment to determine and eliminate the source.^{3, 96, 97, 109, 111, 115, 249, 273-277} **Category IB**
1. Collect environmental samples from potential sources of airborne fungal spores, preferably using a high-volume air sampler rather than settle plates.^{3, 18, 44, 48, 49, 97, 106, 111, 112, 115, 249, 254, 273-277, 292, 312} **Category IB**
 2. If either an environmental source of airborne fungi or an engineering problem with filtration or pressure differentials is identified, promptly perform corrective measures to eliminate the source and route of entry.^{96, 97} **Category IB**
 3. Use an EPA-registered anti-fungal biocide (e.g., copper-8-quinolinolate) for decontaminating structural materials.^{50, 277, 312, 329} **Category IB**
 4. If an environmental source of airborne fungi is not identified, review infection control measures, including engineering controls, to identify potential areas for correction or improvement.^{73, 117} **Category IB**
 5. If possible, perform molecular subtyping of *Aspergillus* spp. isolated from patients and the environment to establish strain identities.^{252, 293-296} **Category II**
- K. If air-supply systems to high-risk areas (e.g., PE rooms) are not optimal, use portable, industrial-grade HEPA filters on a temporary basis until rooms with optimal air-handling systems become available.^{3, 120, 273-277} **Category II**

III. Infection-Control and Ventilation Requirements for PE Rooms

- A. Minimize exposures of severely immunocompromised patients (e.g., solid organ transplant patients or allogeneic neutropenic patients) to activities that might cause aerosolization of fungal spores (e.g., vacuuming or disruption of ceiling tiles).^{9, 20, 109, 272} **Category IB**
- B. Minimize the length of time that immunocompromised patients in PE are outside their rooms for diagnostic procedures and other activities.^{9, 283} **Category IB**
- C. Provide respiratory protection for severely immunocompromised patients when they must leave PE for diagnostic studies and other activities; consult the most recent revision of CDC's *Guidelines for Prevention of Health-Care-Associated Pneumonia* for information regarding the appropriate type of respiratory protection.^{3, 9} **Category II**

- D. Incorporate ventilation engineering specifications and dust-controlling processes into the planning and construction of new PE units. **Category IB, IC**
1. Install central or point-of-use HEPA filters for supply (incoming) air.^{3, 18, 20, 44, 99–104, 120, 254, 316–318, 1432, 1434} **Category IB, IC** (AIA: 5.1, 5.2, 7.2.D)
 2. Ensure that rooms are well sealed by 1) properly constructing windows, doors, and intake and exhaust ports; 2) maintaining ceilings that are smooth and free of fissures, open joints, and crevices; 3) sealing walls above and below the ceiling, and 4) monitoring for leakage and making necessary repairs.^{3, 111, 120, 317, 318} **Category IB, IC** (AIA: 7.2.D3)
 3. Ventilate the room to maintain ≥ 12 ACH.^{3, 9, 120, 241, 317, 318} **Category IC** (AIA: 7.2.D)
 4. Locate air supply and exhaust grilles so that clean, filtered air enters from one side of the room, flows across the patient's bed, and exits from the opposite side of the room.^{3, 120, 317, 318} **Category IC** (AIA: 7.31.D1)
 5. Maintain positive room air pressure (≥ 2.5 Pa [0.01-inch water gauge]) in relation to the corridor.^{3, 35, 120, 317, 318} **Category IB, IC** (AIA: Table 7.2)
 6. Maintain airflow patterns and monitor these on a daily basis by using permanently installed visual means of detecting airflow in new or renovated construction, or using other visual methods (e.g., flutter strips, or smoke tubes) in existing PE units. Document the monitoring results.^{120, 273} **Category IC** (AIA: 7.2.D6)
 7. Install self-closing devices on all room exit doors in protective environments.¹²⁰ **Category IC** (AIA: 7.2.D4)
- E. Do not use laminar air flow systems in newly constructed PE rooms.^{316, 318} **Category II**
- F. Take measures to protect immunocompromised patients who would benefit from a PE room and who also have an airborne infectious disease (e.g., acute VZV infection or tuberculosis).
1. Ensure that the patient's room is designed to maintain positive pressure.
 2. Use an anteroom to ensure appropriate air balance relationships and provide independent exhaust of contaminated air to the outside, or place a HEPA filter in the exhaust duct if the return air must be recirculated.^{120, 317} **Category IC** (AIA: 7.2.D1, A7.2.D)
 3. If an anteroom is not available, place the patient in AII and use portable, industrial-grade HEPA filters to enhance filtration of spores in the room.²¹⁹ **Category II**
- G. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for PE areas and take immediate steps to restore the fixed ventilation system function.^{9, 120, 278} **Category IC** (AIA: 5.1)

IV. Infection-Control and Ventilation Requirements for AII Rooms

- A. Incorporate certain specifications into the planning, and construction or renovation of AII units.^{4, 107, 120, 317, 318} **Category IB, IC**
1. Maintain continuous negative air pressure (2.5 Pa [0.01-inch water gauge]) in relation to the air pressure in the corridor; monitor air pressure periodically, preferably daily, with audible manometers or smoke tubes at the door (for existing AII rooms) or with a permanently installed visual monitoring mechanism. Document the results of monitoring.^{120, 317, 318} **Category IB, IC** (AIA: 7.2.C7, Table 7.2)
 2. Ensure that rooms are well-sealed by properly constructing windows, doors, and air-intake and exhaust ports; when monitoring indicates air leakage, locate the leak and make necessary repairs.^{120, 317, 318} **Category IB, IC** (AIA: 7.2.C3)
 3. Install self-closing devices on all AII room exit doors.¹²⁰ **Category IC** (AIA: 7.2.C4)
 4. Provide ventilation to ensure ≥ 12 ACH for renovated rooms and new rooms, and ≥ 6 ACH for existing AII rooms.^{4, 107, 120} **Category IC** (AIA: Table 7.2)

5. Direct exhaust air to the outside, away from air-intake and populated areas. If this is not practical, air from the room can be recirculated after passing through a HEPA filter.^{4, 120} **Category IC** (AIA: Table 7.2)
- B. Where supplemental engineering controls for air cleaning are indicated from a risk assessment of the AII area, install UVGI units in the exhaust air ducts of the HVAC system to supplement HEPA filtration or install UVGI fixtures on or near the ceiling to irradiate upper room air.⁴ **Category II**
- C. Implement environmental infection-control measures for persons with known or suspected airborne infectious diseases.
 1. Use AII rooms for patients with or suspected of having an airborne infection who also require cough-inducing procedures, or use an enclosed booth that is engineered to provide 1) ≥ 12 ACH; 2) air supply and exhaust rate sufficient to maintain a 2.5 Pa [0.01-inch water gauge] negative pressure difference with respect to all surrounding spaces with an exhaust rate of ≥ 50 ft³/min.; and 3) air exhausted directly outside away from air intakes and traffic or exhausted after HEPA filtration prior to recirculation.^{4, 120, 348–350} **Category IB, IC** (AIA: 7.15.E, 7.31.D23, 9.10, Table 7.2)
 2. Although airborne spread of viral hemorrhagic fever (VHF) has not been documented in a health-care setting, prudence dictates placing a VHF patient in an AII room, preferably with an anteroom to reduce the risk of occupational exposure to aerosolized infectious material in blood, vomitus, liquid stool, and respiratory secretions present in large amounts during the end stage of a patient's illness.^{202–204} **Category II**
 - a. If an anteroom is not available, use portable, industrial-grade HEPA filters in the patient's room to provide additional ACH equivalents for removing airborne particulates.
 - b. Ensure that health-care workers wear face shields or goggles with appropriate respirators when entering the rooms of VHF patients with prominent cough, vomiting, diarrhea, or hemorrhage.²⁰³
 3. Place smallpox patients in negative pressure rooms at the onset of their illness, preferably using a room with an anteroom if available.⁶ **Category II**
- D. **No recommendation is offered** regarding negative pressure or isolation rooms for patients with *Pneumocystis carinii* pneumonia.^{126, 131, 152} **Unresolved issue**
- E. Maintain back-up ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for AII rooms and take immediate steps to restore the fixed ventilation system function.^{4, 120, 278} **Category IC** (AIA: 5.1)

V. Infection-Control and Ventilation Requirements for Operating Rooms

- A. Implement environmental infection-control and ventilation measures for operating rooms.
 1. Maintain positive-pressure ventilation with respect to corridors and adjacent areas.^{7, 120, 356} **Category IB, IC** (AIA: Table 7.2)
 2. Maintain ≥ 15 ACH, of which ≥ 3 ACH should be fresh air.^{120, 357, 358} **Category IC** (AIA: Table 7.2)
 3. Filter all recirculated and fresh air through the appropriate filters, providing 90% efficiency (dust-spot testing) at a minimum.^{120, 362} **Category IC** (AIA: Table 7.3)
 4. In rooms not engineered for horizontal laminar airflow, introduce air at the ceiling and exhaust air near the floor.^{120, 357, 359} **Category IC** (AIA: 7.31.D4)
 5. Do not use UV lights to prevent surgical-site infections.^{356, 364–370} **Category IB**
 6. Keep operating room doors closed except for the passage of equipment, personnel, and patients, and limit entry to essential personnel.^{351, 352} **Category IB**
- B. Follow precautionary procedures for TB patients who also require emergency surgery.^{4, 347, 371} **Category IB, IC**

1. Use an N95 respirator approved by the National Institute for Occupational Safety and Health (NIOSH) without exhalation valves in the operating room.^{347, 372} **Category IC** (Occupational Safety and Health Administration [OSHA]; 29 Code of Federal Regulations [CFR] 1910.134,139)
 2. Intubate the patient in either the AII room or the operating room; if intubating the patient in the operating room, do not allow the doors to open until 99% of the airborne contaminants are removed (Appendix B, Table B.1).^{4, 358} **Category IB**
 3. When anesthetizing a patient with confirmed or suspected TB, place a bacterial filter between the anesthesia circuit and patient's airway to prevent contamination of anesthesia equipment or discharge of tubercle bacilli into the ambient air.^{371, 373}
Category IB
 4. Extubate and allow the patient to recover in an AII room.^{4, 358} **Category IB**
 5. If the patient has to be extubated in the operating room, allow adequate time for ACH to clean 99% of airborne particles from the air (Appendix B, Table B.1) because extubation is a cough-producing procedure.^{4, 358} **Category IB**
- C. Use portable, industrial-grade HEPA filters temporarily for supplemental air cleaning during intubation and extubation for infectious TB patients who require surgery.^{4, 219, 358}
Category II
1. Position the units appropriately so that all room air passes through the filter; obtain engineering consultation to determine the appropriate placement of the unit.⁴
Category II
 2. Switch the portable unit off during the surgical procedure. **Category II**
 3. Provide fresh air as per ventilation standards for operating rooms; portable units do not meet the requirements for the number of fresh ACH.^{120, 215, 219} **Category II**
- D. If possible, schedule infectious TB patients as the last surgical cases of the day to maximize the time available for removal of airborne contamination. **Category II**
- E. **No recommendation is offered** for performing orthopedic implant operations in rooms supplied with laminar airflow.^{362, 364} **Unresolved issue**
- F. Maintain backup ventilation equipment (e.g., portable units for fans or filters) for emergency provision of ventilation requirements for operating rooms, and take immediate steps to restore the fixed ventilation system function.^{68, 120, 278, 372} **Category IB, IC** (AIA: 5.1)

VI. Other Potential Infectious Aerosol Hazards in Health-Care Facilities

- A. In settings where surgical lasers are used, wear appropriate personal protective equipment, including N95 or N100 respirators, to minimize exposure to laser plumes.^{347, 378, 389}
Category IC (OSHA; 29 CFR 1910.134,139)
- B. Use central wall suction units with in-line filters to evacuate minimal laser plumes.^{378, 382, 386, 389}
Category II
- C. Use a mechanical smoke evacuation system with a high-efficiency filter to manage the generation of large amounts of laser plume, when ablating tissue infected with human papilloma virus (HPV) or performing procedures on a patient with extrapulmonary TB.^{4, 382, 389-392}
Category II

D. Recommendations—Water

I. Controlling the Spread of Waterborne Microorganisms

- A. Practice hand hygiene to prevent the hand transfer of waterborne pathogens, and use barrier precautions (e.g., gloves) as defined by other guidelines.^{6, 464, 577, 586, 592, 1364} **Category IA**

- B. Eliminate contaminated water or fluid environmental reservoirs (e.g., in equipment or solutions) wherever possible.^{464, 465} **Category IB**
- C. Clean and disinfect sinks and wash basins on a regular basis by using an EPA-registered product as set by facility policies. **Category II**
- D. Evaluate for possible environmental sources (e.g., potable water) of specimen contamination when waterborne microorganisms (e.g., NTM) of unlikely clinical importance are isolated from clinical cultures (e.g., specimens collected aseptically from sterile sites or, if post-procedural, colonization occurs after use of tap water in patient care).^{607, 610–612} **Category IB**
- E. Avoid placing decorative fountains and fish tanks in patient-care areas; ensure disinfection and fountain maintenance if decorative fountains are used in the public areas of the health-care facility.⁶⁶⁴ **Category IB**

II. Routine Prevention of Waterborne Microbial Contamination Within the Distribution System

- A. Maintain hot water temperature at the return at the highest temperature allowable by state regulations or codes, preferably $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$), and maintain cold water temperature at $< 68^{\circ}\text{F}$ ($< 20^{\circ}\text{C}$).^{3, 661} **Category IC** (States; ASHRAE: 12:2000)
- B. If the hot water temperature can be maintained at $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$), explore engineering options (e.g., install preset thermostatic valves in point-of-use fixtures) to help minimize the risk of scalding.⁶⁶¹ **Category II**
- C. When state regulations or codes do not allow hot water temperatures above the range of 105°F – 120°F (40.6°C – 49°C) for hospitals or 95°F – 110°F (35°C – 43.3°C) for nursing care facilities or when buildings cannot be retrofitted for thermostatic mixing valves, follow either of these alternative preventive measures to minimize the growth of *Legionella* spp. in water systems. **Category II**
 - 1. Periodically increase the hot water temperature to $\geq 150^{\circ}\text{F}$ ($\geq 66^{\circ}\text{C}$) at the point of use.⁶⁶¹ **Category II**
 - 2. Alternatively, chlorinate the water and then flush it through the system.^{661, 710, 711} **Category II**
- D. Maintain constant recirculation in hot-water distribution systems serving patient-care areas.¹²⁰ **Category IC** (AIA: 7.31.E.3)

III. Remediation Strategies for Distribution System Repair or Emergencies

- A. Whenever possible, disconnect the ice machine before planned water disruptions. **Category II**
- B. Prepare a contingency plan to estimate water demands for the entire facility in advance of significant water disruptions (i.e., those expected to result in extensive and heavy microbial or chemical contamination of the potable water), sewage intrusion, or flooding.^{713, 719} **Category IC** (JCAHO: EC 1.4)
- C. When a significant water disruption or an emergency occurs, adhere to any advisory to boil water issued by the municipal water utility.⁶⁴² **Category IB, IC** (Municipal order)
 - 1. Alert patients, families, staff, and visitors not to consume water from drinking fountains, ice, or drinks made from municipal tap water, while the advisory is in effect, unless the water has been disinfected (e.g., by bringing to a rolling boil for ≥ 1 minute).⁶⁴² **Category IB, IC** (Municipal order)
 - 2. After the advisory is lifted, run faucets and drinking fountains at full flow for ≥ 5 minutes, or use high-temperature water flushing or chlorination.^{642, 661} **Category IC, II** (Municipal order; ASHRAE 12:2000)
- D. Maintain a high level of surveillance for waterborne disease among patients after a boil water advisory is lifted. **Category II**

- E. Corrective decontamination of the hot water system might be necessary after a disruption in service or a cross-connection with sewer lines has occurred.
1. Decontaminate the system when the fewest occupants are present in the building (e.g., nights or weekends).^{3, 661} **Category IC** (ASHRAE: 12:2000)
 2. If using high-temperature decontamination, raise the hot-water temperature to 160°F–170°F (71°C–77°C) and maintain that level while progressively flushing each outlet around the system for ≥ 5 minutes.^{3, 661} **Category IC** (ASHRAE: 12:2000)
 3. If using chlorination, add enough chlorine, preferably overnight, to achieve a free chlorine residual of ≥ 2 mg/L (≥ 2 ppm) throughout the system.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
 - a. Flush each outlet until chlorine odor is detected.
 - b. Maintain the elevated chlorine concentration in the system for ≥ 2 hrs (but ≤ 24 hrs).
 4. Use a very thorough flushing of the water system instead of chlorination if a highly chlorine-resistant microorganism (e.g., *Cryptosporidium* spp.) is suspected as the water contaminant. **Category II**
- F. Flush and restart equipment and fixtures according to manufacturers' instructions. **Category II**
- G. Change the pretreatment filter and disinfect the dialysis water system with an EPA-registered product to prevent colonization of the reverse osmosis membrane and downstream microbial contamination.⁷²¹ **Category II**
- H. Run water softeners through a regeneration cycle to restore their capacity and function. **Category II**
- I. If the facility has a water-holding reservoir or water-storage tank, consult the facility engineer or local health department to determine whether this equipment needs to be drained, disinfected with an EPA-registered product, and refilled. **Category II**
- J. Implement facility management procedures to manage a sewage system failure or flooding (e.g., arranging with other health-care facilities for temporary transfer of patients or provision of services), and establish communications with the local municipal water utility and the local health department to ensure that advisories are received in a timely manner upon release.^{713, 719} **Category IC** (JCAHO: EC 1.4; Municipal order)
- K. Implement infection-control measures during sewage intrusion, flooding, or other water-related emergencies.
1. Relocate patients and clean or sterilize supplies from affected areas. **Category II**
 2. If hands are not visibly soiled or contaminated with proteinaceous material, include an alcohol-based hand rub in the hand hygiene process 1) before performing invasive procedures; 2) before and after each patient contact; and 3) whenever hand hygiene is indicated.¹³⁶⁴ **Category II**
 3. If hands are visibly soiled or contaminated with proteinaceous material, use soap and bottled water for handwashing.¹³⁶⁴ **Category II**
 4. If the potable water system is not affected by flooding or sewage contamination, process surgical instruments for sterilization according to standard procedures. **Category II**
 5. Contact the manufacturer of the automated endoscope reprocessor (AER) for specific instructions on the use of this equipment during a water advisory. **Category II**
- L. Remediate the facility after sewage intrusion, flooding, or other water-related emergencies.
1. Close off affected areas during cleanup procedures. **Category II**
 2. Ensure that the sewage system is fully functional before beginning remediation so contaminated solids and standing water can be removed. **Category II**

3. If hard-surface equipment, floors, and walls remain in good repair, ensure that these are dry within 72 hours; clean with detergent according to standard cleaning procedures. **Category II**
 4. Clean wood furniture and materials (if still in good repair); allow them to dry thoroughly before restoring varnish or other surface coatings. **Category II**
 5. Contain dust and debris during remediation and repair as outlined in air recommendations (Air: II G 4, 5). **Category II**
- M. Regardless of the original source of water damage (e.g., flooding versus water leaks from point-of-use fixtures or roofs), remove wet, absorbent structural items (e.g., carpeting, wallboard, and wallpaper) and cloth furnishings if they cannot be easily and thoroughly cleaned and dried within 72 hours (e.g., moisture content $\leq 20\%$ as determined by moisture meter readings); replace with new materials as soon as the underlying structure is declared by the facility engineer to be thoroughly dry.^{18, 266, 278, 1026} **Category IB**

IV. Additional Engineering Measures as Indicated by Epidemiologic Investigation for Controlling Waterborne, Health-Care–Associated Legionnaires Disease

- A. When using a pulse or one-time decontamination method, superheat the water by flushing each outlet for ≥ 5 minutes with water at 160°F – 170°F (71°C – 77°C) or hyperchlorinate the system by flushing all outlets for ≥ 5 minutes with water containing ≥ 2 mg/L (≥ 2 ppm) free residual chlorine using a chlorine-based product registered by the EPA for water treatment (e.g., sodium hypochlorite [chlorine bleach]).^{661, 711, 714, 724, 764, 766} **Category IB** (ASHRAE: 12:2000)
- B. After a pulse treatment, maintain both the heated water temperature at the return and the cold water temperature as per the recommendation (Water: IIA) wherever practical and permitted by state codes, or chlorinate heated water to achieve 1–2 mg/L (1–2 ppm) free residual chlorine at the tap using a chlorine-based product registered by the EPA for water treatment (e.g., sodium hypochlorite [bleach]).^{26, 437, 661, 709, 726, 727} **Category IC** (States; ASHRAE: 12:2000)
- C. Explore engineering or educational options (e.g., install preset thermostatic mixing valves in point-of-use fixtures or post warning signs at each outlet) to minimize the risk of scalding for patients, visitors, and staff. **Category II**
- D. **No recommendation is offered** for treating water in the facility’s distribution system with chlorine dioxide, heavy-metal ions (e.g., copper or silver), monochloramine, ozone, or UV light.^{728–746} **Unresolved issue**

V. General Infection-Control Strategies for Preventing Legionnaires Disease

- A. Conduct an infection-control risk assessment of the facility to determine if patients at risk or severely immunocompromised patients are present.^{3, 431, 432} **Category IB**
- B. Implement general strategies for detecting and preventing Legionnaires disease in facilities that do not provide care for severely immunocompromised patients (i.e., facilities that do not have HSCT or solid organ transplant programs).^{3, 431, 432} **Category IB**
 1. Establish a surveillance process to detect health-care–associated Legionnaires disease.^{3, 431, 432} **Category IB**
 2. Inform health-care personnel (e.g., infection control, physicians, patient-care staff, and engineering) regarding the potential for Legionnaires disease to occur and measures to prevent and control health-care–associated legionellosis.^{437, 759} **Category IB**
 3. Establish mechanisms to provide clinicians with laboratory tests (e.g., culture, urine antigen, direct fluorescence assay [DFA], and serology) for the diagnosis of Legionnaires disease.^{3, 431} **Category IB**

- C. Maintain a high index of suspicion for health-care–associated Legionnaires disease, and perform laboratory diagnostic tests for legionellosis on suspected cases, especially in patients at risk who do not require a PE for care (e.g., patients receiving systemic steroids; patients aged ≥ 65 years; or patients with chronic underlying disease [e.g., diabetes mellitus, congestive heart failure, or chronic obstructive lung disease]).^{3, 395, 417, 423–425, 432, 435, 437, 453}
Category IA
- D. Periodically review the availability and clinicians' use of laboratory diagnostic tests for Legionnaires disease in the facility; if clinicians' use of the tests on patients with diagnosed or suspected pneumonia is limited, implement measures (e.g., an educational campaign) to enhance clinicians' use of the test(s).⁴⁵³ **Category IB**
- E. If one case of laboratory-confirmed, health-care–associated Legionnaires disease is identified, or if two or more cases of laboratory-suspected, health-care–associated Legionnaires disease occur during a 6-month period, certain activities should be initiated.^{405, 408, 431, 453, 739, 759} **Category IB**
1. Report the cases to the state and local health departments where required. **Category IC** (States)
 2. If the facility does not treat severely immunocompromised patients, conduct an epidemiologic investigation, including retrospective review of microbiologic, serologic, and postmortem data to look for previously unidentified cases of health-care–associated Legionnaires disease, and begin intensive prospective surveillance for additional cases.^{3, 405, 408, 431, 453, 739, 759} **Category IB**
 3. If no evidence of continued health-care–associated transmission exists, continue intensive prospective surveillance for ≥ 2 months after the initiation of surveillance.^{3, 405, 408, 431, 453, 739, 759} **Category IB**
- F. If there is evidence of continued health-care–associated transmission (i.e., an outbreak), conduct an environmental assessment to determine the source of *Legionella* spp.^{403–410, 455} **Category IB**
1. Collect water samples from potential aerosolized water sources (Appendix C).¹²⁰⁹ **Category IB**
 2. Save and subtype isolates of *Legionella* spp. obtained from patients and the environment.^{403–410, 453, 763, 764} **Category IB**
 3. If a source is identified, promptly institute water system decontamination measures per recommendations (see Water IV).^{766, 767} **Category IB**
 4. If *Legionella* spp. are detected in ≥ 1 cultures (e.g., conducted at 2-week intervals during 3 months), reassess the control measures, modify them accordingly, and repeat the decontamination procedures; consider intensive use of techniques used for initial decontamination, or a combination of superheating and hyperchlorination.^{3, 767, 768} **Category IB**
- G. If an environmental source is not identified during a Legionnaires disease outbreak, continue surveillance for new cases for ≥ 2 months. Either defer decontamination pending identification of the source of *Legionella* spp., or proceed with decontamination of the hospital's water distribution system, with special attention to areas involved in the outbreak. **Category II**
- H. **No recommendation is offered** regarding routine culturing of water systems in health-care facilities that do not have patient-care areas (i.e., PE or transplant units) for persons at high risk for *Legionella* spp. infection.^{26, 453, 707, 709, 714, 747, 753} **Unresolved issue**
- I. **No recommendation is offered** regarding the removal of faucet aerators in areas for immunocompetent patients. **Unresolved issue**
- J. Keep adequate records of all infection-control measures and environmental test results for potable water systems. **Category II**

VI. Preventing Legionnaires Disease in Protective Environments and Transplant Units

- A. When implementing strategies for preventing Legionnaires disease among severely immunosuppressed patients housed in facilities with HSCT or solid-organ transplant programs, incorporate these specific surveillance and epidemiologic measures in addition to the steps previously outlined (Water: V and Appendix C).
1. Maintain a high index of suspicion for legionellosis in transplant patients even when environmental surveillance cultures do not yield legionellae.^{430, 431} **Category IB**
 2. If a case occurs in a severely immunocompromised patient, or if severely immunocompromised patients are present in high-risk areas of the hospital (e.g., PE or transplant units) and cases are identified elsewhere in the facility, conduct a combined epidemiologic and environmental investigation to determine the source of *Legionella* spp.^{431, 767} **Category IB**
- B. Implement culture strategies and potable water and fixture treatment measures in addition to those previously outlined (Water: V). **Category II**
1. Depending on state regulations on potable water temperature in public buildings,⁷²⁵ hospitals housing patients at risk for health-care–associated legionellosis should either maintain heated water with a minimum return temperature of $\geq 124^{\circ}\text{F}$ [$\geq 51^{\circ}\text{C}$] and cold water at $< 68^{\circ}\text{F}$ [$< 20^{\circ}\text{C}$], or chlorinate heated water to achieve 1–2 mg/L (1–2 ppm) of free residual chlorine at the tap.^{26, 441, 661, 709–711, 726, 727} **Category II**
 2. Periodic culturing for legionellae in potable water samples from HSCT or solid-organ transplant units can be performed as part of a comprehensive strategy to prevent Legionnaires disease in these units.^{9, 431, 710, 769} **Category II**
 3. **No recommendation is offered** regarding the optimal methodology (i.e., frequency or number of sites) for environmental surveillance cultures in HSCT or solid organ transplant units. **Unresolved issue**
 4. In areas with patients at risk, when *Legionella* spp. are not detectable in unit water, remove, clean, and disinfect shower heads and tap aerators monthly by using a chlorine-based, EPA-registered product. If an EPA-registered chlorine disinfectant is not available, use a chlorine bleach solution (500–615 ppm [1:100 v/v dilution]).^{661, 745} **Category II**
- C. If *Legionella* spp. are determined to be present in the water of a transplant unit, implement certain measures until *Legionella* spp. are no longer detected by culture.
1. Decontaminate the water supply as outlined previously (Water: IV).^{3, 9, 661, 766, 767} **Category IB**
 2. Do not use water from the faucets in patient-care rooms to avoid creating infectious aerosols.^{9, 412} **Category IB**
 3. Restrict severely immunocompromised patients from taking showers.^{9, 412} **Category IB**
 4. Use water that is not contaminated with *Legionella* spp. for HSCT patients' sponge baths.^{9, 412} **Category IB**
 5. Provide patients with sterile water for tooth brushing, drinking, and for flushing nasogastric tubing during legionellosis outbreaks.^{9, 412} **Category IB**
- D. Do not use large-volume room air humidifiers that create aerosols (e.g., by Venturi principle, ultrasound, or spinning disk) unless they are subjected to high-level disinfection and filled only with sterile water.^{3, 9, 402, 455} **Category IB**

VII. Cooling Towers and Evaporative Condensers

- A. When planning construction of new health-care facilities, locate cooling towers so that the drift is directed away from the air-intake system, and design the towers to minimize the volume of aerosol drift.^{404, 661, 786} **Category IC** (ASHRAE: 12:2000)

- B. Implement infection-control procedures for operational cooling towers.^{404, 661, 784}
Category IC (ASHRAE: 12:2000)
1. Install drift eliminators.^{404, 661, 784} **Category IC** (ASHRAE: 12:2000)
 2. Use an effective EPA-registered biocide on a regular basis.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
 3. Maintain towers according to manufacturers' recommendations, and keep detailed maintenance and infection control records, including environmental test results from legionellosis outbreak investigations.⁶⁶¹ **Category IC** (ASHRAE: 12:2000)
- C. If cooling towers or evaporative condensers are implicated in health-care-associated legionellosis, decontaminate the cooling-tower system.^{404, 405, 786, 787} **Category IB**

VIII. Dialysis Water Quality and Dialysate

- A. Adhere to current AAMI standards for quality assurance performance of devices and equipment used to treat, store, and distribute water in hemodialysis centers (both acute and maintenance [chronic] settings) and for the preparation of concentrates and dialysate.^{31, 32, 666-668, 789, 791, 800, 807, 809, 1454, 1455} **Category IA, IC** (AAMI: ANSI/AAMI RD5:1992, ANSI/AAMI RD 47:1993)
- B. **No recommendation is offered** regarding whether more stringent requirements for water quality should be imposed in hemofiltration and hemodiafiltration. **Unresolved issue**^{789, 791, 792, 834, 835}
- C. Conduct microbiological testing specific to water in dialysis settings. **Category IA, IC** (AAMI: ANSI/AAMI RD 5: 1992, ANSI/AAMI RD 47: 1993, ANSI/AAMI RD 62:2001)
1. Perform bacteriologic assays of water and dialysis fluids at least once a month and during outbreaks using standard quantitative methods.^{792, 834, 835} **Category IA, IC** (AAMI: ANSI/AAMI RD 62:2001)
 - a. Assay for heterotrophic, mesophilic bacteria (e.g., *Pseudomonas* spp).
 - b. Do not use nutrient-rich media (e.g., blood agar or chocolate agar).
 2. In conjunction with microbiological testing, perform endotoxin testing on product water used to reprocess dialyzers for multiple use.^{789, 791, 806, 811, 816, 829} **Category IA, IC** (AAMI: ANSI/AAMI RD 5:1992, ANSI/AAMI RD 47:1993)
 3. Ensure that water does not exceed the limits for microbial counts and endotoxin concentrations outlined in Table 18.^{789, 791, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD 5:1992, ANSI/AAMI RD 47:1993)
- D. Disinfect water distribution systems in dialysis settings on a regular schedule. Monthly disinfection is recommended.^{666-668, 792, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD62:2001)
- E. Whenever practical, design and engineer water systems in dialysis settings to avoid incorporating joints, dead-end pipes, and unused branches and taps that can harbor bacteria.^{666-668, 792, 800} **Category IA, IC** (AAMI: ANSI/AAMI RD62:2001)
- F. When storage tanks are used in dialysis systems, they should be routinely drained, disinfected with an EPA-registered product, and fitted with an ultrafilter or pyrogenic filter (membrane filter with a pore size sufficient to remove small particles and molecules ≥ 1 kilodalton) installed in the water line distal to the storage tank.⁷⁹² **Category IC** (AAMI: ANSI/AAMI RD62:2001)

IX. Ice Machines and Ice

- A. Do not handle ice directly by hand, and wash hands before obtaining ice. **Category II**
- B. Use a smooth-surface ice scoop to dispense ice.^{680, 863} **Category II**
1. Keep the ice scoop on a chain short enough the scoop cannot touch the floor, or keep the scoop on a clean, hard surface when not in use.^{680, 863} **Category II**
 2. Do not store the ice scoop in the ice bin. **Category II**
- C. Do not store pharmaceuticals or medical solutions on ice intended for consumption; use sterile ice to keep medical solutions cold, or use equipment specifically manufactured for this purpose.^{600, 863} **Category IB**

- D. Machines that dispense ice are preferred to those that require ice to be removed from bins or chests with a scoop.^{687, 869} **Category II**
- E. Limit access to ice-storage chests, and keep the container doors closed except when removing ice.⁸⁶³ **Category II**
- F. Clean, disinfect, and maintain ice-storage chests on a regular basis. **Category II**
 - 1. Follow the manufacturer's instructions for cleaning. **Category II**
 - 2. Use an EPA-registered disinfectant suitable for use on ice machines, dispensers, or storage chests in accordance with label instructions. **Category II**
 - 3. If instructions and EPA-registered disinfectants suitable for use on ice machines are not available, use a general cleaning/disinfecting regimen as outlined in Box 12.⁸⁶³ **Category II**
 - 4. Flush and clean the ice machines and dispensers if they have not been disconnected before anticipated lengthy water disruptions. **Category II**
- G. Install proper air gaps where the condensate lines meet the waste lines. **Category II**
- H. Conduct microbiologic sampling of ice, ice chests, and ice-making machines and dispensers where indicated during an epidemiologic investigation.^{861–863} **Category IB**

X. Hydrotherapy Tanks and Pools

- A. Drain and clean hydrotherapy equipment (e.g., Hubbard tanks, tubs, whirlpools, whirlpool spas, or birthing tanks) after each patient's use, and disinfect equipment surfaces and components by using an EPA-registered product in accordance with the manufacturer's instructions. **Category II**
- B. In the absence of an EPA-registered product for water treatment, add sodium hypochlorite to the water:
 - 1. Maintain a 15-ppm chlorine residual in the water of small hydrotherapy tanks, Hubbard tanks, and tubs.⁸⁸⁹ **Category II**
 - 2. Maintain a 2–5 ppm chlorine residual in the water of whirlpools and whirlpool spas.⁹⁰⁵ **Category II**
 - 3. If the pH of the municipal water is in the basic range (e.g., when chloramine is used as the primary drinking water disinfectant in the community), consult the facility engineer regarding the possible need to adjust the pH of the water to a more acid level before disinfection, to enhance the biocidal activity of chlorine.⁸⁹⁴ **Category II**
- C. Clean and disinfect hydrotherapy equipment after using tub liners. **Category II**
- D. Clean and disinfect inflatable tubs unless they are single-use equipment. **Category II**
- E. **No recommendation is offered** regarding the use of antiseptic chemicals (e.g., chloramine-T) in the water during hydrotherapy sessions. **Unresolved issue**
- F. Conduct a risk assessment of patients prior to their use of large hydrotherapy pools, deferring patients with draining wounds or fecal incontinence from pool use until their condition resolves. **Category II**
- G. For large hydrotherapy pools, use pH and chlorine residual levels appropriate for an indoor pool as provided by local and state health agencies. **Category IC** (States)
- H. **No recommendation is offered** regarding the use in health care of whirlpools or spa equipment manufactured for home or recreational use. **Unresolved issue**

XI. Miscellaneous Medical Equipment Connected to Water Systems

- A. Clean, disinfect, and maintain AER equipment according to the manufacturer's instructions and relevant scientific literature to prevent inadvertent contamination of endoscopes and bronchoscopes with waterborne microorganisms.^{911–915} **Category IB**
 - 1. To rinse disinfected endoscopes and bronchoscopes, use water of the highest quality practical for the system's engineering and design (e.g., sterile water or

- bacteriologically-filtered water [water filtered through 0.1–0.2- μm filters]).^{912, 914, 915, 918} **Category IB**
2. Dry the internal channels of the reprocessed endoscope or bronchoscope using a proven method (e.g., 70% alcohol followed by forced-air treatment) to lessen the potential for the proliferation of waterborne microorganisms and to help prevent biofilm formation.^{671, 921, 923, 925, 928} **Category IB**
- B. Use water that meets nationally recognized standards set by the EPA for drinking water (<500 CFU/mL for heterotrophic plate count) for routine dental treatment output water.^{935, 936, 943, 944} **Category IB, IC** (EPA: 40 CFR 1 Part 141, Subpart G).
- C. Take precautions to prevent waterborne contamination of dental unit water lines and instruments.
1. After each patient, discharge water and air for a minimum of 20–30 seconds from any dental device connected to the dental water system that enters the patient’s mouth (e.g., handpieces, ultrasonic scalers, and air/water syringe).^{936, 937} **Category II**
 2. Consult with dental water-line manufacturers to 1) determine suitable methods and equipment to obtain the recommended water quality; and 2) determine appropriate methods for monitoring the water to ensure quality is maintained.^{936, 946} **Category II**
 3. Consult with the dental unit manufacturer on the need for periodic maintenance of anti-retraction mechanisms.^{937, 946} **Category IB**

E. Recommendations—Environmental Services

I. Cleaning and Disinfecting Strategies for Environmental Surfaces in Patient-Care Areas

- A. Select EPA-registered disinfectants, if available, and use them in accordance with the manufacturer’s instructions.^{2, 974, 983} **Category IB, IC** (EPA: 7 United States Code [USC] § 136 et seq)
- B. Do not use high-level disinfectants/liquid chemical sterilants for disinfection of either noncritical instrument/devices or any environmental surfaces; such use is counter to label instructions for these toxic chemicals.^{951, 952, 961–964} **Category IB, IC** (FDA: 21 CFR 801.5, 807.87.e)
- C. Follow manufacturers’ instructions for cleaning and maintaining noncritical medical equipment. **Category II**
- D. In the absence of a manufacturer’s cleaning instructions, follow certain procedures.
 1. Clean noncritical medical equipment surfaces with a detergent/disinfectant. This may be followed with an application of an EPA-registered hospital disinfectant with or without a tuberculocidal claim (depending on the nature of the surface and the degree of contamination), in accordance with disinfectant label instructions.⁹⁵² **Category II**
 2. Do not use alcohol to disinfect large environmental surfaces.⁹⁵¹ **Category II**
 3. Use barrier protective coverings as appropriate for noncritical equipment surfaces that are 1) touched frequently with gloved hands during the delivery of patient care; 2) likely to become contaminated with blood or body substances; or 3) difficult to clean (e.g., computer keyboards).⁹³⁶ **Category II**
- E. Keep housekeeping surfaces (e.g., floors, walls, and tabletops) visibly clean on a regular basis and clean up spills promptly.⁹⁵⁴ **Category II**
 1. Use a one-step process and an EPA-registered hospital disinfectant/detergent designed for general housekeeping purposes in patient-care areas when 1) uncertainty exists as to the nature of the soil on these surfaces [e.g., blood or body fluid contamination versus routine dust or dirt]; or 2) uncertainty exists regarding the presence or absence of multi-drug resistant organisms on such surfaces.^{952, 983, 986, 987} **Category II**

2. Detergent and water are adequate for cleaning surfaces in nonpatient-care areas (e.g., administrative offices). **Category II**
3. Clean and disinfect high-touch surfaces (e.g., doorknobs, bed rails, light switches, and surfaces in and around toilets in patients' rooms) on a more frequent schedule than minimal touch housekeeping surfaces. **Category II**
4. Clean walls, blinds, and window curtains in patient-care areas when they are visibly dusty or soiled.^{2, 971, 972, 982} **Category II**
- F. Do not perform disinfectant fogging in patient-care areas.^{2, 976} **Category IB**
- G. Avoid large-surface cleaning methods that produce mists or aerosols or disperse dust in patient-care areas.^{9, 20, 109, 272} **Category IB**
- H. Follow proper procedures for effective use of mops, cloths, and solutions. **Category II**
 1. Prepare cleaning solutions daily or as needed, and replace with fresh solution frequently according to facility policies and procedures.^{986, 987} **Category II**
 2. Change the mop head at the beginning of the day and also as required by facility policy, or after cleaning up large spills of blood or other body substances. **Category II**
 3. Clean mops and cloths after use and allow to dry before reuse; or use single-use, disposable mop heads and cloths.^{971, 988-990} **Category II**
- I. After the last surgical procedure of the day or night, wet vacuum or mop operating room floors with a single-use mop and an EPA-registered hospital disinfectant.⁷ **Category IB**
- J. Do not use mats with tacky surfaces at the entrance to operating rooms or infection-control suites.⁷ **Category IB**
- K. Use appropriate dusting methods for patient-care areas designated for immunocompromised patients (e.g., HSCT patients).^{9, 94, 986} **Category IB**
 1. Wet-dust horizontal surfaces daily by moistening a cloth with a small amount of an EPA-registered hospital detergent/disinfectant.^{9, 94, 986} **Category IB**
 2. Avoid dusting methods that disperse dust (e.g., feather-dusting).⁹⁴ **Category IB**
- L. Keep vacuums in good repair, and equip vacuums with HEPA filters for use in areas with patients at risk.^{9, 94, 986, 994} **Category IB**
- M. Close the doors of immunocompromised patients' rooms when vacuuming, waxing, or buffing corridor floors to minimize exposure to airborne dust.^{9, 94, 994} **Category IB**
- N. When performing low- or intermediate-level disinfection of environmental surfaces in nurseries and neonatal units, avoid unnecessary exposure of neonates to disinfectant residues on environmental surfaces by using EPA-registered disinfectants in accordance with manufacturers' instructions and safety advisories.^{974, 995-997} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
 1. Do not use phenolics or any other chemical germicide to disinfect bassinets or incubators during an infant's stay.^{952, 995-997} **Category IB**
 2. Rinse disinfectant-treated surfaces, especially those treated with phenolics, with water.⁹⁹⁵⁻⁹⁹⁷ **Category IB**
- O. When using phenolic disinfectants in neonatal units, prepare solutions to correct concentrations in accordance with manufacturers' instructions, or use premixed formulations.^{974, 995-997} **Category IB, IC** (EPA: 7 USC § 136 et seq.)

II. Cleaning Spills of Blood and Body Substances

- A. Promptly clean and decontaminate spills of blood or other potentially infectious materials.^{967, 998-1004} **Category IB, IC** (OSHA: 29 CFR 1910.1030 §d.4.ii.A)
- B. Follow proper procedures for site decontamination of spills of blood or blood-containing body fluids.^{967, 998-1004} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A)
 1. Use protective gloves and other PPE appropriate for this task.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.3.i, ii)

2. If the spill contains large amounts of blood or body fluids, clean the visible matter with disposable absorbent material, and discard the contaminated materials in appropriate, labeled containment.^{967, 1002, 1003, 1010, 1012} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.B)
3. Swab the area with a cloth or paper towels moderately wetted with disinfectant, and allow the surface to dry.^{967, 1010} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A)
- C. Use EPA-registered hospital disinfectants labeled tuberculocidal or registered germicides on the EPA Lists D and E (products with specific label claims for HIV or hepatitis B virus [HBV]) in accordance with label instructions to decontaminate spills of blood and other body fluids.^{967, 1007, 1010} **Category IC** (OSHA 29 CFR 1910.1030 § d.4.ii.A memorandum 2/28/97; compliance document CPL 2-2.44D [11/99])
- D. An EPA-registered sodium hypochlorite product is preferred, but if such products are not available, generic versions of sodium hypochlorite solutions (e.g., household chlorine bleach) may be used.
 1. Use a 1:100 dilution (500–615 ppm available chlorine) to decontaminate nonporous surfaces after cleaning a spill of either blood or body fluids in patient-care settings.^{1010, 1011} **Category II**
 2. If a spill involves large amounts of blood or body fluids, or if a blood or culture spill occurs in the laboratory, use a 1:10 dilution (5,000–6,150 ppm available chlorine) for the first application of germicide before cleaning.^{954, 1010} **Category II**

III. Carpeting and Cloth Furnishings

- A. Vacuum carpeting in public areas of health-care facilities and in general patient-care areas regularly with well-maintained equipment designed to minimize dust dispersion.⁹⁸⁶
Category II
- B. Periodically perform a thorough, deep cleaning of carpeting as determined by facility policy by using a method that minimizes the production of aerosols and leaves little or no residue.¹¹¹ **Category II**
- C. Avoid use of carpeting in high-traffic zones in patient-care areas or where spills are likely (e.g., burn therapy units, operating rooms, laboratories, and intensive care units).^{111, 1023, 1028}
Category IB
- D. Follow proper procedures for managing spills on carpeting.
 1. Spot-clean blood or body substance spills promptly.^{967, 1010, 1011, 1032} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A, interpretation)
 2. If a spill occurs on carpet tiles, replace any tiles contaminated by blood and body fluids or body substances.¹⁰³² **Category IC** (OSHA 29 CFR 1910.1030 § d.4.ii interpretation)
- E. Thoroughly dry wet carpeting to prevent the growth of fungi; replace carpeting that remains wet after 72 hours.^{9, 1026} **Category IB**
- F. **No recommendation is offered** regarding the routine use of fungicidal or bactericidal treatments for carpeting in public areas of a health-care facility or in general patient-care areas. **Unresolved issue**
- G. Do not use carpeting in hallways and patient rooms in areas housing immunosuppressed patients (e.g., PE areas).^{9, 111} **Category IB**
- H. Avoid the use of upholstered furniture and furnishings in high-risk patient-care areas and in areas with increased potential for body substance contamination (e.g., pediatrics units).⁹
Category II
- I. **No recommendation is offered** regarding whether upholstered furniture and furnishings should be avoided in general patient-care areas. **Unresolved issue**
- J. Maintain upholstered furniture in good repair. **Category II**
 1. Maintain the surface integrity of the upholstery by repairing tears and holes.
Category II

2. If upholstered furniture in a patient's room requires cleaning to remove visible soil or body substance contamination, move that item to a maintenance area where it can be adequately cleaned with a process appropriate for the type of upholstery and the nature of the soil. **Category II**

IV. Flowers and Plants in Patient-Care Areas

- A. Flowers and potted plants need not be restricted from areas for immunocompetent patients.^{515, 702, 1040, 1042} **Category II**
- B. Designate care and maintenance of flowers and potted plants to staff not directly involved with patient care.⁷⁰² **Category II**
- C. If plant or flower care by patient-care staff is unavoidable, instruct the staff to wear gloves when handling the plants and flowers and perform hand hygiene after glove removal.⁷⁰² **Category II**
- D. Do not allow fresh or dried flowers, or potted plants in patient-care areas for immunosuppressed patients.^{9, 109, 515, 1046} **Category II**

V. Pest Control

- A. Develop pest-control strategies, with emphasis on kitchens, cafeterias, laundries, central sterile supply areas, operating rooms, loading docks, construction activities, and other areas prone to infestations.^{1050, 1072, 1075} **Category II**
- B. Install screens on all windows that open to the outside; keep screens in good repair.¹⁰⁷² **Category IB**
- C. Contract for routine pest control service by a credentialed pest-control specialist who will tailor the application to the needs of a health-care facility.¹⁰⁷⁵ **Category II**
- D. Place laboratory specimens (e.g., fixed sputum smears) in covered containers for overnight storage.^{1065, 1066} **Category II**

VI. Special Pathogens

- A. Use appropriate hand hygiene, PPE (e.g., gloves), and isolation precautions during cleaning and disinfecting procedures.^{5, 952, 1130, 1364} **Category IB**
- B. Use standard cleaning and disinfection protocols to control environmental contamination with antibiotic-resistant gram-positive cocci (e.g., methicillin-resistant *Staphylococcus aureus*, vancomycin intermediate-resistant *Staphylococcus aureus*, or vancomycin-resistant *Enterococcus* [VRE]).^{5, 1116–1118} **Category IB**
 1. Pay close attention to cleaning and disinfection of high-touch surfaces in patient-care areas (e.g., bed rails, carts, bedside commodes, bedrails, doorknobs, or faucet handles).^{5, 1116–1118} **Category IB**
 2. Ensure compliance by housekeeping staff with cleaning and disinfection procedures.^{5, 1116–1118} **Category IB**
 3. Use EPA-registered hospital disinfectants appropriate for the surface to be disinfected (e.g., either low- or intermediate-level disinfection) as specified by the manufacturers' instructions.^{974, 1106–1110, 1118} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
 4. When contact precautions are indicated for patient care, use disposable patient-care items (e.g., blood pressure cuffs) whenever possible to minimize cross-contamination with multiple-resistant microorganisms.¹¹⁰² **Category IB**
 5. Follow these same surface cleaning and disinfecting measures for managing the environment of VRSA patients.^{1110, 1116–1118} **Category II**
- C. Environmental-surface culturing can be used to verify the efficacy of hospital policies and procedures before and after cleaning and disinfecting rooms that house patients with VRE.^{5, 1084, 1087, 1088, 1092, 1096} **Category II**

1. Obtain prior approval from infection-control staff and the clinical laboratory before performing environmental surface culturing. **Category II**
 2. Infection-control staff, with clinical laboratory consultation, must supervise all environmental culturing. **Category II**
- D. Thoroughly clean and disinfect environmental and medical equipment surfaces on a regular basis using EPA-registered disinfectants in accordance with manufacturers' instructions.^{952, 974, 1130, 1143} **Category IB, IC** (EPA: 7 USC § 136 et seq.)
- E. Advise families, visitors, and patients about the importance of hand hygiene to minimize the spread of body substance contamination (e.g., respiratory secretions or fecal matter) to surfaces.⁹⁵² **Category II**
- F. Do not use high-level disinfectants (i.e., liquid chemical sterilants) on environmental surfaces; such use is inconsistent with label instructions and because of the toxicity of the chemicals.^{2, 951, 952, 964} **Category IC** (FDA: 21 CFR 801.5, 807.87.e)
- G. Because no EPA-registered products are specific for inactivating *Clostridium difficile* spores, use hypochlorite-based products for disinfection of environmental surfaces in those patient-care areas where surveillance and epidemiology indicate ongoing transmission of *C. difficile*.^{952, 1130, 1141} **Category II**
- H. **No recommendation is offered** regarding the use of specific EPA-registered hospital disinfectants with respect to environmental control of *C. difficile*. **Unresolved issue**
- I. Apply standard cleaning and disinfection procedures to control environmental contamination with respiratory and enteric viruses in pediatric-care units and care areas for immunocompromised patients.^{986, 1158} **Category IC** (EPA: 7 USC § 136 et seq.)
- J. Clean surfaces that have been contaminated with body substances; perform low- to intermediate-level disinfection on cleaned surfaces with an EPA-registered disinfectant in accordance with the manufacturer's instructions.^{967, 974, 1158} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.ii.A; EPA: 7 USC § 136 et seq.)
- K. Use disposable barrier coverings as appropriate to minimize surface contamination. **Category II**
- L. Develop and maintain cleaning and disinfection procedures to control environmental contamination with agents of Creutzfeldt-Jakob disease (CJD), for which no EPA-registered product exists. **Category II**
1. In the absence of contamination with central nervous system tissue, extraordinary measures (e.g., use of 2N sodium hydroxide [NaOH] or applying full-strength sodium hypochlorite) are not needed for routine cleaning or terminal disinfection of a room housing a confirmed or suspected CJD patient.^{951, 1199} **Category II**
 2. After removing gross tissue from the surface, use either 1N NaOH or a sodium hypochlorite solution containing approximately 10,000–20,000 ppm available chlorine (dilutions of 1:5 to 1:3 v/v, respectively, of U.S. household chlorine bleach; contact the manufacturers of commercially available sodium hypochlorite products for advice) to decontaminate operating room or autopsy surfaces with central nervous system or cerebral spinal fluid contamination from a diagnosed or suspected CJD patient.^{951, 1170, 1188, 1191, 1197–1199, 1201} **Category II**
 - a. The contact time for the chemical used during this process should be 30 min–1 hour.^{1191, 1197, 1201}
 - b. Blot up the chemical with absorbent material and rinse the treated surface thoroughly with water.
 - c. Discard the used, absorbent material into appropriate waste containment.
 3. Use disposable, impervious covers to minimize body substance contamination to autopsy tables and surfaces.^{1197, 1201} **Category IB**

- M. Use standard procedures for containment, cleaning, and decontamination of blood spills on surfaces as previously described (Environmental Services: II).⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 §d.4.ii.A)
1. Wear PPE appropriate for a surface decontamination and cleaning task.^{967, 1199}
Category IC (OSHA 29 CFR 1910.1030 §d.3.i, ii)
 2. Discard used PPE by using routine disposal procedures or decontaminate reusable PPE as appropriate.^{967, 1199} **Category IC** (OSHA 29 CFR 1910.1030 §d.3.viii)

F. Recommendations—Environmental Sampling

I. General Information

- A. Do not conduct random, undirected microbiologic sampling of air, water, and environmental surfaces in health-care facilities.^{2, 1214} **Category IB**
- B. When indicated, conduct microbiologic sampling as part of an epidemiologic investigation or during assessment of hazardous environmental conditions to detect contamination and verify abatement of a hazard.^{2, 1214} **Category IB**
- C. Limit microbiologic sampling for quality assurance purposes to 1) biological monitoring of sterilization processes; 2) monthly cultures of water and dialysate in hemodialysis units; and 3) short-term evaluation of the impact of infection-control measures or changes in infection-control protocols.^{2, 1214} **Category IB**

II. Air, Water, and Environmental-Surface Sampling

- A. When conducting any form of environmental sampling, identify existing comparative standards and fully document departures from standard methods.^{945, 1214, 1223, 1224, 1238}
Category II
- B. Select a high-volume air sampling device if anticipated levels of microbial airborne contamination are expected to be low.^{290, 1218, 1223, 1224} **Category II**
- C. Do not use settle plates to quantify the concentration of airborne fungal spores.²⁹⁰
Category II
- D. When sampling water, choose growth media and incubation conditions that will facilitate the recovery of waterborne organisms.⁹⁴⁵ **Category II**
- E. When using a sample/rinse method for sampling an environmental surface, develop and document a procedure for manipulating the swab, gauze, or sponge in a reproducible manner so that results are comparable.¹²³⁸ **Category II**
- F. When environmental samples and patient specimens are available for comparison, perform the laboratory analysis on the recovered microorganisms down to the species level at a minimum and beyond the species level if possible.¹²¹⁴ **Category II**

G. Recommendations—Laundry and Bedding

I. Employer Responsibilities

- A. Employers must launder workers' personal protective garments or uniforms that are contaminated with blood or other potentially infectious materials.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.3.iv)

II. Laundry Facilities and Equipment

- A. Maintain the receiving area for contaminated textiles at negative pressure compared with the clean areas of the laundry in accordance with AIA construction standards in effect during the time of facility construction.^{120, 1260–1262} **Category IC** (AIA: 7.23.B1, B2)
- B. Ensure that laundry areas have handwashing facilities and products and appropriate PPE available for workers.^{120, 967} **Category IC** (AIA: 7.23.D4; OSHA: 29 CFR 1910.1030 § d.2.iii)
- C. Use and maintain laundry equipment according to manufacturers' instructions.^{1250, 1263}
Category II
- D. Do not leave damp textiles or fabrics in machines overnight.¹²⁵⁰ **Category II**
- E. Disinfection of washing and drying machines in residential care is not needed as long as gross soil is removed before washing and proper washing and drying procedures are used.
Category II

III. Routine Handling of Contaminated Laundry

- A. Handle contaminated textiles and fabrics with minimum agitation to avoid contamination of air, surfaces, and persons.^{6, 967, 1258, 1259} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
- B. Bag or otherwise contain contaminated textiles and fabrics at the point of use.⁹⁶⁷
Category IC (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 1. Do not sort or prerinse contaminated textiles or fabrics in patient-care areas.⁹⁶⁷
Category IC (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 2. Use leak-resistant containment for textiles and fabrics contaminated with blood or body substances.^{967, 1258} **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
 - 3. Identify bags or containers for contaminated textiles with labels, color coding, or other alternative means of communication as appropriate.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iv)
- C. Covers are not needed on contaminated textile hampers in patient-care areas. **Category II**
- D. If laundry chutes are used, ensure that they are properly designed, maintained, and used in a manner to minimize dispersion of aerosols from contaminated laundry.^{1253, 1267–1270}
Category IC (AAMI: ANSI/AAMI ST65:2000)
 - 1. Ensure that laundry bags are closed before tossing the filled bag into the chute.
Category II
 - 2. Do not place loose items in the chute. **Category II**
- E. Establish a facility policy to determine when textiles or fabrics should be sorted in the laundry facility (i.e., before or after washing).^{1271, 1272} **Category II**

IV. Laundry Process

- A. If hot-water laundry cycles are used, wash with detergent in water $\geq 160^{\circ}\text{F}$ ($\geq 71^{\circ}\text{C}$) for ≥ 25 minutes.^{2, 120} **Category IC** (AIA: 7.31.E3)
- B. **No recommendation is offered** regarding a hot-water temperature setting and cycle duration for items laundered in residence-style health-care facilities. **Unresolved issue**
- C. Follow fabric-care instructions and special laundering requirements for items used in the facility.¹²⁷⁸ **Category II**
- D. Choose chemicals suitable for low-temperature washing at proper use concentration if low-temperature ($< 160^{\circ}\text{F}$ [$< 71^{\circ}\text{C}$]) laundry cycles are used.^{1247, 1281–1285} **Category II**
- E. Package, transport, and store clean textiles and fabrics by methods that will ensure their cleanliness and protect them from dust and soil during interfacility loading, transport, and unloading.² **Category II**

V. Microbiologic Sampling of Textiles

- A. Do not conduct routine microbiological sampling of clean textiles.^{2, 1286} **Category IB**

- B. Use microbiological sampling during outbreak investigations if epidemiologic evidence suggests a role for health-care textiles and clothing in disease transmission.¹²⁸⁶ **Category IB**

VI. Special Laundry Situations

- A. Use sterilized textiles, surgical drapes, and gowns for situations requiring sterility in patient care.⁷ **Category IB**
- B. Use hygienically clean textiles (i.e., laundered, but not sterilized) in neonatal intensive care units.^{997, 1288} **Category IB**
- C. Follow manufacturers' recommendations for cleaning fabric products including those with coated or laminated surfaces. **Category II**
- D. Do not use dry cleaning for routine laundering in health-care facilities.^{1289–1291} **Category II**
- E. Use caution when considering the use of antimicrobial mattresses, textiles, and clothing as replacements for standard bedding and other fabric items; EPA has not approved public health claims asserting protection against human pathogens for treated articles.¹³⁰⁶ **Category II**
- F. **No recommendation is offered** regarding using disposable fabrics and textiles versus durable goods. **Unresolved issue**

VII. Mattresses and Pillows

- A. Keep mattresses dry; discard them if they become and remain wet or stained, particularly in burn units.^{1310–1315} **Category IB**
- B. Clean and disinfect mattress covers using EPA-registered disinfectants, if available, that are compatible with the cover materials to prevent the development of tears, cracks, or holes in the cover.^{1310–1315} **Category IB**
- C. Maintain the integrity of mattress and pillow covers. **Category II**
 - 1. Replace mattress and pillow covers if they become torn or otherwise in need of repair. **Category II**
 - 2. Do not stick needles into the mattress through the cover. **Category II**
- D. Clean and disinfect moisture-resistant mattress covers between patients using an EPA-registered product, if available.^{1310–1315} **Category IB**
- E. If using a mattress cover completely made of fabric, change these covers and launder between patients.^{1310–1315} **Category IB**
- F. Launder pillow covers and washable pillows in the hot-water cycle between patients or when they become contaminated with body substances.¹³¹⁵ **Category IB**

VIII. Air-Fluidized Beds

- A. Follow manufacturers' instructions for bed maintenance and decontamination. **Category II**
- B. Change the polyester filter sheet at least weekly or as indicated by the manufacturer.^{1317, 1318, 1322, 1323} **Category II**
- C. Clean and disinfect the polyester filter sheet thoroughly, especially between patients, using an EPA-registered product, if available.^{1317, 1318, 1322, 1323} **Category IB**
- D. Consult the facility engineer to determine the proper location of air-fluidized beds in negative-pressure rooms.¹³²⁶ **Category II**

H. Recommendations—Animals in Health-Care Facilities

I. General Infection-Control Measures for Animal Encounters

- A. Minimize contact with animal saliva, dander, urine, and feces.^{1365–1367} **Category II**
- B. Practice hand hygiene after any animal contact.^{2, 1364} **Category IB**
 - 1. Wash hands with soap and water, especially if hands are visibly soiled.¹³⁶⁴
Category IB
 - 2. Use either soap and water or alcohol-based hand rubs when hands are not visibly soiled.¹³⁶⁴ **Category IB**

II. Animal-Assisted Activities, Animal-Assisted Therapy, and Resident Animal Programs

- A. Avoid selection of nonhuman primates and reptiles in animal-assisted activities, animal-assisted therapy, or resident animal programs.^{1360–1362} **Category IB**
- B. Enroll animals that are fully vaccinated for zoonotic diseases and that are healthy, clean, well-groomed, and negative for enteric parasites or otherwise have completed recent antihelminthic treatment under the regular care of a veterinarian.^{1349, 1360} **Category II**
- C. Enroll animals that are trained with the assistance or under the direction of individuals who are experienced in this field.¹³⁶⁰ **Category II**
- D. Ensure that animals are handled by persons trained in providing activities or therapies safely, and who know the animals' health status and behavior traits.^{1349, 1360} **Category II**
- E. Take prompt action when an incident of biting or scratching by an animal occurs during an animal-assisted activity or therapy.
 - 1. Remove the animal permanently from these programs.¹³⁶⁰ **Category II**
 - 2. Report the incident promptly to appropriate authorities (e.g., infection-control staff, animal program coordinator, or local animal control).¹³⁶⁰ **Category II**
 - 3. Promptly clean and treat scratches, bites, or other accidental breaks in the skin.
Category II
- F. Perform an ICRA and work actively with the animal handler prior to conducting an animal-assisted activity or therapy to determine if the session should be held in a public area of the facility or in individual patient rooms.^{1349, 1360} **Category II**
- G. Take precautions to mitigate allergic responses to animals. **Category II**
 - 1. Minimize shedding of animal dander by bathing animals <24 hours before a visit.¹³⁶⁰
Category II
 - 2. Groom animals to remove loose hair before a visit, or using a therapy animal cape.¹³⁵⁸
Category II
- H. Use routine cleaning protocols for housekeeping surfaces after therapy sessions.
Category II
- I. Restrict resident animals, including fish in fish tanks, from access to or placement in patient-care areas, food preparation areas, dining areas, laundry, central sterile supply areas, sterile and clean supply storage areas, medication preparation areas, operating rooms, isolation areas, and PE areas. **Category II**
- J. Establish a facility policy for regular cleaning of fish tanks, rodent cages, bird cages, and any other animal dwellings and assign this cleaning task to a nonpatient-care staff member; avoid splashing tank water or contaminating environmental surfaces with animal bedding.
Category II

III. Protective Measures for Immunocompromised Patients

- A. Advise patients to avoid contact with animal feces and body fluids such as saliva, urine, or solid litter box material.⁸ **Category II**

- B. Promptly clean and treat scratches, bites, or other wounds that break the skin.⁸ **Category II**
- C. Advise patients to avoid direct or indirect contact with reptiles.¹³⁴⁰ **Category IB**
- D. Conduct a case-by-case assessment to determine if animal-assisted activities or animal-assisted therapy programs are appropriate for immunocompromised patients.¹³⁴⁹ **Category II**
- E. **No recommendation is offered** regarding permitting pet visits to terminally ill immunosuppressed patients outside their PE units. **Unresolved issue**

IV. Service Animals

- A. Avoid providing access to nonhuman primates and reptiles as service animals.^{1340, 1362} **Category IB**
- B. Allow service animals access to the facility in accordance with the Americans with Disabilities Act of 1990, unless the presence of the animal creates a direct threat to other persons or a fundamental alteration in the nature of services.^{1366, 1376} **Category IC** (U.S. Department of Justice: 28 CFR § 36.302)
- C. When a decision must be made regarding a service animal's access to any particular area of the health-care facility, evaluate the service animal, the patient, and the health-care situation on a case-by-case basis to determine whether significant risk of harm exists and whether reasonable modifications in policies and procedures will mitigate this risk.¹³⁷⁶ **Category IC** (Justice: 28 CFR § 36.208 and App.B)
- D. If a patient must be separated from his or her service animal while in the health-care facility
 - 1) ascertain from the person what arrangements have been made for supervision or care of the animal during this period of separation; and 2) make appropriate arrangements to address the patient's needs in the absence of the service animal. **Category II**

V. Animals as Patients in Human Health-Care Facilities

- A. Develop health-care facility policies to address the treatment of animals in human health-care facilities.
 1. Use the multidisciplinary team approach to policy development, including public media relations in order to disclose and discuss these activities. **Category II**
 2. Exhaust all veterinary facility, equipment, and instrument options before undertaking the procedure. **Category II**
 3. Ensure that the care of the animal is supervised by a licensed veterinarian. **Category II**
- B. When animals are treated in human health-care facilities, avoid treating animals in operating rooms or other patient-care areas where invasive procedures are performed (e.g., cardiac catheterization laboratories, or invasive nuclear medicine areas). **Category II**
- C. Schedule the animal procedure for the last case of the day for the area, at a time when human patients are not scheduled to be in the vicinity. **Category II**
- D. Adhere strictly to standard precautions. **Category II**
- E. Clean and disinfect environmental surfaces thoroughly using an EPA-registered product in the room after the animal is removed. **Category II**
- F. Allow sufficient ACH to clean the air and help remove airborne dander, microorganisms, and allergens [Appendix B, Table B.1.]). **Category II**
- G. Clean and disinfect using EPA-registered products or sterilize equipment that has been in contact with animals, or use disposable equipment. **Category II**
- H. If reusable medical or surgical instruments are used in an animal procedure, restrict future use of these instruments to animals only. **Category II**

VI. Research Animals in Health-Care Facilities

- A. Use animals obtained from quality stock, or quarantine incoming animals to detect zoonotic diseases. **Category II**
- B. Treat sick animals or remove them from the facility. **Category II**
- C. Provide prophylactic vaccinations, as available, to animal handlers and contacts at high risk. **Category II**
- D. Ensure proper ventilation through appropriate facility design and location.¹³⁹⁵ **Category IC** (U.S. Department of Agriculture [USDA]: 7 USC 2131)
 - 1. Keep animal rooms at negative pressure relative to corridors.¹³⁹⁵ **Category IC** (USDA: 7 USC 2131)
 - 2. Prevent air in animal rooms from recirculating elsewhere in the health-care facility.¹³⁹⁵ **Category IC** (USDA: 7 USC 2131)
- E. Keep doors to animal research rooms closed. **Category II**
- F. Restrict access to animal facilities to essential personnel. **Category II**
- G. Establish employee occupational health programs specific to the animal research facility, and coordinate management of postexposure procedures specific for zoonoses with occupational health clinics in the health-care facility.^{1013, 1378} **Category IC** (U.S. Department of Health and Human Services [DHHS]: BMBL; OSHA: 29 CFR 1910.1030.132-139)
- H. Document standard operating procedures for the unit.¹⁰¹³ **Category IC** (DHHS: BMBL)
- I. Conduct routine employee training on worker safety issues relevant to the animal research facility (e.g., working safely with animals and animal handling).^{1013, 1393} **Category IC** (DHHS: BMBL; OSHA: 29 CFR 1910.1030.132-139)
- J. Use precautions to prevent the development of animal-induced asthma in animal workers.¹⁰¹³ **Category IC** (DHHS: BMBL)

I. Recommendations—Regulated Medical Waste

I. Categories of Regulated Medical Waste

- A. Designate the following as major categories of medical waste that require special handling and disposal precautions: 1) microbiology laboratory wastes [e.g., cultures and stocks of microorganisms]; 2) bulk blood, blood products, blood, and bloody body fluid specimens; 3) pathology and anatomy waste; and 4) sharps [e.g., needles and scalpels].² **Category II**
- B. Consult federal, state, and local regulations to determine if other waste items are considered regulated medical wastes.^{967, 1407, 1408} **Category IC** (States; Authorities having jurisdiction [AHJ]; OSHA: 29 CFR 1910.1030 §g.2.1; U.S. Department of Transportation [DOT]: 49 CFR 171-180; U.S. Postal Service: CO23.8)

II. Disposal Plan for Regulated Medical Wastes

- A. Develop a plan for the collection, handling, predisposal treatment, and terminal disposal of regulated medical wastes.^{967, 1409} **Category IC** (States; AHJ; OSHA: 29 CFR 1910.1030 §g.2.i)
- B. Designate a person or persons to be responsible for establishing, monitoring, reviewing, and administering the plan. **Category II**

III. Handling, Transporting, and Storing Regulated Medical Wastes

- A. Inform personnel involved in the handling and disposal of potentially infective waste of the possible health and safety hazards; ensure that they are trained in appropriate handling and disposal methods.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § g.2.i)
- B. Manage the handling and disposal of regulated medical wastes generated in isolation areas by using the same methods as for regulated medical wastes from other patient-care areas.² **Category II**
- C. Use proper sharps disposal strategies.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)

1. Use a sharps container capable of maintaining its impermeability after waste treatment to avoid subsequent physical injuries during final disposal.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)
 2. Place disposable syringes with needles, including sterile sharps that are being discarded, scalpel blades, and other sharp items into puncture-resistant containers located as close as practical to the point of use.⁹⁶⁷ **Category IC** (OSHA: 29 CFR 1910.1030 § d.4.iii.A)
 3. Do not bend, recap, or break used syringe needles before discarding them into a container.^{6, 967, 1415} **Category IC** (OSHA: 29 CFR 1910.1030 § d.2.vii and § d.2.vii.A)
- D. Store regulated medical wastes awaiting treatment in a properly ventilated area that is inaccessible to vertebrate pests; use waste containers that prevent the development of noxious odors. **Category IC** (States; AHJ)
- E. If treatment options are not available at the site where the medical waste is generated, transport regulated medical wastes in closed, impervious containers to the on-site treatment location or to another facility for treatment as appropriate. **Category IC** (States; AHJ)

IV. Treatment and Disposal of Regulated Medical Wastes

- A. Treat regulated medical wastes by using a method (e.g., steam sterilization, incineration, interment, or an alternative treatment technology) approved by the appropriate authority having jurisdiction (AHJ) (e.g., states, Indian Health Service [IHS], Veterans Affairs [VA]) before disposal in a sanitary landfill. **Category IC** (States, AHJ)
- B. Follow precautions for treating microbiological wastes (e.g., amplified cultures and stocks of microorganisms).¹⁰¹³ **Category IC** (DHHS: BMBL)
1. Biosafety level 4 laboratories must inactivate microbiological wastes in the laboratory by using an approved inactivation method (e.g., autoclaving) before transport to and disposal in a sanitary landfill.¹⁰¹³ **Category IC** (DHHS: BMBL)
 2. Biosafety level 3 laboratories must inactivate microbiological wastes in the laboratory by using an approved inactivation method (e.g., autoclaving) or incinerate them at the facility before transport to and disposal in a sanitary landfill.¹⁰¹³ **Category IC** (DHHS: BMBL)
- C. Biosafety levels 1 and 2 laboratories should develop strategies to inactivate amplified microbial cultures and stocks onsite by using an approved inactivation method (e.g., autoclaving) instead of packaging and shipping untreated wastes to an offsite facility for treatment and disposal.^{1013, 1419–1421} **Category II**
- D. Laboratories that isolate select agents from clinical specimens must comply with federal regulations for the receipt, transfer, management, and appropriate disposal of these agents.¹⁴¹² **Category IC** (DHHS: 42 CFR 73 § 73.6)
- E. Sanitary sewers may be used for the safe disposal of blood, suctioned fluids, ground tissues, excretions, and secretions, provided that local sewage discharge requirements are met and that the state has declared this to be an acceptable method of disposal.¹⁴¹⁴ **Category II**

V. Special Precautions for Wastes Generated During Care of Patients with Rare Diseases

- A. When discarding items contaminated with blood and body fluids from VHF patients, contain these regulated medical wastes with minimal agitation during handling.^{6, 203} **Category II**
- B. Manage properly contained wastes from areas providing care to VHF patients in accordance with recommendations for other isolation areas (Regulated Medical Waste: III B).^{2, 6, 203} **Category II**
- C. Decontaminate bulk blood and body fluids from VHF patients using approved inactivation methods (e.g., autoclaving or chemical treatment) before disposal.^{6, 203} **Category IC, II** (States; AHJ)

- D. When discarding regulated medical waste generated during the routine (i.e., non-surgical) care of CJD patients, contain these wastes and decontaminate them using approved inactivation methods (e.g., autoclaving or incineration) appropriate for the medical waste category (e.g., blood, sharps, pathological waste).^{2, 6, 948, 1199} **Category IC, II** (States; AHJ)
- E. Incinerate medical wastes (e.g., central nervous system tissues or contaminated disposable materials) from brain autopsy or biopsy procedures of diagnosed or suspected CJD patients.^{1197, 1201} **Category IB**

Part III. References

Note: The bold item in parentheses indicated the citation number or the location of this reference listed in the MMWR version of this guideline.

1. Simmons BP. Guideline for hospital environmental control. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1981.
2. **(270)** Garner JS, Favero MS. Guideline for handwashing and hospital environmental control. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, 1985. Document No. 99-1117 (Also available at *Infect Control* 1986; 7: 231-43.)
3. **(27)** CDC. Guidelines for prevention of nosocomial pneumonia. *MMWR* 1997;46(No. RR-1):1-79.
4. **(34)** CDC. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities. *MMWR* 1994;43(No. RR-13):1-132.
5. **(318)** CDC. Recommendations for preventing the spread of vancomycin resistance. Recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR* 1995;44(No. RR-12):1-13.
6. **(36)** Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53-80.
7. **(114)** Mangram AJ, Horan TC, Pearson ML, Silver LC, Jarvis WR, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of surgical site infection, 1999. *Infect Control Hosp Epidemiol* 1999;20:247-80.
8. **(396)** CDC. USPHS/IDSA guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus. *MMWR* 1999;48(No. RR-10):1-66.
9. **(37, Appendix; 5)** CDC. CDC/IDSA/ASBMT guidelines for the prevention of opportunistic infections in hematopoietic stem cell transplant recipients. *MMWR* 2000;49(No. RR-10):1-128.
10. Garner JS. The CDC Hospital Infection Control Practice Advisory Committee. *Am J Infect Control* 1993;21:160-2.
11. Bennett JV, Brachman PS, eds. The inanimate environment. In: Rhame FS. *Hospital Infections*, 4th ed. Philadelphia, PA: Lippincott-Raven, 1998;299-324.
12. Weber DJ, Rutala WA. Environmental issues and nosocomial infections. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*, 3rd ed. Baltimore, MD: Williams & Wilkins, 1997;491-514.
13. Greene VW. Microbiological contamination control in hospitals. *Hospitals JAHA* 1969;43:78-88.
14. American Hospital Association. *Hospital statistics, 2000 ed.* Historical trends in utilization, personnel, and finances for selected years from 1946 through 1998 [Table]. Chicago, IL: Health Forum LLC, 2000;2-3.
15. McKee B. Neither bust nor boom. *Architecture* 1998. Available at: www.britannica.com/bcom/magazine/article/0,5744,39579,00.html
16. Crowell CL. Better, not bigger: construction costs soar on wings of patient demand, construction and design survey finds. *Mod Healthc* 1999;29:23-6, 28-34, 36-8.
17. **(9)** Sarubbi FA Jr, Kopf BB, Wilson NO, McGinnis MR, Rutala WA. Increased recovery of *Aspergillus flavus* from respiratory specimens during hospital construction. *Am Rev Respir Dis* 1982;125:33-8.
18. **(2)** Arnow PM, Sadigh MC, Weil D, Chudy R. Endemic and epidemic aspergillosis associated with inhospital replication of *Aspergillus* organisms. *J Infect Dis* 1991;164:998-1002.

19. (38) Flynn PM, Williams BG, Hethrington SV, Williams BF, Giannini MA, Pearson TA. *Aspergillus terreus* during hospital renovation [letter]. *Infect Control Hosp Epidemiol* 1993;14:363–5.
20. (48) Weems JJ Jr, Davis BJ, Tablan OC, Kaufman L, Martone WJ. Construction activity: an independent risk factor for invasive aspergillosis and zygomycosis in patients with hematologic malignancy. *Infect Control* 1987;8:71–5.
21. (10) Streifel AJ, Stevens PP, Rhame FS. In-hospital source of airborne *Penicillium* species spores. *J Clin Microbiol* 1987;25:1–4.
22. Noskin GA, Stosor V, Cooper J, Peterson L. Recovery of vancomycin-resistant enterococci on fingertips and environmental surfaces. *Infect Control Hosp Epidemiol* 1995;16:577–81.
23. Manian FA, Meyer L, Jenne J. *Clostridium difficile* contamination of blood pressure cuffs: a call for a closer look at gloving practices in the era of universal precautions. *Infect Control Hosp Epidemiol* 1996;17:180–2.
24. McFarland LV, Mulligan NE, Kwok RYY, et al. Nosocomial acquisition of *Clostridium difficile* infection. *N Engl J Med* 1989;320:204–10.
25. Nath SK, Thomely JH, Kelly M, et al. A sustained outbreak of *Clostridium difficile* in a general hospital: persistence of a toxigenic clone in four units. *Infect Control Hosp Epidemiol* 1994;15:382–9.
26. (165) Johnson JT, Yu VL, Best MG, et al. Nosocomial legionellosis in surgical patients with head and neck cancer: implications for epidemiological reservoir and mode of transmission. *Lancet* 1985;2:298–300.
27. Blatt SP, Parkinson MD, Pace E, et al. Nosocomial Legionnaires' disease: aspiration as a primary mode of disease acquisition. *Am J Med* 1993;95:16–22.
28. Bert F, Maubec E, Bruneau B, Berry P, Lambert-Zechovsky N. Multi-resistant *Pseudomonas aeruginosa* associated with contaminated tap-water in a neurosurgery intensive care unit. *J Hosp Infect* 1998;39:53–62.
29. Muylldermans G, de Smet F, Perrard D, et al. Neonatal infections with *Pseudomonas aeruginosa* associated with a water-bath used to thaw fresh frozen plasma. *J Hosp Infect* 1998;39:309–14.
30. Buttery JP, Alabaster SJ, Heine FG, et al. Multi-resistant *Pseudomonas aeruginosa* outbreak in a pediatric oncology ward related to bath toys. *Pediatr Infect Dis J* 1998;17:509–13.
31. (224) Bolan G, Reingold AL, Carson LA, et al. Infections with *Mycobacterium chelonae* in patients receiving dialysis and using processed hemodialyzers. *J Infect Dis* 1985;152:1013–9.
32. (225) Lowry PW, Beck-Sague CM, Bland LA, et al. *Mycobacterium chelonae* infection among patients receiving high-flux dialysis in a hemodialysis clinic in California. *J Infect Dis* 1990;161:85–90.
33. Schaal KP. Medical and microbiological problems arising from airborne infection in hospitals. *J Hosp Infect* 1991;18 (Suppl A):451–9.
34. Osterholm MT, Hedberg CW, Moore KA. Epidemiology of infectious diseases. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;156–67.
35. (3) Streifel AJ. Design and maintenance of hospital ventilation systems and prevention of airborne nosocomial infections. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999:1211–21.
36. Bodey GP, Vartivarian S. Aspergillosis. *Eur J Clin Microbiol Infect Dis* 1989;8:413–37.
37. Latgé JP. *Aspergillus fumigatus* and aspergillosis. *Clin Microbiol Rev* 1999;12:310–50.
38. Derouin F. Special issue on aspergillosis. *Pathol Biol* 1990;42:625–736.
39. Dixon DD, Walsh TJ. Human pathogenesis. In: Bennett JW, Klich MA, eds. *Aspergillus*, biology and industrial application. Boston, MA: Butterworth-Heinemann, 1992;249–67.
40. Kurup VP, Kumar A. Immunodiagnosis of aspergillosis. *Clin Microbiol Rev* 1991;4:439–56.
41. Latgé JP, Paris S, Sarfati J, et al. Infectivity of *Aspergillus fumigatus*. In: Vanden Bossche H, Stevens DA, Odds FC, eds. *Host-Fungus Interplay*. Bethesda, MD: National Foundation for Infectious Diseases, 1997;99–110.
42. Schaffner A. Host defense in aspergillosis. In: Bennett E, Hay RJ, Peterson PK, eds. *New Strategies in Fungal Disease*. Edinburgh, United Kingdom: Churchill Livingstone, 1992;98–112.
43. Vanden Bossche H, Mackenzie DWR, Cauwenbergh G, eds. *Aspergillus* and aspergillosis. New York, NY: Plenum Press, 1988.
44. (82) Sherertz RJ, Belani A, Kramer BS, et al. Impact of air filtration on nosocomial *Aspergillus* infections: unique risk of bone marrow transplant recipients. *Am J Med* 1987;83:709–18.
45. Young RC, Bennett JE, Vogel CL, Carbone PP, DeVita VT. Aspergillosis: the spectrum of the disease in 98 patients. *Medicine* 1970;49:147–73.
46. Rhame FS. Lessons from the Roswell Park bone marrow transplant aspergillosis outbreak. *Infect Control* 1985;6:345–6.

47. Rotsein C, Cummings KM, Tidings J, et al. An outbreak of invasive aspergillosis among allogeneic bone marrow transplants: a case-control study. *Infect Control* 1985;6:347-55.
48. (83) Aisner J, Schimpff SC, Bennett JE, Young VM, Wiernik PH. Aspergillus infections in cancer patients: association with fireproofing materials in a new hospital. *JAMA* 1976;235:411-2.
49. (64) Arnow PM, Anderson RL, Mainous PD, Smith EJ. Pulmonary aspergillosis during hospital renovation. *Am Rev Respir Dis* 1978;118:49-53.
50. (61) Streifel AJ, Lauer JL, Vesley D, Juni B, Rhame FS. *Aspergillus fumigatus* and other thermotolerant fungi generated by hospital building demolition. *Appl Environ Microbiol* 1983;46:375-8.
51. Hopkins CC, Weber DJ, Rubin RH. Invasive aspergillosis infection: possible non-ward common source within the hospital environment. *J Hosp Infect* 1989;13:19-25.
52. Denning DW. Invasive aspergillosis. *Clin Infect Dis* 1998;26:781-805.
53. Manuel RJ, Kibbler CC. The epidemiology and prevention of invasive aspergillosis. *J Hosp Infect* 1998;39:95-109.
54. Kennedy HF, Simpson EM, Wilson N, Richardson MD, Michie JR. *Aspergillus flavus* endocarditis in a child with neuroblastoma. *J Infect* 1998;36:126-7.
55. (75) McCarty JM, Flam MS, Pullen G, Jones R, Kassel SH. Outbreak of primary cutaneous aspergillosis related to intravenous arm boards. *J Pediatr* 1986;108(Pt.1):721-4.
56. Goldberg B, Eversmann WW, Eitzen EM Jr. Invasive aspergillosis of the hand. *J Hand Surg* 1982;7:38-42.
57. Grossman ME, Fithian EC, Behrens C, Bissinger J, Fracaro M, Neu HC. Primary cutaneous aspergillosis in six leukemic children. *J Am Acad Dermatol* 1985;12:313-8.
58. Panke TW, McManus AT, Spebar MJ. Infection of a burn wound by *Aspergillus niger*: gross appearance simulating ecthyma gangrenosa. *Am J Clin Pathol* 1979;72:230-2.
59. Fraser DW, Ward JL, Ajello L, Plikaytis BD. Aspergillosis and other systemic mycoses: the growing problem. *JAMA* 1979;242:1631-5.
60. Iwen PC, Reed EC, Armitage JO, et al. Nosocomial invasive aspergillosis in lymphoma patients treated with bone marrow or peripheral stem cell transplants. *Infect Control Hosp Epidemiol* 1993;14:131-9.
61. Cordonnier C, Bernaudin JF, Bierling P, Huet Y, Vernant JP. Pulmonary complications occurring after allogeneic bone marrow transplantation: a study of 130 consecutively transplanted patients. *Cancer* 1986;58:1047-54.
62. (76) Klimowski LL, Rotstein C, Cummings KM. Incidence of nosocomial aspergillosis in patients with leukemia over a twenty-year period. *Infect Control Hosp Epidemiol* 1989;10:299-305.
63. (79) Walmsley S, Devi S, King S, Schneider R, Richardson S, Ford-Jones L. Invasive *Aspergillus* infections in a pediatric hospital: a ten-year review. *Pediatr Infect Dis* 1993;12:673-82.
64. (57) Pannuti CS, Gingrich RD, Pfaller MA, Wenzel RP. Nosocomial pneumonia in adult patients undergoing bone marrow transplantation: a 9-year study. *J Clin Oncol* 1991;9:77-84.
65. (58) Wingard JR, Beals SU, Santos GW, Mertz WG, Saral R. *Aspergillus* infections in bone marrow transplant recipients. *Bone Marrow Transplant* 1987;2:175-81.
66. Humphreys H, Johnson EM, Warnock DW, Willatts SM, Winter RJ, Speller DCE. An outbreak of aspergillosis in a general ITU. *J Hosp Infect* 1991;18:167-77.
67. Sessa A, Meroni M, Battini G, et al. Nosocomial outbreak of *Aspergillus fumigatus* infection among patients in a renal unit? *Nephrol Dial Transplant* 1996;11:1322-4.
68. (134) Anderson K, Morris G, Kennedy H, et al. Aspergillosis in immunocompromised pediatric patients: associations with building hygiene, design, and indoor air. *Thorax* 1996;51:256-61.
69. (39) Tabbara KF, al Jabarti A. Hospital construction-associated outbreak of ocular aspergillosis after cataract surgery. *Ophthalmology* 1998;105:522-26.
70. Ferre A, Domingo P, Alonso C, Franquet T, Gurgui M, Verger G. Invasive pulmonary aspergillosis: A study of 33 cases. *Med Clin (Barc)* 1998;110:421-5. (Spanish)
71. Ewig S, Paar WD, Pakos E et al. Nosocomial ventilator-associated pneumonias caused by *Aspergillus fumigatus* in non-immunosuppressed, non-neutropenic patients. *Pneumologie* 1998;52:85-90. (German)
72. Singer S, Singer D, Ruchel R, Mergeryan H, Schmidt U, Harms K. Outbreak of systemic aspergillosis in a neonatal intensive care unit. *Mycoses* 1998;41:223-7.
73. (88) Allo MD, Miller J, Townsend T, Tan C. Primary cutaneous aspergillosis associated with Hickman intravenous catheters. *N Engl J Med* 1987;317:1105-8.
74. Boon AP, Adams DH, Buckels J, McMaster P. Cerebral aspergillosis in liver transplantation. *J Clin Pathol* 1990;43:114-8.

75. Pla MP, Berenguer J, Arzuaga JA, Banares R, Polo JR, Bouza E. Surgical wound infection by *Aspergillus fumigatus* in liver transplant recipients. *Diagn Microbiol Infect Dis* 1992;15:703–6.
76. Kanj SS, Welty-Wolf K, Madden J, et al. Fungal infections in lung and heart-lung transplant recipients: report of 9 cases and review of the literature. *Medicine* 1996;75:142–56.
77. (77) Pfundstein J. *Aspergillus* infection among solid organ transplant recipients: a case study. *J Transpl Coord* 1997;7:187–9.
78. Brenier-Pinchart MP, Lebeau B, Devouassoux G, et al. *Aspergillus* and lung transplant recipients: a mycologic and molecular epidemiologic study. *J Heart Lung Transplant* 1998;17:972–9.
79. (59) Gerson SL, Talbot GH, Hurwitz S, Strom B, Lusk EJ, Cassileth PA. Prolonged granulocytopenia: the major risk factor for invasive pulmonary aspergillosis in patients with leukemia. *Ann Intern Med* 1984;100:345–51.
80. Weber SF, Peacock JE Jr, Do KA, Cruz JM, Powell BL, Capizzi RL. Interaction of granulocytopenia and construction activity as risk factors for nosocomial invasive filamentous fungal disease in patients with hematologic disorders. *Infect Control Hosp Epidemiol* 1990;11:235–42.
81. Rees JR, Pinner RW, Hajjeh RA, Brandt ME, Reingold AL. The epidemiological features of invasive mycotic infections in the San Francisco Bay area, 1992–1993: results of population-based laboratory active surveillance. *Clin Infect Dis* 1998;27:1138–47.
82. McNeil MM, Nash SL, Hajjeh RA, Conn LA, Plikaytis BD. Trends in mortality due to invasive mycoses in the United States [abstract]. In: Program & Abstracts of the International Conference on Emerging Infectious Diseases. Atlanta, GA, 1998. Abstract No. S7.3.
83. Wald A, Leisenring W, van Burik JA, Bowden RA. Epidemiology of *Aspergillus* infections in a large cohort of patients undergoing bone marrow transplantation. *J Infect Dis* 1997;175:1459–66.
84. Gurwith MJ, Stinson EB, Remington JS. *Aspergillus* infection complicating cardiac transplantation: Report of five cases. *Arch Intern Med* 1971;128:541–5.
85. Weiland D, Ferguson RM, Peterson PK, Snover DC, Simmons RL, Najarian JS. Aspergillosis in 25 renal transplant patients. *Ann Surg* 1983;198:622–9.
86. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987;106:209–16.
87. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988;296:1–10.
88. Gustafson TL, Schaffner W, Lavelly GB, Stratton CW, Johnson HK, Hutcheson RH. Invasive aspergillosis in renal transplant recipients: correlation with corticosteroid therapy. *J Infect Dis* 1983;148:230–8.
89. Denning DW, Stevens DA. Antifungal and surgical treatment of invasive aspergillosis: review of 2121 published cases. *Rev Infect Dis* 1990;12:1147–201.
90. Weinberger M, Elattaar I, Marshall D, et al. Patterns of infection in patients with aplastic anemia and the emergence of *Aspergillus* as a major cause of death. *Medicine* 1992;71:24–43.
91. Noble WC, Clayton YM. Fungi in the air of hospital wards. *J Gen Microbiol* 1963;32:397–402.
92. Solomon WR, Burge HP, Boise JR. Airborne *Aspergillus fumigatus* levels outside and within a large clinical center. *J Allergy Clin Immunol* 1978;62:56–60.
93. Streifel AJ, Rhame FS. Hospital air filamentous fungal spore and particle counts in a specially designed hospital. In: *Indoor Air '93: Proceedings of the Sixth International Conference on Indoor Air and Climate*, Vol. 4. Helsinki, Finland:161–5.
94. (40) Rhame FS, Streifel AJ, Kersey JH Jr, McGlave PB. Extrinsic risk factors for pneumonia in the patient at high risk for infection. *Am J Med* 1984;76(5A):42–52.
95. (78) Rhame FS, Streifel A, Stevens P, et al. Endemic *Aspergillus* airborne spore levels are a major risk factor for aspergillosis in bone marrow transplant patients [abstract]. In: Abstracts of the 25th Interscience Conference on Antimicrobial Agents and Chemotherapy 1985. Abstract No. 147.
96. (60) Lentino JR, Rosenkranz MA, Michaels JA, Kurup VP, Rose HD, Rytel MW. Nosocomial aspergillosis: a retrospective review of airborne disease secondary to road construction and contaminated air conditioners. *Am J Epidemiol* 1982;116:430–7.
97. (49) Krasinski K, Holzman RS, Hanna B, Greco MA, Graff M, Bhogal M. Nosocomial fungal infection during hospital renovation. *Infect Control* 1985;6:278–82.
98. (29) Gage AA, Dean DC, Schimert G, Minsley N. *Aspergillus* infection after cardiac surgery. *Arch Surg* 1970;101:384–87.

99. **(95)** Siegler L, Kennedy MJ. *Aspergillus*, *Fusarium*, and other opportunistic moniliaceous fungi. In: Murray PR, Baron EJ, Pfaller MA, Tenoer FC, Tenover FC, Tenover FC, eds. Manual of clinical microbiology, 7th ed. Washington, DC: American Society for Microbiology Press, 1999;1212–41.
100. **(70)** Overberger PA, Wadowsky RM, Schaper MM. Evaluation of airborne particulates and fungi during hospital renovation. *Am Ind Hyg Assoc J* 1995;56:706–12.
101. **(96)** Breton P, Germaud P, Morin O, Audoin AF, Milpied N, Harousseau JL. Unusual pulmonary mycoses in patients with hematologic disease. *Rev Pneumol Clin* 1998;54:253–7. (French)
102. **(97)** Guarro J, Nucci M, Akiti T, Gené J, Barreiro MDGC, Gonçalves RT. Fungemia due to *Fusarium sacchari* in an immunosuppressed patient. *J Clin Microbiol* 2000;38:419–21.
103. **(98)** Burton JR, Zachery JB, Bessin R, et al. Aspergillosis in four renal transplant patients: diagnosis and effective treatment with amphotericin B. *Ann Intern Med* 1972;77:383–8.
104. **(80)** Kyriakides GK, Zinneman HHA, Hall WH, et al. Immunologic monitoring and aspergillosis in renal transplant patients. *Am J Surg* 1976;131:246–52.
105. Simmons RB, Price DL, Noble JA, Crow SA, Ahearn DG. Fungal colonization of air filters from hospitals. *Am Ind Hyg Assoc J* 1997;58:900–4.
106. **(4)** Pittet D, Huguenin T, Dharan S, et al. Unusual case of lethal pulmonary aspergillosis in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1996;154(2 Pt 1):541–4.
107. **(104)** Mahoney DH Jr, Steuber CP, Starling KA, Barrett FF, Goldberg J, Fernbach DJ. An outbreak of aspergillosis in children with acute leukemia. *J Pediatr* 1979;95:70–2.
108. Ruutu P, Valtonen V, Tiitonen L, et al. An outbreak of invasive aspergillosis in a hematologic unit. *Scand J Infect Dis* 1987;19:347–51.
109. **(51)** Walsh TJ, Dixon DM. Nosocomial aspergillosis: environmental microbiology, hospital epidemiology, diagnosis, and treatment. *Eur J Epidemiol* 1989;5:131–42.
110. Buffington J, Reporter R, Lasker BA, et al. Investigation of an epidemic of invasive aspergillosis: utility of molecular typing with the use of random amplified polymorphic DNA probes. *Pediatr Infect Dis J* 1994;13:386–93.
111. **(44)** Gerson SL, Parker P, Jacobs MR, Creger R, Lazarus HM. Aspergillosis due to carpet contamination [letter]. *Infect Control Hosp Epidemiol* 1994;15:221–3.
112. **(84)** Fox BC, Chamberlin L, Kulich P, Rae EJ, Webster LR. Heavy contamination of operating room air by *Penicillium* species: identification of the source and attempts at decontamination. *Am J Infect Control* 1990;18:300–6.
113. Chazalet V, Debeauvais J-P, Sarfati J, et al. Molecular typing of environmental and patient isolates of *Aspergillus fumigatus* from various hospital settings. *J Clin Microbiol* 1998;36:1494–500.
114. Loudon KW, Coke AP, Burnie JP, Shaw AJ, Oppenheim BA, Morris CQ. Kitchens as a source of *Aspergillus niger* infection. *J Hosp Infect* 1996;32:191–8.
115. **(81)** Abzug MJ, Gardner S, Glode MP, Cymanski M, Roe MH, Odom LF. Heliport-associated nosocomial mucormycoses [letter]. *Infect Control Hosp Epidemiol* 1992;13:325–6.
116. Alvarez M, Lopez Ponga B, Rayon C, et al. Nosocomial outbreak caused by *Scedosporium prolificans* (*inflatum*): four fatal cases in leukemic patients. *J Clin Microbiol* 1995;33:3290–5.
117. **(89)** Schleupner CJ, Hamilton JR. A pseudoepidemic of pulmonary fungal infections related to fiberoptic bronchoscopy. *Infect Control* 1980;1:38–42.
118. Jackson L, Klotz SA, Normand RE. A pseudoepidemic of *Sporothrix cyanescens* pneumonia occurring during renovation of a bronchoscopy suite. *J Med Vet Mycol* 1990;28:455–9.
119. **(30)** Vargo JA, Ginsberg MM, Mizrahi M. Human infestation by the pigeon mite: a case report. *Am J Infect Control* 1983;11:24–5.
120. **(1)** American Institute of Architects. Guidelines for design and construction of hospital and health care facilities, 2001. Washington, DC: American Institute of Architects Press, 2001.
121. Diamond RD. *Cryptococcus neoformans*. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of Infectious Diseases, 5th Ed. Philadelphia, PA: Churchill Livingstone, 2000;2707–18.
122. Deepe GS Jr. *Histoplasma capsulatum*. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of Infectious Diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2718–33.
123. Brodsky AL, Gregg MB, Loewenstein MS, et al. Outbreak of histoplasmosis associated with the 1970 earth day activities. *Am J Med* 1973;54:333–42.
124. Ward JI, Weeks M, Allen D, et al. Acute histoplasmosis: clinical, epidemiologic, and serologic findings of an outbreak associated with exposure to a fallen tree. *Am J Med* 1979;66:587–95.

125. Galgiani JN. Coccidioidomycoses. In: Remington JS, Swartz MN, eds. Current clinical topics in infectious disease. Malden, MA: Blackwell Science, 1997;188–204.
126. **(111)** Gerberding JL. Nosocomial transmission of opportunistic infections. *Infect Control Hosp Epidemiol* 1998;19:574–7.
127. Hughes WT. Natural mode of acquisition for de novo infection with *Pneumocystis carinii*. *J Infect Dis* 1982;145:842–8.
128. Olsson M, Sukura A, Lindberg LA, et al. Detections of *Pneumocystis carinii* DNA by filtration of air. *Scand J Infect Dis* 1996;28:279–82.
129. Bartlett MS, Vermund SH, Jacobs R, et al. Detection of *Pneumocystis carinii* DNA in air samples: likely environmental risk to susceptible persons. *J Clin Microbiol* 1997;35:2511–3.
130. Lundgren B, Elvin K, Rothman LP, Ljungstrom I, Lidman C, Lundgren JD. Transmission of *Pneumocystis carinii* from patients to hospital staff. *Thorax* 1997;52:422–4.
131. **(112)** Vargas SL, Ponce CA, Gigliotti F, et al. Transmission of *Pneumocystis carinii* DNA from a patient with *P. carinii* pneumonia to immunocompetent contact health care workers. *J Clin Microbiol* 2000;38:1536–8.
132. **(113)** Walzer PD. *Pneumocystis carinii*. In: Mandell GL, Bennett JE, Dolin R. eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2781–95.
133. CDC. Screening for tuberculosis and tuberculosis infection in high-risk populations: recommendations of the Advisory Committee for Elimination of Tuberculosis. *MMWR* 1995;44(No. RR-11):18–34.
134. CDC. Targeted tuberculin testing and treatment of latent tuberculosis infection. *MMWR* 2000;49(No. RR-6):1–51.
135. D'Agata EMC, Wise S, Stewart A, Lefkowitz LB Jr. Nosocomial transmission of *Mycobacterium tuberculosis* from an extrapulmonary site. *Infect Control Hosp Epidemiol* 2001;22:10–2.
136. CDC. Summary of notifiable diseases, United States 2001. *MMWR* 2001;50(53):1–108.
137. Haas DW. *Mycobacterium tuberculosis*. In: Mandell GL, Bennett JE, Dolin R. eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2576–607.
138. American Public Health Association. Tuberculosis. In: Chin J, ed. Control of communicable diseases manual, 17th ed. Washington, DC: American Public Health Association Press, 2000:521–30.
139. American Thoracic Society/CDC. Treatment of tuberculosis. *Am J Respir Crit Care Med* 2003;167:603–62.
140. Matlow AG, Jarrison A, Monteath A, Roach P, Balfe JW. Nosocomial transmission of tuberculosis (TB) associated with care of an infant with peritoneal TB. *Infect Control Hosp Epidemiol* 2000;21:222–3.
141. Jensen PA. Airborne *Mycobacterium* spp. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. Manual of environmental microbiology. Washington, DC: American Society for Microbiology Press, 1997;676–81.
142. **(41)** Wells WF. Aerodynamics of droplet nuclei. In: Airborne contagion and air hygiene. Cambridge, MA: Harvard University Press, 1955;13–9.
143. White A. Relation between quantitative nasal cultures and dissemination of staphylococci. *J Lab Clin Med* 1961;58:273–7.
144. Huijsmans-Evers AG. Results of routine tests for the detection of dispersers of *Staphylococcus aureus*. *Arch Chir Neerl* 1978;30:141–50.
145. Boyce JM, Opal SM, Potter-Bynoe G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a healthcare worker with chronic sinusitis. *Clin Infect Dis* 1993;17:496–504.
146. Hambraeus A, Sanderson HF. The control of ventilation of airborne bacterial transfer between hospital patients, and its assessment by means of a particle tracer. 3. Studies with an airborne-particle tracer in an isolation ward for burned patients. *J Hyg (Lond)* 1972;70:299–312.
147. Nakashima AK, Allen JR, Martone WJ, et al. Epidemic bullous impetigo in a nursery due to a nasal carrier of *Staphylococcus aureus*: role of epidemiology and control measures. *Infect Control* 1984;5:326–31.
148. Bethune DW, Blowers R, Parker M, Pask EA. Dispersal of *Staphylococcus aureus* by patients and surgical staff. *Lancet* 1965;1:480–3.
149. Sherertz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the *Staphylococcus aureus* — virus interaction revisited. *Ann Intern Med* 1996;124:539–47.
150. Gryska PF, O'Dea AE. Postoperative streptococcal wound infection: the anatomy of an epidemic. *JAMA* 1970;213:1189–91.
151. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A *Streptococcus* traced to a vaginal carrier. *J Infect Dis* 1978;138:287–92.

152. Berkelman RL, Martin D, Graham DR. Streptococcal wound infection caused by a vaginal carrier. *JAMA* 1982;247:2680–2.
153. McIntyre DM. An epidemic of *Streptococcus pyrogenes* puerpural and postoperative sepsis with an unusual site — the anus. *Am J Obstet Gynecol* 1968;101:308–14.
154. Gaynes RP, Horan TC. Surveillance of nosocomial infections. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1285–318.
155. Wenzel RP, Veazey JM Jr, Townsend TR. Role of the inanimate environment in hospital-acquired infections. In: Cundy KR, Ball W, eds. *Infection control in healthcare facilities: microbiological surveillance*. Baltimore, MD: University Park Press, 1977;71–98.
156. Mortimer EA Jr, Wolinsky E, Gonzaga AJ, Rammelkamp CH Jr. Role of airborne transmission in staphylococcal infections. *Br Med J* 1966;5483:319–22.
157. Youngs ER, Roberts C, Kramer JM, Gilbert RJ. Dissemination of *Bacillus cereus* in a maternity unit. *J Infect* 1985;10:228–32.
158. Richardson AJ, Rothburn MM, Roberts C. Pseudo-outbreak of *Bacillus* species: related to fiberoptic bronchoscopy. *J Hosp Infect* 1986;7:208–10.
159. Bryce EA, Smith JA, Tweeddale M, Andruschak BJ, Maxwell MR. Dissemination of *Bacillus cereus* in an intensive care unit. *Infect Control Hosp Epidemiol* 1993;14:459–62.
160. Lie PY-F, Ke S-C, Chen S-L. Use of pulsed-field gel electrophoresis to investigate a pseudo-outbreak of *Bacillus cereus* in a pediatric unit. *J Clin Microbiol* 1997;35:1533–5.
161. McDonald LC, Walker M, Carson L, et al. Outbreak of *Acinetobacter* spp. bloodstream infections in a nursery associated with contaminated aerosols and air conditioners. *Pediatr Infect Dis J* 1998;17:716–22.
162. Leclair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980;302:450–3.
163. Gustafson TL, Lavelly GB, Brawner ERJ, Hutcheson RHJ, Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982;70:550–6.
164. Josephson A, Gombert ME. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988;158:238–41.
165. Sawyer MJ, Chamberlin CJ, Wu YN, Aintablian N, Wallace MR. Detection of varicella-zoster virus DNA in air samples from hospital rooms. *J Infect Dis* 1994;169:91–4.
166. Menkhous NA, Lamphear B, Linnemann CC. Airborne transmission of varicella-zoster virus in hospitals. *Lancet* 1990;226:1315.
167. CDC. Prevention of varicella: recommendations of the advisory committee on immunization practices (ACIP). *MMWR* 1996;45(No. RR-11):1–36.
168. Davis RM, Orenstein WA, Frank JAJ, et al. Transmission of measles in medical settings: 1980 through 1984. *JAMA* 1986;255:1295–8.
169. Watkins NM, Smith RPJ, St Germain DL, Mackay DN. Measles infection in a hospital setting. *Am J Infect Control* 1987;15:201–6.
170. Revera ME, Mason WH, Ross LA, Wright HT Jr. Nosocomial measles infection in a pediatric hospital during a community-wide epidemic. *J Pediatr* 1991;119:183–6.
171. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings — United States, 1985–1989. *Am J Med* 1991;91(suppl):320S–4S.
172. Patriarca PA, Weber JA, Parker RA, et al. Efficacy of influenza vaccine in nursing homes: reduction in illness and complications during influenza A (H3N2) epidemics. *JAMA* 1985;253:1136–9.
173. Arden NH, Patriarca PA, Fasano MB, et al. The roles of vaccination and amantadine prophylaxis in controlling an outbreak of influenza A (H3N2) in a nursing home. *Arch Intern Med* 1988;148:865–8.
174. CDC. Influenza A outbreaks — Louisiana, August 1993. *MMWR* 1993;42:132–4.
175. Drinka PJ, Gravenstein S, Krause P, Schilling M, Miller BA, Shult P. Outbreaks of influenza A and B in a highly immunized nursing home population. *J Family Practice* 1997;45:509–14.
176. Schilling M, Povinelli L, Krause P, et al. Efficacy of zanamivir for chemoprophylaxis of nursing home influenza outbreaks. *Vaccine* 1998;16:1771–4.
177. Hall CB. Nosocomial viral infections: perennial weeds on pediatric wards. *Am J Med* 1981;70:670–6.
178. Whimbey E, Elting LS, Couch RB, et al. Influenza A virus infections among hospitalized adult bone marrow transplant recipients. *Bone Marrow Transpl* 1994;13:437–40.
179. Evans ME, Hall KL, Berry SE. Influenza control in acute care hospitals. *Am J Infect Control* 1997;25:357–62.

180. Munoz FM, Campbell JR, Atmar RL, et al. Influenza A virus outbreak in a neonatal intensive care unit. *Pediatr Infect Dis J* 1999;18:811–5.
181. Alford RH, Kasel JA, Gerone PJ, Knight V. Human influenza resulting from aerosol inhalation. *Proc Soc Exp Biol Med* 1966;122:800–4.
182. Moser MR, Bender TR, Margolis HS, Noble GR, Kendal AP, Ritter DG. An outbreak of influenza aboard a commercial airliner. *Am J Epidemiol* 1979;110:1–6.
183. Chanock RW, Kim HW, Vargosko AJ, et al. Respiratory syncytial virus 1: virus recovery and other observations during 1960 — outbreak of bronchiolitis, pneumonia, and other minor respiratory illness in children. *JAMA* 1961;176:647–53.
184. Gardner DS, Court SDM, Brocklebank JT, et al. Virus cross-infection in paediatric wards. *Br Med J* 1973;2:571–75.
185. Sawyer LA, Murphy JJ, Kaplan JE, et al. 25–30 nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988;127:1261–71.
186. Baxby D. Poxviruses. In: Belshe RB, ed. *Textbook of human virology*, 2nd ed. St. Louis, MO: Mosby Year Book, 1991;930–46.
187. Neff JM. Variola (smallpox) and monkeypox viruses. In: Mandell GL, Bennett JE, Dolin R. eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;1555–6.
188. Wehrle PF, Posch J, Richter KH, Henderson DA. An airborne outbreak of smallpox in a German hospital and its significance with respect to other recent outbreaks in Europe. *Bull WHO* 1970;43:669–79.
189. Hawkes N. Science in Europe: smallpox death in Britain challenges presumption of laboratory safety. *Science* 1979;203:855–6.
190. Eickhoff TC. Airborne nosocomial infection: a contemporary perspective. *Infect Control Hosp Epidemiol* 1994;15:663–72.
191. Nuzum EO, Rossi CA, Stephenson EH, LeDuc JW. Aerosol transmission of Hantaan and related viruses to laboratory rats. *Am J Trop Med Hyg* 1988;38:636–40.
192. CDC. Hantavirus infection — southwestern United States: Interim recommendations for risk reduction. *CDC. MMWR* 1993;42(No. RR-11):1–13.
193. Vitek CR, Breiman RF, Ksiazek TG, et al. Evidence against person-to-person transmission of hantavirus to health care workers. *Clin Infect Dis* 1996;22:824–6.
194. Wells RM, Young J, Williams RJ, et al. Hantavirus transmission in the United States. *Emerg Infect Dis* 1997;3:361–5.
195. Chaparro J, Vega J, Terry W, et al. Assessment of person-to-person transmission of hantavirus pulmonary syndrome in a Chilean hospital setting. *J Hosp Infect* 1998;40:281–5.
196. Nolte KB, Foucar K, Richmond JY. Hantaviral biosafety issues in the autopsy room and laboratory: Concerns and recommendations. *Hum Pathol* 1996;27:1253–4.
197. Stephenson EH, Larson EW, Dominik JW. Effect of environmental factors on aerosol-induced Lassa virus infection. *J Med Virol* 1984;14:295–303.
198. Monath TP. Lassa fever: review of epidemiology and epizootiology. *Bull World Health Organ* 1975;52:577–92.
199. Monath TP, Casals J. Diagnosis of Lassa fever and the isolation and management of patients. *Bull WHO* 1975;52:707–15.
200. Zweighaft RM, Fraser DW, Hattwick MA, et al. Lassa fever: response to an imported case. *N Engl J Med* 1977;297:803–7.
201. Cooper CB, Gransden WR, Webster M, et al. A case of Lassa fever: experience at St Thomas' hospital. *Br Med J (Clin Res Ed)* 1982;285:1003–5.
202. **(108)** Monath TP. Yellow fever: Victor, victoria? Conqueror, conquest? Epidemics and research in the last forty years and prospects for the future. *Am J Trop Med Hyg* 1991;45:1–43.
203. **(109)** CDC. Update: management of patients with suspected viral hemorrhagic fever — United States. *MMWR* 1995;44:475–9.
204. **(110)** Weber DJ, Rutala WA. Risks and prevention of nosocomial transmission rare zoonotic diseases. *Clin Infect Dis* 2001;32:446–56.
205. Decker MD, Schaffner W. Nosocomial diseases of healthcare workers spread by the airborne or contact routes (other than tuberculosis). In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1101–26.

206. **(46)** Fridkin SK, Kremer FB, Bland LA, Padhye A, McNeil MM, Jarvis WR. *Acremonium kiliense* endophthalmitis that occurred after cataract extraction in an ambulatory surgical center and was traced to an environmental reservoir. *Clin Infect Dis* 1996;22:222–7.
207. Loeb M, Wilcox L, Thornley D, et al. *Bacillus* species pseudobacteremia following hospital construction. *Can J Infect Control* 1995;10:37–40.
208. Olle-Goig JE, Canela-Soler J. An outbreak of *Brucella melitensis* infection by airborne transmission among laboratory workers. *Am J Public Health* 1987;77:335–8.
209. Kiel FW, Khan MY. Brucellosis among hospital employees in Saudi Arabia. *Infect Control Hosp Epidemiol* 1993;14:268–72.
210. Staszkiwicz J, Lewis CM, Colville J, Zervos M, Band J. Outbreak of *Brucella melitensis* among microbiology laboratory workers in a community hospital. *J Clin Microbiol* 1991;20:287–90.
211. Fiori PL, Mastrandrea S, Rappelli P, Cappuccinelli P. *Brucella abortus* infection acquired in microbiology laboratories. *J Clin Microbiol* 2000;38:2005–6.
212. Spinelli JS, Ascher MS, Brooks DL, et al. Q fever crisis in San Francisco: controlling a sheep zoonosis in a lab animal facility. *Lab Anim* 1981;10:29–38.
213. **(Table 1)** American Conference of Governmental Industrial Hygienists (ACGIH). HVAC components, functions and malfunctions (topic 8-4). In: *Industrial ventilation: a Manual of recommended practice*, 24th ed. Cincinnati, OH : American Conference of Governmental Industrial Hygienists, Inc., 2001.
214. **(35)** American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. *Ventilation for Indoor Air Quality*. ASHRAE Standard 62-1999. Atlanta, GA: ASHRAE 1999;1–27.
215. **(133)** Burroughs HEB. Sick building syndrome: fact, fiction, or facility? In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Health Care Organizations, 1997;3–13.
216. American Society of Heating, Refrigerating and Air-conditioning Engineers, Inc. Gravimetric and dust spot procedures for testing air cleaning devices used in general ventilation for removing particulate matter. ANSI/ASHRAE Standard 52–1–1999. Atlanta, GA: ASHRAE 1999;1–25.
217. Robinson TJ, Ouellet AE. Filters and filtration. *ASHRAE J* 1999;65–70.
218. Dryden GE, Dryden SR, Brown DG, Schatzle KC, Godzeski C. Performance of bacteria filters. *Respir Care* 1980;25:1127–35.
219. **(33)** Rutala WA, Jones SM, Worthington JM, Reist PC, Weber DJ. Efficacy of portable filtration units in reducing aerosolized particles in the size range of *Mycobacterium tuberculosis*. *Infect Control Hosp Epidemiol* 1995;16:391–8.
220. **(5)** U.S. Environmental Protection Agency, Office of Air and Radiation and U.S. Department of Health & Human Services, National Institute of Occupational Safety and Health. *Building air quality: a guide for building owners and facilities managers*. Washington, DC: USEPA, 1991. EPA/400/1-91/033, or NIOSH 91-114. Available at: www.cdc.gov/niosh/baqtoc.html
221. **(28)** Ko G, Burge HA, Muileberg M, Rudnick S, First M. Survival of mycobacteria on HEPA filter material. *J Am Biol Safety Assoc* 1998;3:65–78.
222. **(6)** Rao CY, Burge HA, Chang JCS. Review of quantitative standards and guidelines for fungi in indoor air. *J Air & Waste Manage Assoc* 1996;46:899–906.
223. Riley RL, Wells WF, Mills CC, Nyka W, McLean RL. Air hygiene in tuberculosis: quantitative studies of infectivity and control in a pilot ward. *Am Rev Tuberc* 1957;75:420–31.
224. Riley RL, Nardell EA. Cleaning the air: the theory and application of UV air disinfection. *Am Rev Respir Dis* 1989;139:1286–94.
225. Riley RL. Ultraviolet air disinfection for control of respiratory contagion. In: Kundsinn RB, ed. *Architectural design and indoor microbial pollution*. New York, NY: Oxford University Press, 1988;174–97.
226. Willmon TL, Hollaender A, Langmuir AD. Studies of the control of acute respiratory diseases among naval recruits. I. A review of a four-year experience with ultraviolet irradiation and dust suppressive measures, 1943 to 1947. *Am J Hyg* 1948;48:227–32.
227. Wells WF, Wells MW, Wilder TS. The environmental control of epidemic contagion. I. An epidemiologic study of radiant disinfection of air in day schools. *Am J Hyg* 1942;35:97–121.
228. Perkins JE, Bahlke AM, Silverman HF. Effect of ultra-violet irradiation of classrooms on spread of measles in large rural central schools. *Am J Public Health Nations Health* 1947;37:529–37.
229. Nagy R. Application and measurement of ultraviolet radiation. *Am Ind Hyg Assoc J* 1964;25:274–81.
230. Illuminating Engineering Society. *IES Lighting handbook*, 4th ed. New York, NY: Illuminating Engineering Society, 1966;25–7.

231. Riley RL. Indoor spread of respiratory infection by recirculation of air. *Bull Physiopathol Respir* 1979;15:699–705.
232. Menzies D, Pasztor J, Rand T, Bourbeau J. Germicidal ultraviolet irradiation in air conditioning systems: effect on office worker health and wellbeing — a pilot study. *Occup Environ Med* 1999;56:397–402.
233. Riley RL, Permutt S. Room air disinfection by ultraviolet irradiation of upper air: air mixing and germicidal effectiveness. *Arch Environ Health* 1971;22:208–19.
234. Nicas M, Miller SL. A multi-zone model evaluation of the efficacy of upper-room air ultraviolet germicidal irradiation. *Appl Occup Environ Hyg* 1999;14:317–28.
235. Kethley TW, Branch K. Ultraviolet lamps for room air disinfection: effect of sampling location and particle size of bacterial aerosol. *Arch Environ Health* 1972;25:205–14.
236. Riley RL, Knight M, Middlebrook G. Ultraviolet susceptibility of BCG and virulent tubercle bacilli. *Am Rev Respir Dis* 1976;113:413–8.
237. Collins FM. Relative susceptibility of acid-fast and non-acid-fast bacteria to ultraviolet light. *Appl Microbiol* 1971;21:411–3.
238. Riley RL, Permutt S, Kaufman JE. Convection, air mixing, and ultraviolet air disinfection in rooms. *Arch Environ Health* 1971;22:200–7.
239. Nardell EA. Fans, filters, or rays? Pros and cons of the current environmental tuberculosis control technologies. *Infect Control Hosp Epidemiol* 1993;14:681–5.
240. ECRI. Health devices evaluation of mobile high efficiency filter air cleaners (MHEFACs). *ECRI* 1997;26:367–88.
241. **(103)** American Society of Heating, Refrigerating, and Air-Conditioning Engineers. 1999 ASHRAE Handbook: heating, ventilating, and air-conditioning applications. Chapter 7: Health care facilities. Atlanta, GA: ASHRAE, 1999;7.1–7.13.
242. Elovitz KM. Understanding what humidity does and why. *ASHRAE J* 1999;April:84–90.
243. Orme I. Patient impact. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997:43–52.
244. Gundermann KO. Spread of microorganisms by air-conditioning systems — especially in hospitals. *Ann NY Acad Sci* 1980;209–17.
245. Arundel AV, Sterling EM, Biggin JH, Sterling TD. Indirect health effects of relative humidity in indoor environments. *Environ Health Perspect* 1986;65:351–61.
246. U.S. Environmental Protection Agency. *Ventilation and air quality in offices*. Washington, DC: EPA Document #402-F-94-003, Revision: July 1990.
247. Hermans RD, Streifel AJ. Ventilation design. In: Bierbaum PJ, Lippman M, eds. *Workshop on engineering controls for preventing airborne infections in workers in health care and related facilities*. Cincinnati, OH: NIOSH and CDC, 1993;107–46.
248. Memarzadeh F, Jiang J. A methodology for minimizing risk from airborne organisms in hospital isolation rooms. *ASHRAE Trans* 2000;106:731–47.
249. **(11)** Hansen W. The need for an integrated indoor air quality program. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;xiii – xviii.
250. **(12)** Bartley J. Ventilation. In: Pfeiffer J, ed. *APIC Text of infection control and epidemiology*. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC), 2000;77.1–77.11.
251. Levine AS, Siegel SE, Schreiber AD, et al. Protected environments and prophylactic antibiotics. *N Engl J Med* 1973;288:477–483.
252. **(90)** Denning DW, Clemons KV, Hanson LH, Stevens DA. Restriction endonuclease analysis of total cellular DNA of *Aspergillus fumigatus* isolates of geographically and epidemiologically diverse origin. *J Infect Dis* 1990;162:1151–8.
253. Rhame FS. Prevention of nosocomial aspergillosis. *J Hosp Infect* 1991;18 (Suppl. A):466–72.
254. **(85)** Barnes RA, Rogers TR. Control of an outbreak of nosocomial aspergillosis by laminar air-flow isolation. *J Hosp Infect* 1989;14:89–94.
255. Roy M-C. The operating theater: a special environmental area. In: Wenzel RP, ed. *Prevention and control of nosocomial infections*, 3rd ed. Baltimore, MD: William & Wilkins, 1997;515–38.
256. Pavelchak N, DePersis RP, London M, et al. Identification of factors that disrupt negative air pressurization of respiratory isolation rooms. *Infect Control Hosp Epidemiol* 2000;21:191–5.

257. Anderson K. *Pseudomonas pyocyanea* disseminated from an air cooling apparatus. *Med J Austr* 1959;529–32.
258. Shaffer JG, McDade JJ. Airborne *Staphylococcus aureus*: a possible source in air control equipment. *Arch Environ Health* 1963;5:547–51.
259. Morey PR. Building-related illness with a focus on fungal issues. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;15–25.
260. Streifel AJ. Recognizing IAQ risk and implementing an IAQ program. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations; Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;75–91.
261. Morey PR. Appendix B. Fungal growth checklist. In: Hansen W. ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Health Care Organizations Publications, 1997;129–35.
262. Brock DL, Jiankang J, Rinaldi MG, Wickes BL, Huycke MM. Outbreak of invasive *Aspergillus* infection in surgical patients, associated with a contaminated air-handling system. *Clin Inf Dis* 2003;37:786–93.
263. (31) National Air Duct Cleaners Association. General specifications for the cleaning of commercial HVAC systems. Washington, DC: NADCA, 2002. Publication No. NAD-06. Available at: www.nadca.com/standards/standards.asp
264. (32) U.S. Environmental Protection Agency. Use of disinfectants and sanitizers in heating, ventilation, air conditioning, and refrigeration systems [letter]. March 14, 2002. Available at: www.epa.gov/oppad001/hvac.htm
265. U.S. Environmental Protection Agency. Should you have the air ducts in your home cleaned? Washington, DC: EPA, 1997. EPA Document No. 402-K-97-002.
266. (159) Vujanovic V, Smoragiewicz W, Krzysztyniak K. Airborne fungal ecological niche determination as one of the possibilities for indirect mycotoxin risk assessment in indoor air. *Environ Toxicol* 2001;16:1–8.
267. Soules WJ. Airflow management techniques. *Clean Rooms* 1993;2:18–20.
268. Lawson CN. Commissioning hospitals for compliance. *ASHRAE Trans* 1993;99(2).
269. Wadowsky R, Benner S. Distribution of the genus *Aspergillus* in hospital room air conditioners. *Infect Control* 1987;8:516–8.
270. Streifel AJ. Aspergillosis and construction. In: Kundsinn RB, ed. Architectural design and indoor microbial pollution. New York, NY: Oxford University Press, 1988;198–217.
271. Streifel AJ, Vesley D, Rhame FS. Occurrence of transient high levels of airborne fungal spores. Proceedings of the 6th Conference on Indoor Air Quality and Climate. Toronto, ON: 1990.
272. (73) Morey R, Williams C. Porous insulation in buildings: a potential source of microorganisms. Proceedings - Indoor Air '90, 5th International Conference. Toronto, ON: 1990;1–6.
273. (13) Bartley J. Construction and renovation. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc., 2000; 72.1–72.11.
274. (14) Harvey MA. Critical-care-unit design and furnishing: Impact on nosocomial infections. *Infect Control Hosp Epidemiol* 1998;19:597–601.
275. (15) National Association of Children's Hospitals and Related Institutions. Patient Care Focus Groups 1998. Assessing organizational readiness for infection control issues related to construction, renovation, and physical plant projects.
276. (50) Bartley JM. APIC State-of-the-art report: the role of infection control during construction in health care facilities. *Am J Infect Control* 2000;28:156–9.
277. (16) Carter CD, Barr BA. Infection control issues in construction and renovation. *Infect Control Hosp Epidemiol* 1997;18:587–96.
278. (47) Streifel AJ. Maintenance and engineering. In: Pfeiffer J, ed. APIC Text of Infection Control and Epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc., 2000;76.1-76.8.
279. Kennedy V, Barnard B, Hackett B. Use of a risk matrix to determine the level of barrier protection during construction activities. Atlanta, GA : Peachtree Publications, 1997;27–8.
280. Morey PR. Building-related illness with a focus on fungal issues. In: Hansen W, ed. A guide to managing indoor air quality in health care organizations. Oakbrook Terrace, IL: Joint Commission on Accreditation of Healthcare Organizations Publications, 1997;15–25.

281. (67) Bartley J, ed. Infection control tool kit series – construction and renovation. Washington, DC: Association for Professionals in Infection Control and Epidemiology, 1999.
282. Bryce EA, Walker M, Scharf S, et al. An outbreak of cutaneous aspergillosis in a tertiary-care hospital. *Infect Control Hosp Epidemiol* 1996;17:170–2.
283. (62) Thio CL, Smith D, Merz WG, et al. Refinements of environmental assessment during an outbreak investigation of invasive aspergillosis in a leukemia and bone marrow transplant unit. *Infect Control Hosp Epidemiol* 2000;21:18–23.
284. (65) Kuehn TH, Gacek B, Yang CH, et al. Final report: ASHRAE 804-RP Phase I identification of contaminants, exposures effects, and control options for construction/renovation activities. Atlanta, GA:ASHRAE, Inc. 1995.
285. Kennedy HF, Michie JR, Richardson MD. Air sampling for *Aspergillus* spp. during building activity in a paediatric hospital ward. *J Hosp Infect* 1995;31:322–25.
286. Leenders ACAP, van Belkum A, Behrendt M, Luijendijk AD, Verbrugh HA. Density and molecular epidemiology of *Aspergillus* in air and relationship to outbreaks of *Aspergillus* infection. *J Clin Microbiol* 1999;37:1752–7.
287. Rath PM, Ansorg R. Value of environmental sampling and molecular typing of aspergilli to assess nosocomial sources of aspergillosis. *J Hosp Infect* 1997;37:47–53.
288. (71) Streifel AJ, Marshall JW. Parameters for ventilation controlled environments in hospitals. In: Design, Construction, and Operation of Healthy Buildings. Atlanta, GA: ASHRAE Press, 1998.
289. (348) Streifel AJ. Air cultures for fungi. In: Gilchrist M, ed. Clinical microbiology procedures handbook. Washington, DC: American Society for Microbiology Press, 1992;11.8.1–11.8.7.
290. American Conference of Governmental Industrial Hygienists (ACGIH). 2000 Threshold limit Values and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists, Inc., 2000;1–184.
291. U.S. Department of Labor, Occupational Safety and Health Administration. Air contaminants standard. 29 CFR 1910.1000, §1910.1000, Tables Z-1, Z-3. *Federal Register* 1993;58:35338–51.
292. (86) Leenders A, vanBelkum A, Janssen S, et al. Molecular epidemiology of apparent outbreaks of invasive *Aspergillus* in a hematology ward. *J Clin Microbiol* 1996;34:345–51.
293. (91) James MJ, Lasker BA, McNeil MM, Shelton M, Warnock DW, Reiss E. Use of a repetitive DNA probe to type clinical and environmental isolates of *Aspergillus flavus* from a cluster of cutaneous infections in a neonatal intensive care unit. *J Clin Microbiol* 2000;38:3612–8.
294. (92) Skladny H, Buchheidt D, Baust C, et al. Specific detection of *Aspergillus* species in blood and bronchoalveolar lavage samples of immunocompromised patients by two-step PCR. *J Clin Microbiol* 1999;37:3865–71.
295. (93) Symoens F, Bouchara J-P, Heinemann S, Nolard N. Molecular typing of *Aspergillus terreus* isolates by random amplification of polymorphic DNA. *J Hosp Infect* 2000;44:273–80.
296. (94) Diaz-Guerra TM, Mellado E, Cuenca-Estrella M, Gaztelurrutia L, Villate Navarro JI, Rodríguez Tudela JL. Genetic similarity among one *Aspergillus flavus* strain isolated from a patient who underwent heart surgery and two environmental strains obtained from the operating room. *J Clin Microbiol* 2000;38:2419–22.
297. Buttner MP, Stetzenbach LD. Monitoring airborne fungal spores in an experimental indoor environment to evaluate sampling methods and the effects of human activity on air sampling. *Appl Environ Microbiol* 1993;59:219–26.
298. Sayer WJ, Shean DB, Ghosseiri J. Estimation of airborne fungal flora by the Anderson sampler versus the gravity settling culture plate. *J Allerg* 1969;44:214–27.
299. Hay RJ, Clayton YM, Goodley JM. Fungal aerobiology: how, when and where? *J Hosp Infect* 1995;30(Suppl):S352–7.
300. Morris G, Kokki MH, Anderson K, Richardson MD. Sampling of *Aspergillus* spores in air. *J Hosp Infect* 2000;44:81–92.
301. Iwen PC, Davis JC, Reed EC, Winfield BA, Hinrichs SH. Airborne fungal spore monitoring in a protective environment during hospital construction and correlation with an outbreak of invasive aspergillosis. *Infect Control Hosp Epidemiol* 1994;15:303–6.
302. Pegues DA, Lasker BA, McNeil MM, Hamm PM, Lundal JL, Kubak BM. Cluster of cases of invasive aspergillosis in a transplant intensive care unit: evidence of person-to-person airborne transmission. *Clin Infect Dis* 2002;34:412–6.

303. Goodley JM, Clayton YM, Hay RJ. Environmental sampling for aspergilli during building construction on a hospital site. *J Hosp Infect* 1994;26:27–35.
304. (72) American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE). The HVAC commissioning process. Atlanta, GA: ASHRAE, 1996;1–48. ASHRAE Guideline 1.
305. (63) Mermel LA, Josephson SL, Giorgio CH, Dempsey J, Parenteau S. Association of Legionnaires' disease with construction: contamination of potable water? *Infect Control Hosp Epidemiol* 1995;16:76–81.
306. Loo VG, Bertrand C, Dixon C, et al. Control of construction-associated nosocomial aspergillosis in an antiquated hematology unit. *Infect Control Hosp Epidemiol* 1996;17:360–4.
307. (68) Ottney TC. Particle management for HVAC systems. *ASHRAE J* 1993;35:26–28, 30, 32, 34.
308. Rautiala S, Reponen T, Nevalainen A, et al. Control of exposure to airborne viable microorganisms during remediation of moldy buildings: report of three case studies. *Am Ind Hyg Assoc J* 1998;59:455–60.
309. (69) Finkelstein LE, Mendelson MH. Infection control challenges during hospital renovation. *Am J Nursing* 1997;97:60–1.
310. Hruszkewycz V, Ruben B, Hypes CM, Bostic GD, Staszkiwicz J, Band JD. A cluster of pseudofungemia associated with hospital renovation adjacent to the microbiology laboratory. *Infect Control Hosp Epidemiol* 1992;13:147–50.
311. Laurel VL, Meier PA, Astorga A, Dolan D, Brockett R, Rinaldi MG. Pseudoepidemic of *Aspergillus niger* infections traced to specimen contamination in the microbiology laboratory. *J Clin Microbiol* 1999;37:1612–6.
312. (66) Opal SM, Asp AA, Cannady PB Jr, Morse PL, Burton LJ, Hammer II PG. Efficacy of infection control measures during a nosocomial outbreak of disseminated aspergillosis associated with hospital construction. *J Infect Dis* 1986;153: 634–7.
313. Fitzpatrick F, Prout S, Gilleece A, Fenelon LE, Murphy OM. Nosocomial aspergillosis during building work — a multidisciplinary approach. *J Hosp Infect* 1999;42:170–1.
314. Garrett DO, Jochimsen E, Jarvis W. Invasive *Aspergillus* spp. infections in rheumatology patients. *J Rheumatol* 1999;26:146–9.
315. Larsson L, Larsson PF. Analysis of chemical markers as a means of characterizing airborne microorganisms in indoor environments: a case study. *Indoor Built Environ* 2001;10:232–7.
316. (99) Buckner CD, Clift RA, Sanders JE, et al. Protective environment for marrow transplant recipients: a prospective study. *Ann Intern Med* 1978;89:893–901.
317. (100) Murray WA, Streifel AJ, O'Dea TJ, Rhame FS. Ventilation for protection of immune compromised patients. *ASHRAE Trans* 1988;94:1185–91.
318. (101) Streifel AJ, Vesley D, Rhame FS, Murray B. Control of airborne fungal spores in a university hospital. *Environment International* 1989;12:441–4.
319. Perry S, Penland WZ. The portable laminar flow isolator: new unit for patient protection in a germ-free environment. In: *Recent Results in Cancer Research*. New York, NY: Springer-Verlag, 1970.
320. Hayden CS, Fischbach, M, Johnston OE. A model for calculating air leakage in negative pressure isolation areas. Cincinnati, OH: DHHS, 1997. NIOSH Report ECTR 212-05c.
321. DeLuga GF. Differential airflow, pressure, have key relationship in pressurization. *Lab Design* 1997;2:6–7.
322. Rhame FS. Nosocomial aspergillosis: How much protection for which patients? *Infect Control Hosp Epidemiol* 1989;10:296–8.
323. Hofflin JM, Potasman I, Baldwin JC, Oyster PE, Stinson EB, Remington JS. Infectious complications in heart transplant recipients receiving cyclosporine and corticosteroids. *Ann Intern Med* 1987;106:209–16.
324. Schulman LL, Smith CR, Drusin R, Rose EA, Enson Y, Reemtsma K. Respiratory complications of cardiac transplantation. *Am J Med Sci* 1988;296:1–10.
325. Dummer JS, Ho M. Risk factors and approaches to infections in transplant recipients. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;3126–35.
326. Dummer JS, Ho M. Infections in solid organ transplant recipients. In: Mandell GL, Bennett JE, Dolin R, eds. *Principles and practice of infectious diseases*, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;3148–58.
327. Walsh TR, Guttendorf J, Dummer S, et al. The value of protective isolation procedures in cardiac transplant recipients. *Ann Thorac Surg* 1989;47:539–45.
328. Streifel AJ. Health-care IAQ: guidance for infection control. *HPAC Heating/Piping/Air Cond Eng* 2000; Oct:28–30, 33, 34, 36.

329. (87) Yeager CC. Copper and zinc preservatives. In: Block SS, ed. Disinfection, sterilization, and preservation, 4th ed. Philadelphia, PA: Lea & Febiger, 1991;358–61.
330. Cookson ST, Jarvis WR. Prevention of nosocomial transmission of *Mycobacterium tuberculosis*. Infect Dis Clin North Am 1997;11:367–409.
331. (42) CDC. Nosocomial transmission of multidrug-resistant tuberculosis among HIV-infected persons: Florida and New York, 1988–1991. MMWR 1991;40:585–91.
332. (43) CDC. Outbreak of multidrug-resistant tuberculosis at a hospital — New York City, 1991. MMWR 1993;42:427–34.
333. (7) Beck-Sague CM, Dooley SW, Hutton MD, et al. Hospital outbreak of multidrug-resistant *Mycobacterium tuberculosis*. JAMA 1992;268:1280–6.
334. (17) Coronado VG, Beck-Sague CM, Hutton MD, et al. Transmission of multidrug-resistant *Mycobacterium tuberculosis* among persons with human immunodeficiency virus infection in an urban hospital: Epidemiologic and restriction fragment length polymorphism analysis. J Infect Dis 1993;168:1052–5.
335. (18) Coronado VG, Valway S, Finelli L, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis* among intravenous drug users with human immunodeficiency virus infection [abstract]. In: Abstracts of the Third Annual Meeting of the Society for Hospital Epidemiology of America. Chicago, IL: Infect Control Hosp Epidemiol 1993;14:428.
336. (8) Dooley SW, Villarino ME, Lawrence M, et al. Tuberculosis in a hospital unit for patients infected with the human immunodeficiency virus (HIV): evidence of nosocomial transmission. JAMA 1992;267:2632–4.
337. (19) Edlin BR, Tokars JI, Grieco MH, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome: epidemiologic studies and restriction fragment length polymorphism analysis. N Engl J Med 1992;326:1514–22.
338. (20) Fischl MA, Uttamchandani RB, Daikos GL, et al. An outbreak of tuberculosis caused by multiple-drug-resistant tubercle bacilli among patients with HIV infection. Ann Intern Med 1992;117:177–83.
339. (21) Ikeda ARM, Birkhead GS, DeFerdinando Jr GT, et al. Nosocomial tuberculosis: an outbreak of a strain resistant to seven drugs. Infect Control Hosp Epidemiol 1995;16:152–9.
340. (22) Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. Res Microbiol 1992;144:117–22.
341. (23) Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. Am J Infect Control 1995;23:146–51.
342. (24) Jereb JA, Klevens RM, Privett TD, et al. Tuberculosis in health care workers at a hospital with an outbreak of multidrug-resistant *Mycobacterium tuberculosis*. Arch Intern Med 1995;155:854–9.
343. (25) Moran GJ, McCabe F, Morgan MT, Talan DA. Delayed recognition and infection control for tuberculosis patients in the emergency department. Ann Emerg Med 1995;26:283–9.
344. (26) Pearson ML, Jereb JA, Frieden TR, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*: a risk to hospitalized patients and health-care workers. Ann Intern Med 1992;117:191–6.
345. Tokars JI, Jarvis WR, Edlin BR, et al. Tuberculin skin testing of hospital employees during an outbreak of multidrug-resistant tuberculosis in human immunodeficiency virus (HIV) infected patients. Infect Control Hosp Epidemiol 1992;13:509–10.
346. Macher JM. The use of germicidal lamps to control tuberculosis in healthcare facilities. Infect Control Hosp Epidemiol 1993;14:723–9.
347. (129) U.S. Department of Labor, Occupational Safety and Health Administration. Respiratory Protection, 29 CFR 1910.139. Federal Register 1998;63:1152–300.
348. (105) Ehrenkranz NJ, Kicklighter JL. Tuberculosis outbreak in a general hospital: evidence for airborne spread of infection. Ann Intern Med 1972;77:377–82.
349. (106) Calder RA, Duclos P, Wilder MH, et al. *Mycobacterium tuberculosis* transmission in a health clinic. Bull Int Union Tuberc Lung Dis 1991;66:103–6.
350. (107) Jereb JA, Burwen DR, Dooley SW, et al. Nosocomial outbreak of tuberculosis in a renal transplant unit: application of a new technique for restriction fragment length polymorphism analysis of *Mycobacterium tuberculosis* isolates. J Infect Dis 1993;168:1219–24.
351. (127) Ayliffe GAJ. Role of the environment of the operating suite in surgical wound infection. Rev Infect Dis 1991;13(suppl):S800–S804.
352. (128) Choux M, Genitori L, Lang D, Lena G. Shunt implantation: reducing the incidence of shunt infection. J Neurosurg 1992;77:875–80.

353. Edmiston CE Jr, Sinski S, Seabrook GR, Simons D, Goheen MP. Airborne particulates in the OR environment. *AORN J* 1999;69:1169–72.
354. Duhaime AC, Bonner K, McGowan KL, Schut L, Sutton LN, Plotkin S. Distribution of bacteria in the operating room environment and its relation to ventricular shunt infections: a prospective study. *Childs Nerv Syst* 1991;7:211–4.
355. Everett WD, Kipp H. Epidemiologic observations of operating room infections resulting from variations in ventilation and temperature. *Am J Infect Control* 1991;19:277–82.
356. **(115)** Lidwell OM. Clean air at operation and subsequent sepsis in the joint. *Clin Orthop* 1986;211:91–02.
357. **(116)** Nichols RL. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 3rd ed. Boston, MA: Little, Brown and Company, 1992;461–73.
358. **(117)** Clark RP, Reed PJ, Seal DV, Stephenson ML. Ventilation conditions and air-borne bacteria and particles in operating theatres: proposed safe economies. *J Hyg (Lond)* 1985;95:325–35.
359. **(119)** Laufman H. The operating room. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 2nd ed. Boston, MA/Toronto, ON: Little, Brown and Company, 1986;315–23.
360. Pittet D, Ducle G. Infectious risk factors related to operating rooms. *Infect Control Hosp Epidemiol* 1994;15:456–62.
361. Hambræus A. Aerobiology in the operating room — a review. *J Hosp Infect* 1988;11(suppl. A):68–76.
362. **(118)** Babb JR, Lynam P, Ayliffe GAJ. Risk of airborne transmission in an operating theater containing four ultraclean air units. *J Hosp Infect* 1995;31:159–68.
363. Velesco, E, Thuler LCS, Martins CAS, deCastroDias LM, Conalves VMSC. Risk factors for infectious complications after abdominal surgery for malignant disease. *Am J Infect Control* 1996;24:1–6.
364. **(120)** National Academy of Sciences, National Research Council, Division of Medical Sciences, Ad Hoc Committee on Trauma. Postoperative wound infections: the influence of ultraviolet irradiation of the operating room and of various other factors. *Ann Surg* 1964;160 (suppl.):1–192.
365. **(121)** Charnley J. A clean-air operating enclosure. *Br J Surg* 1964;51:202–5.
366. **(122)** Lidwell OM, Lowbury EJJ, Whyte W, Blowers R, Stanley SJ, Lowe D. Effect of ultraclean air in operating rooms on deep sepsis in the joint after total hip or knee replacement: a randomized study. *Br Med J* 1982;285:10–4.
367. **(123)** Hill C, Flamant R, Mazas F, Evrard J. Prophylactic cefazolin versus placebo in total hip replacement: report of a multicentre double-blind randomized trial. *Lancet* 1981;1:795–6.
368. **(124)** Ha’eri GB, Wiley AM. Total hip replacement in a laminar flow environment with special reference to deep infections. *Clin Orthop* 1980;148:163–8.
369. **(125)** Collins DK, Steinhaus K. Total hip replacement without deep infection in a standard operating room. *J Bone Joint Surg* 1976;58A:446–50.
370. **(126)** Taylor GD, Bannister GC, Leeming JP. Wound disinfection with ultraviolet radiation. *J Hosp Infect* 1995;30:85–93.
371. **(130)** Langevin PB, Rand KH, Layton AJ. The potential for dissemination of *Mycobacterium tuberculosis* through the anesthesia breathing circuit. *Chest* 1999;115:1107–14.
372. **(131)** U.S. Department of Labor, Occupational Safety and Health Administration. Occupational exposure to tuberculosis: proposed rule. (29 CFR 1910). *Federal Register* 1997;62:54159–209.
373. **(132)** Aranha-Creado H, Prince D, Greene K, Brandwein H. Removal of *Mycobacterium* species by breathing circuit filters. *Infect Control Hosp Epidemiol* 1997;18:252–254.
374. Anesthesiology Society of America. *Infection Control for Practice of Anesthesiology*. 1999. Available at: www.asahq.org/Profinfo/Infection/Infection_TOC.html
375. McCarthy JF. Risk factors for occupational exposures in healthcare professionals. In: Hansen W, ed. *A guide to managing indoor air quality in health care organizations*. Oakbrook Terrace IL: Joint Commission on Accreditation of Healthcare Organizations, 1997;27–41.
376. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 85-126-1932;1988.
377. National Institute for Occupational Safety and Health. NIOSH Health Hazard Evaluation and Technical Assistance Report: HETA 88-101-2008;1990.
378. **(135)** National Institute for Occupational Safety and Health. Control of smoke from laser/electric surgical procedures. DHHS (NIOSH) Publication 96-128;1996. Available at: www.cdc.gov/niosh/hc11.html
379. Taravella MJ, Weinberg A, Blackburn P, May M. Do intact viral particles survive excimer laser ablation? *Arch Ophthalmol* 1997;115:1028–30.

380. Hagen KB, Kettering JD, Aprecio RM, et al. Lack of virus transmission by the excimer laser plume. *Am J Ophthalmol* 1997;124:206–11.
381. Kunachak S, Sithisarn P, Kulapaditharom B. Are laryngeal papilloma virus-infected cells viable in the plume derived from a continuous mode carbon dioxide laser, and are they infectious? A preliminary report on one laser mode. *J Laryng Otol* 1996;110:1031–3.
382. **(137)** Hughes PS, Hughes AP. Absence of human papillomavirus DNA in the plume of erbium:YAG laser-treated warts. *J Am Acad Dermatol* 1998;38:426–8.
383. Garden JM, O'Bannion K, Sheinitz LS, et al. Papillomavirus in the vapor of carbon dioxide laser treated verrucae. *JAMA* 1988;125:1199–202.
384. Sawchuck WS, Weber JP, Lowry DR, et al. Infectious papillomavirus in the vapour of warts treated with carbon dioxide laser or electrocoagulation: detection and protection. *J Am Acad Dermatol* 1989;21:41–9.
385. Baggish MS, Poiesz BJ, Joret D, et al. Presence of human immunodeficiency virus DNA in laser smoke. *Lasers Surg Med* 1991;11:197–203.
386. **(138)** Capizzi PJ, Clay RP, Battey MJ. Microbiologic activity in laser resurfacing plume and debris. *Lasers Surg Med* 1998;23:172–4.
387. McKinley IB Jr, Ludlow MO. Hazards of laser smoke during endodontic therapy. *J Endodont* 1994;20:558.
388. Favero MS, Bolyard EA. Microbiologic considerations: disinfection and sterilization strategies and the potential for airborne transmission of bloodborne pathogens. *Surg Clin North Am* 1995;75:1071–89.
389. **(136)** Association of periOperative Registered Nurses. Recommended practices for laser safety in practice settings. In: Standards, Recommended Practices and Guidelines. Denver CO; AORN;2003;301–5.
390. **(139)** ECRI. Surgical smoke evacuation systems. *Health Devices* 1997;26:132–72.
391. **(140)** ECRI. Update evaluation: Surgical smoke evacuation systems. *Health Devices* 1999;28:333–62.
392. **(141)** ECRI. Stationary surgical smoke evacuation systems. *Health Devices* 2001;30:73–86.
393. American National Standards Institute. ANSI National standard for safe use of lasers in health care facilities. ANSI Z136.3-1996.
394. Kaufman AF, McDade J, Patton C, et al. Pontiac fever: isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981;114:337–47.
395. **(192)** Marston BJ, Lipman HB, Breiman RF. Surveillance for Legionnaires' disease: risk factors for morbidity and mortality related to infection with *Legionella*. *Arch Intern Med* 1994;154:2417–22.
396. **(Appendix; 4)** Hoge CW, Breiman RF. Advances in the epidemiology and control of *Legionella* infections. *Epidemiol Rev* 1991;13:329–40.
397. Breiman RF, Butler JC. Legionnaires' disease: clinical, epidemiological, and public health perspectives. *Semin Respir Infect* 1998;13:84–9.
398. Yu, VL. *Legionella pneumophila* (Legionnaires' disease). In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2424–35.
399. Muder RR. Other *Legionella* species. In: Mandell GL, Bennett JE, Dolin R, eds. Principles and practice of infectious diseases, 5th ed. Philadelphia, PA: Churchill Livingstone, 2000;2435–41.
400. Yu VL. Could aspiration be the major mode of transmission for *Legionella*? *Am J Med* 1993;95:13–5.
401. Jimenez P, Torres A, Rodriguez-Roisin R, et al. Incidence and etiology of pneumonia acquired during mechanical ventilation. *Crit Care Med* 1989;17:882–5.
402. **(220)** Zuravleff JJ, Yu VL, Shonnard JW, Rihs JD, Best M. *Legionella pneumophila* contamination of a hospital humidifier: demonstration of aerosol transmission and subsequent subclinical infection in exposed guinea pigs. *Am Rev Respir Dis* 1983;128:657–61.
403. **(202)** Mastro TD, Fields BS, Breiman RF, Campbell J, Plikaytis BD, Spika JS. Nosocomial Legionnaires' disease and use of medication nebulizers. *J Infect Dis* 1991;163:667–70.
404. **(203)** Dondero TJ Jr, Rendtorff RC, Mallison GF, et al. An outbreak of Legionnaires' disease associated with a contaminated air-conditioning cooling tower. *N Engl J Med* 1980;302:365–70.
405. **(199)** Garbe PL, Davis BJ, Weisfield JS, et al. Nosocomial Legionnaires' disease: epidemiologic demonstration of cooling towers as a source. *JAMA* 1985;254:521–4.
406. **(204)** O'Mahony MC, Stanwell-Smith RE, Tillett HE, et al. The Stafford outbreak of Legionnaires' disease. *Epidemiol Infect* 1990;104:361–80.
407. **(205)** Breiman RF, Fields BS, Sanden G, Volmer L, Meier A, Spika J. An outbreak of Legionnaires' disease associated with shower use: possible role of amoebae. *JAMA* 1990;263:2924–6.

408. (200) Hanrahan JP, Morse DL, Scharf VB, et al. A community hospital outbreak of legionellosis: transmission by potable hot water. *Am J Epidemiol* 1987;125:639–9.
409. (206) Breiman RF, VanLoock FL, Sion JP, et al. Association of “sink bathing” and Legionnaires’ disease [abstract]. In: Abstracts of the 91st Meeting of the American Society for Microbiology, 1991.
410. (207) Struelens MJ, Maes N, Rost F, et al. Genotypic and phenotypic methods for the investigation of a nosocomial *Legionella pneumophila* outbreak and efficacy of control measures. *J Infect Dis* 1992;166:22–30.
411. Terranova W, Cohen ML, Fraser DW. Outbreak of Legionnaires’ disease diagnosed in 1977. *Lancet* 1978;2:122–4.
412. (219) Marrie TJ, Haldane D, MacDonald S, et al. Control of endemic nosocomial Legionnaires’ disease by using sterile potable water for high risk patients. *Epidemiol Infect* 1991;107:591–605.
413. Nechwatal R, Ehret W, Klatte OJ, et al. Nosocomial outbreak of legionellosis in a rehabilitation center: demonstration of potable water as a source. *Infection* 1993;21:235–40.
414. Hoebe CJP, Cluitmanans JJM, Wagenvoort JHT. Two fatal cases of nosocomial *Legionella pneumophila* pneumonia associated with a contaminated cold water supply. *Eur J Clin Microbiol Infect Dis* 1998;17:740–9.
415. Helms CM, Viner JP, Sturm RH, et al. Comparative features of pneumococcal, *Mycoplasma*, and Legionnaires’ disease pneumonias. *Ann Intern Med* 1979;90:543–7.
416. Yu V, Kroboth FJ, Shonnard J, Brown A, McDearman S, Magnussen M. Legionnaires’ disease: new clinical perspectives from a prospective pneumonia study. *Am J Med* 1982;73:357–61.
417. (194) Jimenez ML, Aspa J, Padilla B, et al. Fiberoptic bronchoscopic diagnosis of pulmonary disease in 151 HIV-infected patients with pneumonitis. *Eur J Clin Microbiol Infect Dis* 1991;10:491–6.
418. Lowry PW, Blankenship RJ, Gridley W, et al. A cluster of *Legionella* sternal wound infections due to postoperative topical exposure to contaminated tap water. *N Engl J Med* 1991;324:109–12.
419. Shah A, Check F, Baskin S. Legionnaires’ disease and acute renal failure: case report and review. *Clin Infect Dis* 1992;14:204–7.
420. Lowry PW, Tompkins LS. Nosocomial legionellosis: a review of pulmonary and extrapulmonary syndromes. *Am J Infect Control* 1993;21:21–7.
421. Schlanger G, Lutwick LI, Kurzman M, et al. Sinusitis caused by *L. pneumophila* in a patient with acquired immune deficiency syndrome. *Am J Med* 1984;77:957–60.
422. Tompkins LS, Roessler BJ, Redd SC, et al. *Legionella* prosthetic-valve endocarditis. *N Engl J Med* 1988;318:530–5.
423. (195) Bock BV, Kirby BD, Edelstein PH, et al. Legionnaires’ disease in renal transplant recipients. *Lancet* 1978;1:410–3.
424. (196) Kirby BD, Snyder KM, Meyer RD, Finegold SM. Legionnaires’ disease: report of 65 nosocomially acquired cases and review of the literature. *Medicine* 1980;59:188–205.
425. (197) Brady MT. Nosocomial Legionnaires’ disease in a children’s hospital. *J Pediatr* 1989;115:46–50.
426. Horie H, Kawakami H, Minoshima K, et al. Neonatal Legionnaires’ disease: histologic findings in an autopsied neonate. *Acta Pathol Jpn* 1992;42:427–31.
427. Roig J, Aguilar X, Ruiz J, et al. Comparative study of *Legionella pneumophila* and other nosocomial pneumonias. *Chest* 1991;99:344–50.
428. Redd SC, Schuster DM, Quan J, et al. Legionellosis cardiac transplant recipients: results of a nationwide survey. *J Infect Dis* 1988;158:651–3.
429. Seu P, Winston DJ, Olthoft KM, et al. Legionnaires’ disease in liver transplant recipients. *Infect Dis Clin Pract* 1993;2:109–13.
430. (215) Chow JW, Yu VL. *Legionella*: a major opportunistic pathogen in transplant recipients. *Semin Respir Infect* 1998;13:132–9.
431. (189) Kool JL, Fiore AE, Kioski CM, et al. More than ten years of unrecognized nosocomial transmission of Legionnaires’ disease among transplant patients. *Infect Control Hosp Epidemiol* 1998;19:898–904.
432. (190) Le Saux NM, Sekla L, McLeod J, et al. Epidemic of nosocomial Legionnaires’ disease in renal transplant recipients: a case-control and environmental study. *Can Med Assoc J* 1989;140:1047–53.
433. Berendt RF, Young HW, Allen RG, Knutsen GL. Dose-response of guinea pigs experimentally infected with aerosols of *Legionella pneumophila*. *J Infect Dis* 1980;141:186–92.
434. Marston BJ, Plouffe JF, File TM, et al. Incidence of community-acquired pneumonia requiring hospitalization — results of a population-based active surveillance study in Ohio. *Arch Intern Med* 1997;157:1709–18.

435. **(198)** Muder RR, Yu VL, McClure JK, Kroboth FJ, Kominos SD, Lumish RN. Nosocomial Legionnaires' disease uncovered in a prospective pneumonia study: implications for underdiagnosis. *JAMA* 1983;249:3184–8.
436. Brennen C, Vickers JP, Yu VL, Puntereri A, Yee YC. Discovery of occult *Legionella pneumonia* in a long-stay hospital: Results of prospective serologic survey. *Br Med J* 1987;295:306–7.
437. **(166)** Marrie TJ, MacDonald S, Clarke K, Haldane D. Nosocomial Legionnaires' disease: lessons from a four-year prospective study. *Am J Infect Control* 1991;19:79–85.
438. Stout JE, Yu VL. Current concepts: legionellosis. *N Engl J Med* 1997;337:682–7.
439. Vergis EN, Yu VL. Macrolides are ideal for empiric therapy of community-acquired pneumonia in the immunocompromised host. *Semin Respir Infect* 1998;13:322–8.
440. Sopena N, Sabria-Leal M, Pedro-Botet ML, et al. Comparative study of the clinical presentation of *Legionella pneumonia* and other community-acquired pneumonias. *Chest* 1998;113:1195–200.
441. **(217)** Hirani NA, MacFarlane JT. Impact of management guidelines on the outcome of severe community acquired pneumonia. *Thorax* 1997;52:17–21.
442. Lieberman D, Porath A, Schlaeffer F, Boldur L. *L. pneumophila* species community-acquired pneumonia: a review of 56 hospitalized patients. *Chest* 1996;109:1243–9.
443. Ewig S, Bauer T, Hasper E, et al. Value of routine microbial investigation in community-acquired pneumonia treated in a tertiary care center. *Respiration* 1996;63:164–9.
444. Marrie TJ, Peeling RW, Fine MJ, et al. Ambulatory patients with community-acquired pneumonia: the frequency of atypical agents and clinical course. *Am J Med* 1996;101:508–15.
445. Benin AI, Benson RF, Besser RE. Trends in Legionnaires' disease, 1980–1998: declining mortality and new patterns of diagnosis. *Clin Infect Dis* 2002;35:1039–46.
446. Fliermans CD, Cherry WB, Orrison LH, Smith SJ, Tison DL, Pope DH. Ecologic distribution of *Legionella pneumophila*. *Appl Environ Microbiol* 1981;41:9–16.
447. Morris GK, Patton CM, Feeley JC, et al. Isolation of the Legionnaires' disease bacterium from environmental samples. *Ann Intern Med* 1979;90:664–6.
448. Hsu SC, Martin R, Wentworth BB. Isolation of *Legionella* species from drinking water. *Appl Environ Microbiol* 1984;48:830–2.
449. Tison DL, Seidler RJ. *Legionella* incidence and density in potable drinking water. *Appl Environ Microbiol* 1983;45:337–9.
450. Parry MF, Stampleman L, Hutchinson JH, et al. Waterborne *Legionella bozemanii* and nosocomial pneumonia in immunosuppressed patients. *Ann Intern Med* 1985;103:205–10.
451. England AC, Fraser DW. Sporadic and epidemic nosocomial legionellosis in the United States: epidemiologic features. *Am J Med* 1981;70:707–11.
452. Cohen ML, Broome CV, Paris AL, et al. Fatal nosocomial Legionnaires' disease: clinical and epidemiological characteristics. *Ann Intern Med* 1979;90:611–3.
453. **(193)** Haley CE, Cohen ML, Halter J, Meyer RD. Nosocomial Legionnaires' disease: a continuing common-source epidemic at Wadsworth Medical Center. *Ann Intern Med* 1979;90:583–6.
454. Stout JE, Yu VL, Vickers RM, Shonnard J. Potable water supply as the hospital reservoir for Pittsburgh pneumonia agent. *Lancet* 1982;1:471–2.
455. **(201)** Arnow PM, Chou T, Weil D, Shapiro EN, Kretzschmar C. Nosocomial Legionnaires' disease caused by aerosolized tap water from respiratory devices. *J Infect Dis* 1982;146:460–7.
456. Farrell ID, Barker JE, Miles EP, Hutchinson JCP. A field study of the survival of *Legionella pneumophila* in a hospital hot-water system. *Epidemiol Infect* 1990;104:381–7.
457. Stout JE, Yu VL, Best MG. Ecology of *Legionella pneumophila* within water distribution systems. *Appl Environ Microbiol* 1985;49:221–8.
458. Sanden GN, Fields BS, Barbaree JM, et al. Viability of *Legionella pneumophila* in chlorine-free water at elevated temperatures. *Curr Microbiol* 1989;61–5.
459. Schulze-Röbbecke R, Rodder M, Exner M. Multiplication and killing temperatures of naturally occurring legionellae. *Zbl Bakt Hyg B* 1987;184:495–500.
460. Habicht W, Muller HE. Occurrence and parameters of frequency of *Legionella* in warm water systems of hospitals and hotels in Lower Saxony. *Zbl Bakt Hyg B* 1988;186:79–88.
461. Ciesielski CA, Blaser MJ, Wang WL. Role of stagnation and obstruction of water flow in isolation of *Legionella pneumophila* from hospital plumbing. *Appl Environ Microbiol* 1984;48:984–7.
462. Rowbotham TJ. Preliminary report on the pathogenicity of *Legionella pneumophila* for freshwater and soil amoebae. *J Clin Path* 1980;33:179–83.

463. Fields BS, Sanden GN, Barbaree JM, et al. Intracellular multiplication of *Legionella pneumophila* in amoebae isolated from hospital hot water tanks. *Curr Microbiol* 1989;18:131–7.
464. (142) Villarino ME, Stevens LE, Schable B, et al. Risk factors for epidemic *Xanthomonas maltophilia* infection/colonization in intensive care unit patients. *Infect Control Hosp Epidemiol* 1992;13:201–6.
465. (147) Burdge DR, Nakielna EM, Noble MA. Case-control and vector studies of nosocomial acquisition of *Pseudomonas cepacia* in adult patients with cystic fibrosis. *Infect Control Hosp Epidemiol* 1993;14:127–30.
466. Stephenson JR, Heard SR, Richards MA, Tabaqchali S. Gastrointestinal colonization and septicaemia with *Pseudomonas aeruginosa* due to contaminated thymol mouthwash in immunocompromised patients. *J Hosp Infect* 1985;6:369–78.
467. Kolmos HJ, Thusen B, Neilsen SV, Lohmann M, Kristoffersen K, Rosdahl VT. Outbreak of infection in a burns unit due to *Pseudomonas aeruginosa* originating from contaminated tubing used for irrigating patients. *J Hosp Infect* 1993;24:11–21.
468. Vanholder R, Vanhaecke E, Ringoir S. Waterborne *Pseudomonas* septicemia. *ASAIO Trans* 1990;36:M215–6.
469. Ehni WF, Reller LB, Ellison RT III. Bacteremia in granulocytopenic patients in a tertiary-care general hospital. *Rev Infect Dis* 1991;13:613–9.
470. Gallagher PG, Watanakunakorn C. *Pseudomonas* bacteremia in a community teaching hospital, 1980–1984. *Rev Infect Dis* 1989;11:846–52.
471. Centers for Disease Control. Nosocomial infection and pseudoinfection from contaminated endoscopes and bronchoscopes — Wisconsin and Missouri. *MMWR* 1991;40:675–8.
472. Kerr JR, Moore JE, Curran MD, et al. Investigation of a nosocomial outbreak of *Pseudomonas aeruginosa* pneumonia in an intensive care unit by random amplification of polymorphic DNA assay. *J Hosp Infect* 1995;30:125–31.
473. Brewer SC, Wunderink RG, Jones CB, Leeper KV. Ventilator-associated pneumonia due to *Pseudomonas aeruginosa*. *Chest* 1996;109:1019–22.
474. Rello J, Jubert P, Valles J, et al. Evaluation of outcome for intubated patients with pneumonia due to *Pseudomonas aeruginosa*. *Clin Infect Dis* 1996;23:973–8.
475. Henderson A, Kelly W, Wright M. Fulminant primary *Pseudomonas aeruginosa* pneumonia and septicaemia in previously well adults. *Intensive Care Med* 1992;18:430–2.
476. Torres A, Serra-Battles J, Ferrer A, et al. Severe community acquired pneumonia: epidemiology and prognostic factors. *Am Rev Respir Dis* 1991;144:312–8.
477. Pedersen SS, Koch C, Høiby N, Rosendal K. An epidemic spread of multiresistant *Pseudomonas aeruginosa* in a cystic fibrosis center. *J Antimicrob Chemother* 1986;17:505–6.
478. Kubesch P, Dörk T, Wulbrand U, et al. Genetic determinants of airways' colonization with *Pseudomonas aeruginosa* in cystic fibrosis. *Lancet* 1993;341:189–93.
479. Koch C, Høiby N. Pathogenesis of cystic fibrosis. *Lancet* 1993;341:1065–9.
480. Worlitzsch D, Wolz C, Botzenart K, et al. Molecular epidemiology of *Pseudomonas aeruginosa* – urinary tract infections in paraplegic patients. *Zentrabl Hyg Umweltmed* 1989;189:175–84.
481. Glenister H, Holton J, Teall A. Urinary tract pressure recording equipment as a source for infection. *J Hosp Infect* 1985;6:224–6.
482. Ferroni A., Nguyen L, Pron B, Quense G, Brusset MC, Berche P. Outbreak of nosocomial urinary tract infections due to *Pseudomonas aeruginosa* in a paediatric surgical unit associated with tap water contamination. *J Hosp infect* 1998;39:301–7.
483. Marrie TJ, Major H, Gurwith M, et al. Prolonged outbreak of nosocomial urinary tract infection with a single strain of *Pseudomonas aeruginosa*. *Can Med Assoc J* 1978;119:593–8.
484. Moore B, Forman A. An outbreak of urinary *Pseudomonas aeruginosa* infection acquired during urological operations. *Lancet* 1966;2:929–31.
485. Anderson RJ, Schafer LA, Olin DB, Eickhoff TC. Septicemia in renal transplant recipients. *Arch Surg* 1973;106:692–4.
486. Fang G, Brennen C, Wagener M, et al. Use of ciprofloxacin versus use of aminoglycosides for therapy of complicated urinary tract infection: prospective, randomized clinical and pharmacokinetic study. *Antimicrob Agents Chemother* 1991;35:1849–55.
487. Dorff GJ, Beimer NF, Rosenthal DR, Rytel MW. *Pseudomonas* septicemia: illustrated evolution of its skin lesions. *Arch Intern Med* 1971;128:591–5.
488. Teplitz C. Pathogenesis of *Pseudomonas vasculitis* and septic lesions. *Arch Pathol* 1965;80:297–307.

489. Roberts R, Tarpay MM, Marks MI, Nitschke R. Erysipelas-like lesions and hyperesthesia as manifestations of *Pseudomonas aeruginosa* sepsis. *JAMA* 1982;248:2156–7.
490. Duncan BW, Adzick NS, deLorimier AA, et al. Necrotizing fasciitis in childhood. *J Pediatr Surg* 1992;27:668–71.
491. McManus AT, Mason AD Jr, McManus WF, Pruitt BA Jr. Twenty-five year review of *Pseudomonas aeruginosa* bacteremia in a burn center. *Eur J Clin Microbiol* 1985;4:219–23.
492. Tredget EE, Shankowsky HA, Joffe AM, et al. Epidemiology of infections with *Pseudomonas aeruginosa* in burn patients: the role of hydrotherapy. *Clin Infect Dis* 1992;15:941–9.
493. Schlech WF III, Simosen N, Sumarah R, Martin RS. Nosocomial outbreak of *Pseudomonas aeruginosa* folliculitis associated with a physiotherapy pool. *Can Med Assoc J* 1986;134:909–13.
494. Fang G, Keys TF, Gentry LO, et al. Prosthetic valve endocarditis resulting from nosocomial bacteremia: a prospective, multicenter study. *Ann Intern Med* 1993;119:560–7.
495. Cohen PS, Maguire JH, Weinstein L. Infective endocarditis caused by gram-negative bacteria: a review of the literature, 1945–1977. *Prog Cardiovasc Dis* 1980;22:205–42.
496. Wise BL, Mathis JL, Jawetz E. Infections of the central nervous system due to *Pseudomonas aeruginosa*. *J Neurosurg* 1969;31:432–4.
497. Bray DA, Calcaterra TC. *Pseudomonas* meningitis complicating head and neck surgery. *Laryngoscope* 1976;86:1386–90.
498. Schein OD, Wasson PJ, Boruchoff SA, Kenyon KR. Microbial keratitis associated with contaminated ocular medications. *Am J Ophthalmol* 1988;105:361–5.
499. Procope JA. Delayed-onset *Pseudomonas* keratitis after radial keratotomy. *J Cataract Refract Surg* 1997;23:1271–2.
500. Sapico FL, Montgomerie JZ. Vertebral osteomyelitis in intravenous drug abusers: report of three cases and review of the literature. *Rev Infect Dis* 1980;2:196–206.
501. Tindel JR, Crowder JG. Septic arthritis due to *Pseudomonas aeruginosa*. *JAMA* 1971;218:559–61.
502. Martone WJ, Tablan OC, Jarvis WR. The epidemiology of nosocomial epidemic *Pseudomonas cepacia* infections. *Eur J Epidemiol* 1987;3:222–32.
503. Goldmann DA, Klingler JD. *Pseudomonas cepacia*: biology, mechanisms of virulence, epidemiology. *J Pediatr* 1986;108:806–12.
504. Widmer AF, Wenzel RP, Trilla A, et al. Outbreak of *Pseudomonas aeruginosa* infections in a surgical intensive care unit: probable transmission via hands of a health care worker. *Clin Infect Dis* 1993;16: 372–6.
505. Döring G, Hörz M, Ortelt J, et al. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993;110:427–36.
506. Hollyoak V, Allison D, Summers J. *Pseudomonas aeruginosa* wound infection associated with a nursing home whirlpool bath. *CDR Review* 1995;5:R100–2.
507. Grundmann H, Kropec A, Hartung D, Berner R, Daschner F. *Pseudomonas aeruginosa* in a neonatal intensive care unit: reservoirs and ecology of the nosocomial pathogen. *J Infect Dis* 1993;168:943–7.
508. Martino P, Venditti M, Papa G, Orefici G, Serra P. Water supply as a source of *Pseudomonas aeruginosa* in a hospital for hematological malignancies. *Bollettino dell Istituto Sieroterapico Milanese* 1985;64:109–14.
509. Ayliffe GAJ, Babb JR, Collins BJ, Lowbury EJ, Newsom SWB. *Pseudomonas aeruginosa* in hospital sinks. *Lancet* 1974;2:578–81.
510. Kluyver AJ. *Pseudomonas aureofaciens* nov. spec and its pigments. *J Bacteriol* 1956;72:406–11.
511. Romling U, Fiedler B, Bosshammer J, et al. Epidemiology of chronic *Pseudomonas aeruginosa* infections in cystic fibrosis. *J Infect Dis* 1994;170:1616–21.
512. Jones F, Bartlett CL. Infections associated with whirlpools and spas. *Soc Appl Bacteriol Symp Series* 1985;14:61S–6S.
513. Casewell MW, Slater NG, Cooper JE. Operating theater water-baths as a cause of *Pseudomonas* septicemia. *J Hosp Infect* 1981;2:237–47.
514. Rechsteiner J, Landheer JE, de Jong J, van Kregten E, Lindner JG. [Kidney lithotripter as a possible source of hospital infection]. *Nederlands Tijdschrift voor Geneeskunde* 1988;132:1849–59. (Dutch)
515. (308) Taplin D, Mertz PM. Flower vases in hospitals as reservoirs for pathogens. *Lancet* 1973;2: 1279–1281.
516. Kaiser AB. Humidifiers and *Pseudomonas* infections. *N Engl J Med* 1970;283:708.

517. Levin MH, Olson B, Nathan C, et al. *Pseudomonas* in the sinks in an intensive care unit: relation to patients. *J Clin Pathol* 1984;37:424–7.
518. Paszko-Kolva C, Yamamoto H, Shahamat M, Sawyer TK, Morris G, Colwell RR. Isolation of amoebae and *Pseudomonas* and *Legionella* spp. from eyewash stations. *Appl Environ Microbiol* 1991;57:163–7.
519. Struelens MJ, Rost F, Deplano A, et al. *Pseudomonas aeruginosa* and *Enterobacteriaceae* bacteremia after biliary endoscopy: an outbreak investigation using DNA macrorestriction analysis. *Am J Med* 1993;95:489–98.
520. Blanc DS, Parret T, Janin B, Raselli P, Francioli P. Nosocomial infections and pseudoinfections from contaminated bronchoscopes: two-year follow up using molecular markers. *Infect Control Hosp Epidemiol* 1997;18:134–6.
521. Boukadida J, de Montalembert M, Gaillard JL, et al. Outbreak of gut colonization by *Pseudomonas aeruginosa* in immunocompromised children undergoing total digestive decontamination: analysis by pulsed-field electrophoresis. *J Clin Microbiol* 1991;29:2068–71.
522. Grigis A, Goglio A, Parea M, Gnechi F, Minetti B, Barbui T. Nosocomial outbreak of severe *Pseudomonas aeruginosa* infections in haematological patients. *Eur J Epidemiol* 1993;9:390–5.
523. Gupta AK, Shashi S, Mohan M, Lamba IM, Gupta R. Epidemiology of *Pseudomonas aeruginosa* infections in a neonatal intensive care unit. *J Trop Pediatr* 1993;39:32–6.
524. Sader HS, Pignatari AC, Leme IL, et al. Epidemiologic typing of multiply drug-resistant *Pseudomonas aeruginosa* isolated from an outbreak in an intensive care unit. *Diagn Microbiol Infect Dis* 1993;17:13–8.
525. Krecmery V, Trupl J. Nosocomial outbreak of meropenem-resistant *Pseudomonas aeruginosa* infections in a cancer center. *J Hosp Infect* 1994;28:209–18.
526. Jumaa P, Chattopadhyay B. Outbreak of gentamicin, ciprofloxacin-resistant *Pseudomonas aeruginosa* in an intensive care unit, traced to contaminated quivers. *J Hosp Infect* 1994;28:209–18.
527. Carson LA, Favero MS, Bond WW, Petersen NJ. Morphological, biochemical, and growth characteristics of *Pseudomonas cepacia* from distilled water. *Appl Microbiol* 1973;25:476–83.
528. Bassett DC, Stokes KJ, Thomas WR. Wound infection with *Pseudomonas multivorans*: a waterborne contaminant of disinfectant solutions. *Lancet* 1970;1:1188–91.
529. Wishart MM, Riley TV. Infection with *Pseudomonas maltophilia*: hospital outbreak due to contaminated disinfectant. *Med J Aust* 1976;2:710–2.
530. Conly JM, Klass L, Larson L. *Pseudomonas cepacia* colonization and infection in intensive care units. *Can Med Assoc J* 1986;134:363–6.
531. Bosshammer J, Fielder B, Gudowis P, von der Hardt H, Romling U, Tummler B. Comparative hygienic surveillance of contamination with pseudomonads in a cystic fibrosis ward over a 4-year period. *J Hosp Infect* 1995;31:261–74.
532. Hutchinson GR, Parker S, Pryor JA, et al. Home-use nebulizers: a potential primary source of *B. cepacia* and other colistin-resistant, gram-negative bacteria in patients with cystic fibrosis. *J Clin Microbiol* 1996;34:584–7.
533. Pegues DA, Carson LA, Anderson RL, et al. Outbreak of *Pseudomonas cepacia* bacteremia in oncology patients. *Clin Infect Dis* 1993;16:407–11.
534. CDC. Nosocomial *Burkholderia cepacia* infection and colonization with intrinsically contaminated mouthwash — Arizona, 1998. *MMWR* 1998;47:926–8.
535. Berthelot P, Grattard F, Mahul P, et al. Ventilator temperature sensors: an unusual source of *Pseudomonas cepacia* in nosocomial infection. *J Hosp Infect* 1993;25:33–43.
536. Khardori N, Elting L, Wong E, et al. Nosocomial infections due to *Xanthomonas maltophilia* (*Pseudomonas maltophilia*) in patients with cancer. *Rev Infect Dis* 1990;12:997–1003.
537. Oie S, Oomaki M, Yorioka K, et al. Microbial contamination of “sterile water” used in Japanese hospitals. *J Hosp Infect* 1998;38:61–5.
538. Crane LR, Tagle LC, Palutke WA. Outbreak of *Pseudomonas paucimobilis* in an intensive care facility. *JAMA* 1981;246:985–7.
539. Lemaitre D, Elaichouni A, Hundhausen M, et al. Tracheal colonization with *Sphingomonas paucimobilis* in mechanically-ventilated neonates due to contaminated ventilator temperature probes. *J Hosp Infect* 1996;32:199–206.
540. Maki DG, Klein BS, McCormick RD, et al. Nosocomial *Pseudomonas pickettii* bacteremias traced to narcotic tampering. A case for selective drug screening of health care personnel. *JAMA* 1991;265:981–6.
541. Maroye P, Doermann HP, Rogues AM, Gachie JP, Mégraud F. Investigation of an outbreak of *Ralstonia pickettii* in a paediatric hospital by RAPD. *J Hosp Infection* 2000;44:267–72.

542. McNeil MM, Solomon SL, Anderson RL, et al. Nosocomial *Pseudomonas pickettii* colonization associated with a contaminated respiratory therapy solution in a special care nursery. *J Clin Microbiol* 1985;22:903–7.
543. Lamka KG, LeChevallier MW, Seidler RJ. Bacterial contamination of drinking water supplies in a modern rural neighborhood. *Appl Environ Microbiol* 1980;39:734–8.
544. Nakashima AK, McCarthy MA, Martone WJ, Anderson RL. Epidemic septic arthritis caused by *Serratia marcescens* and associated with a benzalkonium chloride antiseptic. *J Clin Microbiol* 1987;25:1014–8.
545. Nakashima AK, Highsmith AK, Martone WJ. Survival of *Serratia marcescens* in benzalkonium chloride and in multiple-dose medication vials: relationship to epidemic septic arthritis. *J Clin Microbiol* 1987;25:1019–21.
546. Bosi C, Davin-Regli A, Charrel R, Rocca B, Monnet D, Bollet C. *Serratia marcescens* nosocomial outbreak due to contamination of hexetidine solution. *J Hosp Infect* 1996;33:217–24.
547. Ehrenkranz NJ, Bolyard EA, Wiener M, Cleary TJ. Antibiotic-sensitive *Serratia marcescens* infections complicating cardiopulmonary operations: contaminated disinfectant as a reservoir. *Lancet* 1980;2:1289–92.
548. Cimolai N, Trombley C, Wensley D, LeBlanc J. Heterogeneous *Serratia marcescens* genotypes from a nosocomial pediatric outbreak. *Chest* 1997;111:194–7.
549. Hartstein AI, Rashad AL, Liebler JM, et al. Multiple intensive care unit outbreaks of *Acinetobacter calcoaceticus* subspecies anitratus respiratory infection and colonization associated with contaminated, reusable ventilator circuits and resuscitation bags. *Am J Med* 1988;85:624–31.
550. Stone JW, Das BC. Investigation of an outbreak of infection with *Acinetobacter calcoaceticus* in a special care baby unit. *J Hosp Infect* 1986;7:42–8.
551. Vandenbroucke-Grauls CMJE, Kerver AJH, Rommes JH, Jansen R, den Dekker C, Verhoef J. Endemic *Acinetobacter anitratus* in a surgical intensive care unit: mechanical ventilators as reservoir. *Eur J Clin Microbiol Infect Dis* 1988;7:485–9.
552. Cefai C, Richards J, Gould FK, McPeake P. An outbreak of *Acinetobacter* respiratory infection resulting from incomplete disinfection of ventilatory equipment. *J Hosp Infect* 1990;15:177–82.
553. Gervich DH, Grout CS. An outbreak of nosocomial *Acinetobacter* infections from humidifiers. *Am J Infect Control* 1985;13:210–5.
554. Castle M, Tenney JH, Weinstein MP, Eickhoff TC. Outbreak of a multiply resistant *Acinetobacter* in a surgical intensive care unit. *Heart Lung* 1978;7:641–4.
555. Smith PW, Massanari RM. Room humidifiers as a source of *Acinetobacter* infections. *JAMA* 1977;237:795–7.
556. Snyderman DR, Maloy MF, Brock SM, Lyons RW, Rubin SJ. Pseudobacteremia: false-positive blood cultures from mist tent contamination. *Am J Epidemiol* 1977;106:154–9.
557. Rosenthal SL. Sources of *Pseudomonas* and *Acinetobacter* species found in human culture materials. *Am J Clin Pathol* 1974;62:807–11.
558. Allen KD, Green HT. Hospital outbreak of multi-resistant *Acinetobacter anitratus*: an airborne mode of spread. *J Hosp Infect* 1987;9:169–75.
559. Crombach WHJ, Dijkshoorn L, van Noort-Klaassen M, Niessen J, van Knippenbert-Gordebeke G. Control of an epidemic spread of multi-resistant *Acinetobacter calcoaceticus* in a hospital. *Intensive Care Med* 1989;15:166–170.
560. Catalano M, Quelle LS, Jeric PE, Di Martino A, Maimone SM. Survival of *Acinetobacter baumannii* on bed rails during an outbreak and during sporadic cases. *J Hosp Infect* 1999;42:27–35.
561. D'Agata EMC, Venkataraman L, DeGirolami P, Samore M. Molecular epidemiology of ceftazidime-resistant gram-negative bacilli on inanimate surfaces and their role in cross-transmission during non-outbreak periods. *J Clin Microbiol* 1999;37:3065–7.
562. Jawad A, Snelling AM, Heritage J, Hawkey PM. Exceptional desiccation tolerance of *Acinetobacter radioresistens*. *J Hosp Infect* 1998;39:235–40.
563. Jawad A, Seifert H, Snelling AM, Heritage J, Hawkey PM. Survival of *Acinetobacter baumannii* on dry surfaces: comparison of outbreak and sporadic isolates. *J Clin Microbiol* 1998;36:1938–41.
564. Getschell-White, SI, Donowitz LG, Groschel DHM. The inanimate environment of an intensive care unit as a potential source of nosocomial bacteria: evidence for long survival of *Acinetobacter calcoaceticus*. *Infect Control Hosp Epidemiol* 1989;10:402–6.
565. Loiwal V, Kumar A, Gupta P, Gomber S, Ramchandran VG. *Enterobacter aerogenes* outbreak in a neonatal intensive care unit. *Pediatr Int* 1999;41:157–61.

566. Matsaniotis NS, Syriopoulou VP, Theodoridou MC, Tzanetou KG, Mostrou GI. *Enterobacter* sepsis in infants and children due to contaminated intravenous fluids. *Infect Control* 1984;5:471-7.
567. Zembrzuska-Sadlowska E. The dangers of infections of the hospitalized patients with the microorganisms present in preparations and in the hospital environment. *Acta Pol Pharm* 1995;52:173-8.
568. Felts SK, Schaffner W, Melly MA, Koenig MG. Sepsis caused by contaminated intravenous fluids. *Ann Intern Med* 1972;77:881-90.
569. Modi N, Damjanovic V, Cooke RW. Outbreak of cephalosporin resistant *Enterobacter cloacae* infection in a neonatal intensive care unit. *Arch Dis Child* 1987;62:148-51.
570. Graham DR, Wu E, Highsmith AK, Ginsburg ML. An outbreak of pseudobacteremia caused by *Enterobacter cloacae* from a phlebotomist's vial of thrombin. *Ann Intern Med* 1981;95:585-8.
571. Andersen BM, Sorlie D, Hotvedt R, et al. Multiply beta-lactam-resistant *Enterobacter cloacae* infections linked to the environmental flora in a unit for cardiothoracic and vascular surgery. *Scand J Infect Dis* 1989;21:181-91.
572. Wisplinghoff H, Perbix W, Seifert H. Risk factors for nosocomial bloodstream infections due to *Acinetobacter baumannii*: a case-control study of adult burn patients. *Clin Infect Dis* 1999;28:59-66.
573. Crowe M, Towner KJ, Humphreys H. Clinical and epidemiological features of an outbreak of *Acinetobacter* infection in an intensive therapy unit. *J Med Microbiol* 1995;43:55-62.
574. National Nosocomial Infections Surveillance (NNIS) Report: Data summary from October 1986-April 1996, issued May 1996. *Am J Infect Control* 1996;24:380-8.
575. Bergogne-Bérézin E, Joly-Guillou ML. Hospital infection with *Acinetobacter* spp.: an increasing problem. *J Hosp Infect* 1991;18 (suppl A):250-5.
576. Fagon JY, Chastre J, Hance AJ, Montravers P, Novara A, Gibert C. Nosocomial pneumonia in ventilated patients: a cohort study evaluating attributable mortality and hospital stay. *Am J Med* 1993;94: 281-8.
577. (143) Seifert H, Strate A, Pulverer G. Nosocomial bacteremia due to *Acinetobacter baumannii*: clinical features, epidemiology, and predictors of mortality. *Medicine* 1995;74:340-9.
578. Cisneros JM, Reyes MJ, Pachón J, et al. Bacteremia due to *Acinetobacter baumannii*: epidemiology, clinical findings, and prognostic features. *Clin Infect Dis* 1996;22:1026-32.
579. Schaberg DR, Culver DH, Gaynes RP. Major trends in the microbial ecology of nosocomial infections. *Am J Med* 1991;91(suppl 3B):72S-5S.
580. Wang CC, Chu ML, Ho LJ, Hwang RC. Analysis of plasmid pattern in pediatric intensive care outbreaks of nosocomial infection due to *Enterobacter cloacae*. *J Hosp Infect* 1991;19:33-40.
581. Acolet D, Ahmet Z, Houang E, Hurley R, Kaufman ME. *Enterobacter cloacae* in a neonatal intensive care unit: account of an outbreak and its relationship to use of third generation cephalosporins. *J Hosp Infect* 1994;28:273-86.
582. Mayhall CG, Lamb VA, Gayle WE Jr, Haynes BW Jr. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139:166-71.
583. John JF Jr, Sharbaugh RJ, Bannister ER. *Enterobacter cloacae*: bacteremia, epidemiology, and antibiotic resistance. *Rev Infect Dis* 1982;4:13-28.
584. McDonald C, Banerjee SN, Jarvis WR, NNIS. Seasonal variation of *Acinetobacter* infections: 1987-1996. *Clin Infect Dis* 1999;29:1133-7.
585. Beck-Sague CM, Jarvis WR, Brook JH, et al. Epidemic bacteremia due to *Acinetobacter baumannii* in five intensive care units. *Am J Epidemiol* 1990;132:723-33.
586. (144) Yu VL. *Serratia marcescens*: historical perspective and clinical review. *N Engl J Med* 1979;300:887-93.
587. Wenger PN, Tokars JI, Brennan P, et al. An outbreak of *Enterobacter hormaechei* infection and colonization in an intensive care nursery. *Clin Infect Dis* 1997;24:1243-4.
588. Buxton AE, Anderson RL, Wedegar D, Atlas E. Nosocomial respiratory tract infection and colonization with *Acinetobacter calcoaceticus*. *Am J Med* 1978;65:507-13.
589. French GL, Casewell MW, Roncoroni AJ, Knight S, Philipps I. A hospital outbreak of antibiotic-resistant *Acinetobacter anitratus*: epidemiology and control. *J Hosp Infect* 1980;1:125-31.
590. Guenter SH, Hendley JO, Wenzel RP. Gram-negative bacilli as nontransient flora on the hands of hospital personnel. *J Clin Microbiol* 1987;25:488-90.
591. Dreyfuss D, Djedaini K, Weber P, et al. Prospective study of nosocomial pneumonia and of patient and circuit colonization during mechanical ventilation with circuit changes every 48 hours versus no change. *Am Rev Respir Dis* 1991;143:738-43.

592. **(145)** Go SE, Urban C, Burns J, et al. Clinical and molecular epidemiology of *Acinetobacter* infections sensitive only to polymixin B and sublactam. *Lancet* 1994;344:1329–32.
593. Musa EK, Desai N, Casewell MW. The survival of *Acinetobacter calcoaceticus* inoculated on fingertips and on formica. *J Hosp Infect* 1990;15:219–27.
594. Jawad A, Heritage J, Snelling AM, Gascoyne-Binzi DM, Hawkey PM. Influence of relative humidity and suspending menstrua on survival of *Acinetobacter* spp. on dry surfaces. *J Clin Microbiol* 1996;34:2881–7.
595. Mulin B, Talon D, Viel JF, et al. Risk factors for nosocomial colonization with multiresistant *Acinetobacter baumannii*. *Eur J Clin Microbiol Infect Dis* 1995;14:569–76.
596. O'Brien RJ. The epidemiology of nontuberculous mycobacterial disease. *Clin Chest Med* 1989;10:407–18.
597. Böttger EC, Teske A, Kirschner P, et al. Disseminated "*Mycobacterium genavense*" infection in patients with AIDS. *Lancet* 1992;340:76–80.
598. Wallace RJ Jr, Brown BA, Griffith DE. Nosocomial outbreaks/pseudo-outbreaks caused by nontuberculous mycobacteria. *Ann Rev Microbiol* 1998;52:453–90.
599. Chapman JS, Dewlett HJ, Potts WE. Cutaneous reactions to unclassified mycobacterial antigens: a study of children in household contact with patients who excrete unclassified mycobacteria. *Am Rev Respir Dis* 1962;86:547–52.
600. **(245)** Crow HE, Corpe RF, Smith CE. Is serious pulmonary disease caused by nonphotochromogenic ("atypical") acid-fast mycobacteria communicable? *Dis Chest* 1961;39:372–81.
601. Kuritsky JM, Bullen MG, Broome CV, Silcox VA, Good RC, Wallace, RJ Jr. Sternal wound infections and endocarditis due to organisms of the *Mycobacterium fortuitum* complex. *Ann Intern Med* 1983;98:938–9.
602. Laussucq S, Baltch AL, Smith RP, et al. Nosocomial *Mycobacterium fortuitum* colonization from a contaminated ice machine. *Am Rev Respir Dis* 1988;138:891–4.
603. Panwalker AP, Fuhse E. Nosocomial *Mycobacterium gordonae* pseudo-infection from contaminated ice machines. *Infect Control* 1986;7:67–70.
604. Wallace RJ Jr, Musser JM, Hull SI, et al. Diversity and sources of rapidly growing mycobacteria associated with infections following cardiac surgery. *J Infect Dis* 1989;159:708–16.
605. Burns DN, Wallace RJ Jr, Schultz ME, et al. Nosocomial outbreak of respiratory tract colonization with *Mycobacterium fortuitum*: demonstration of the usefulness of pulsed-field gel electrophoresis in an epidemiologic investigation. *Am Rev Respir Dis* 1991;144:1153–9.
606. Lessing MPA, Walker MM. Fatal pulmonary infection due to *Mycobacterium fortuitum*. *J. Clin Pathol* 1993;46:271–2.
607. **(149)** Hoy J, Rolston K, Hopfer RL. Pseudoepidemic of *Mycobacterium fortuitum* in bone marrow cultures. *Am J Infect Control* 1987;15:268–71.
608. Lockwood WW, Friedman C, Bus N, Pierson C, Gaynes R. An outbreak of *Mycobacterium terrae* in clinical specimens associated with a hospital potable water supply. *Am Rev Respir Dis* 1989;140:1614–7.
609. Sniadack DH, Ostroff SM, Karlix MA, et al. A nosocomial pseudo-outbreak of *Mycobacterium xenopi* due to a contaminated water supply: lessons in prevention. *Infect Control Hosp Epidemiol* 1993;14:636–41.
610. **(148)** Cox R, deBorja K, Bach MC. A pseudo-outbreak of *Mycobacterium chelonae* infections related to bronchoscopy. *Infect Control Hosp Epidemiol* 1997;18:136–7.
611. **(150)** Stine TM, Harris AA, Levin S, Rivera N, Kaplan, RL. A pseudoepidemic due to atypical mycobacteria in a hospital water supply. *JAMA* 1987;258:809–11.
612. **(151)** Bennett SN, Peterson DE, Johnson DR, Hall WN, Robinson-Dunn B, Dietrich S. Bronchoscopy-associated *Mycobacterium xenopi* pseudo-infections. *Am J Respir Crit Care Med* 1994;150:245–50.
613. Chadha R, Grover M, Sharma A, et al. An outbreak of post-surgical wound infections due to *Mycobacterium abscessus*. *Pediatr Surg Int* 1998;13:406–10.
614. Von Reyn CF, Maslow JN, Barber TW, Falkinham JO III, Arbeit RD. Persistent colonization of potable water as a source of *Mycobacterium avium* infection in AIDS. *Lancet* 1994;343:1137–41.
615. du Moulin GC, Stottmeier KD, Pelletier PA, Tsang AY, Hedley-Whyte J. Concentration of *Mycobacterium avium* by hospital hot water systems. *JAMA* 1988;260:1599–601.
616. Peters M, Müller C, Rüscher-Gerdes S, et al. Isolation of atypical mycobacteria from tap water in hospitals and homes: Is this a possible source of disseminated MAC infection in AIDS patients? *J Infect* 1995;31:39–44.
617. Soto LE, Bobadilla M, Villalobos Y, et al. Post-surgical nasal cellulitis outbreak due to *Mycobacterium chelonae*. *J Hosp Infect* 1991;19:99–106.
618. Wenger JD, Spika JS, Smithwick RW, et al. Outbreak of *Mycobacterium chelonae* infection associated with use of jet injectors. *JAMA* 1990;264:373–6.

619. Safranek TJ, Jarvis WR, Carson LA, et al. *Mycobacterium chelonae* wound infections after plastic surgery employing contaminated gentian violet skin-marking solution. *N Eng J Med* 1987;317:197–201.
620. Gremillion DH, Mursch SB, Lerner CJ. Injection site abscesses caused by *Mycobacterium chelonae*. *Infect Control* 1983;4:25–8.
621. Begg N, O'Mahoney M, Penny P, Richardson AE. *Mycobacterium chelonae* associated with a hospital hydrotherapy pool. *Community Med* 1986;8:348–50.
622. Aubuchon C, Hill JJ Jr, Graham DR. Atypical mycobacterial infection of soft tissue associated with use of a hot tub. A case report. *J Bone Joint Surg* 1986;68-A:766–8.
623. Kirk J, Kaminski GW. *Mycobacterium marinum* infection. *Aust J Dermatol* 1976;17:111–6.
624. Ross BC, Johnson PDR, Oppedisano F, et al. Detection of *Mycobacterium ulcerans* in environmental samples during an outbreak of ulcerative disease. *Appl Environ Microbiol* 1997;63:4135–8.
625. Tokars JI, McNeil MM, Tablan OC, et al. *Mycobacterium gordonae* pseudoinfection associated with a contaminated antimicrobial solution. *J Clin Microbiol* 1990;28:2765–9.
626. Arnow PM, Bakir M, Thompson K, Bova JL. Endemic contamination of clinical specimens by *Mycobacterium gordonae*. *Clin Infect Dis* 2000;31:472–6.
627. Wright EP, Collins CH, Yates MD. *Mycobacterium xenopi* and *Mycobacterium kansasii* in a hospital water supply. *J Hosp Infect* 1985;6:175–8.
628. du Moulin GC, Stottmeier KD. Waterborne mycobacteria: an increasing threat to health. *ASM News* 1986;10:525–9.
629. Engel HWB, Berwald LG. The occurrence of *Mycobacterium kansasii* in tapwater. *Tubercle* 1980;61:21–6.
630. Kubalek I, Mysak J. The prevalence of environmental mycobacteria in drinking water supply systems in a demarcated region in Czech Republic in the period 1984–1989. *Eur J Epidemiol* 1996;12:471–4.
631. Fox C, Smith F, Brogan O, et al. Non-tuberculous mycobacteria in a hospital's piped water supply. *J Hosp Infect* 1992;21:152–4.
632. Aronson T, Holtzman A, Glover N, et al. Comparison of large restriction fragments of *Mycobacterium avium* isolates recovered from AIDS and non-AIDS patients with those of isolates from potable water. *J Clin Microbiol* 1999;37:1008–12.
633. Carson LA, Bland LA, Cusick LB, et al. Prevalence of nontuberculous mycobacteria in water supplies of hemodialysis centers. *Appl Environ Microbiol* 1988;54:3122–5.
634. Carson LA, Petersen NJ, Favero MS, Aguero SM. Growth characteristics of atypical mycobacteria in water and their comparative resistance to disinfectants. *Appl Environ Microbiol* 1978;36:839–46.
635. Taylor RH, Falkinham III JO, Norton CD, LeChevallier MW. Chlorine, chloramine, chlorine dioxide, and ozone susceptibility of *Mycobacterium avium*. *Appl Environ Microbiol* 2000;66:1702–5.
636. Schulze-Röbbecke R, Fischeder R. Mycobacteria in biofilms. *Zbl Hyg* 1989;188:385–90.
637. Schulze-Röbbecke R, Feldmann C, Fischeder R, Janning B, Exner M, Wahl G. Dental units: an environmental study of sources of potentially pathogenic mycobacteria. *Tubercle Lung Dis* 1995;76:318–23.
638. Meisel JL, Perera DR, Meligro C, Rublin CE. Overwhelming watery diarrhea associated with *Cryptosporidium* in an immunosuppressed patient. *Gastroenterology* 1976;70:1156–60.
639. Nime FA, Page DL, Holscher MA, Yardley JH. Acute enterocolitis in a human being infected with the protozoan *Cryptosporidium*. *Gastroenterology* 1976;70:592–8.
640. Goldstein ST, Juranek DD, Ravenholt O, et al. Cryptosporidiosis: an outbreak associated with drinking water despite state-of-the-art treatment. *Ann Intern Med* 1996;124:459–68.
641. Rose JB. Enteric waterborne protozoa: hazard and exposure assessment. In: Craun GF, ed. *Safety of water disinfection: balancing chemical and microbial risks*. Washington, DC: ILSI Press, 1993;115–26.
642. (157) Juranek DD, Addiss D, Bartlett ME, et al. Cryptosporidiosis and public health: workshop report. *J AWWA* 1995;87:69–80.
643. DuPont HL, Chappell CL, Sterling CR, Okhuysen PC, Rose JB, Jakubowski W. The infectivity of *Cryptosporidium parvum* in healthy volunteers. *N Engl J Med* 1995;332:855–9.
644. Okhuysen PC, Chappell CL, Crabb JH, Sterling CR, DuPont HL. Virulence of three distinct *Cryptosporidium parvum* isolates for healthy adults. *J Infect Dis* 1999;180:1275–81.
645. Chappell CL, Okhuysen PC, Sterling CR, Wang C, Jakubowski W, DuPont HL. Infectivity of *Cryptosporidium parvum* in healthy adults with pre-existing anti-*C. parvum* serum immunoglobulin G. *Am J Trop Med* 1999;60:157–64.

646. Meinhardt PL, Casemore DP, Miller KB. Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiol Rev* 1996;18:118–36.
647. Rose JB. Occurrence and significance of *Cryptosporidium* in water. *JAWWA* 1988;80:53–8.
648. Rose JB, Gerba CP, Jakubowski W. Survey of potable water supplies for *Cryptosporidium* and *Giardia*. *Environ Sci Technol* 1991;25:1393–400.
649. LeChevallier MW, Norton WD, Lee RG. *Giardia* and *Cryptosporidium* spp. in filtered drinking water supplies. *Appl Environ Microbiol* 1991;57:2617–21.
650. Mackenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of *Cryptosporidium* infection transmitted through the public drinking water supply. *N Engl J Med* 1994;331:161–7.
651. Atherton F, Newman CP, Casemore DP. An outbreak of waterborne cryptosporidiosis associated with a public water supply in the UK. *Epidemiol Infect* 1995;115:123–31.
652. Hayes EB, Matte TD, O'Brien TR, et al. Large community outbreak of cryptosporidiosis due to contamination of a filtered public water supply. *N Engl J Med* 1989;320:1372–5.
653. Neill MA, Rice SK, Ahmad NV, Flanigan TP. Cryptosporidiosis: an unrecognized cause of diarrhea in elderly hospitalized patients. *Clin Infect Dis* 1996;22:168–70.
654. Rutala WA, Weber DJ. Water as a reservoir of nosocomial pathogens. *Infect Control Hosp Epidemiol* 1997;18:609–16.
655. Chadwick P. The epidemiological significance of *Pseudomonas aeruginosa* in hospital sinks. *Can J Public Health* 1976;67:323–8.
656. Cordes LG, Wiesenthal AM, Gorman GW, et al. Isolation of *Legionella pneumophila* from hospital shower heads. *Ann Intern Med* 1981;94:195–7.
657. Bollin GE, Plouffe JF, Para MF, Hackman B. Aerosols containing *Legionella pneumophila* generated by shower heads and hot-water faucets. *Appl Environ Microbiol* 1985;50:1128–31.
658. Weber DJ, Rutala WA, Blanchet CN, Jordan M, Gergen MF. Faucet aerators: a source of patient colonization with *Stenotrophomonas maltophilia*. *Am J Infect Control* 1999;27:59–63.
659. Kappstein I, Grundmann H, Hauer T, Niemeyer C. Aerators as a reservoir of *Acinetobacter junii*: an outbreak of bacteraemia in paediatric oncology patients. *J Hosp Infect* 2000;44:27–30.
660. Dennis PJL, Wright AE, Rutter DA, Death JE, Jones BPC. *Legionella pneumophila* in aerosols from shower baths. *J Hyg (Camb)* 1984;93:349–53.
661. (153) American Society of Heating, Refrigerating, and Air-Conditioning Engineers. ASHRAE Guideline 12-2000: minimizing the risk of legionellosis associated with building water systems. Atlanta, GA: ASHRAE, Inc., 2000;1–16.
662. Newsom SWB. Microbiology of hospital toilets. *Lancet* 1972;2:700–3.
663. Gerba CP, Wallis C, Melnick JL. Microbiological hazards of household toilets: droplet production and the fate of residual organisms. *Appl Microbiol* 1975;30:229–37.
664. (152) Hlady WG, Mullen RC, Mintz CS, Shelton BG, Hopkins RS, Daikos GL. Outbreak of Legionnaires' disease linked to a decorative fountain by molecular epidemiology. *Am J Epidemiol* 1993;138:555–62.
665. Rees JC, Allen KD. Holy water — A risk factor for hospital-acquired infection. *J Hosp Infect* 1996;32:51–5.
666. (226) Favero MS, Petersen NJ, Boyer KM, Carson LA, Bond WW. Microbial contamination of renal dialysis systems and associated risks. *ASAIO Trans* 1974;20:175–83.
667. (227) Favero MS, Petersen NJ, Carson LA, Bond WW, Hindman SH. Gram-negative bacteria in hemodialysis systems. *Health Lab Sci* 1975;12:321–34.
668. (228) Favero MS, Petersen NJ. Microbiologic guidelines for hemodialysis systems. *Dialysis Transplant* 1979;6:34–6.
669. Griffiths PA, Babb JR, Bradley CR, Fraise AP. Glutaraldehyde-resistant *Mycobacterium chelonae* from endoscope washer disinfectors. *J Appl Microbiol* 1997;82:519–26.
670. Phillips G, McEwan H, Butler J. Quality of water in washer-disinfectors. *J Hosp Infect* 1995;31:152–4.
671. (259) Cooke RPD, Whymant-Morris A, Umasankar RS, Goddard SV. Bacteria-free water for automatic washer-disinfectors: an impossible dream? *J Hosp Infect* 1998;48:63–5.
672. Humphreys H, Lee JV. Water quality for endoscopy washer-disinfectors. *J Hosp Infect* 1999;42:76–8.
673. Muscarella LF. Are all sterilization processes alike? *AORN J* 1998;67:966–70, 973–6.
674. U.S. Food and Drug Administration. MAUDE database. Available at: www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfMAUDE/search.CFM

675. Alvarado CJ, Stolz SM, Maki DG. Nosocomial infections from contaminated endoscopes: a flawed automated endoscope washer — an investigation using molecular epidemiology. *Am J Med* 1991;91 (Suppl 3b):272–80.
676. Abrutyn E, Goodhart GL, Roos K, Anderson R, Buxton A. *Acinetobacter calcoaceticus* outbreak associated with peritoneal dialysis. *Am J Epidemiol* 1978;107:328–35.
677. Mader JT, Reinartz JA. Peritonitis during peritoneal dialysis — the role of the preheating water bath. *J Chronic Dis* 1978;31:635–41.
678. Kosatsky T, Kleeman J. Superficial and systemic illness related to a hot tub. *Am J Med* 1985;79:10–2.
679. McGuckin MB, Thorpe RJ, Abrutyn E. Hydrotherapy: An outbreak of *Pseudomonas aeruginosa* wound infections related to Hubbard tank treatments. *Arch Phys Med Rehabil* 1981;62:283–5.
680. (243) Koepke GH, Christopher RP. Contamination of whirlpool baths during treatment of infected wounds. *Arch Phys Med Rehabil* 1965;46:261–3.
681. Miller JK, LaForest NT, Hedberg M, Chapman V. Surveillance and control of Hubbard tank bacterial contaminants. *Phys Ther* 1972;50:1482–6.
682. Nelson RM, Reed JR, Kenton DM. Microbiological evaluation of decontamination procedures for hydrotherapy tanks. *Phys Ther* 1972;52:919–23.
683. Page CF. The whirlpool bath and cross-infection. *Arch Phys Med Rehabil* 1954;35:97–8.
684. Newsom SWB. Hospital infection from contaminated ice. *Lancet* 1968;2:620–2.
685. Ravn P, Lundgren JD, Kjaeldgaard P, et al. Nosocomial outbreak of cryptosporidiosis in AIDS patients. *Br Med J* 1991;302:277–80.
686. Bangsberg JM, Uldum S, Jensen JS, Bruun BG. Nosocomial legionellosis in three heart-lung transplant patients: case reports and environmental observations. *Eur J Clin Microbiol Infect Dis* 1995;14:99–104.
687. (246) Stout JE, Yu VL, Muraca P. Isolation of *Legionella pneumophila* from the cold water of hospital ice machines: implications for origin and transmission of the organism. *Infect Control* 1985;6:141–6.
688. Cross DF, Benchimol A, Dimond EG. The faucet aerator — a source of *Pseudomonas* infection. *N Engl J Med* 1966;274:1430–1.
689. *Chryseobacterium (Flavobacterium) meningosepticum* outbreak associated with colonization of water taps in a neonatal intensive care unit. *J Hosp Infect* 2001;47:188–92.
690. Brown DG, Baublis J. Reservoirs of *Pseudomonas* in an intensive care unit for newborn infants: Mechanisms of control. *J Pediatr* 1977;90:453–7.
691. Perryman FA, Flournoy DJ. Prevalence of gentamicin- and amikacin-resistant bacteria in sink drains. *J Clin Microbiol* 1980;12:79–83.
692. Doring G, Horz M, Ortelt J, Grupp H, Wolz C. Molecular epidemiology of *Pseudomonas aeruginosa* in an intensive care unit. *Epidemiol Infect* 1993;110:427–36.
693. Teres D, Schweers P, Bushnell LS, Hedley-Whyte J, Feingold DS. Sources of *Pseudomonas aeruginosa* infection in a respiratory/surgical intensive care unit. *Lancet* 1973;1:415–7.
694. Barbeau J, Tanguay R, Faucher E, et al. Multiparametric analysis of waterline contamination in dental units. *Appl Environ Microbiol* 1996;62:3954–9.
695. Atlas RM, Williams JF, Huntington MK. *Legionella* contamination of dental-unit waters. *Appl Environ Microbiol* 1995;61:1208–13.
696. Fayle SA, Pollard MA. Decontamination of dental unit water systems: a review of current recommendations. *Br Dent J* 1996;181:369–72.
697. Pien FD, Bruce AE. Nosocomial *Ewingella americana* bacteremia in an intensive care unit. *Arch Intern Med* 1986;146:111–2.
698. Stiles GM, Singh L, Imazaki G, Stiles QR. Thermodilution cardiac output studies as a cause of prosthetic valve bacterial endocarditis. *J Thorac Cardiovasc Surg* 1984;88:1035–7.
699. Tyndall RL, Lyle MM, Ironside KS. The presence of free-living amoebae in portable and stationary eye wash stations. *Am Ind Hyg Assoc J* 1987;48:933–4.
700. Bowman EK, Vass AA, Mackowski R, Owen BA, Tyndall RL. Quantitation of free-living amoebae and bacterial populations in eyewash stations relative to flushing frequency. *Am Ind Hyg Assoc J* 1996;57:626–33.
701. Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986;7:82–5.
702. (309) Kates SG, McGinley KJ, Larson EL, Leyden JJ. Indigenous multiresistant bacteria from flowers in hospital and nonhospital environments. *Am J Infect Control* 1991;19:156–61.

703. Zanetti F, Stampi S, De L, et al. Water characteristics associated with the occurrence of *Legionella pneumophila* in dental units. *Eur J Oral Sci* 2000;108:22–8.
704. Peel MM, Calwell JM, Christopher PJ, Harkness JL, Rouch GJ. *Legionella pneumophila* and water temperatures in Australian hospitals. *Aust NZ J Med* 1985;15:38–41.
705. Groothuis DG, Veenendaal HR, Dijkstra HL. Influence of temperature on the number of *Legionella pneumophila* in hot water systems. *J Appl Bacteriol* 1985;59:529–36.
706. Plouffe JF, Webster LR, Hackman B. Relationship between colonization of a hospital building with *Legionella pneumophila* and hot water temperatures. *Appl Environ Microbiol* 1983;46:769–70.
707. (12) Alary Ma, Joly JR. Factors contributing to the contamination of hospital water distribution systems by Legionellae. *J Infect Dis* 1992;165:565–9.
708. U.K. Health & Safety Executive. The control of legionellosis in hot and cold water systems. Supplement to the control of Legionellosis, including Legionnaires' disease. London: Health & Safety Executive Office, 1998;1–4.
709. (167) Marrie TJ, Haldane D, Bezanson G, Peppard R. Each water outlet is a unique ecologic niche for *Legionella pneumophila*. *Epidemiol Infect* 1992;108:261–70.
710. (154) Snyder MB, Siwicki M, Wireman J, et al. Reduction of *Legionella pneumophila* through heat flushing followed by continuous supplemental chlorination of hospital hot water. *J Infect Dis* 1990;162:127–32.
711. (155) Ezzeddine H, Van Ossel C, Delmee M, Wauters G. *Legionella* spp. in a hospital hot water system: effect of control measures. *J Hosp Infect* 1989;13:121–31.
712. Reichert M. Automatic washers/disinfectors for flexible endoscopes. *Infect Control Hosp Epidemiol* 1991;12:497–9.
713. (45) Joint Commission on Accreditation of Healthcare Organizations. Hospital accreditation standards, 2001: environment of care. Oakbrook Terrace, IL: JCAHO Press, 2001;193–220.
714. (161) Best M, Yu VL, Stout J, Goetz A, Muder RR, Taylor F. *Legionellaceae* in the hospital water supply: epidemiologic link with disease and evaluation of a method for control of nosocomial Legionnaires' disease and Pittsburgh pneumonia. *Lancet* 1983;2:307–10.
715. CDC. Emergency Response Planning and Coordination. Available at: www.cdc.gov/nceh/emergency/emergency.htm
716. McGlown KJ, Fottler MD. The impact of flooding on the delivery of hospital services in the southeastern United States. *Health Care Manage Rev* 1996;21:55–71.
717. Fisher, HL. Emergency evacuation of the Denver Veteran's Administration Medical Center. *Milit Med* 1986;151:154–61.
718. Peters, MS. Hospitals respond to water loss during the midwest floods of 1993: preparedness and improvisation. *J Emerg Med* 1996;14:345–50.
719. (156) Joint Commission on Accreditation of Healthcare Organizations. Comprehensive Accreditation manual for hospitals: the official handbook (CAH00SJ). Oakbrook Terrace, IL: JCAHO Press, 2000.
720. Hargreaves J, Shireley L, Hansen S, et al. Bacterial contamination associated with electronic faucets: a new risk for healthcare facilities. *Infect Control Hosp Epidemiol* 2001;22:202–5.
721. (158) CDC. National surveillance of dialysis-associated diseases in the United States, 1997. Atlanta, GA: Public Health Service, U.S. Department of Health and Human Services, 1998.
722. Stout JE, Best ME, Yu VL. Susceptibility of members of the family *Legionellaceae* to thermal stress: implications for heat eradication methods in water distribution systems. *Appl Environ Microbiol* 1986;52:396–9.
723. Bornstein N, Vieilly C, Nowiki M, Paucod JC, Fleurette J. Epidemiological evidence of legionellosis transmission through domestic hot water supply systems and possibilities of control. *Isr J Med Sci* 1986;13:39–40.
724. (162) Meenhorst PL, Reingold AL, Groothuis DG, et al. Water-related nosocomial pneumonia caused by *Legionella pneumophila* serogroups 1 and 10. *J Infect Dis* 1985;152:356–64.
725. (216) Mandel AS, Sprauer MA, Sniadack DH, Ostroff SM. State regulation in hospital water temperature. *Infect Control Hosp Epidemiol* 1993;14:642–5.
726. (168) Department of Health. The control of *Legionella* in health care premises: a code of practice. London: HMSO, 1991.
727. (169) Helms CM, Massanari RM, Wenzel RP, et al. Legionnaires' disease associated with a hospital water system: a five-year progress report on continuous hyperchlorination. *JAMA* 1988;259:2423–7.

728. (170) Edelstein PH, Whittaker RE, Kreiling RL, Howell, CL. Efficacy of ozone in eradication of *Legionella pneumophila* from hospital plumbing fixtures. *Appl Environ Microbiol* 1982;44:1330–4.
729. (171) Muraca P, Stout JE, Yu, VL. Comparative assessment of chlorine, heat, ozone, and UV light for killing *Legionella pneumophila* within a model plumbing system. *Appl Environ Microbiol* 1987;53:447–53.
730. (172) Domingue EL, Tyndall RL, Mayberry WR, Pancorbo OC. Effects of three oxidizing biocides on *Legionella pneumophila* serogroup 1. *Appl Environ Microbiol* 1988;54:741–7.
731. (173) Landeen LK, Yahya MT, Gerba CP. Efficacy of copper and silver ions and reduced levels of free chlorine in inactivation of *Legionella pneumophila*. *Appl Environ Microbiol* 1989;55:3045–50.
732. (174) Matulonis U, Rosenfeld CS, Shaddock RK. Prevention of *Legionella* infections in bone marrow transplant unit: multifaceted approach to decontamination of a water system. *Infect Control Hosp Epidemiol* 1993;14:571–83.
733. (175) Liu Z, Stout JE, Tedesco L, et al. Controlled evaluation of copper-silver ionization in eradicating *Legionella pneumophila* from a hospital water distribution system. *J Infect Dis* 1994;169:919–22.
734. (176) Margolin AB. Control of microorganisms in source water and drinking water. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenback LD, Walter MV, eds. *Manual of environmental microbiology*. Washington, DC: American Society for Microbiology Press, 1997;195–202.
735. (177) Freije MR. *Legionella* control in health care facilities: a guide for minimizing risk. HC Information Resources, Inc. 1996;65–75.
736. (178) Yu-sen E, Lin R, Vidic D, Stout JE, Yu VL. *Legionella* in water distribution systems. *J AWWA* 1998;90:112–21.
737. (179) Biurrun A, Caballero L, Pelaz C, Leon E, Gago A. Treatment of a *Legionella pneumophila*-colonized water distribution system using copper-silver ionization and continuous chlorination. *Infect Control Hosp Epidemiol* 1999;20:426–8.
738. (180) Goetz A, Yu VL. Copper-silver ionization: cautious optimism for *Legionella* disinfection and implications for environmental culturing. *Am J Infect Control* 1997;25:449–51.
739. (181) Stout JE, Lin YS, Goetz AM, Muder RR. Controlling *Legionella* in hospital water systems: experience with the superheat-and-flush method and copper-silver ionization. *Infect Control Hosp Epidemiol* 1998;19:911–4.
740. (182) Walker JT, Mackerness CW, Mallon D, Makin T, Williets T, Keevil CW. Control of *Legionella pneumophila* in a hospital water system by chlorine dioxide. *J Ind Microbiol* 1995;15:384–90.
741. (183) Hambidge A. Reviewing efficacy of alternative water treatment techniques. *Health Estate* 2001;55:23–5.
742. (184) Rohr U, Senger M, Selenka F, Turley R, Wilhelm M. Four years of experience with silver-copper ionization for control of *Legionella* in a German university hospital hot water plumbing system. *Clin Infect Dis* 1999;29:1507–11.
743. (185) Cunliffe DA. Inactivation of *Legionella pneumophila* by monochloramine. *J Appl Bacteriol* 1990;68:453–9.
744. (186) Kirmeyer GJ, Foust GW, Pierson GL, Simmler JJ, LeChevalier MW. Optimizing chloramine treatment. Denver, CO: American Water Works Research Foundation, 1993.
745. (187) Kool JL, Carpenter JC, Fields BS. Effect of monochloramine disinfection of municipal drinking water on risk of nosocomial Legionnaires' disease. *Lancet* 1999;353:272–7.
746. (188) Kool JL, Bergmire-Sweat D, Butler JC, et al. Hospital characteristics associated with colonization of water systems by *Legionella* and risk of nosocomial Legionnaires' disease: a cohort study of 15 hospitals. *Infect Control Hosp Epidemiol* 1999;20:798–805.
747. (213) Yu VL, Beam TR Jr, Lumish RM, et al. Routine culturing for *Legionella* in the hospital environment may be a good idea: a three-hospital prospective study. *Am J Med Sci* 1987;294:97–9.
748. Allegheny County Health Department. Approaches to prevention and control of *Legionella* infection in Allegheny County health care facilities. Pittsburgh, PA: Allegheny County Health Department, 1997;1–13.
749. Goetz AM, Stout JE, Jacobs SL, et al. Nosocomial Legionnaires' disease discovered in community hospitals following cultures of the water system: seek and ye shall find. *Am J Infect Control* 1998;26:8–11.
750. Maryland Department of Health and Mental Hygiene. Report of the Maryland Scientific Working Group to study *Legionella* in the water systems in healthcare institutions. Available at: www.dhmd.state.md.us/html/legionella.htm
751. Yu VL. Nosocomial legionellosis: Current epidemiologic issues. In: Remington JS, Swartz MN, eds. *Current clinical topics in infectious diseases*. New York, NY: McGraw-Hill, 1986;239–53.

752. Vickers RM, Yu VL, Hanna SS. Determinants of *Legionella pneumophila* contamination of water distribution systems: 15-hospital prospective study. *Infect Control* 1987;8:357–63.
753. **(214)** Tobin JO, Swann RA, Bartlett CLR. Isolation of *Legionella pneumophila* from water systems: methods and preliminary results. *Br Med J* 1981;282:515–7.
754. Marrie TJ, Bezanson G, Fox J, Kuehn R, Haldane D, Birbridge S. Dynamics of *Legionella pneumophila* in the potable water of one floor of a hospital. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;238–40.
755. Plouffe JF, Para MF, Maher WE, Hackman B, Webster L. Subtypes of *Legionella pneumophila* serogroup 1 associated with different attack rates. *Lancet* 1983;2:649–50.
756. Fraser DW. Sources of legionellosis. In: Thornsberry C, Balows A, Feeley JC, Jakubowski W, eds. *Legionella: Proceedings of the 2nd International Symposium*. Washington, DC: American Society for Microbiology Press, 1994:277–80.
757. Dourmon E, Bibb WF, Rajagopalan P, Desplaces N, McKinney RM. Monoclonal antibody reactivity as a virulence marker for *Legionella pneumophila* serogroup 1 strain. *J Infect Dis* 1992;165:569–73.
758. Brundrett GW. Guides on avoiding Legionnaires' disease. In: *Legionella and building services*. Oxford, UK: Butterworth Heineman, 1992;346–73.
759. **(191)** Kugler JW, Armitage JO, Helms CM, et al. Nosocomial Legionnaires' disease: occurrence in recipients of bone marrow transplants. *Am J Med* 1983;74:281–8.
760. Lepine LA, Jernigan DB, Butler JC, et al. A recurrent outbreak of nosocomial Legionnaires' disease detected by urinary antigen testing: evidence for long-term colonization of a hospital plumbing system. *Infect Control Hosp Epidemiol* 1998;19:905–10.
761. Barbaree JM. Selecting a subtyping technique for use in investigations of legionellosis epidemics. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;169–72.
762. Joly JR, McKinney RM, Tobin JO, Bibb WF, Watkins ID, Ramsay D. Development of a standardized subgrouping scheme for *Legionella pneumophila* serogroup 1 using monoclonal antibodies. *J Clin Microbiol* 1986;23:768–71.
763. **(209)** Schoonmaker D, Helmberger T, Birkhead G. Comparison of ribotyping and restriction enzyme analysis using pulsed-field gel electrophoresis for distinguishing *Legionella pneumophila* isolates obtained during a nosocomial outbreak. *J Clin Microbiol* 1992;30:1491–8.
764. **(163)** Johnston JM, Latham RH, Meier FA, et al. Nosocomial outbreak of Legionnaires' disease: molecular epidemiology and disease control measures. *Infect Control* 1987;853–8.
765. Best MG, Goetz A, Yu VL. Heat eradication measures for control of nosocomial Legionnaires' disease: Implementation, education, and cost analysis. *Infect Control* 1984;12:26–30.
766. **(164)** Muraca PW, Yu VL, Goetz A. Disinfection of water distribution systems for *Legionella*: a review of application procedures and methodologies. *Infect Control Hosp Epidemiol* 1990;11:79–88.
767. **(210)** Knirsch CA, Jakob K, Schoonmaker D, et al. An outbreak of *Legionella micdadei* pneumonia in transplant patients: evaluation, molecular epidemiology, and control. *Am J Med* 2000;108:290–5.
768. **(211)** CDC. Sustained transmission of nosocomial Legionnaires' Disease — Arizona and Ohio. *MMWR* 1997;46:416–21.
769. **(218)** Patterson WJ, Hay J, Seal DV, McLuckie JC. Colonization of transplant unit water supplies with *Legionella* and protozoa: precautions required to reduce the risk of legionellosis. *J Hosp Infect* 1997;37:7–17.
770. U.S. Department of Labor, Occupational Safety and Health Administration. OSHA technical manual, Section III, Chapter 7. Legionellosis. Available at: www.osha-slc.gov/dts/osta/otm/otm_iii/otm_iii_7.html
771. Rudnick JR, Beck-Sague CM, Anderson RL, Schable B, Miller JM, Jarvis WR. Gram-negative bacteremia in open-heart surgery patients traced to probable tap-water contamination of pressure-monitoring equipment. *Infect Control Hosp Epidemiol* 1996;17:281–5.
772. Miller RP. Cooling towers and evaporative condensers. *Ann Intern Med* 1979;90:667–70.
773. Butler JC, Breiman RF. Legionellosis. In: Evans AS, Brachman PS, eds. *Bacterial infections of humans*, 3rd ed. New York, NY: Plenum Medical, 1998;355–76.
774. Witherell LE, Novick LF, Stone KM, et al. *Legionella* in cooling towers. *J Environ Health* 1986;49:134–9.
775. Cordes LG, Fraser DW, Skaliy P, et al. Legionnaires' disease outbreak at an Atlanta, Georgia country club: evidence for spread from an evaporative condenser. *Am J Epidemiol* 1980;111:425–31.

776. Kaufmann AF, McDade JE, Patton CM, et al. Pontiac fever: isolation of the etiologic agent (*Legionella pneumophila*) and demonstration of its mode of transmission. *Am J Epidemiol* 1981;114:337–47.
777. Morton S, Bartlett CLR, Bibby LF, Hutchinson DM, Dyer JV, Dennis PJ. Outbreak of Legionnaires' disease from a cooling water system in a power station. *Br J Indust Med* 1986;43:630–5.
778. Friedman S, Spitalny K, Barbaree J, Faur Y, McKinney R. Pontiac fever outbreak associated with a cooling tower. *Am J Public Health* 1987;77:568–72.
779. Addiss DG, Davis JP, LaVenture M, Wand PJ, Hutchinson MA, McKinney RM. Community-acquired Legionnaires' disease associated with a cooling tower: evidence for longer-distance transport of *Legionella pneumophila*. *Am J Epidemiol* 1989;130:557–68.
780. Keller DW, Hajjeh R, DeMaria A Jr, et al. Community outbreak of Legionnaires' disease: an investigation confirming the potential for cooling towers to transmit *Legionella* species. *Clin Inf Dis* 1996;22:257–61.
781. Pastoris MC, Ciceroni L, Lo Monaco R, et al. Molecular epidemiology of an outbreak of Legionnaires' disease associated with a cooling tower in Genova-Sestri Ponente, Italy. *Eur J Clin Microbiol Infect Dis* 1997;16:883–92.
782. Brown CM, Nuorti PJ, Breiman RF, et al. A community outbreak of Legionnaires' disease linked to hospital cooling towers: an epidemiological method to calculate dose of exposure. *Inter J Epidemiol* 1999;28:353–9.
783. Broadbent CR. *Legionella* in cooling towers: Practical research, design, treatment, and control guidelines. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;217–22.
784. (222) Bhopal RS, Barr G. Maintenance of cooling towers following two outbreaks of Legionnaires' disease in a city. *Epidemiol Infect* 1990;104:29–38.
785. CDC. Suggested health and safety guidelines for public spas and hot tubs. Atlanta, GA: Centers for Disease Control, 1985. Publication No. 99–960.
786. (221) World Health Organization. Environmental aspects of the control of Legionellosis, 14th ed. Copenhagen, Denmark: World Health Organization, 1986. Schriftenr Ver Wasser Boden Lufthyg 1993;91:249–52. (German)
787. (223) World Health Organization. Epidemiology, prevention, and control of legionellosis: memorandum from a WHO meeting. *Bull WHO* 1990;68:155–64.
788. Association for the Advancement of Medical Instrumentation. American National Standard Hemodialysis Systems ANSI/AAMI RD5-1981, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1982.
789. (229) Association for the Advancement of Medical Instrumentation. American National Standard Hemodialysis Systems ANSI/AAMI RD5-1992, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1993.
790. Association for the Advancement of Medical Instrumentation. Reuse of hemodialyzers ROH-1986, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1986.
791. (230) Association for the Advancement of Medical Instrumentation. American National Standard Reuse of hemodialyzers ANSI/AAMI RD47-1993, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 1993.
792. (236) Association for the Advancement of Medical Instrumentation. Water treatment equipment for hemodialysis applications. ANSI/AAMI RD62-2001, Association for the Advancement of Medical Instrumentation. Arlington, VA: AAMI, 2001.
793. Tokars JI, Miller ER, Alter MJ, Arduino MJ. National surveillance of dialysis associated diseases in the United States, 1997. *Semin Dialysis* 2000;13:75–85.
794. Hindman SH, Carson LA, Petersen NJ, et al. Pyrogenic reactions during hemodialysis caused by extramural endotoxin. *Lancet* 1975;2:732–4.
795. Stamm JE, Engelhard WE, Parson JE. Microbiological study of water softener resins. *Appl Microbiol* 1969;18:376–86.
796. Alter MJ, Favero MS, Miller JK, Coleman BJ, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1988. *ASAIO Trans* 1990;36:107–18.
797. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1990. *ASAIO J* 1993;39:71–80.
798. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1991. *ASAIO J* 1993;39:966–75.

799. Tokars JI, Alter MJ, Favero MS, Moyer LA, Bland LA. National surveillance of dialysis-associated diseases in the United States, 1993. *ASAIO J* 1996;42:219–29.
800. **(231)** Petersen NJ, Boyer KM, Carson LA, Favero MS. Pyrogenic reactions from inadequate disinfection of a dialysis unit distribution system. *Dialysis Transpl* 1978;7:52–7.
801. Gazenfeldt-Gazit E, Elaihou HE. Endotoxin antibodies in patients on maintenance hemodialysis. *Israel J Med Sci* 1969;5:1032–6.
802. Laude-Sharp M, Canoff M, Simard L, Pusineri C, Kazatchkine M, Haeffner-Cavaillon N. Induction of IL-1 during hemodialysis: transmembrane passage of intact endotoxin (LPS). *Kidney Int* 1990;38:1089–94.
803. Arduino MJ, Bland LA, McAllister SK, Favero MS. The effects of endotoxin contaminated dialysate and polysulfone or cellulosic membranes on the release of TNF α during simulated dialysis. *Artif Organs* 1995;19:880–6.
804. Greisman SE, Hornick RB. Comparative pyrogenic reactivity of rabbit and man to bacterial endotoxin. *Proc Soc Exp Biol Med* 1969;131:1154–8.
805. Weary ME, Donohue G, Pearson FC, Story K. Relative potencies of four reference endotoxin standards as measured by the *Limulus* amoebocyte lysate and USP rabbit pyrogen tests. *Appl Environ Microbiol* 1980;40:1148–51.
806. **(239)** Bland LA, Ridgeway MR, Aguerro SM, Carson LA, Favero MS. Potential bacteriologic and endotoxin hazards associated with liquid bicarbonate concentrate. *ASAIO Trans* 1987;33:542–5.
807. **(232)** Dawids SG, Vejlsgaard R. Bacteriological and clinical evaluation of different dialysate delivery systems. *Acta Med Scand* 1976;199:151–5.
808. Favero MS, Alter MJ, Tokars JI, Arduino MJ. Dialysis-associated infections and their control. In: Bennett JV, Brachman PS, eds. *Hospital infections* 4th ed. Philadelphia, PA: Lippincott-Raven, 1998;357–80.
809. **233** Kidd EE. Bacterial contamination of dialyzing fluid of artificial kidney. *Br Med J* 1964;1:880–2.
810. Jones DM, Tobin BM, Harlow GR, et al. Bacteriological studies of the modified kiil dialyzer. *Br Med J* 1970;3:135–7.
811. **(240)** Raji L, Shapiro FL, Michael AF. Endotoxemia in febrile reactions during hemodialysis. *Kidney Int* 1973;4:57–60.
812. Vanholder R, Van Haecke E, Veys N, et al. Endotoxin transfer through dialysis membranes: small versus large-pore membranes. *Nephrol Dial Transplant* 1992;7:333–9.
813. Evans RC, Holmes CJ. In vitro study of the transfer of cytokine-inducing substances across selected high-flux hemodialysis membranes. *Blood Purif* 1991;9:92–101.
814. Lonnemann G, Behme TC, Lenzer B, et al. Permeability of dialyzer membranes to TNF α -inducing substances derived from water bacteria. *Kidney Int* 1992;42:61–8.
815. Ureña P, Herbelin A, Zingraff J, et al. Permeability of cellulosic and non-cellulosic membranes to endotoxin subunits and cytokine production during in-vitro hemodialysis. *Nephrol Dial Transplant*. 1992;7:16–28.
816. **(241)** Bommer J, Becker KP, Urbaschek R. Potential transfer of endotoxin across high-flux polysulfone membranes. *J Am Soc Nephrol* 1996;7:883–8.
817. Yamagami S, Adachi T, Sugimura, T, et al. Detection of endotoxin antibody in long-term dialysis patients. *Int J Artif Organs* 1990;13:205–10.
818. Arduino MJ. CDC investigations of noninfectious outbreaks of adverse events in hemodialysis facilities, 1979–1999. *Semin Dialysis* 2000;13:86–91.
819. Roth V, Jarvis WR. Outbreaks of infection and/or pyrogenic reactions in dialysis patients. *Semin Dialysis* 2000;13:92–100.
820. Gordon SM, Tipple MME, Bland LA, Jarvis WR. Pyrogenic reactions associated with reuse of disposable hollow-fiber hemodialyzers. *JAMA* 1988;260:2077–81.
821. Alter MJ, Tokars JI, Arduino MJ. Nosocomial infections in hemodialysis units — strategies for control. In: Owen WF, Periera BJG, Sayegh MH, eds. *Dialysis and transplantation: a companion to Brenner and Rector's "The Kidney."* Orlando, FL: WB Saunders Company, 1999;337–57.
822. Bernick JJ, Port FK, Favero MS, Brown DG. Bacterial and endotoxin permeability of hemodialysis membranes. *Kidney Int* 1979;16:491–6.
823. Bommer J, Becker KP, Urbaschek R, Ritz E, Urbaschek B. No evidence for endotoxin transfer across high flux polysulfone membranes. *Clin Nephrol* 1987;27:278–82.
824. Schindler R, Lonnemann G, Schaeffer J, et al. The effect of ultrafiltered dialysate on the cellular content of interleukin-1 receptor antagonist in patients on chronic hemodialysis. *Nephron* 1994;68:229–33.

825. Akrum RAE, Frolich M, Gerritsen AF, et al. Improvement of chronic inflammatory state in hemodialysis patients by the use of ultrapure water for dialysate. *J Am Soc Nephrol* 1997;8:226A.
826. Quellhorst E. Methods of Hemodialysis. *Nieren U Hochdruck* 1998;27:35–41.
827. Baz M, Durand C, Ragon A, et al. Using ultrapure water in hemodialysis delays carpal tunnel syndrome. *Int J Artif Organs* 1991;14:681–5.
828. Schwalbe S, Holzhauser M, Schaeffer J, et al. β 2-Microglobulin associated amyloidosis: a vanishing complication of long-term hemodialysis? *Kidney Int* 1997;52:1077–83.
829. (242) Arduino MJ, Favero MS. Microbiologic aspects of hemodialysis: water quality for hemodialysis. AAMI Monograph WQD-1998. Arlington, VA: Association for the Advancement of Medical Instrumentation, 1998.
830. Leyboldt JK, Schmidt B, Gurland, HJ. Measurement of backfiltration rates during hemodialysis with highly permeable membranes. *Blood Purif* 1991;9:74–84.
831. Carson LA, Bland LA, Cusick LB, Collin S, Favero MS, Bolan G. Factors affecting endotoxin levels in fluids associated with hemodialysis procedures. In: Novitsky TJ, Watson SW, eds. Detection of bacterial endotoxins with the Limulus Amoebocyte Lysate Test. New York, NY: Alan R. Liss, 1987;223–4.
832. Anderson RL, Holland BW, Carr JK, Bond WW, Favero MS. Effect of disinfectants on pseudomonads colonized on the interior surface of PVC pipes. *Am J Public Health* 1990;80:17–21.
833. Bland LA, Favero MS. Microbial contamination control strategies for hemodialysis. *JCAHO Plant Tech Manage Series* 1989;3:30–6.
834. (237) Bland LA. Microbiological and endotoxin assays of hemodialysis fluids. *Adv Renal Replacement Ther* 1995;2:70–9.
835. (238) Arduino MJ, Bland LA, Aguero SM, Carson LA, Ridgeway M, Favero MS. Comparison of microbiologic assay methods for hemodialysis fluids. *J Clin Microbiol* 1991;29:592–4.
836. Association for the Advancement of Medical Instrumentation. American national standard water treatment equipment for hemodialysis applications. ANSI/AAMI RD62-1999. Arlington, VA: Association for the Advancement of Medical Instrumentation, 1999.
837. Arduino MJ. How should dialyzers be reprocessed? *Semin Dialysis* 1998;11:282–4.
838. Jochimsen EM, Frenette C, Delorme M, et al. A cluster of bloodstream infections and pyrogenic reactions among hemodialysis patients traced to dialysis machine waste-handling option units. *Am J Nephrol* 1998;18:485–9.
839. Wang SA, Levine RB, Carson LA, et al. An outbreak of gram-negative bacteremia in hemodialysis patients traced to hemodialysis machine waste drain ports. *Infect Control Hosp Epidemiol* 1999;20:746–51.
840. National Institutes of Health. U.S. Renal Diseases Survey: 1999 Annual Data Report. Bethesda, MD: National Institute of Diabetes, Digestive and Kidney Diseases, Division of Kidney, Urologic, and Hematologic Diseases, 1999.
841. Monsen T, Olofson C, Ronnmark M, Wistrom J. Clonal spread of staphylococci among patients with peritonitis associated with continuous ambulatory peritoneal dialysis. *ASAIO J* 2000;57:613–8.
842. Band JD, Ward JI, Fraser DW, et al. Peritonitis due to a *Mycobacterium chelonae*-like organism associates with intermittent chronic peritoneal dialysis. *J Infect Dis* 1982;145:9–17.
843. Monsen T, Crabtree JH, Siddiqui RA, et al. Dialysis catheter infection related peritonitis: incidence and time dependent risk. *ASAIO J* 1999;45:574–80.
844. Vera G, Lew SQ. *Mycobacterium fortuitum* peritonitis in two patients receiving continuous ambulatory peritoneal dialysis. *Am J Nephrol* 1999;19:586–9.
845. Soriano F, Rodriguez-Tudela JL, Gomez-Garces JL, Velo M. Two possibly related cases of *Mycobacterium fortuitum* peritonitis in continuous ambulatory peritoneal dialysis. *Eur J Clin Microbiol* 1989;8:895–7.
846. Szeto CC, Li PK, Leung CB, Yu AW, Lui SF, Lai NK. *Xanthomonas maltophilia* peritonitis in uremic patients receiving ambulatory peritoneal dialysis. *Am J Kidney Dis* 1997;29:991–5.
847. Panlilio AL, Beck-Sague CM, Siegel JD, et al. Infections and pseudoinfections due to povidone-iodine solution contaminated with *Pseudomonas cepacia*. *Clin Infect Dis* 1992;14:1078–83.
848. Riebel W, Frantz N, Adelstein D, Spanguolo PJ. *Corynebacterium JK*: a cause of nosocomial device-related infection. *Rev Infect Dis* 1986;8:42–9.
849. Radix AE, Bieluch VM, Graeber CW. Peritonitis caused by *Monilia sitophila* in a patient undergoing peritoneal dialysis. *Int J Artif Organs* 1996;19:218–20.
850. Banerjee S, Marwaha RK, Bajwa RP. Fungal peritonitis complicating peritoneal dialysis. *Indian Pediatr* 1995;32:693–7.

851. Bergeson E, Denis R, Cartier P. Peritoneal dialysis: peritonitis and catheter infections. *Annales de Chirurgie* 1996;50:606–12. (French)
852. Troidle L, Kliger AS, Goldie SJ, et al. Continuous peritoneal dialysis-associated peritonitis of nosocomial origin. *Perit Dialysis International* 1996;16:505–10.
853. Smith CA. Reduced incidence of peritonitis by utilizing “flush before fill” in APD. *Adv Perit Dialysis* 1997;13:224–6.
854. Valeri A, Radhakrishnan J, Vernocchi L, Carmichael LD, Stern L. The epidemiology of peritonitis in acute peritoneal dialysis: a comparison between open- and closed drainage systems. *Am J Kidney Dis* 1993;21:300–9.
855. Stamm WE, Colelle JJ, Anderson RL, Dixon RE. Indwelling arterial catheters as a source of nosocomial bacteremia: an outbreak caused by *Flavobacterium* species. *N Engl J Med* 1975;292:1099–102.
856. Schimpff SC. Gram negative bacteremia. *Support Care Cancer* 1993;1:5–18.
857. Graman PS, Quinlan GA, Rank JA. Nosocomial legionellosis traced to contaminated ice. *Infect Control Hosp Epidemiol* 1997;18:637–40.
858. Gahrn-Hansen B, Uldum SA, Schmidt J, Nielsen B, Birkeland SA, Jorgensen KA. [Nosocomial *Legionella pneumophila* infection in a nephrology department]. *Ugeskrift for Laeger* 1995;157:590–4. (German)
859. Wilson IG, Hogg GM, Barr JG. Microbiological quality of ice in hospital and community. *J Hosp Infect* 1997;36:171–80.
860. Spencer RC. The emergence of epidemic, multiple-antibiotic-resistant *Stenotrophomonas (Xanthomonas) maltophilia* and *Burkholderia (Pseudomonas) cepacia*. *J Hosp Infect* 1995;30(suppl):453–64.
861. (248) Cannon RO, Poliner JR, Hirschhorn RB, et al. A multistate outbreak of Norwalk virus gastroenteritis associated with consumption of commercial ice. *J Infect Dis* 1991;164:860–3.
862. (249) Khan AS, Moe CL, Glass RI, et al. Norwalk virus-associated gastroenteritis traced to ice consumption aboard a cruise ship in Hawaii: comparison and application of molecular method-based assays. *J Clin Microbiol* 1994;32:318–22.
863. (244) CDC. Outbreak of viral gastroenteritis — Pennsylvania and Delaware. *MMWR* 1987;36:709–11.
864. Quick R, Paugh K, Addiss D, Kobayashi J, Baron R. Restaurant-associated outbreak of giardiasis. *J Infect Dis* 1992;166:673–6.
865. Hedberg CW, White KE, Johnson JA, et al. An outbreak of *Salmonella enteritidis* infection at a fast food restaurant: implications for foodhandler-associated transmission. *J Infect Dis* 1991;164:1135–40.
866. Burnett IA, Weeks GR, Harris DM. A hospital study of ice-making machines: their bacteriology, design, usage, and upkeep. *J Hosp Infect* 1994;28:305–13.
867. Petersen NJ. Don't culture the ice machines. *Hosp Infect Control* 1982;9:8–9.
868. CDC. Sanitary care and maintenance of ice chests and ice machines. Atlanta, GA: CDC, 1979. Publication No. 00-2384.
869. (247) Manangan LP, Anderson RL, Arduino MJ, Bond WW. Sanitary care and maintenance of ice-storage chests and ice-making machines in healthcare facilities. *Am J Infect Control* 1998;26:111–2.
870. Anonymous. Ice as a source of infection. *CDR Weekly* 1993;3:241.
871. Cardaney CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacteria contamination. *J Burn Care Rehab* 1985;6:230–2.
872. Gruber RP, Laub DR, Vistnes LM. The effect of hydrotherapy on the clinical course and pH of experimental cutaneous chemical burns. *Plastic Reconstruct Surg* 1975;55:200–4.
873. Mansell RE, Borchardt KA. Disinfecting hydrotherapy equipment. *Arch Phys Med Rehabil* 1974;55:318–20.
874. Hall J, Skevington SM, Maddison PH, Chapman K. A randomized and controlled trial of hydrotherapy in rheumatoid arthritis. *Arthritis Care Res* 1996;9:206–15.
875. Gross A, Cutright DE, Bhaskar SN. Effectiveness of pulsating water jet lavage in treatment of contaminated crush injuries. *Am J Surgery* 1972;124:373–7.
876. Rodeheaver GT, Paltry D, Thacker JG, Edgerton MT, Edlich RF. Wound cleansing by high pressure irrigation. *Surg Gynecol Obstetr* 1975;141:357–62.
877. Saxe A, Goldestein E, Dixon S, Ostrup R. Pulsatile lavage in the management of postoperative wound infections. *Am Surgeon* 1980;46:391–7.
878. Weller K. In search of efficacy and efficiency: an alternative to conventional wound cleansing modalities. *Ostomy/Wound Manage* 1991;37:23–8.
879. Solomon SL. Host factors in whirlpool-associated *Pseudomonas aeruginosa* skin disease. *Infect Control* 1985;6:402–6.

880. Hicks CB, Chulay JD. Bacteremic *Citrobacter freundii* cellulitis associated with tub immersion in a patient with the nephrotic syndrome. *Mil Med* 1988;153:400–1.
881. Mayhall CG, Lamb VA, Gayle WE, Haynes BW. *Enterobacter cloacae* septicemia in a burn center: epidemiology and control of an outbreak. *J Infect Dis* 1979;139:166–71.
882. Marrie TJ, Gass RSR, Yates L. *Legionella pneumophila* in a physiotherapy pool. *Eur J Clin Microbiol* 1987;6:212–3.
883. Havelaar AH, Berwald LG, Groothuis DG, Baas JG. Mycobacteria in semi-public swimming pools and whirlpools. *Ztb Bakteriol Mikrobiol Hyg [B]* 1985;180:505–14.
884. Favero MS. Whirlpool spa-associated infections: are we really in hot water? *Am J Public Health* 1984;74:653–5.
885. Ratnam S, Hogan K, March SB, Butler RW. Whirlpool-associated folliculitis caused by *Pseudomonas aeruginosa*: report of an outbreak and review. *J Clin Microbiol* 1986;23:655–9.
886. Stone HH, Kolb LD. The evolution and spread of gentamicin-resistant *Pseudomonas*. *J Trauma* 1971;11:586–9.
887. Richard P, LeFlock R, Chamoux C, Pannier M, Espaze E, Richet H. *Pseudomonas aeruginosa* outbreak in a burn unit: role of antimicrobials in the emergence of multiply resistant strains. *J Infect Dis* 1994;170:377–83.
888. Berrouane YF, McNutt L-A, Buschelman BJ, et al. Outbreak of severe *Pseudomonas aeruginosa* infections caused by a contaminated drain in a whirlpool bathtub. *Clin Infect Dis* 2000;31:1331–7.
889. (250) Schmidt OW, Cooney MK, Foy HM. Adeno-associated virus in adenovirus type 3 conjunctivitis. *Infect Immun* 1975;11:1362–70.
890. DeJonckheere JF. Hospital hydrotherapy pools treated with ultraviolet light: bad bacteriological quality and presence of thermophilic *Naegleria*. *J Hyg (Lond)* 1982;88:205–14.
891. American Physical Therapy Association. Hydrotherapy/therapeutic pool infection control guidelines. Alexandria, VA: APTA, 1995;112.
892. CDC. Disinfection of hydrotherapy pools and tanks. Atlanta, GA: Centers for Disease Control, Public Health Service, U.S. Department of Health and Human Services, 1974. Publication No. HHS 00-2383.
893. Price D, Ahearn DG. Incidence and persistence of *Pseudomonas aeruginosa* in whirlpools. *J Clin Microbiol* 1988;26:1650–4.
894. (252) White CG. Chemistry of chlorination. In: Handbook of chlorination and alternative disinfectants, 3rd ed. New York, NY: Van Nostrand Reinhold, 1992;184–249.
895. Mayhall CG. Burn patients. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC), 2000;32.1–32.8.
896. Smith RF, Blasi D, Dayton SL, Chipps DD. Effects of sodium hypochlorite on the microbial flora of burns and normal skin. *J Trauma* 1974;14:938–44.
897. Cardany CR, Rodeheaver GT, Horowitz JH, Kenney JG, Edlich RF. Influence of hydrotherapy and antiseptic agents on burn wound bacterial contamination. *J Burn Care Rehabil* 1985;6:230–2.
898. Steve L, Goodhart P, Alexander J. Hydrotherapy burn treatment: use of chloramine-T against resistant microorganisms. *Arch Phys Med Rehabil* 1979;60:301–3.
899. Golland A. Basic hydrotherapy. *Physiotherapy* 1981;67:258–62.
900. Edlich RF, Becker DG, Phung D, McClelland WA, Day SG. Water treatment of hydrotherapy exercise pools. *J Burn Care Rehabil* 1988;9:9510–5.
901. Penny PT. Hydrotherapy pools of the future — the avoidance of health problems. *J Hosp Infect* 1991;18:535–42.
902. CDC. Swimming pools: safety and disease control through proper design and operation. Atlanta, GA: U.S. Department of Health and Human Services, 1976. Publication No. HHS No. 88–8319.
903. Linneman CC Jr. Nosocomial infections associated with physical therapy, including hydrotherapy. In: Mayhall CG, ed. Hospital epidemiology and infection control, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;931–6.
904. Aspinall ST, Graham R. Two sources of contamination of a hydrotherapy pool by environmental organisms. *J Hosp Infect* 1989;14:285–92.
905. (251) McCandlish R, Renfrew M. Immersion in water during labor and birth: the need for evaluation. *Birth* 1993;20:79–85.
906. Hawkins S. Water vs conventional births: Infection rates compared. *Nursing Times* 1995;91:38–40.

907. Vochem M, Vogt M, Doring G. Sepsis in a newborn due to *Pseudomonas aeruginosa* from a contaminated tub bath. *N Engl J Med* 2001;345:378–9.
908. Eriksson M, Ladfors L, Mattsson LA, Fall O. Warm tub bath during labor: a study of 1385 women with prelabor rupture of the membranes after 34 weeks of gestation. *Acta Obstet Gynaecol Scand* 1996;75:642–4.
909. Rush J, Burlock S, Lambert K, Loosley-Millman M, Hutchinson B, Enkin M. The effects of whirlpool baths in labor: a randomized, controlled trial. *Birth* 1996;23:136–3.
910. Davis BJ. Whirlpool operation and the prevention of infection. *Infect Control* 1985;6:394–7.
911. (253) Muscarella LF. Automatic flexible endoscope reprocessors. *Gastrointest Endosc Clin N Am* 2000;10:245–57.
912. (254) Muscarella LF. Anticipated reliability of liquid chemical sterilants [letter]. *Am J Infect Control* 1998;26:155–6.
913. (255) Muscarella LF. Dejà vu... all over again? The importance of instrument drying [letter]. *Infect Control Hosp Epidemiol* 2000;21:628–9.
914. (256) Gubler JGH, Salfinger M, von Graevenitz A. Pseudoepidemic of nontuberculous mycobacteria due to a contaminated bronchoscope cleaning machine: report of an outbreak and review of the literature. *Chest* 1992;101:1245–9.
915. (257) Fraser VJ, Jones M, Murray PR, Medoff G, Zhang Y, Wallace RJ Jr. Contamination of flexible fiberoptic bronchoscopes with *Mycobacterium chelonae* linked to an automated bronchoscope disinfection machine. *Am Rev Respir Dis* 1992;145:853–5.
916. Maloney S, Welbel S, Daves B, et al. *Mycobacterium abscessus* pseudo-infection traced to an automated endoscope washer: utility of epidemiologic and laboratory investigation. *J Infect Dis* 1994;169:1166–9.
917. Merighi A, Contato E, Scagliarini R, et al. Quality improvement in gastrointestinal endoscopy: microbiologic surveillance of disinfection. *Gastrointest Endosc* 1996;43:457–62.
918. (258) Muscarella LF. Application of environmental sampling to flexible endoscope reprocessing: the importance of monitoring the rinse water. *Infect Control Hosp Epidemiol* 2002;23:285–9.
919. Mitchell DH, Hicks LJ, Chiew R, Montanaro JC, Chen SC. Pseudoepidemic of *Legionella pneumophila* serogroup 6 associated with contaminated bronchoscopes. *J Hosp Infect* 1997;37:19–23.
920. Ido K, Ishino Y, Ota Y, et al. Deficiencies of automatic endoscopic reprocessors: a method to achieve high-grade disinfection of endoscopes. *Gastrointest Endosc* 1996;44:583–6.
921. (260) Allen JJ, Allen MO, Olsen MM, et al. *Pseudomonas* infection of the biliary system resulting from the use of a contaminated endoscope. *Gastroenterology* 1987;92:759–63.
922. Agerton T, Valway S, Gore B, et al. Transmission of a highly drug-resistant strain (Strain W-1) of *Mycobacterium tuberculosis*: community outbreak and nosocomial transmission via a contaminated bronchoscope. *JAMA* 1997;278:1073–7.
923. (261) Michele TM, Cronin WA, Graham NMH, et al. Transmission of *Mycobacterium tuberculosis* by a fiberoptic bronchoscope: identification by DNA fingerprinting. *JAMA* 1997;278:1093–5.
924. Bronowicki J-P, Venard V, Botte C, et al. Patient-to-patient transmission of hepatitis C virus during colonoscopy. *N Engl J Med* 1997;337:237–40.
925. (262) U.S. Food and Drug Administration, CDC. FDA and CDC Public health advisory: infection from endoscopes inadequately reprocessed by an automated endoscope reprocessing system. September 10, 1999. Available at: www.fda.gov/cdrh/safety.html
926. Rey JF. Endoscopic disinfection. A worldwide problem. *J Clin Gastroenterol* 1999;28:291–7.
927. Wang HC, Liaw YS, Yang PC, Kuo SH, Luh KT. A pseudoepidemic of *Mycobacterium chelonae* infection caused by contamination of a fiberoptic bronchoscope suction channel. *Eur Respir J* 1995;8:1259–62.
928. (263) Alvarado CJ, Reichelderfer M. APIC guideline for infection prevention and control in flexible endoscopy. *Am J Infect Control* 2000;28:138–55.
929. Van Klingeren B, Pullen W. Glutaraldehyde resistant mycobacteria from endoscope washers. *J Hosp Infect* 1993;25:147–9.
930. Flournoy DJ, Petrone RL, Voth DW. A pseudo-outbreak of *Methylobacterium mesophilica* isolated from patients undergoing bronchoscopy. *Eur J Clin Microbiol Infect Dis* 1992;11:240–3.
931. Reeves DS, Brown NM. Mycobacterial contamination of fiberoptic bronchoscopes. *J Hosp Infect* 1995;30(suppl):S531–S536.
932. Kelstrup J, Funder-Nielsen T, Theilade J. Microbial aggregate contamination of water lines in dental equipment and its control. *Acta Path Scand* 1977;85:177–83.

933. Challacombe SJ, Fernandes LL. Detecting *Legionella pneumophila* in water systems: a comparison of various dental units. *J Am Dent Assoc* 1995;126:603–8.
934. Singh R, Stine OC, Smith DL, Spitznagel JK Jr, Labib ME, Williams HN. Microbial diversity of biofilms in dental unit water systems. *Appl Environ Microbiol* 2003;69:3412–20.
935. (264) CDC. Statement from the Centers for Disease Control and Prevention (CDC) regarding biofilm and dental unit water quality. Atlanta, GA: U.S. Public Health Service, Department of Health and Human Services, 1999. Available at: www.cdc.gov/nccdphp/oh/ic-fs-biofilm.htm
936. (265) CDC. Recommended infection control practices for dentistry, 1993. *MMWR* 1993;42(No. RR-8):1–12.
937. (268) Bagga BS, Murphy RA, Anderson AW, Punwani I. Contamination of dental unit cooling water with oral microorganisms and its prevention. *J Am Dent Assoc* 1984;109:712–6.
938. Scheid RC, Rosen S, Beck FM. Reduction of CFUs in high-speed handpiece water lines over time. *Clin Prev Dent* 1990;12:9–12.
939. Williams JF, Johnston AM, Johnson B, Huntington MK, Mackenzie CD. Microbial contamination of dental unit waterlines: prevalence, intensity, and microbiological characteristics. *J Am Dent Assoc* 1993;124:59–65.
940. Santiago JI, Huntington MK, Johnston AM, Quinn RS, Williams JF. Microbial contamination of dental unit waterlines: short- and long-term effects of flushing. *Gen Dent* 1994;42:528–35.
941. Williams HN, Johnson A, Kelley JI, et al. Bacterial contamination of the water supply in newly installed dental units. *Quintessence Int* 1995;26:331–7.
942. CDC. Guideline for infection control in dental health-care settings. *MMWR* 2003;52:in press.
943. (266) Office of Safety and Asepsis Procedures Research Foundation. Position paper on dental unit waterlines. Annapolis, MD: OSAPRF, 2000. Available at: www.osap.org/issues/pages/water/duwl.htm
944. (267) U.S. Environmental Protection Agency. National primary drinking water regulations, 40 CFR 1, Part 141, Subpart G;1999. At: www.epa.gov/safewater/mcl.html
945. (344) Eaton AD, Clesceri LS, Greenberg AE, eds. Standard methods for the examination of water and wastewater, 20th ed. Washington, DC: American Public Health Association, 1998;9–1 through 9–41.
946. (269) Shearer BG. Biofilm and the dental office. *J Am Dent Assoc* 1996;127:181–9.
947. Maki DG, Alvarado CJ, Hassemer CA, Zilz MA. Relation of the inanimate hospital environment to endemic nosocomial infection. *N Engl J Med* 1982;307:1562–6.
948. Danforth D, Nicolle LE, Hume K, Alfieri N, Sims H. Nosocomial infections on nursing units with floors cleaned with a disinfectant compared with detergent. *J Hosp Infect* 1987;10:229–35.
949. Spaulding EH. Role of chemical disinfection in the prevention of nosocomial infections. In: Brachman PS, Eickhoff TC, eds. *Proceedings of the International Conference on Nosocomial Infections, 1970*. Chicago, IL: American Hospital Association, 1971;247–54.
950. Spaulding EH. Chemical disinfection and antisepsis in the hospital. *J Hosp Res* 1972;9:5–31.
951. (273) Favero MS, Bond WW. Chemical disinfection of medical and surgical materials. In: Block SS, ed. *Disinfection, sterilization, and preservation, 5th ed.* Philadelphia, PA: Lippincott Williams & Wilkins, 2001;881–917.
952. (274) Rutala WA. APIC guideline for selection and use of disinfectants. *Am J Infect Control* 1996;24:313–42.
953. Agolini G, Russo A, Clementi M. Effect of phenolic and chlorine disinfectants on hepatitis C virus binding and infectivity. *Am J Infect Control* 1999;27:236–9.
954. (279) Favero MS, Bond WW. Sterilization, disinfection, and antisepsis in the hospital. In: Balows A, Hausler WJ Jr, Herrmann KL, Isenberg HD, Shadomy HJ, eds. *Manual of clinical microbiology, 5th ed.* Washington, DC: American Society for Microbiology, 1991;183–200.
955. Nyström B. Bioburden of non-disposable surgical instruments and operating room textiles. In: Gaughran ERL, Morrissey RF, eds. *Sterilization of medical products, Vol II*. Montreal, Québec: Multiscience Publications Ltd., 1981;156–63.
956. Nyström B. Disinfection of surgical instruments. *J Hosp Infect* 1981;2:3636–8.
957. Rutala WA, Weber DJ. FDA labeling requirements for disinfection of endoscopes: a counterpoint. *Infect Control Hosp Epidemiol* 1995;16:231–5.
958. Parker HH IV, Johnson RD. Effectiveness of ethylene oxide for sterilization of dental handpieces. *J Dent* 1995;1:1–3.

959. Alfa MJ, DeGagne P, Olson N, Puchalski T. Comparison of ion plasma, vaporized hydrogen peroxide, and 100% ethylene oxide sterilizers to the 12/88 ethylene oxide gas sterilizer. *Infect Control Hosp Epidemiol* 1996;17:92–100.
960. Rutala WA, Gergen MF, Jones JF, Weber DJ. Levels of microbial contamination on surgical instruments. *Am J Infect Control* 1998;26:143–5.
961. (275) Stingeni L, Lapomarda V, Lisi P. Occupational hand dermatitis in hospital environments. *Contact Dermatitis* 1995;33:172–6.
962. (276) Ashdown BC, Stricof DD, May ML, Sherman SJ, Carmody RF. Hydrogen peroxide poisoning causing brain infarction: neuroimaging findings. *AM J Roentgenol* 1998;170:1653–5.
963. (277) Busch A, Werner E. [Animal tolerance to peracetic acid. 1. Experimental results following the application of peracetic acid solutions on the skin of pigs]. *Monatshefte für Veterinaermedizin* 1974;29:494–8. (German)
964. (278) U.S. Food and Drug Administration. Medical devices: adequate directions for use. 21 CFR Part 801.5, 807.87.e.
965. U.S. Food and Drug Administration (FDA) and U.S. Environmental Protection Agency (EPA). Memorandum of understanding between the Food and Drug Administration, Public Health Service, Department of Health and Human Services, and the Environmental Protection Agency: Notice regarding matters of mutual responsibility — regulation of liquid chemical germicides intended for use on medical devices, 1993. Available from FDA, Center for Devices and Radiological Health (CDRH), Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850, or EPA, Registration Division, Antimicrobial Program Branch, 401 M St., SW, Washington DC 20460.
966. U.S. Food and Drug Administration (FDA). Interim measures for the registration of antimicrobial products/liquid chemical germicides with medical device use claims under the memorandum of understanding between EPA and FDA, 1994. CDRH Facts on Demand, Shelf #851, p. 14; June 30, 1994. Available from FDA, CDRH, Office of Health and Industry Programs, Division of Small Manufacturers Assistance, Rockville MD 20850.
967. (293) U.S. Department of Labor, Occupational Safety and Health Administration. Occupational Exposure to Bloodborne Pathogens: final rule (29 CFR 1910.1030). *Federal Register* 1991;56:64004–182.
968. Collins BJ. The hospital environment: how clean should a hospital be? *J Hosp Infect* 1988;11 (Suppl A): 53–6.
969. Van den Berg RWA, Claahsen HL, Niessen M, Muytjens HL, Liem K, Voss A. *Enterobacter cloacae* outbreak in the NICU related to disinfected thermometers. *J Hosp Infect* 2000;45:29–34.
970. Spaulding EH. Alcohol as a surgical disinfectant. *AORN J* 1964;2:67–71.
971. (282) Ayliffe GAJ, Collins BJ, Lowbury EJJ, Babb JR, Lilly HA. Ward floors and other surfaces as reservoirs of hospital infection. *J Hyg (Camb)* 1967;65:515–37.
972. (283) Dancer SJ. Mopping up hospital infection. *J Hosp Infect* 1999;43:85–100.
973. Gable TS. Bactericidal effectiveness of floor cleaning methods in a hospital environment. *Hospitals JAHA* 1966;40:107–11.
974. (271) U.S. Environmental Protection Agency. Federal Insecticide, Fungicide, and Rodenticide Act. 7 USC 6 § 136 et seq.; 1972. Available at: www4.law.cornell.edu/uscode/7/ch6schII.html
975. Petersen NJ, Marshall JH, Collins DE. Why wash walls in hospital isolation rooms? *Health Lab Sci* 1973;10:23–7.
976. (285) Mallison GF. Decontamination, disinfection, and sterilization. *Nurs Clin North Am* 1980;15:757–67.
977. Ayliffe GAJ, Collins BJ, Lowbury EJJ. Cleaning and disinfection of hospital floors. *Br Med J* 1966;2:442–5.
978. Vesley D, Pryor AK, Walter WG, Shaffer JG. A cooperative microbiological evaluation of floor-cleaning procedures in hospital patient rooms. *Health Lab Sci* 1970;7:256–64.
979. Daschner J, Rabbenstein G, Langmaack H. [Flächenkontamination zur Verhütung und Bekämpfung von Drakenhausinfektionen.] *Deutsche Medizinische Wochenschrift* 1980;10:325–9. (German)
980. Dharan S, Mourouga P, Copin P, Bessmer G, Tschanz B, Pittet D. Routine disinfection of patients' environmental surfaces: Myth or reality? *J Hosp Infect* 1999;42:113–7.
981. Palmer PH, Yeoman DM. A study to assess the value of disinfectants when washing ward floors. *Med J Australia* 1972;2:1237–9.
982. (284) Schmidt EA, Coleman DL, Mallison GF. Improved system for floor cleaning in health care facilities. *Appl Environ Microbiol* 1984;47:942–6.

983. (272) Mallison GF. Hospital disinfectants for housekeeping: floors and tables. *Infect Control* 1984;5:537.
984. Vesley D, Klapes NA, Benzow K, Le CT. Microbiological evaluation of wet and dry floor sanitization systems in hospital patient rooms. *Appl Environ Microbiol* 1987;53:1042–5.
985. Werry C, Lawrence JM, Sanderson PJ. Contamination of detergent cleaning solutions during hospital cleaning. *J Hosp Infect* 1988;11:44–9.
986. (280) Chou T. Environmental services. In: APIC Text of Infection Control and Epidemiology. Pfeiffer J, ed. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc.;2000;73.1–73.8.
987. (281) Rutala WA, Weber D. General information on cleaning, disinfection, and sterilization. In: Pfeiffer J, ed. APIC Text of infection control and epidemiology. Washington, DC: Association for Professionals in Infection Control and Epidemiology, Inc (APIC);2000:55.1–55.6.
988. (286) Walter CW, Kundsinn RB. The floor as a reservoir of hospital infections. *Surg Gynec Obstet* 1960;111:412–22.
989. (287) Scott E, Bloomfield SF. The survival and transfer of microbial contamination via cloths, hands and utensils. *J Appl Bacteriol* 1990;68:271–8.
990. (288) Scott E, Bloomfield SF. Investigations of the effectiveness of detergent washing, drying and chemical disinfection on contamination of cleaning cloths. *J Appl Bacteriol* 1990;68:279–83.
991. Givan KF, Black BL, Williams PF. Multiplication of *Pseudomonas* species in phenolic germicidal detergent solution. *Can J Pub Health* 1971;62:72.
992. Thomas MEM, Piper E, Maurer IM. Contamination of an operating theater by gram-negative bacteria: examination of water supplies, cleaning methods, and wound infections. *J Hyg (Camb)* 1972;70:63–73.
993. Medcraft JW, Hawkins JM, Fletcher BN, Dadswell JV. Potential hazard from spray cleaning of floors in hospital wards. *J Hosp Infect* 1987;9:151–7.
994. (289) Brown DG, Schaltzle K, Gable T. The hospital vacuum cleaner: mechanism for redistributing microbial contaminants. *J Environ Health* 1980;42:192–6.
995. (290) Wysowski DK, Flynt JW, Goldfield M, et al. Epidemic hyperbilirubinemia and use of a phenolic disinfectant detergent. *Pediatrics* 1978;61:165–70.
996. (291) Doan HM, Keith L, Shennan AT. Phenol and neonatal jaundice. *Pediatrics* 1979;64:324–5.
997. (292) American Academy of Pediatrics, American College of Obstetricians and Gynecologists. Infection control. In: Guidelines for perinatal care, 4th ed. Evanston, IL: AAP, ACOG, 1997;269–74.
998. (294) Spire B, Montagnier L, Barré-Sinoussi F, Chermann JC. Inactivation of lymphadenopathy associated virus by chemical disinfectants. *Lancet* 1984;2:899–901.
999. (295) Martin LS, McDougal JS, Loskoski SL. Disinfection and inactivation of the human T lymphotropic virus type-III/lymphadenopathy-associated virus. *J Infect Dis* 1985;152:400–3.
1000. (296) Hanson PJ, Gor D, Jeffries DJ, Collins JV. Chemical inactivation of HIV on surfaces. *Br Med J* 1989;298:862–4.
1001. (297) Bloomfield SF, Smith-Burchnell CA, Dalglish AG. Evaluation of hypochlorite-releasing disinfectants against the human immunodeficiency virus (HIV). *J Hosp Infect* 1990;15:273–8.
1002. (298) Druce JD, Jardine D, Locarnini SA, Birch CJ. Susceptibility of HIV to inactivation by disinfectants and ultraviolet light. *J Hosp Infect* 1995;30:167–80.
1003. (299) Van Bueren J, Simpson RA, Salman H, Farrelly HD, Cookson BD. Inactivation of HIV-1 by chemical disinfectants: sodium hypochlorite. *Epidemiol Infect* 1995;115:567–79.
1004. (300) Prince DL, Prince HN, Thraehart O, et al. Methodological approaches to disinfection of human hepatitis viruses. *J Clin Microbiol* 1993;31:3296–304.
1005. Tabor E, Gerety RJ. A survey of formalin inactivation of hepatitis A virus, hepatitis B virus, and a non-A, non-B hepatitis agent. In: Second International Max von Pettenkofer Symposium on Viral Hepatitis. Munich, Germany: October 1982.
1006. Thraehart O, Kuwert EK, Scheiermann N, et al. Comparison of the morphological alteration and disintegration test (MADT) and the chimpanzee infectivity test for determination of hepatitis B virucidal activity of chemical disinfectants. *Zentralbl Bakteriol Mikrobiol Hyg (B)* 1982;176:472–84.
1007. (303) U.S. Department of Labor, Occupational Safety and Health Administration. EPA-registered disinfectants for HIV/HBV. [Memorandum 2/28/97]. Available at: www.osha-slc.gov/OshDoc/Interp_data/I19970228C.html
1008. U.S. Environmental Protection Agency. Lists A, B, C, D, E, and F: EPA registered disinfectants, sanitizers and sterilants. Available at: www.epa.gov/oppad001/chemregindex.htm

1009. U.S. Environmental Protection Agency. Protocols for testing the efficacy of disinfectants against hepatitis B. *Federal Register* 2000;65:51828–30.
1010. (301) CDC. Recommendations for prevention of HIV transmission in health-care settings. *MMWR* 1987;36(No.2S):1S–18S.
1011. (304) Weber DJ, Barbee SL, Sobsey MD, Rutala WA. The effect of blood on the antiviral activity of sodium hypochlorite, a phenolic, and a quaternary ammonium compound. *Infect Control Hosp Epidemiol* 1999;20:821–7.
1012. (302) Sattar SA, Springthorpe VS. Survival and disinfectant inactivation of the human immunodeficiency virus: a critical review. *Rev Infect Dis* 1991;13:430–47.
1013. (400) CDC, National Institutes of Health. Biosafety in microbiological and biomedical laboratories, 4th ed. Washington, DC: U.S. Government Printing Office, 1999.
1014. Lee R. The advantages of carpets in mental hospitals. *Ment Hosp* 1965;16:324–5.
1015. Simmons D, Reizenstein J, Grant M. Considering carpets in hospital use. *Dimensions* 1982;June:18–21.
1016. Willmott M. The effect of a vinyl floor surface and a carpeted floor surface upon walking in elderly hospital in-patients. *Age Ageing* 1986;15:119–20.
1017. Shaffer J, Key I. The microbiological effects of carpeting on the hospital environment. *Hospitals JAHA* 1966;40:126–39.
1018. Walter W, Stober A. Quantitative and qualitative microbial studies of hospital carpets. *J Environ Health* 1967;30:293–300.
1019. Anderson RL. Biological evaluation of carpeting. *Appl Microbiol* 1969;18:180–7.
1020. Lanese RR, Keller MD, Macpherson CR, Covey RC. A study of microflora on tiled and carpeted surfaces in a hospital nursery. *Am J Public Health* 1973;63:174–8.
1021. Bonde GJ. Bacterial flora of synthetic carpets in hospitals. *Health Lab Sci* 1973;10:308–18.
1022. Rylander R, Myrback K, Verner-Carlson B, Ohrstrom M. Bacteriological investigation of wall-to-wall carpeting. *Am J Public Health* 1974;64:163–8.
1023. (305) Suzuki A, Namba Y, Matsuura M, Horisawa A. Bacterial contamination of floors and other surfaces in operating rooms: a five-year survey. *J Hyg (Camb)* 1984;93:559–66.
1024. Skoutelis AT, Westenfelder GO, Beckerdite M, Phair JP. Hospital carpeting and epidemiology of *Clostridium difficile*. *Am J Infect Control* 1993;22:212–7.
1025. Anderson RL, Mackel DC, Stoler BS, Mallison GF. Carpeting in hospitals: an epidemiological evaluation. *J Clin Microbiol* 1982;15:408–15.
1026. (160) Vesper S, Dearborn DG, Yike I, et al. Evaluation of *Stachybotrys chartarum* in the house of an infant with pulmonary hemorrhage: quantitative assessment before, during, and after remediation. *J Urban Health* 2000;77:68–85.
1027. Bakker PGH, Faogali JL. The effect of carpet on the number of microbes in the hospital environment. *N Zeal Med J* 1977;85:88–92.
1028. (306) Richet H, McNeil M, Pewters W, et al. *Aspergillus flavus* in a bone marrow transplant unit (BMTU): Pseudofungemia traced to hallway carpeting [abstract]. In: Abstracts of the 89th Annual Meeting of the American Society for Microbiology, 1989.
1029. CDC. Respiratory illness associated with carpet cleaning at a hospital clinic — Virginia. *MMWR* 1983;32:378,383–4.
1030. Maley MP. Bacterial threats to new hospitals. *Lancet* 1997;350:223–4.
1031. U.S. Environmental Protection Agency. Office of Toxic Substances. Efficacy data requirements. Supplemental recommendations DIS/TSS-2;1/25/79.
1032. (307) U.S. Department of Labor, Occupational Safety and Health Administration. OSHA Standards Interpretation and Compliance Letters;6/10/94: Decontamination of a plush carpet surface after a spill. Available at: www.osha-slc.gov/OshDoc/Interp_data/I19940610.html
1033. Richards K. New York City: special suites — Memorial Sloan Kettering. *Interiors* 1998;157:56–9.
1034. Noskin BA, Bednarz P, Suriano T, Reiner S, Peterson LR. Persistent contamination of fabric-covered furniture by vancomycin-resistant enterococci: implications for upholstery selection in hospitals. *Am J Infect Control* 2000;28:311–3.
1035. Sanderson PJ, Alshafi KM. Environmental contamination by organisms causing urinary tract infection. *J Hosp Infect* 1995;29: 301–3.
1036. Babe KS Jr, Arlian LG, Confer PD, Kim R. House dust mite (*Dermatophagoides farinae* and *Dermatophagoides pteronyssinus*) prevalence in the rooms and hallways of a tertiary care hospital. *J Allergy Clin Immunol* 1995;95:801–5.

1037. Custovic A, Fletcher A, Pickering CAC, et al. Domestic allergens in public places III: house dust mite, cat, dog, and cockroach allergens in British hospitals. *Clin Exper Allergy* 1998;28:53–9.
1038. Ansorg R, Thomssen R, Stubbe P. *Erwinia* species causing fatal septicemia in a newborn. *Med Microbiol Immunol (Berl)* 1974;159:161–70.
1039. Trust TJ, Bartlett KH. Isolation of *Pseudomonas aeruginosa* and other bacterial species from ornamental aquarium plants. *Appl Environ Microbiol* 1976;31:992–4.
1040. (310) Bartzokas CA, Holley MP, Sharp CA. Bacteria in flower vase water: incidence and significance in general ward practice. *Br J Surg* 1975;62:295–7.
1041. Watson AG, Koons CE. *Pseudomonas* on the chrysanthemums. *Lancet* 1973;2:91.
1042. (311) Siegman-Igra Y, Shalem A, Berger SA, Livio S, Michaeli D. Should potted plants be removed from hospital wards? *J Hosp Infect* 1986;7:82–5.
1043. Rosenzweig AL. Contaminated flower vases. *Lancet* 1973;2:568–9.
1044. Johansen KS, Laursen H, Wilhjelmsen BJ. Flower vases as reservoirs of pathogens. *Lancet* 1974;1:359.
1045. Rogues AM, Quesnel C, Revel P, Saric J, Gachie JP. Potted plants as a potential reservoir of *Fusarium* species. *J Hosp Infect* 1997;35:163–4.
1046. (312) Lass-Flörl C, Rath P, Niederwieser D, et al. *Aspergillus terreus* infections in haematological malignancies: molecular epidemiology suggests association with in-hospital plants. *J Hosp Infect* 2000;46:31–5.
1047. Levine OS, Levine MM. Houseflies (*Musca domestica*) as mechanical vectors of shigellosis. *Rev Infect Dis* 1991;13:688–96.
1048. Šrámová H, Daniel M, Absolonová V, Dědičová D, Jedličková Z, Lhotová H, Petráš P, Subertová V. Epidemiological role of arthropods detectable in health facilities. *J Hosp Infect* 1992;20:281–92.
1049. Tan SW, Yap KL, Lee HL. Mechanical transport of rotavirus by the legs and wings of *Musca domestica* (Diptera: *Muscidae*). *J Med Entomol* 1997;34:527–31.
1050. (313) Burgess NR. Hospital design and cockroach control. *Trans R Soc Trop Med Hyg* 1984;78:293–4.
1051. Ash N, Greenberg B. Vector potential for the German cockroach (Diptera: *Blattellidae*) in dissemination of *Salmonella enteritidis* serotype typhimurium. *J Med Entomol* 1980;17:417–23.
1052. Fotedar R, Banerjee U. Nosocomial fungal infections — study of the possible role of cockroaches (*Blattella germanica*) as vectors. *Acta Trop* 1992;50:339–43.
1053. Rosef O, Kapperud G. House flies (*Musca domestica*) as possible vectors of *Campylobacter fetus* subsp. jejuni. *Appl Environ Microbiol* 1983;45:381–3.
1054. Forsey T, Darougar S. Transmission of chlamydiae by the housefly. *Br J Ophthalmol* 1981;65:147–50.
1055. Grübel P, Hoffman JS, Chong FK, Burstein NA, Mepani C, Cave DR. Vector potential of houseflies (*Musca domestica*) for *Helicobacter pylori*. *J Clin Microbiol* 1997;35:1300–3.
1056. Oothuman P, Jeffery J, Aziz, AHA, Bakar EA, Jegathesan M. Bacterial pathogens isolated from cockroaches trapped from pediatric wards in peninsular Malaysia. *Trans R Soc Trop Med Hyg* 1989;83:133–5.
1057. Beatson SH. Pharaoh's ants as pathogen vectors in hospitals. *Lancet* 1972;1:425–7.
1058. LeGuyader A, Rivault C, Chaperon J. Microbial organisms carried by brown-banded cockroaches in relation to their spatial distribution in a hospital. *Epidemiol Infect* 1989;102:485–92.
1059. Fotedar R, Banerjee U, Shriniwas, Verma A. Cockroaches (*Blattella germanica*) as carriers of microorganisms of medical importance in hospitals. *Epidemiol Infect* 1991;107:181–7.
1060. Fotedar R, Banerjee U, Singh S, Shriniwas, Verma AK. The housefly (*Musca domestica*) as a carrier of pathogenic microorganisms in a hospital environment. *J Hosp Infect* 1992;20:209–15.
1061. Fotedar R, Shriniwas, Banerjee U, Samantray JC, Nayar E, Verma A. Nosocomial infections: cockroaches as possible vectors of drug-resistant *Klebsiella*. *J Hosp Infect* 1991;18:155–9.
1062. Devi SJN, Murray CJ. Cockroaches (*Blatta* and *Periplaneta* species) as reservoirs of drug-resistant salmonellas. *Epidemiol Infect* 1991;107:357–64.
1063. Cotton MF, Wasserman E, Pieper CH, et al. Invasive disease due to extended spectrum beta-lactamase-producing *Klebsiella pneumoniae* in a neonatal unit: the possible role of cockroaches. *J Hosp Infect* 2000;44:13–7.
1064. Baker LF. Pests in hospitals. *J Hosp Infect* 1981;2:5–9.
1065. (316) Allen BW. Excretion of viable tubercle bacilli by *Blatta orientalis* (the oriental cockroach) following ingestion of heat-fixed sputum smears: a laboratory investigation. *Trans R Soc Trop Med Hyg* 1987;81:98–9.

1066. (317) Laszlo A. Technical guide: sputum examination for tuberculosis by direct microscopy in low income countries, 5th ed. Paris, France: International Union Against Tuberculosis and Lung Disease, 2000. Available at: www.iatld.org/html/body_guides.htm
1067. Cohen D, Green M, Block C, et al. Reduction of transmission of shigellosis by control of houseflies (*Musca domestica*). *Lancet* 1991;337:993–7.
1068. Daniel M, Šrámová H, Zálabská E. *Lucilia sericata* (Diptera: Calliphoridae) causing hospital-acquired myiasis of a traumatic wound. *J Hosp Infect* 1994;28:149–52.
1069. Jacobson JA, Kolts RL, Conti M, Burke JP. Hospital-acquired myiasis. *Infect Control* 1980;1:319–20.
1070. Mielke U. Nosocomial myiasis. *J Hosp Infect* 1997;37:1–5.
1071. Sherman RA. Wound myiasis in urban and suburban United States. *Arch Intern Med* 2000;160:2004–14.
1072. (314) Lukin LG. Human cutaneous myiasis in Brisbane: a prospective study. *Med J Aust* 1989;150:237–40.
1073. Watkins M, Wyatt T. A ticklish problem: pest infestation in hospitals. *Prof Nurse* 1989;4:389–92.
1074. Schoninger S. Pest control and extermination in health care facilities. *Prof Sanit Manage* 1978;9:24–7.
1075. (315) Bruesch J. Institutional pest management: current trends. *Exec Housekeep Today* 1994;15:6–12.
1076. Tenover FC. VRSA, VISA, GISA: the dilemma behind the name game. *Clin Microbiol Newsletter* 2000;22:49–53.
1077. CDC. National Nosocomial Infections Surveillance System (NNIS): Semiannual report. December 1999. Available at: www.cdc.gov/ncidod/hip/SURVEILL/NNIS.HTM
1078. Hartstein AI, Mulligan ME. Methicillin-resistant *Staphylococcus aureus*. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;347–64.
1079. Walsh TJ, Vlahov D, Hansen SL, et al. Prospective microbiologic surveillance in control of nosocomial methicillin-resistant *Staphylococcus aureus*. *Infect Control* 1987;8:7–14.
1080. Walsh TJ, Auger F, Tatem BA, Hansen SL, Standford HJ. Novobiocin and rifampin in combination against methicillin-resistant *Staphylococcus aureus*: an in vitro comparison with vancomycin plus rifampin. *J Antimicrob Chemother* 1986;17:75–82.
1081. McNeil MM, Solomon SL. The epidemiology of MRSA. *Antimicrobiol Newsl* 1985;2:49–56.
1082. Oie S, Kamiya A. Survival of methicillin-resistant *Staphylococcus aureus* (MRSA) on naturally contaminated dry mops. *J Hosp Infect* 1996;34:145–9.
1083. Arnow PM, Allyn PA, Nichols EM, Hill DL, Pezzlo M, Bartlett RH. Control of methicillin-resistant *Staphylococcus aureus* in a burn unit: role of nurse staffing. *J Trauma* 1982;22:954–9.
1084. (329) Karanfil LV, Murphy M, Josephson A, et al. A cluster of vancomycin-resistant *Enterococcus faecium* in an intensive care unit. *Infect Control Hosp Epidemiol* 1992;13:195–200.
1085. Handwerker S, Raucher B, Altarac D, et al. Nosocomial outbreak due to *Enterococcus faecium* highly resistant to vancomycin, penicillin, and gentamicin. *Clin Infect Dis* 1993;16:750–5.
1086. Boyle JF, Soumakis SA, Rendo A, et al. Epidemiologic analysis and genotypic characterization of a nosocomial outbreak of vancomycin-resistant enterococci. *J Clin Microbiol* 1993;31:1280–5.
1087. (330) Boyce JM, Opal SM, Chow JW, et al. Outbreak of multidrug-resistant *Enterococcus faecium* with transferable vanB class vancomycin resistance. *J Clin Microbiol* 1994;32:1148–53.
1088. (331) Rhinehart E, Smith NE, Wennerstein C, et al. Rapid dissemination of beta-lactamase-producing, aminoglycoside-resistant *Enterococcus faecalis* among patients and staff on an infant-toddler surgical ward. *N Engl J Med* 1990;323:1814–8.
1089. Crossley K, Landesman B, Zaske D. An outbreak of infections caused by strains of *Staphylococcus aureus* resistant to methicillin and aminoglycosides. II. epidemiologic studies. *J Infect Dis* 1979;139:280–7.
1090. Peacock JE Jr, Marsik FJ, Wenzel RP. Methicillin-resistant *Staphylococcus aureus*: introduction and spread within a hospital. *Ann Intern Med* 1980;93:526–32.
1091. Walsh TJ, Hansen SL, Tatem BA, Auger F, Standford HJ. Activity of novobiocin against methicillin-resistant *Staphylococcus aureus*. *J Antimicrob Chemother* 1985;15:435–40.
1092. (332) Livornese LL Jr, Sias S, Samel C, et al. Hospital-acquired infection with vancomycin-resistant *Enterococcus faecium* transmitted by electronic thermometers. *Ann Intern Med* 1992;117:112–6.
1093. Gould FK, Freeman R. Nosocomial infection with microsphere beds. *Lancet* 1993;342:241–2.

1094. Morris JG, Shay DK, Hebden JN, et al. *Enterococci* resistant to multiple antimicrobial agents, including vancomycin: establishment of endemicity in a university medical center. *Ann Intern Med* 1995;123:250–9.
1095. Edmond MB, Ober JS, Weinbaum DL, et al. Vancomycin-resistant *Enterococcus faecium* bacteremia: risk factors for infection. *Clin Infect Dis* 1995;20:1126–33.
1096. (333) Zervos MJ, Kauffman CA, Therasse PM, Bergman AG, Mikesell TS, Schaberg DR. Nosocomial infection by gentamicin-resistant *Streptococcus faecalis*: an epidemiologic study. *Ann Intern Med* 1987;106:687–91.
1097. Zervos MJ, Dembinski S, Mikesell T, Schaberg DR. High-level resistance to gentamicin in *Streptococcus faecalis*: risk factors and evidence for exogenous acquisition of infection. *J Infect Dis* 1986;153: 1075–83.
1098. Bonilla HF, Zervos MA, Lyons MJ, et al. Colonization with vancomycin-resistant *Enterococcus faecium*: comparison of a long-term-care unit with an acute-care hospital. *Infect Control Hosp Epidemiol* 1997;18:333–9.
1099. Bonilla HF, Zervos MJ, Kauffman CA. Long-term survival of vancomycin-resistant *Enterococcus faecium* on a contaminated surface. *Infect Control Hosp Epidemiol* 1996;17:770–1.
1100. Boyce JM, Bermel LA, Zervos MJ, et al. Controlling vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1995;16:634–7.
1101. Boyce JM, Potter-Bynoe G, Chenevert C, King T. Environmental contamination due to methicillin-resistant *Staphylococcus aureus*: possible infection control implications. *Infect Control Hosp Epidemiol* 1997;18:622–7.
1102. (328) Layton MC, Perez M, Heald P, Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993;14:369–75.
1103. Collins SM, Hacek DM, Degen LA, Wright MP, Noskin GA, Peterson LR. Contamination of the clinical microbiology laboratory with vancomycin-resistant enterococci and multidrug-resistant *Enterobacteriaceae*: implications for hospital and laboratory workers. *J Clin Microbiol* 2001;39:3772–4.
1104. Bonten MJM, Hayden MK, Nathan C, et al. Epidemiology of colonisation of patients and environment with vancomycin-resistant enterococci. *Lancet* 1996;348:1615–9.
1105. Wendt C, Wiesenthal B, Dietz E, Rüden H. Survival of vancomycin-resistant and vancomycin-susceptible enterococci on dry surfaces. *J Clin Microbiol* 1998;36:3734–46.
1106. (323) Bradley CR, Fraise AP. Heat and chemical resistance of enterococci. *J Hosp Infect* 1996;34:191–6.
1107. (324) Anderson RL, Carr JH, Bond WW, Favero MS. Susceptibility of vancomycin-resistant enterococci to environmental disinfectants. *Infect Control Hosp Epidemiol* 1997;18:195–9.
1108. (325) Saurina G, Landman D, Quale JM. Activity of disinfectants against vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1997;18:345–7.
1109. (326) Rutala WA, Stiegel MM, Sarubbi FA, Weber DJ. Susceptibility of antibiotic-susceptible and antibiotic-resistant hospital bacteria to disinfectants. *Infect Control Hosp Epidemiol* 1997;18:417–21.
1110. (327) Schulster LM, Anderson RL. Susceptibility of glycopeptide-intermediate resistant *Staphylococcus aureus* (GISA) to surface disinfectants, hand washing chemicals, and a skin antiseptic [abstract Y-3]. In: Abstracts of the 98th General Meeting, American Society for Microbiology. 1998.
1111. Armstrong-Evans M, Litt M, McArthur MA, et al. Control of transmission of vancomycin-resistant *Enterococcus faecium* in a long-term-care facility. *Infect Control Hosp Epidemiol* 1999;20:312–17.
1112. Global Consensus Conference: Final Recommendations. Global Consensus Conference on Infection Control Issues Related to Antimicrobial Resistance. *Am J Infect Control* 1999;27:503–13.
1113. Freeman R, Kearns AM, Lightfoot NF. Heat resistance of nosocomial enterococci. *Lancet* 1994;345:64–5.
1114. CDC. *Staphylococcus aureus* resistant to vancomycin — United States. *MMWR* 2002;51:565–7.
1115. CDC. Vancomycin resistant *Staphylococcus aureus* — Pennsylvania, 2002. *MMWR* 2002;51:902.
1116. (320) Weber DJ, Rutala WA. Role of environmental contamination in the transmission of vancomycin-resistant enterococci. *Infect Control Hosp Epidemiol* 1997;18:306–9.
1117. (321) Lai KK, Kelley AL, Melvin ZS, Belliveau PP, Fontecchio SA. Failure to eradicate vancomycin-resistant enterococci in a university hospital and the cost of barrier precautions. *Infect Control Hosp Epidemiol* 1998;19:647–2.

1118. (322) Byers KE, Durbin LJ, Simonton BM, Anglim AM, Adal KA, Farr BM. Disinfection of hospital rooms contaminated with vancomycin-resistant *Enterococcus faecium*. *Infect Control Hosp Epidemiol* 1998;19:261–4.
1119. Siegel DL, Edelstein PH, Nachamkin I. Inappropriate testing for diarrheal diseases in the hospital. *JAMA* 1990;263:979–82.
1120. Yannelli B, Gurevich I, Schoch PE, Cunha BA. Yield of stool cultures, ova and parasite tests, and *Clostridium difficile* determination in nosocomial diarrhea. *Am J Infect Control* 1988;16:246–9.
1121. Gerding DN, Olson MM, Peterson LR, et al. *Clostridium difficile*-associated diarrhea and colitis in adults: a prospective case-controlled epidemiologic study. *Arch Intern Med* 1986;146:95–100.
1122. Svenungsson B, Burman LG, Jalakas-Pörnnull K, Lagergren Å, Struwe J, Åkerlund T. Epidemiology and molecular characterization of *Clostridium difficile* strains from patients with diarrhea: low disease incidence and evidence of limited cross-infection in a Swedish teaching hospital. *J Clin Microbiol* 2003;41:4031–7.
1123. Barlett JG. Antibiotic-associated colitis. *Dis Mon* 1984;30:1–55.
1124. Pierce PF Jr, Wilson R, Silva J Jr, et al. Antibiotic-associated pseudomembranous colitis: an epidemiologic investigation of a cluster of cases. *J Infect Dis* 1982;145:269–74.
1125. Aronsson B, Möllby, Nord C-E. Antimicrobial agents and *Clostridium difficile* in acute enteric disease: epidemiologic data from Sweden, 1980–1982. *J Infect Dis* 1985;151:476–81.
1126. Thibault A, Miller MA, Gaese C. Risk factors for the development of *Clostridium difficile*-associated diarrhea during a hospital outbreak. *Infect Control Hosp Epidemiol* 1991;12:345–8.
1127. McFarland LV, Surawicz CM, Stamm WE. Risk factors for *Clostridium difficile* carriage and *Clostridium difficile*-associated diarrhea in a cohort of hospitalized patients. *J Infect Dis* 1990;162:678–84.
1128. Zadik PM, Moore AP. Antimicrobial associations of an outbreak of diarrhoea due to *Clostridium difficile*. *J Hosp Infect* 1998;39:189–93.
1129. Johnson S, Homann SR, Bettin KM, et al. Treatment of asymptomatic *Clostridium difficile* carriers (fecal excretors) with vancomycin or metronidazole: a randomized, placebo controlled trial. *Ann Intern Med* 1992;117:297–302.
1130. (319) Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J Jr. *Clostridium difficile*-associated diarrhea and colitis. *Infect Control Hosp Epidemiol* 1995;16:459–77.
1131. Titov L, Lebedkova N, Shabanov A, Tang YJ, Cohen SH, Silva J Jr. Isolation and molecular characterization of *Clostridium difficile* strains from patients and the hospital environment in Belarus. *J Clin Microbiol* 2000;38:1200–2.
1132. Mulligan ME, Rolfe RD, Finegold SM, George WL. Contamination of a hospital environment by *Clostridium difficile*. *Curr Microbiol* 1979;3:173–5.
1133. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr. Epidemiology of antibiotic-associated colitis: isolation of *Clostridium difficile* from the hospital environment. *Am J Med* 1981;70:906–8.
1134. Malamou-Ladas H, Farrell SO, Nash JO, Tabaqchali S. Isolation of *Clostridium difficile* from patients and the environment of hospital wards. *J Clin Pathol* 1983;6:88–92.
1135. Kaatz GW, Gitlin SD, Schaberg DR, et al. Acquisition of *Clostridium difficile* from the hospital environment. *Am J Epidemiol* 1988;127:1289–94.
1136. Cohen SH, Tang YJ, Muenzer J, Gumerlock PH, Silva J Jr. Isolation of various genotypes of *Clostridium difficile* from patients and the environment in an oncology ward. *J Infect Dis* 1997;889–93.
1137. Savage AM. Nosocomial spread of *Clostridium difficile*. *Infect Control* 1983;4:31–3.
1138. Brooks SE, Veal RO, Kramer M, Dore L, Schupf N, Adachi M. Reduction in the incidence of *Clostridium difficile*-associated diarrhea in an acute care hospital and a skilled nursing facility following replacement of electronic thermometers with single-use disposables. *Infect Control Hosp Epidemiol* 1992;13:98–103.
1139. Johnson S, Gerding DN, Olson MM, et al. Prospective, controlled study of vinyl glove use to interrupt *Clostridium difficile* nosocomial transmission. *Am J Med* 1990;88:137–40.
1140. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of *Clostridium difficile* infection. *New Engl J Med* 1989;320:204–10.
1141. Mayfield JL, Leet T, Miller J, Mundy LM. Environmental control to reduce transmission of *Clostridium difficile*. *Clin Inf Dis* 2000;31:995–1000.

1142. Wilcox MH, Fawley WN, Wigglesworth N, Parnell P, Verity P, Freeman J. Comparison of the effect of detergent versus hypochlorite cleaning on environmental contamination and incidence of *Clostridium difficile* infection. *J Hosp Infect* 2003;54:109–14.
1143. (334) Worsley MA. Infection control and prevention of *Clostridium difficile* infection. *J Antimicrobial Chemother* 1998;41 (Suppl. C):59–66.
1144. von Rheinbaben F, Schünemann S, Groß T, Wolff MH. Transmission of viruses via contact in a household setting: experiments using bacteriophage ϕ X174 as a model virus. *J Hosp Infect* 2000;46:61–6.
1145. Hall CB, Douglas G Jr, Gelman JM. Possible transmission by fomites of respiratory syncytial virus. *J Infect Dis* 1980;141:98–102.
1146. Brady MT, Evans J, Cuartas J. Survival and disinfection of parainfluenza viruses on environmental surfaces. *Am J Infect Control* 1990;18:18–23.
1147. Hendley JO, Wenzel RP, Gwaltney JM Jr. Transmission of rhinovirus colds by self-inoculation. *N Engl J Med* 1973;288:1361–4.
1148. Butz AM, Fosarelli P, Dick J, Cusack T, Yolken R. Prevalence of rotavirus on high-risk fomites in day-care facilities. *Pediatrics* 1993;92:202–5.
1149. Wilde J, Van R, Pickering LK, Eiden J, Yolken R. Detection of rotaviruses in the day care environment — detection by reverse transcriptase polymerase chain reaction. *J Infect Dis* 1992;166:507–11.
1150. Chapin M, Yatabe J, Cherry JD. An outbreak of rotavirus gastroenteritis on a pediatric unit. *Am J Infect Control* 1983;11:88–91.
1151. Appleton H, Higgins PG. Viruses and gastroenteritis in infants. *Lancet* 1975;i:1297.
1152. Abad FX, Villena C, Guix S, Caballero S, Pintó RM, Bosch A. Potential role of fomites in the vehicular transmission of human astroviruses. *Appl Environ Microbiol* 2001;67:3904–7.
1153. Chadwick PR, Beards G, Brown D, et al. Management of hospital outbreaks of gastro-enteritis due to small round structured viruses. Report of the Public Health Laboratory Service, Viral Gastroenteritis Working Group. *J Hosp Infect* 2000;45:1–10.
1154. Spender QW, Lewis D, Price EH. Norwalk-like viruses: study of an outbreak. *Arch Dis Child* 1986;61:142–7.
1155. Storr J, Rice S, Phillips AD, Price E, Walker Smith JA. Clinical associations of Norwalk-like virus in the stools of children. *J Pediatr Gastroenterol Nutr* 1986;5:576–80.
1156. Russo PL, Spelman DW, Harrington GA, et al. Hospital outbreak of Norwalk-like virus. *Infect Control Hosp Epidemiol* 1997;17:1374–8.
1157. Springthorpe VS, Grenier JL, Lloyd-Evans N, Sattar SA. Chemical disinfection of human rotaviruses: efficacy of commercially-available products in suspension tests. *J Hyg (Camb)* 1986;97:139–61.
1158. (335) Lloyd-Evans N, Springthorpe VS, Sattar SA. Chemical disinfection of human rotavirus-contaminated inanimate surfaces. *J Hyg (Camb)* 1986;97:163–73.
1159. CDC. Interim recommendations for cleaning and disinfection of the SARS patient environment. At: www.cdc.gov/ncidod/sars/cleaningpatientenviro.htm
1160. Brown P, Gajdusek DC. The human spongiform encephalopathies: Kuru, Creutzfeldt-Jakob disease, and Gerstmann-Sträussler-Scheinker syndrome. *Curr Top Microbiol Immunol* 1991;172:1–20.
1161. Will RG. Epidemiology of Creutzfeldt-Jakob disease. *Br Med Bull* 1993;49:960–70.
1162. Holman RC, Khan AS, Belay ED, Schonberger LB. Creutzfeldt-Jakob disease in the United States, 1979–1994: using national mortality data to assess the possible occurrence of variant cases. *Emerg Infect Dis* 1996;2:333–7.
1163. Will RG, Ironside JW, Zeidler M, et al. A new variant of Creutzfeldt-Jakob disease in the U.K. *Lancet* 1996;347:921–5.
1164. Lasmézas CI, Deslys JP, Demaimay R, et al. BSE transmission to macaques. *Nature* 1996;381:743–4.
1165. Collinge J, Sidle KCL, Heads J, Ironside J, Hill AF. Molecular analysis of prion strain variation and the aetiology of “new variant” CJD. *Nature* 1996;383:685–90.
1166. Bruce ME, Will RG, Ironside JW, et al. Transmission to mice indicates that “new variant” CJD is caused by the BSE agent. *Nature* 1997;389:498–501.
1167. Prusiner SB. Biology and genetics of prion diseases. *Ann Rev Microbiol* 1994;48:655–86.
1168. Prusiner SB. Human prion diseases. In: Zuckerman AJ, Banatvala JE, Pattison JR, eds. Principles and practice of clinical virology, 3rd ed. Chichester, UK: John Wiley & Sons, 1995;703–29.
1169. Prusiner SB. Prions. *Proc Natl Acad Sci USA* 1998;95:13363–83.

1170. **(337)** Kimberlin RH, Walker CA, Millson GC, et al. Disinfection studies with two strains of mouse-passaged scrapie agent. Guidelines for Creutzfeldt-Jakob and related agents. *J Neurol Sci* 1983;59:349–55.
1171. Sklaviadis TK, Manuelidis L, Manuelidis EE. Physical properties of the Creutzfeldt-Jakob disease agent. *J Virol* 1989;63:1212–22.
1172. Brown P, Gajdusek DC, Gibbs CJ Jr, Asher DM. Potential epidemic of Creutzfeldt-Jakob disease from human growth hormone therapy. *N Engl J Med* 1985;12:728–33.
1173. Brown P, Preece MA, Will RG. “Friendly fire” in medicine: hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992;340:24–7. Brown P, Preece MA, Will RG. “Friendly fire” in medicine: hormones, homografts and Creutzfeldt-Jakob disease. *Lancet* 1992;340:24–7.
1174. Frasier D, Foley TP Jr. Creutzfeldt-Jakob disease in recipients of pituitary hormones. *J Clin Endocrinol Metabol* 1994;78:1277–9.
1175. Centers for Disease Control. Epidemiologic notes and reports: rapidly progressive dementia in a patient who received a cadaveric dura mater graft. *MMWR* 1987;36:49–50, 55.
1176. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a patient receiving cadaveric dura mater graft. *MMWR* 1987;36:324–5.
1177. Centers for Disease Control. Epidemiologic notes and reports update: Creutzfeldt-Jakob disease in a second patient who received a cadaveric dura mater graft. *MMWR* 1989;38:37–8, 43.
1178. CDC. Creutzfeldt-Jakob disease in patients who received a cadaveric dura mater graft — Spain, 1985–1992. *MMWR* 1993;42:560–3.
1179. Martinez-Lage JF, Poza M, Sola J, et al. Accidental transmission of Creutzfeldt-Jakob disease by dural cadaveric grafts. *J Neurol Neurosurg Psychiatry* 1994;57:1091–4.
1180. CDC. Creutzfeldt-Jakob disease associated with cadaveric dura mater grafts — Japan, January 1979–May 1997. *MMWR* 1997;46:1066–9.
1181. Lang CLG, Heckmann JG, Neundörfer B. Creutzfeldt-Jakob disease via dural and corneal transplants. *J Neurol Sci* 1998;160:128–39.
1182. Nevin S, McMenemey WH, Behrman S, Jones DP. Subacute spongiform encephalopathy — a subacute form of encephalopathy attributable to vascular dysfunction (spongiform cerebral atrophy). *Brain* 1960;83:519–69.
1183. Bernoulli C, Siegfried J, Baumgartner G, et al. Danger of accidental person-to-person transmission of Creutzfeldt-Jakob disease by surgery. *Lancet* 1977;1:478–9.
1184. Will RG, Matthews WB. Evidence for case-to-case transmission of Creutzfeldt-Jakob disease. *J Neurol Neurosurg Psychiatry* 1982;45:235–8.
1185. El Hachimi KH, Chaunu M-P, Cervenakova L, Brown P, Foncin J-F. Putative neurosurgical transmission of Creutzfeldt-Jakob disease with analysis of donor and recipient: agent strains. *Comp Rendus Acad Sci Iii: Science de la vie* 1997;320:319–28.
1186. Brown P, Gibbs CJ, Amyx HL, et al. Chemical disinfection of Creutzfeldt-Jakob disease virus. *N Engl J Med* 1982;306:1279–82.
1187. Brown P, Rohwer RG, Gajdusek DC. Newer data on the inactivation of scrapie virus or Creutzfeldt-Jakob disease virus in brain tissue. *J Infect Dis* 1986;153:1145–8.
1188. **(338)** Rosenberg RN, White CL, Brown P, Gajdusek DC, Volpe JJ, Dyck PJ. Precautions in handling tissues, fluids, and other contaminated materials from patients with documented or suspected Creutzfeldt-Jakob disease. *Ann Neurol* 1986;19:75–7.
1189. Taylor DM. Resistance of the ME7 scrapie agent to peracetic acid. *Vet Microbiol* 1991;27:19–24.
1190. Taguchi F, Tamai Y, Uchida K, et al. Proposal for a procedure for complete inactivation of the Creutzfeldt-Jakob disease agent. *Arch Virol* 1991;119:297–301.
1191. **(339)** Taylor D. Inactivation of the unconventional agents of scrapie, bovine spongiform encephalopathy, and Creutzfeldt-Jakob disease. *J Hosp Infect* 1991;18 (Suppl A):141–6.
1192. Favero MS. Current issues in hospital hygiene and sterilization technology. *J Infect Control (Asia Pacific Edition)* 1998;1:8–10.
1193. Ricketts MN, Cashman NR, Stratton EE, El Saadany S. Is Creutzfeldt-Jakob disease transmitted in blood? *Emerg Infect Dis* 1997;3:155–63.
1194. Will RG, Kimberlin RH. Creutzfeldt-Jakob disease and the risk from blood or blood products. *Vox Sang* 1998;75:178–80.
1195. Evatt B, Austin H, Barnhart E, et al. Surveillance for Creutzfeldt-Jakob disease among persons with hemophilia. *Transfusion* 1998;38:817–20.

1196. Patry D, Curry B, Easton D, Mastrianni JA, Hogan DB. Creutzfeldt-Jakob disease (CJD) after blood product transfusion from a donor with CJD. *Neurology* 1998;50:1872–3.
1197. (340) Budka H, Aguzzi A, Brown P, et al. Tissue handling in suspected Creutzfeldt-Jakob disease (CJD) and other human spongiform encephalopathies (prion diseases). *Brain Pathol* 1995;5:319–22.
1198. (341) Ironside JW, Bell JE. The “high-risk” neuropathological autopsy in AIDS and Creutzfeldt-Jakob disease: principles and practice. *Neuropathol Appl Neurobiol* 1996;22:388–93.
1199. (336) Rutala WA, Weber DJ. Creutzfeldt-Jakob disease: recommendations for disinfection and sterilization. *Clin Infect Dis* 2001;32:1348–56.
1200. Joint Commission for the Accreditation of Healthcare Organizations. Exposure to Creutzfeldt-Jakob Disease. Sentinel Alert, Issue 20; June 2001. Available at: www.jcaho.org/edu_pub/sealert/sea20.html
1201. (342) World Health Organization. WHO Infection control guideline for transmissible spongiform encephalopathies: report of a WHO consultation. Geneva, Switzerland: WHO, 1999. Available at: www.who.int/emc-documents/tse/whocdscsraph2003c.html
1202. Litsky BY. Results of bacteriological surveys highlight problem areas in hospitals. *Hospital Management* 1966;101:82–8.
1203. Eickhoff TC. Microbiologic sampling. *Hospitals* 1970;44:86–7.
1204. American Hospital Association Committee on Infections Within the Hospitals. Statement on microbiologic sampling in the hospital 1974;48:125–6.
1205. Rafferty KM, Pancoast SJ. Brief report: bacteriological sampling of telephones and other hospital staff-hand contact objects. *Infect Control* 1984;5:533–5.
1206. Haley RW, Shachtman RS. The emergence of infection control programs in U.S. hospitals: an assessment, 1976. *Am J Epidemiol* 1980;111:574–91.
1207. Mallison GF, Haley RW. Microbiologic sampling of the inanimate environment in U.S. hospitals, 1976–1977. *Am J Med* 1981;70:941–6.
1208. Gröschel DHM. Air sampling in hospitals. *Ann NY Acad Sci* 1980;353:230–40.
1209. (208, Appendix; 6) Barbaree JM, Gorman GW, Martin WT, Fields BS, Morrill WE. Protocol for sampling environmental sites for legionellae. *Appl Environ Microbiol* 1987;53:1454–8.
1210. Eickhoff TC. Microbiologic sampling of the hospital environment. *Health Lab Sci* 1974;11:73–5.
1211. Isenberg HD. Significance of environmental microbiology in nosocomial infections and the care of hospitalized patients. In: Lorian V, ed. Significance of medical microbiology in the care of patients. Baltimore, MD: Williams & Wilkins, 1977;220–34.
1212. McGowan JE Jr, Weinstein RA. The role of the laboratory in control of nosocomial infection. In: Bennett JV, Brachman PS, eds. *Hospital infections*, 4th ed. Philadelphia, PA: Lippincott Raven, 1998;143–64.
1213. Turner AG, Wilkins JR, Craddock JG. Bacterial aerosolization from an ultrasonic cleaner. *J Clin Microbiol* 1975;1:289–93.
1214. (343) Bond WW, Schulster LM. Microbiological culturing of environmental and medical-device surfaces. In: Isenberg HD, Miller JM, Bell M, eds. *Clinical microbiology procedures handbook*, section 11. Washington, DC: American Society for Microbiology Press, 2004 (in press).
1215. Cole EC, Cook CE. Characterization of infectious aerosols in health care facilities: an aid to effective engineering controls and preventive strategies. *Am J Infect Control* 1998;26:452–64.
1216. Nevalainen A, Willeke K, Liebhaber F, Pastuszka J, Burge H, Henningson E. Bioaerosol sampling. In: Willeke K, Baron PA, eds. *Aerosol management*. New York, NY: Van Nostrand Reinhold, 1993;471–92.
1217. Cox CS. *The aerobiological pathway of microorganisms*. Chichester UK: John Wiley & Sons, 1987.
1218. (349) Wolf HW, Skaliy P, Hall LB, et al. Sampling microbiological aerosols. Public Health Service publication No. 686. Government Printing Office, Washington, DC: 1964.
1219. Zeterberg JM. A review of respiratory virology and the spread of virulent and possible antigenic viruses via air conditioning systems. *Ann Allergy* 1973;31:228–34.
1220. Randall CW, Ledbetter JO. Bacterial air pollution from activated sludge units. *Am Ind Hyg Assoc J* 1966;Nov/Dec:506–19.
1221. Salem H, Gardner DE. Health aspects of bioaerosols. In: Lighthart B, Mohr AJ, eds. *Atmospheric microbial aerosols, theory and applications*. New York, NY: Chapman and Hall, 1985;304–30.
1222. Sattar SA, Ijaz MK. Spread of viral infections by aerosols. *Crit Rev Environ Control* 1987;17:89–131.
1223. (345) Buttner MP, Willeke K, Grinshpun SA. Sampling and analysis of airborne microorganisms. In: Hurst CJ, Knudsen GR, McInerney MJ, Stetzenbach LD, Walter MV, eds. *Manual of environmental microbiology*. Washington, DC: American Society for Microbiology Press, 1997;629–40.

1224. **(346)** Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. In: NIOSH Manual of Analytical Methods; Cincinnati OH; CDC; 1998: p. 82–112. Available at: www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf
1225. Jolley AE. The value of surveillance cultures on neonatal intensive care units. *J Hosp Infect* 1993;25:153–9.
1226. Hardy KA, McGowan KL, Fisher MC, Schidlow DV. *Pseudomonas cepacia* in the hospital setting: lack of transmission between cystic fibrosis patients. *J Pediatr* 1986;109:51–4.
1227. Hambræus A, Lagerqvist-Widh A, Zettersten U, Engberg S, Sedin G, Sjöberg L. Spread of *Klebsiella* in a neonatal ward. *Scand J Infect Dis* 1991;23:189–94.
1228. Humphreys H, Peckham D, Patel P, Knox A. Airborne dissemination of *Burkholderia (Pseudomonas) cepacia* from adult patients with cystic fibrosis. *Thorax* 1994;49:1157–9.
1229. Pankhurst CL, Harrison VE, Philpott-Howard J. Evaluation of contamination of the dentist and dental surgery environment with *Burkholderia (Pseudomonas) cepacia* during treatment of children with cystic fibrosis. *Int J Paediatr Dent* 1995;5:243–7.
1230. Weber DO, Gooch JJ, Wood WR, Britt EM, Kraft RO. Influence of operating room surface contamination on surgical wounds: a prospective study. *Arch Surg* 1976;111:484–8.
1231. Pfeiffer EH, Wittig JR, Dunkelberg H, Werner HP. Hygienic and bacteriological comparative studies in 50 hospitals. V. Bacterial contamination of hospital surfaces. *Zentralbl Bakteriol [B]* 1978;167:11–21. (German)
1232. Sattar SA, Lloyd-Evans N, Springthorpe VS. Institutional outbreaks of rotavirus diarrhea: potential role of fomites and environmental surfaces as vehicles for virus transmission. *J Hyg (Camb)* 1986;96:277–89.
1233. Smith SM, Eng RH, Padberg FT Jr. Survival of nosocomial pathogenic bacteria at ambient temperature. *J Med* 1996;27:293–302.
1234. Craythorn JM, Barbour AG, Matsen JM, Britt MR, Garibaldi RA. Membrane filter contact technique for bacteriological sampling of moist surfaces. *J Clin Microbiol* 1980;12:250–5.
1235. Scott E, Bloomfield SF, Barlow CG. A comparison of contact plate and calcium alginate swab techniques for quantitative assessment of bacteriological contamination of environmental surfaces. *J Appl Bacteriol* 1984;56:317–20.
1236. Poletti L, Pasquarella C, Pitzurra M, Savino A. Comparative efficiency of nitrocellulose membranes versus RODAC plates in microbial sampling on surfaces. *J Hosp Infect* 1999;41:195–201.
1237. Russell AD. Factors influencing the efficacy of antimicrobial agents. In: Russell AD, Hugo WB, Ayliffe GAJ, eds. Principles and practices of disinfection, preservation and sterilization. Oxford, UK: Blackwell Science, 1999;95–123.
1238. **(347)** International Organization for Standardization (ISO). Sterilization of medical devices — microbiological methods, Part 1. ISO Standard 11737-1. Paramus, NJ: International Organization for Standardization, 1995.
1239. Favero MS, Gabis DA, Vesley D. Environmental monitoring procedures. In: Speck ML, ed. Compendium of methods for the microbiological examination of foods, 2nd ed. Washington, DC: American Public Health Association; 1984;47–61.
1240. Favero MS, Bond WW, Petersen NJ, Berquist KR, Maynard JE. Detection methods for study of the stability of hepatitis B antigen on surfaces. *J Infect Dis* 1974;129:210–2.
1241. Favero MS, McDade JJ, Robertsen JA, Hoffmann RK, Edwards RW. Microbiological sampling of surfaces. *J Appl Bacteriol* 1968;31:336–43.
1242. Petersen NJ, Collins DE, Marshall JH. Evaluation of skin cleansing procedures using the wipe-rinse technique. *Health Lab Sci* 1974;11:182–97.
1243. Schalkowsky S, Hall LB, Kline RC. Potential effects of recent findings on spacecraft sterilization requirements. *Space Life Sci* 1969;1:520–30.
1244. Hall LB, Lyle RG. Foundations of planetary quarantine. *Environ Biol Med* 1971;1:5–8.
1245. Rutala WA, Weber DJ. Uses of inorganic hypochlorite (bleach) in health-care facilities. *Clin Microbiol Rev* 1997;10:597–610.
1246. Mallison GF. Central services and linens and laundry. In: Bennett JV, Brachman PS, eds. Hospital infections. Boston, MA: Little, Brown, & Co, 1986;251–6.
1247. **(365)** Blaser MJ, Smith PE, Cody HJ, Wang W-LL, LaForce FM. Killing of fabric-associated bacteria in hospital laundry by low-temperature washing. *J Infect Dis* 1984;149:48–57.
1248. Centers for Disease Control. Outbreak of viral hepatitis in the staff of a pediatric ward — California. *MMWR* 1977;28:77–9.

1249. Shah PC, Krajden S, Kane J, Summerbell RC. Tinea corporis caused by *Microsporum canis*: report of a nosocomial outbreak. *Eur J Epidemiol* 1988;4:33–7.
1250. (353) Barrie D, Hoffman PN, Wilson JA, Kramer JM. Contamination of hospital linen by *Bacillus cereus*. *Epidemiol Infect* 1994;113:297–306.
1251. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella gastroenteritis* to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994;15:22–6.
1252. Pasternak J, Richtmann R, Ganme APP, et al. Scabies epidemic: price and prejudice. *Infect Control Hosp Epidemiol* 1994;15:540–2.
1253. (357) Association for the Advancement of Medical Instrumentation. Processing of reusable surgical textiles for use in health care facilities: ANSI/AAMI recommended practice ST65. Arlington, VA: Association for the Advancement of Medical Instrumentation, 2000;16.
1254. Association of Operating Room Nurses. Recommended practices for surgical attire: AORN standards and recommended practices. *AORN J* 1995;62:141–2.
1255. Loh W, Ng VV, Holton J. Bacterial flora on the white coats of medical students. *J Hosp Infect* 2000;45:65–8.
1256. Belkin NL. Use of scrubs and related apparel in healthcare facilities. *Am J Infect Control* 1997;25:401–4.
1257. Belkin NL. Home laundering of soiled surgical scrubs: surgical site infections and the home environment. *Am J Infect Control* 2000;29:58–64.
1258. (355) Joint Committee on Healthcare Laundry Guidelines. Guidelines for healthcare linen service. Hallendale, FL: Textile Rental Service Association of America, 1999.
1259. (356) Greene VW. Microbiological contamination control in hospitals: part 6 — roles of central service and the laundry. *Hospitals JAHA* 1970;44:98–103.
1260. (350) Wagner RA. Partitioned laundry improves bacteria control. *Hospitals JAHA* 1966;40:148–51.
1261. (351) Hambræus A, Malmborg AS. Is a bed centre in a hospital a hygienic hazard? *J Hyg (Camb)* 1982;88:143–7.
1262. (352) McDonald LL, Pugliese G. Textile processing service. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1031–4.
1263. (354) Legnani PP, Leoni E. Factors affecting the bacteriological contamination of commercial washing machines. *Zentralbl Hyg Umweltmed* 1997;200:319–33.
1264. Maki DG, Alvarado C, Hassemer C. Double-bagging of items from isolation rooms is unnecessary as an infection control measure: a comparative study of surface contamination with single- and double-bagging. *Infect Control* 1986;7:535–7.
1265. Garner JS, Simmons BP. CDC guideline for isolation precautions in hospitals. *Infect Control* 1983;4:245–325 and *Am J Infect Control* 1984;12:103–63.
1266. Weinstein SA, Gantz NM, Pelletier C, Hibert D. Bacterial surface contamination of patients' linen: isolation precautions versus standard care. *Am J Infect Control* 1989;17:264–7.
1267. (358) Hughes HG. Chutes in hospitals. *Can Hosp* 1964;41:56–7, 87.
1268. (359) Michaelsen GS. Designing linen chutes to reduce spread of infectious organisms. *Hospitals JAHA* 1965;39 (3):116–9.
1269. (360) Hoch KW. Laundry chute cleaning recommendations [letter]. *Infect Control* 1982;3:360.
1270. (361) Whyte W, Baird G, Annand R. Bacterial contamination on the surface of hospital linen chutes. *J Hyg (Camb)* 1969;67:427–35.
1271. (363) Walter WG, Schillinger JE. Bacterial survival in laundered fabrics. *Appl Microbiol* 1975;29:368–73.
1272. (362) Taylor LJ. Segregation, collection, and disposal of hospital laundry and waste. *J Hosp Infect* 1988;11 (Suppl. A):57–63.
1273. Barrie D. How hospital linen and laundry services are provided. *J Hosp Infect* 1994;27:219–35.
1274. Riggs CH, Sherrill JC. Textile laundering technology. Hallendale FL: Textile Rental Service Association; 1999;92–7.
1275. Mouton RP, Bekkers JH. Bacteriological results of routine procedures in a hospital laundry. *Folia Med Neerl* 1967;10:71–6.
1276. Nicholes PS. Bacteria in laundered fabrics. *Am J Public Health* 1970;60:2175–80.
1277. Arnold L. A sanitary study of commercial laundry practices. *Am J Public Health* 1938;28:839–44.

1278. (364) Belkin NL. Aseptics and aesthetics of chlorine bleach: can its use in laundering be safely abandoned? *Am J Infect Control* 1998;26:149–51.
1279. Jordan WE, Jones DV. Antiviral effectiveness of chlorine bleach in household laundry use. *Am J Dis Child* 1969;117:313–6.
1280. Hittman Associates, Inc. Energy efficient water use in hospitals [Final summary report (H-W8000-78-756FR)]. Prepared for the University of California, Lawrence Berkeley Laboratory, 1979. Contract No. P.O. 4627702.
1281. (366) Jaska JM, Fredell DL. Impact of detergent systems on bacterial survival on laundered fabrics. *Appl Environ Microbiol* 1980;39:743–8.
1282. (367) Battles DR, Vesley D. Wash water temperature and sanitation in the hospital laundry. *J Environ Health* 1981;43:244–50.
1283. (368) Christian RR, Manchester JT, Mellor MT. Bacteriological quality of fabrics washed at lower-than-standard temperatures in a hospital laundry facility. *Appl Environ Microbiol* 1983;45:591–7.
1284. (369) Smith JA, Neil KR, Davidson CG, Davidson RW. Effect of water temperature on bacterial killing in laundry. *Infect Control* 1987;8:204–9.
1285. (370) Tompkins DS, Johnson P, Fittall BR. Low-temperature washing of patients' clothing: effects of detergent with disinfectant and a tunnel drier on bacterial survival. *J Hosp Infect* 1988;12:51–8.
1286. (371) Ayliffe GAJ, Collins BJ, Taylor LJ. Laundering. In: Wright PSG, ed. *Hospital-acquired Infection: principles and Prevention*. Bristol, UK: 1982;101–6.
1287. Koller W, Wewalka G. A new method for microbiological evaluation of disinfecting laundering processes. *Zbl Bakt Hyg I Abt Orig B* 1982;176:463–71.
1288. (372) Meyer CL, Eitzen HE, Schreiner RL, Gfell MA, Moye L, Kleiman MB. Should linen in newborn intensive care units be autoclaved? *Pediatrics* 1981;67:362–4.
1289. (373) Wagg RE. Disinfection of textiles in laundering and dry cleaning. *Chem Ind* 1965;44:1830–4.
1290. (374) Bates CJ, Wilcox MH, Smith TL, Spencer RC. The efficacy of a hospital dry cleaning cycle in disinfecting material contaminated with bacteria and viruses. *J Hosp Infect* 1993;23:255–62.
1291. (375) Oehnel E. Drycleaning in the hospital laundry. *Can Hosp* 1971;September:66–7.
1292. DiGacomo JC, Odom JW, Ritoto PC, Swan KC. Cost containment in the operating room: use of reusables versus disposable clothing. *Am Surg* 1992;58:654–6.
1293. American Society for Testing Materials. Standard test method for resistance of materials used in protective clothing to penetration by synthetic blood. ASTM, 1998;F1670–98.
1294. American Society for Testing Materials. Standard test method for resistance of materials used in protective clothing to penetration by bloodborne pathogens using phi-X174 bacteriophage penetration as a test system. ASTM 1997;F1671–976.
1295. Belkin NL. Are “impervious” surgical gowns really liquid-proof? *Bull Am Col Surgeons* 1999;84:19–36.
1296. Belkin NL. OR gowns — even a “pass” can fail. *AORN J* 1999;70:302–4.
1297. Laufman H, Belkin NL, Meyer KK. A critical review of a century's progress in surgical apparel: how far have we come? *J Am Col Surgeons* 2000;191:554–68.
1298. Leonas KK, Jinkins RS. The relationship of selected fabric characteristics and the barrier effectiveness of surgical gown fabrics. *Am J Infect Control* 1997;25:16–23.
1299. Meyer KK, Beck WC. Gown-glove interface: a possible solution to the danger zone. *Infect Control Hosp Epidemiol* 1995;16:488–90.
1300. McCullough EA. Methods for determining the barrier efficacy of surgical gowns. *Am J Infect Control* 1993;21:368–74.
1301. Pissiotis CA, Komborozos V, Papoutsis C, Skrekas G. Factors that influence the effectiveness of surgical gowns in the operating theater. *Eur J Surg* 1997;163:597–604.
1302. Association of Operating Room Nurses (AORN). Recommended practices for use and selection of barrier materials for surgical gowns and drapes. Association of Operating Room Nurses. *AORN J* 1996;63:650, 653–4.
1303. Belkin NL, Koch FT. OR barrier materials — Necessity or extravagance? *AORN J* 1998;67:443–5.
1304. Rutala WA, Weber DJ. A review of single-use and reusable gowns and drapes in health care. *Infect Control Hosp Epidemiol* 2001;22:248–57.
1305. Murphy L. Cost/benefit study of reusable and disposable OR draping materials. *J Healthc Mater Manage* 1993;11:44–8.

1306. (376) U.S. Environmental Protection Agency. Consumer products treated with pesticides. Office of Pesticide Programs. Available at: www.epa.gov/opp00001/citizens/treatart.htm
1307. Kalyon BD, Olgun U. Antibacterial efficacy of triclosan-incorporated polymers. *Am J Infect Control* 2001;29:124–5.
1308. U.S. Environmental Protection Agency. Clarification of treated articles exemption. Availability of draft PR notice. *Federal Register* 1998;63:19256–8.
1309. Mayer CE. FTC Challenges antibacterial product. Washington, DC: Washington Post, September 17, 1999;A09.
1310. (377) Fujita K, Lilly HA, Kidson A, Ayliffe GAJ. Gentamicin-resistant *Pseudomonas aeruginosa* infection from mattresses in a burns unit. *Br Med J* 1981;283:219–20.
1311. (378) Grubb DJ, Watson KC. *Pseudomonas* septicaemia from plastic mattresses [letter]. *Lancet* 1982;1:518.
1312. (379) Sherertz RJ, Sullivan ML. An outbreak of infections with *Acinetobacter calcoaceticus* in burn patients: contamination of patients' mattresses. *J Infect Dis* 1985;151:252–8.
1313. (380) Ndawula EM, Brown L. Mattresses as reservoirs of epidemic methicillin-resistant *Staphylococcus aureus* [letter]. *Lancet* 1991;337:488.
1314. (381) O'Donoghue MAT, Allen KD. Costs of an outbreak of wound infections in an orthopaedic ward. *J Hosp Infect* 1992;22:73–9.
1315. (382) Weernink A, Severin WPJ, Thernberg T, Dijkshoorn L. Pillows, an unexpected source of *Acinetobacter*. *J Hosp Infect* 1995;29:189–99.
1316. Newsome TW, Johns LA, Pruitt BA Jr. Use of an air-fluidized bed in the care of patients with extensive burns. *Am J Surg* 1972;124:52–6.
1317. (383) Scheidt A, Drusin LM. Bacteriologic contamination in an air-fluidized bed. *J Trauma* 1983;23:241–2.
1318. (384) Freeman R, Gould FK, Ryan DW, Chamberlain J, Sisson PR. Nosocomial infection due to enterococci attributed to a fluidized microsphere bed: the value of pyrolysis mass spectrometry. *J Hosp Infect* 1994;27:187–93.
1319. Sharbaugh RJ, Hargest TS. Bactericidal effect of the air-fluidized bed. *Am Surgeon* 1971;37:583–6.
1320. Sharbaugh RJ, Hargest TS, Wright FA. Further studies on the bactericidal effect of the air-fluidized bed. *Am Surgeon* 1973;39:253–6.
1321. Winters WD. A new perspective of microbial survival and dissemination in a prospectively contaminated air-fluidized bed model. *Am J Infect Control* 1990;18:307–15.
1322. (385) Clancy MJ. Nosocomial infection and microsphere beds [letter]. *Lancet* 1993;342:680–1.
1323. (386) Clancy MJ. Nosocomial infection due to enterococci attributed to a fluidized microsphere bed [letter]. *J Hosp Infect* 1994;28:324–5.
1324. Vesley D, Hankinson SE, Lauer JL. Microbial survival and dissemination associated with an air-fluidized therapy unit. *Am J Infect Control* 1986;14:35–40.
1325. Bolyard EA, Townsend TR, Horan T. Airborne contamination associated with in-use air-fluidized beds: a descriptive study. *Am J Infect Control* 1987;15:75–8.
1326. (387) Jacobsen E, Gurevich I, Cunha BA. Air-fluidized beds and negative-pressure isolation rooms [letter]. *Am J Infect Control* 1993;21:217–8.
1327. Cooper JE. Pets in hospitals. *Br Med J* 1976;1:698–700.
1328. Egerton JR. Pets and zoonoses. *Med J Aust* 1982;2:311.
1329. Yamauchi T. Pet programs in hospitals. *Pediatr Infect Dis J* 1993;12:707.
1330. Khan MA, Farrag N. Animal-assisted activity and infection control implications in a healthcare setting. *J Hosp Infect* 2000;46:4–11.
1331. Weber DJ, Rutala WA. Epidemiology and prevention of nosocomial infections associated with animals in the hospital. In: Mayhall CG, ed. *Hospital epidemiology and infection control*, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 1999;1399–421.
1332. Acha PN, Szyfres B. Zoonoses and communicable diseases common to man and animals, 2nd ed. Washington, DC: Pan American Health Organization, 1987. Scientific publication No. 503.
1333. Elliot DL, Tolle SW, Goldberg L, Miller JB. Pet-associated illness. *N Engl J Med* 1985;313:985–95.
1334. Marx MB. Parasites, pets, and people. *Primary Care* 1991;18:153–65.
1335. Goldstein EJ. Household pets and human infections. *Infect Dis Clin North Am* 1991;5:117–30.
1336. Chomel BB. Zoonoses of house pets other than dogs, cats, and birds. *Pediatr Infect Dis J* 1992;11:479–87.

1337. Gnann JW Jr, Bressler GS, Bodet CA III, Avent CK. Human blastomycosis after a dog bite. *Ann Intern Med* 1983;98:48–9.
1338. Garcia VF. Animal bites and *Pasteurella* infections. *Pediatr Rev* 1997;18:127–30.
1339. Crowder HR, Dorn CR, Smith RE. Group A *Streptococcus* in pets and group A streptococcal disease in man. *Int J Zoonoses* 1978;5:45–54.
1340. (397) CDC. Reptile-associated salmonellosis — selected states, 1996–1998. *MMWR* 1999;48:1009–13.
1341. Devriese LA, Ieven M, Goossens H, et al. Presence of vancomycin-resistant enterococci in farm and pet animals. *Antimicrob Agent Chemother* 1996;40:2285–7.
1342. Scott GM, Thomson R, Malone-Lee J, Ridgway GL. Cross-infection between animals and man: Possible feline transmission of *Staphylococcus aureus* infection in humans? *J Hosp Infect* 1988;12:29–34.
1343. Weinberg A. Ecology and epidemiology of zoonotic pathogens. *Infect Dis Clin North Am* 1991;5:1–6.
1344. Yu V, Meissner C. Zoonoses. In: Schaechter M, Medoff G, Schlessinger D, eds. *Mechanisms of microbial diseases*. Baltimore, MD: Williams & Wilkins, 1989;749–64.
1345. Ryan KJ. Some bacteria causing zoonotic diseases. In: Sherris JC, ed. *Medical microbiology*, 2nd ed. New York, NY: Elsevier, 1990;489–98.
1346. Chang HJ, Miller HL, Watkins N, et al. An epidemic of *Malassezia pachydermatis* in an intensive care nursery associated with colonization of healthcare workers' pet dog. *N Engl J Med* 1998;338:706–11.
1347. Drusin LM, Ross BG, Rhodes KH, Krause AN, Scott RA. Nosocomial ringworm in a neonatal intensive care unit: a nurse and her cat. *Infect Control Hosp Epidemiol* 2000;21:605–7.
1348. Richet HM, Craven PC, Brown JM, et al. A cluster of *Rhodococcus (Gordona) bronchialis* sternal-wound infections after coronary-artery bypass surgery. *N Engl J Med* 1991;324:104–9.
1349. (394) Saylor K. Pet visitation program. *J Gerontol Nurs* 1998;24:36–8.
1350. Corson SA, O'Leary Corson E. Pets as mediators of therapy. *Curr Psychiatr Ther* 1978;18:195–205.
1351. Fick KM. The influence of an animal on social interactions of nursing home residents in a group setting. *Am J Occup Ther* 1993;47:529–34.
1352. Gunby P. Patient progressing well? He must have a pet. *JAMA* 1979;241:438.
1353. Culliton BJ. Take two pets and call me in the morning. *Science* 1987;237:1560–1.
1354. Wilkes CN, Shalko TK, Trahan M. Pet Rx: Implications for good health. *Health Educ* 1989;20:6–9.
1355. Doyle K, Kukowski T. Utilization of pets in a hospice program. *Health Educ* 1989;20:10–1.
1356. Teeter LM. Pet therapy program. *J Amer Vet Med Assoc* 1997;210:1435–8.
1357. Gammonley J, Yates J. Pet projects: animal assisted therapy in nursing homes. *J Gerontol Nurs* 1991;17:12–5.
1358. (395) Draper RJ, Gerber GJ, Layng EM. Defining the role of pet animals in psychotherapy. *Psychiat J Univ Ottawa* 1990;15:169–72.
1359. Allen DT. Effects of dogs on human health. *J Amer Vet Med Assoc* 1997;210:1136–9.
1360. (391) Delta Society. *Standards of practice for animal-assisted activities and animal-assisted therapy*. Renton, WA: Delta Society, 1996.
1361. (392) Fox JG. Transmissible drug resistance in *Shigella* and *Salmonella* isolated from pet monkeys and their owners. *J Med Primatol* 1975;4:165–71.
1362. (393) Ostrowski SR, Leslie MJ, Parrott T, Abelt S, Piercy PE. B-virus from pet macaque monkeys: an emerging threat in the United States? *Emerg Infect Dis* 1998;4:117–21.
1363. CDC. How to prevent transmission of intestinal roundworms from pets to people. Available at: www.cdc.gov/ncidod/diseases/roundworm/roundworm.htm
1364. (146) Boyce JM, Pittet D. Guideline for hand hygiene in health-care settings: recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force. *Infect Control Hosp Epidemiol* 2002;23 (Suppl):S1–S40.
1365. (388) American Academy of Allergy, Asthma, and Immunology. Tips to remember: indoor allergens. Available at: www.aaaai.org/patients/publicedmat/tips/indoorallergens.stm
1366. (389) Duncan SL, APIC Guideline Committee. APIC State-of-the-art report: the implications of service animals in healthcare settings. *Am J Infect Control* 2000;28:170–80.
1367. (390) Murray AB, Ferguson A, Morrison BJ. The frequency and severity of cat allergy vs. dog allergy in atopic children. *J Allergy Clin Immunol* 1983;72:145–9.
1368. Hodson T, Custovic A, Simpson A, Chapman M, Woodcock A, Green R. Washing the dog reduces dog allergen levels, but the dog needs to be washed twice a week. *J Allergy Clin Immunol* 1999;103:581–5.
1369. Brickel CN. The therapeutic roles of cat mascots with a hospital based population: a staff survey. *Gerontologist* 1979;19:368–72.

1370. Thomas W, Stermer M. Eden alternative principles hold promise for the future of long-term care. *Balance* 1999;3:14–7.
1371. Tavormina CE. Embracing the Eden alternative in long-term care environments. *Geriatr Nurs* 1999;20:158–61.
1372. Brook I, Fish CH, Schantz PM, Cotton DD. Toxocariasis in an institution for the mentally retarded. *Infect Control* 1981;2:317–9.
1373. Huminer D, Symon R, Groskopf I, et al. Seroepidemiological study of toxocariasis and strongyloidiasis in adult mentally retarded institutionalized subjects. *Am J Trop Med Hyg* 1992;46:278–81.
1374. Huminer D, Pitlik SD, Block C, Kaufman L, Amit S, Rosenfeld JB. Aquarium-borne *Mycobacterium marinum* skin infection: report of a case and review of the literature. *Arch Dermatol* 1986;122:698–703.
1375. Lewis FMT, Marsh BJ, von Reyn CF. Fish tank exposure and cutaneous infections due to *Mycobacterium marinum*: tuberculin skin testing, treatment, and prevention. *Clin Infect Dis* 2003;37:390–7.
1376. (398) U.S. Department of Justice. Americans with Disabilities Act. Public Law 101-336 (28 CFR 36.101 et seq.). Title III, Public Accommodations Operated by Private Entities, Sect. 302, Prohibition of Discrimination by Public Accommodations;42 USC 12101 et seq. July 26, 1990.
1377. U.S. Department of Justice, Civil Rights Division, Disability Rights Section. Commonly asked questions about service animals in places of business, 1996.
1378. Schachter J, Sung M, Meyer KF. Potential danger of Q fever in a university hospital environment. *J Infect Dis* 1971;123:301–4.
1379. Konkle DM, Nelson KN, Lunn DP. Nosocomial transmission of *Cryptosporidium* in a veterinary hospital. *J Vet Intern Med* 1997;11:340–3.
1380. House JK, Mainar-Jaime RC, Smith BP, House AM, Kamiya DY. Risk factors for nosocomial *Salmonella* infection among hospitalized horses. *J Am Vet Med Assoc* 1999;214:1511–6.
1381. Weese JS, Staempfli HR, Prescott JF. Isolation of environmental *Clostridium difficile* from a veterinary teaching hospital. *J Vet Diagn Invest* 2000;12:449–52.
1382. Boerlin P, Eugster S, Gaschen F, Straub R, Schawalder P. Transmission of opportunistic pathogens in a veterinary teaching hospital. *Vet Microbiol* 2001;82:347–9.
1383. Schott HC II, Ewart SL, Walker RD, et al. An outbreak of salmonellosis among horses at a veterinary teaching hospital. *J Am Vet Med Assoc* 2001;218:1152–9,1170.
1384. Kim LM, Morley PS, Traub-Dargatz JL, Salman MD, Gentry-Weeks C. Factors associated with *Salmonella* shedding among equine colic patients at a veterinary teaching hospital. *J Am Vet Med Assoc* 2001;218:740–8.
1385. Seguin JC, Walker RD, Caron JP, et al. Methicillin-resistant *Staphylococcus aureus* outbreak in a veterinary teaching hospital: potential human-to-animal transmission. *J Clin Microbiol* 1999;37:1459–63.
1386. Shen DT, Crawford TB, Gorham JR, et al. Inactivation of equine infectious anemia virus by chemical disinfectants. *Am J Vet Res* 1977;38:1217–9.
1387. Scott FW. Virucidal disinfectants and feline viruses. *Am J Vet Res* 1980;41:410–4.
1388. Brown TT. Laboratory evaluation of selected disinfectants as virucidal agents against porcine parvovirus, pseudorabies virus, and transmissible gastroenteritis virus. *Am J Vet Res* 1981;42:1033–6.
1389. Saknimit M, Inatsuki I, Sugiyama Y, et al. Virucidal efficacy of physico-chemical treatments against coronaviruses and parvoviruses of laboratory animals. *Exp Anim* 1988;37:341–5.
1390. Bruins G, Dyer JA. Environmental considerations of disinfectants used in agriculture. *Rev Sci Tech* 1995;14:81–94.
1391. Quinn PJ, Markey BK. Disinfection and disease prevention in veterinary medicine. In: Block SS, ed. *Disinfection, sterilization, and preservation*, 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2001;1069–103.
1392. Fox JG, Lipman NS. Infections transmitted by large and small laboratory animals. *Infect Dis Clin North Am* 1991;5:131–63.
1393. (401) U.S. Department of Labor, Occupational Safety and Health Administration. Personal Protective Equipment for General Industry, Final Rule (29 CFR 1910.132, 1910.138). *Federal Register* 1994;59:16334–64.
1394. U.S. Department of Agriculture. Public Law 89-544 (The Animal Welfare Act of 1966). 7 USC § 2131-2156.

1395. (399) U.S. Department of Agriculture. Public Law 99-198 Food Security Act of 1985, Subtitle F - Animal Welfare. 7 USC § 2131.
1396. Althaus H, Sauerwald M, Schrammeck E. Waste from hospitals and sanatoria. *Zbl Bakteriol Hyg I Abt Orig B* 1983;178:1–29.
1397. Kalnowski G, Wiegand H, Henning R. The microbial contamination of hospital waste. *Zbl Bakteriol Hyg I Abt Orig B* 1983;178:364–79.
1398. Mose JR, Reinthaler F. Microbial contamination of hospital waste and household refuse. *Zbl Bakteriol Hyg I Abt Orig B* 1985;181:98–110.
1399. Collins CH, Kennedy DA. The microbiological hazards of municipal and clinical wastes. *J Appl Bacteriol* 1992;73:1–6.
1400. Rutala WA, Odette RL, Samsa GP. Management of infectious waste by U.S. hospitals. *JAMA* 1989;262:1635–40.
1401. Agency for Toxic Substances and Disease Registry. The public health implications of medical waste: a report to Congress. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, 1990.
1402. Hedrick ER. Infectious waste management — will science prevail? *Infect Control Hosp Epidemiol* 1988;9:488–90.
1403. Keene J. Medical waste management: public pressure vs. sound science. *Hazard Mat Control* 1989;Sept/Oct:29–36.
1404. Keene J. Medical waste: a minimal hazard. *Infect Control Hosp Epidemiol* 1991;12:682–5.
1405. Rutala WA, Weber DJ. Mismatch between science and policy. *N Engl J Med* 1991;325:578–82.
1406. U.S. Environmental Protection Agency. EPA guide for infectious waste management. Washington, DC: U.S. Government Printing Office, 1986. EPA Publication No. 530SW86014.
1407. (402) U.S. Department of Transportation. Hazardous materials regulations. 49 CFR Parts 171–180, Division 6.2; and Hazardous Materials: Revision to Standards for Infectious Substances and Genetically-Modified Microorganisms: Proposed Rule. *Federal Register* 1998;63:46843–59.
1408. (403) U.S. Postal Service. C 023.8.0 Infectious substances (Hazard Class 6, Division 6.2) et seq. At: <http://pe.usps.gov/text/dmm/c023.htm>
1409. (404) Greene R, Miele DJ, Slavik NS. Technical assistance manual: state regulatory oversight of medical waste treatment technologies, 2nd ed: a report of the State and Territorial Association on Alternative Treatment Technologies, 1994.
1410. U.S. Environmental Protection Agency (EPA). 40 CFR Part 60. Standards of performance for new stationary sources and emission guidelines for existing sources: hospital/medical/infectious waste incinerators; Final Rule. *Federal Register* 1997;62:48347–91.
1411. CDC/National Institutes of Health. Biosafety in microbiological and biomedical laboratories. Washington, DC: U.S. Government Printing Office, 1985. DHHS Publication No. (CDC) 93–8395.
1412. (409) U.S. Department of Health and Human Services, Office of Inspector General, CDC. Possession, use, and transfer of select agents and toxins, interim final rule (42 CFR Part 73). *Federal Register*, December 13, 2002;67(240):76885–905.
1413. U.S. Department of Agriculture, Animal and Plant Health Inspection Service. Agricultural Bioterrorism Protection Act of 2002; Possession, use, and transfer of biological agents and toxins: interim final rule (9 CFR Part 121). *Federal Register*. December 13, 2002;67(240):76907–38.
1414. (410) CDC. Recommendations on infective waste. Atlanta, GA: Office of Biosafety and Hospital Infections Program, 1988;1–6.
1415. (405) CDC. National Institute for Occupational Safety and Health. NIOSH Alert: Preventing needlestick injuries in health care settings. Cincinnati, OH: DHHS, 1999. DHHS-NIOSH Publication No. 2000–108.
1416. Rutala WA, Stiegel MM, Sarubbi FA. Decontamination of laboratory microbiological waste by steam sterilization. *Appl Environ Microbiol* 1982;43:1311–6.
1417. Lauer JL, Battles DR, Vesley D. Decontaminating infectious laboratory waste by autoclaving. *Appl Environ Microbiol* 1982;44:690–4.
1418. Palenik CJ, Cumberlander ND. Effects of steam sterilization on the contents of sharps containers. *Am J Infect Control* 1993;21:28–33.
1419. (406) Weber AM, Boudreau Y, Mortimer VD. Stericycle, Inc., Morton, WA. HETA 98-0027-2709. NIOSH, CDC, Cincinnati, OH, 1998.

1420. (407) Johnson KR, Braden CR, Cairns KL, et al. Transmission of *Mycobacterium tuberculosis* from medical waste. *JAMA* 2000;284:1683–8.
1421. (408) Emery R, Sprau D, Lao YJ, Pryor W. Release of bacterial aerosols during infectious waste compaction: An initial hazard evaluation for health care workers. *Am Ind Hyg Assoc J* 1992;53:339–45.
1422. National Committee for Clinical Laboratory Standards (NCCLS). Protection of laboratory workers from instrument biohazards and infectious disease transmitted by blood, body fluids, and tissue. Approved guideline. 1997, NCCLS Document M29-A (ISBN1-56238-339-6).
1423. Snyder JW, Check W. Bioterrorism threats to our future: the role of the clinical microbiology laboratory in detection, identification, and confirmation of biological agents. Report of the October 27–29, 2000 Colloquium, American Academy of Microbiology, American College of Microbiology. Available at: www.asm.org
1424. CDC. List of select agents and biological toxins. At: www.cdc.gov/od/sap/docs/salist.pdf
1425. Bond WW. Survival of hepatitis B virus in the environment. *JAMA* 1984;252:397–8.
1426. Slade JE, Pike EB, Eglin RP, Colbourne JS, Kurtz JB. The survival of human immunodeficiency virus in water, sewage, and sea water. *Water Sci Technol* 1989;21:55–9.
1427. Geertsma RE, Van Asten JAAM. Sterilization of prions. *Zentr Steril* 1995;3:385–94.
1428. (52) Johnson MW, Mitch WE, Heller AH, Spector R. The impact of an educational program on gentamicin use in a teaching hospital. *Am J Med* 1982;73:9–14.
1429. (53) Soumerai SB, Salem-Schatz S, Avorn J, Casteris CS, Ross-Degnan D, Popovsky MA. A controlled trial of educational outreach to improve blood transfusion practice. *JAMA* 1993;270:961–6.
1430. (54) Eisenberg JM. An education program to modify laboratory use by house staff. *J Med Educ* 1977;52:578–81.
1431. (55) Rello J, Quintana E, Ausina V, Puzo V, Puzo C, Net A, Prats G. Risk factors for *Staphylococcus aureus* nosocomial pneumonia in critically ill patients. *Am Rev Respir Dis* 1990;142:1320–4.
1432. (56) McWhinney PHM, Kibbler CC, Hamon MD, et al. Progress in the diagnosis and management of aspergillosis in bone marrow transplantation: 13 years' experience. *Clin Infect Dis* 1993;17:397–404.
1433. (74) Aisner J, Murillo J, Schimpff SC, Steere AC. Invasive aspergillosis in acute leukemia: Correlation with nose cultures and antibiotic use. *Ann Intern Med* 1979;90:4–9.
1434. (102) Rhamé FS. Endemic nosocomial filamentous fungal disease: a proposed structure for conceptualizing and studying the environmental hazard. *Infect Control* 1986;7S:124–5.
1435. (Table 1) Mutchler JE. Principles of ventilation. In: NIOSH. The industrial environment — its evaluation and control. Washington, DC: U.S. Department of Health, Education, and Welfare, Public Health Service, NIOSH, 1973. Publication #74-117. Available at: www.cdc.gov/niosh/74-117.html
1436. Breiman RF, Cozen W, Fields BS, et al. Role of air sampling in investigation of an outbreak of Legionnaires' disease associated with exposure to aerosols from an evaporative condenser. *J Infect Dis* 1990;161:1257–61.
1437. (Appendix; 9) CDC. Procedures for the recovery of *Legionella* from the environment. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service, CDC, 1992;1–13.
1438. Costerton JW, Lewandowski Z, Caldwell DE, Korber DR, Lappin-Scott HM. Microbial biofilms. *Ann Rev Microbiol* 1995;49:711–45.
1439. LeChevallier MW, Babcock TM, Lee RG. Examination and characterization of distribution system biofilms. *Appl Environ Microbiol* 1987;53:2714–24.
1440. Nagy LA, Olson BH. Occurrence and significance of bacteria, fungi, and yeasts associated with distribution pipe surfaces. *Proceeds of the Water Quality Technology Conference*. Portland, OR: 1985;213–38.
1441. Maki DG, Martin WT. Nationwide epidemic of septicemia caused by contaminated infusion products. IV growth of microbial pathogens in fluids for intravenous infusion. *J Infect Dis* 1975;131:267–72.
1442. Costerton JW, Stewart PS, Greenberg EP. Bacterial biofilms: a common cause of persistent infections. *Science* 1999;284:1318–22.
1443. Costerton JW, Khoury AE, Ward KH, Anwar H. Practical measures to control device-related bacterial infections. *Int J Artif Organs* 1993;16:765–70.
1444. Nickel JC, Costerton JW, McLean RJC, Olson M. Bacterial biofilms: influence on the pathogenesis, diagnosis, and treatment of urinary tract infections. *J Antimicrobial Chemother* 1994;33 (Suppl. A):31–41.
1445. LeChevallier MW, Cawthon CD, Lee RG. Inactivation of biofilm bacteria. *Appl Environ Microbiol* 1988;54:2492–9.

1446. Anwar J, Strap JL, Costerton JW. Establishment of aging biofilms: possible mechanism of bacterial resistance to antimicrobial therapy. *Antimicrobiol Agents Chemotherapy* 1992;36:1347–51.
1447. Stewart PS. Biofilm accumulation model that predicts antibiotic resistance to *Pseudomonas aeruginosa* biofilms. *Antimicrobiol Agents Chemotherapy* 1994;38:1052–8.
1448. Chen X, Stewart PS. Chlorine penetration into artificial biofilm is limited by a reaction-diffusion interaction. *Environ Sci Technol* 1996;30:2078–83.
1449. Huang C-T, Yu FP, McFeters GA, Stewart PS. Non-uniform spatial patterns of respiratory activity with biofilms during disinfection. *Appl Environ Microbiol* 1995;61:2252–6.
1450. Donlan RM, Pipes WO. Selected drinking water characteristics and attached microbial population density. *JAWWA* 1988;80:70–6.
1451. Reasoner DJ, Geldreich EE. A new medium for the enumeration and subculture of bacteria from potable water. *Appl Environ Microbiol* 1985;49:1–7.
1452. Pass T, Wright R, Sharp B, Harding GB. Culture of dialysis fluids on nutrient-rich media for short periods at elevated temperatures underestimates microbial contamination. *Blood Purif* 1996;14:136–45.
1453. Arduino MJ, Bland LA, Aguero SM, et al. Effects of incubation time and temperature on microbiologic sampling procedures for hemodialysis fluids. *J Clin Microbiol* 1991;29:1462–5.
1454. (234) Klein E, Pass T, Harding GB, Wright R, Million C. Microbial and endotoxin contamination in water and dialysate in the central United States. *Artif Organs* 1990;14:85–94.
1455. (235) Man N-K, Degremont A, Darbord J-C, Collet M, Vaillant P. Evidence of bacterial biofilm in tubing from hydraulic pathway of hemodialysis system. *Artif Organs* 1998;22:596–600.
1456. Pearson FC, Weary ME, Sargent HE, et al. Comparison of several control standard endotoxins to the National Reference Standard Endotoxin — an HIMA collaborative study. *Appl Environ Microbiol* 1985;50:91–3.
1457. (Appendix; 1) Arnow PM, Weil D, Para MF. Prevalence of significance of *Legionella pneumophila* contamination of residential hot-tap water systems. *J Infect Dis* 1985;152:145–51.
1458. (Appendix; 2) Shelton BG, Morris GK, Gorman GW. Reducing risks associated with *Legionella* bacteria in building water systems. In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;279–81.
1459. (Appendix; 3) Joly JR. Monitoring for the presence of *Legionella*: where, when, and how? In: Barbaree JM, Breiman RF, Dufour AP, eds. *Legionella: current status and emerging perspectives*. Washington, DC: American Society for Microbiology Press, 1993;211–6.
1460. (Appendix; 7) Brenner DJ, Feeley JC, Weaver RE. Family VII. *Legionellaceae*. In: Krieg NR, Holt JG, eds. *Bergey's manual of systemic bacteriology*, volume 1. Baltimore, MD: Williams & Wilkins, 1984;279–88.
1461. (Appendix; 8) Katz SM, Hammel JM. The effect of drying, heat, and pH on the survival of *Legionella pneumophila*. *Ann Clin Lab Sci* 1987;17:150–6.
1462. (Box 2) Alary MA, Joly JR. Comparison of culture methods and an immunofluorescence assay for the detection of *Legionella pneumophila* in domestic hot water devices. *Curr Microbiol* 1992;25:19–25.
1463. (Box 2) Vickers RM, Stout JE, Yu VL. Failure of a diagnostic monoclonal immunofluorescent reagent to detect *Legionella pneumophila* in environmental samples. *Appl Environ Microbiol* 1990;56:2912–4.
1464. (Box 2) Flournoy DJ, Belobraydic KA, Silberg SL, Lawrence CH, Guthrie PJ. False positive *Legionella pneumophila* direct immunofluorescence monoclonal antibody test caused by *Bacillus cereus* spores. *Diag Microbiol Infect Dis* 1988;9:123–5.
1465. (Box 2) Bej AK, Majbubani MH, Atlas RM. Detection of viable *Legionella pneumophila* in water by polymerase chain reaction and gene probe methods. *Appl Environ Microbiol* 1991;57:597–600.
1466. Schulze-Röbbecke R, Jung KD, Pullman H, Hundgeburth J. Control of *Legionella pneumophila* in a hospital hot water system. *Zbl Hyg* 1990;190:84–100.
1467. Colbourne JS, Pratt DJ, Smith MG, Fisher-Hoch SP, Harper D. Water fittings as sources of *Legionella pneumophila* in a hospital plumbing system. *Lancet* 1984;1:210–3.
1468. U.S. Environmental Protection Agency. National interim primary drinking water regulations: control of trihalomethanes in drinking water: final rules. *Federal Register* 1979;44:68624–705.
1469. U.S. Environmental Protection Agency. National interim primary drinking water regulations: Trihalomethanes. *Federal Register* 1983;48:8406–14.

Part IV. Appendices

Appendix A. Glossary of Terms

Acceptable indoor air quality: air in which there are no known contaminants at harmful concentrations as determined by knowledgeable authorities and with which a substantial majority ($\geq 80\%$) of the people exposed do not express dissatisfaction.

ACGIH: American Conference of Governmental Industrial Hygienists.

Action level: the concentration of a contaminant at which steps should be taken to interrupt the trend toward higher, unacceptable levels.

Aerosol: particles of respirable size generated by both humans and environmental sources and that have the capability of remaining viable and airborne for extended periods in the indoor environment.

AIA: American Institute of Architects, a professional group responsible for publishing the *Guidelines for Design and Construction of Hospitals and Healthcare Facilities*, a consensus document for design and construction of health-care facilities endorsed by the U.S. Department of Health and Human Services, health-care professionals, and professional organizations.

Air changes per hour (ACH): the ratio of the volume of air flowing through a space in a certain period of time (the airflow rate) to the volume of that space (the room volume). This ratio is expressed as the number of air changes per hour (ACH).

Air mixing: the degree to which air supplied to a room mixes with the air already in the room, usually expressed as a mixing factor. This factor varies from 1 (for perfect mixing) to 10 (for poor mixing). It is used as a multiplier to determine the actual airflow required (i.e., the recommended ACH multiplied by the mixing factor equals the actual ACH required).

Airborne transmission: a means of spreading infection when airborne droplet nuclei (small particle residue of evaporated droplets $\leq 5 \mu\text{m}$ in size containing microorganisms that remain suspended in air for long periods of time) are inhaled by the susceptible host.

Air-cleaning system: a device or combination of devices applied to reduce the concentration of airborne contaminants (e.g., microorganisms, dusts, fumes, aerosols, other particulate matter, and gases).

Air conditioning: the process of treating air to meet the requirements of a conditioned space by controlling its temperature, humidity, cleanliness, and distribution.

Allogeneic: non-twin, non-self. The term refers to transplanted tissue from a donor closely matched to a recipient but not related to that person.

Ambient air: the air surrounding an object.

Anemometer: a flow meter which measures the wind force and velocity of air. An anemometer is often used as a means of determining the volume of air being drawn into an air sampler.

Anteroom: a small room leading from a corridor into an isolation room. This room can act as an airlock, preventing the escape of contaminants from the isolation room into the corridor.

ASHE: American Society for Healthcare Engineering, an association affiliated with the American Hospital Association.

ASHRAE: American Society of Heating, Refrigerating, and Air-Conditioning Engineers Inc.

Autologous: self. The term refers to transplanted tissue whose source is the same as the recipient, or an identical twin.

Automated cyclor: a machine used during peritoneal dialysis which pumps fluid into and out of the patient while he/she sleeps.

Biochemical oxygen demand (BOD): a measure of the amount of oxygen removed from aquatic environments by aerobic microorganisms for their metabolic requirements. Measurement of BOD is used to determine the level of organic pollution of a stream or lake. The greater the BOD, the greater

the degree of water pollution. The term is also referred to as Biological Oxygen Demand (BOD).

Biological oxygen demand (BOD): an indirect measure of the concentration of biologically degradable material present in organic wastes (pertaining to water quality). It usually reflects the amount of oxygen consumed in five days by biological processes breaking down organic waste (BOD5).

Biosafety level: a combination of microbiological practices, laboratory facilities, and safety equipment determined to be sufficient to reduce or prevent occupational exposures of laboratory personnel to the microbiological agents they work with. There are four biosafety levels based on the hazards associated with the various microbiological agents.

BOD5: the amount of dissolved oxygen consumed in five days by biological processes breaking down organic matter.

Bonneting: a floor cleaning method for either carpeted or hard surface floors that uses a circular motion of a large fibrous disc to lift and remove soil and dust from the surface.

Capped spur: a pipe leading from the water recirculating system to an outlet that has been closed off ("capped"). A capped spur cannot be flushed, and it might not be noticed unless the surrounding wall is removed.

CFU/m³: colony forming units per cubic meter (of air).

Chlamydospores: thick-walled, typically spherical or ovoid resting spores asexually produced by certain types of fungi from cells of the somatic hyphae.

Chloramines: compounds containing nitrogen, hydrogen, and chlorine. These are formed by the reaction between hypochlorous acid (HOCl) and ammonia (NH₃) and/or organic amines in water. The formation of chloramines in drinking water treatment extends the disinfecting power of chlorine. The term is also referred to as Combined Available Chlorine.

Cleaning: the removal of visible soil and organic contamination from a device or surface, using either the physical action of scrubbing with a surfactant or detergent and water, or an energy-based process (e.g., ultrasonic cleaners) with appropriate chemical agents.

Coagulation-flocculation: coagulation is the clumping of particles that results in the settling of impurities. It may be induced by coagulants (e.g., lime, alum, and iron salts). Flocculation in water and wastewater treatment is the agglomeration or clustering of colloidal and finely-divided suspended matter after coagulation by gentle stirring by either mechanical or hydraulic means, such that they can be separated from water or sewage.

Commissioning (a room): testing a system or device to ensure that it meets the pre-use specifications as indicated by the manufacturer or predetermined standard, or air sampling in a room to establish a pre-occupancy baseline standard of microbial or particulate contamination. The term is also referred to as benchmarking at 77°F (25°C).

Completely packaged: functionally packaged, as for laundry.

Conidia: asexual spores of fungi borne externally.

Conidiophores: specialized hyphae that bear conidia in fungi.

Conditioned space: that part of a building that is heated or cooled, or both, for the comfort of the occupants.

Contaminant: an unwanted airborne constituent that may reduce the acceptability of air.

Convection: the transfer of heat or other atmospheric properties within the atmosphere or in the airspace of an enclosure by the circulation of currents from one region to another, especially by such motion directed upward.

Cooling tower: a structure engineered to receive accumulated heat from ventilation systems and equipment and transfer this heat to water, which then releases the stored heat to the atmosphere through evaporative cooling.

Critical item (medical instrument): a medical instrument or device that contacts normally sterile areas of the body or enters the vascular system. There is a high risk of infection from such devices if they are microbiologically contaminated prior to use. These devices must be sterilized before use.

Dead legs: areas in the water system where water stagnates. A dead leg is a pipe or spur, leading from the water recirculating system to an outlet that is used infrequently, resulting in inadequate flow of

water from the recirculating system to the outlet. This inadequate flow reduces the perfusion of heat or chlorine into this part of the water distribution system, thereby adversely affecting the disinfection of the water system in that area.

Deionization: removal of ions from water by exchange with other ions associated with fixed charges on a resin bed. Cations are usually removed and H^+ ions are exchanged; OH^- ions are exchanged for anions.

Detritus: particulate matter produced by or remaining after the wearing away or disintegration of a substance or tissue.

Dew point: the temperature at which a gas or vapor condenses to form a liquid; the point at which moisture begins to condense out of the air. At dew point, air is cooled to the point where it is at 100% relative humidity or saturation.

Dialysate: the aqueous electrolyte solution, usually containing dextrose, used to make a concentration gradient between the solution and blood in the hemodialyzer (dialyzer).

Dialyzer: a device that consists of two compartments (blood and dialysate) separated by a semipermeable membrane. A dialyzer is usually referred to as an artificial kidney.

Diffuser: the grille plate that disperses the air stream coming into the conditioned air space.

Direct transmission: involves direct body surface-to-body surface contact and physical transfer of microorganisms between a susceptible host and an infected/colonized person, or exposure to cloud of infectious particles within 3 feet of the source; the aerosolized particles are $>5 \mu m$ in size.

Disability: as defined by the Americans with Disabilities Act, a disability is any physical or mental impairment that substantially limits one or more major life activities, including but not limited to walking, talking, seeing, breathing, hearing, or caring for oneself.

Disinfection: a generally less lethal process of microbial inactivation (compared to sterilization) that eliminates virtually all recognized pathogenic microorganisms but not necessarily all microbial forms (e.g., bacterial spores).

Drain pans: pans that collect water within the HVAC system and remove it from the system. Condensation results when air and steam come together.

Drift: circulating water lost from the cooling tower in the form as liquid droplets entrained in the exhaust air stream (i.e., exhaust aerosols from a cooling tower).

Drift eliminators: an assembly of baffles or labyrinth passages through which the air passes prior to its exit from the cooling tower. The purpose of a drift eliminator is to remove entrained water droplets from the exhaust air.

Droplets: particles of moisture, such as are generated when a person coughs or sneezes, or when water is converted to a fine mist by a device such as an aerator or shower head. These particles may contain infectious microorganisms. Intermediate in size between drops and droplet nuclei, these particles tend to quickly settle out from the air so that any risk of disease transmission is generally limited to persons in close proximity to the droplet source.

Droplet nuclei: sufficiently small particles ($1-5 \mu m$ in diameter) that can remain airborne indefinitely and cause infection when a susceptible person is exposed at or beyond 3 feet of the source of these particles.

Dual duct system: an HVAC system that consists of parallel ducts that produce a cold air stream in one and a hot air stream in the other.

Dust: an air suspension of particles (aerosol) of any solid material, usually with particle sizes $\leq 100 \mu m$ in diameter.

Dust-spot test: a procedure that uses atmospheric air or a defined dust to measure a filter's ability to remove particles. A photometer is used to measure air samples on either side of the filter, and the difference is expressed as a percentage of particles removed.

Effective leakage area: the area through which air can enter or leave the room. This does not include supply, return, or exhaust ducts. The smaller the effective leakage area, the better isolated the room.

Endotoxin: the lipopolysaccharides of gram-negative bacteria, the toxic character of which resides in the lipid portion. Endotoxins generally produce pyrogenic reactions in persons exposed to these

bacterial components.

Enveloped virus: a virus whose outer surface is derived from a membrane of the host cell (either nuclear or the cell's outer membrane) during the budding phase of the maturation process. This membrane-derived material contains lipid, a component that makes these viruses sensitive to the action of chemical germicides.

Evaporative condenser: a wet-type, heat-rejection unit that produces large volumes of aerosols during the process of removing heat from conditioned space air.

Exhaust air: air removed from a space and not reused therein.

Exposure: the condition of being subjected to something (e.g., infectious agents) that could have a harmful effect.

Fastidious: having complex nutritional requirements for growth, as in microorganisms.

Fill: that portion of a cooling tower which makes up its primary heat transfer surface. Fill is alternatively known as "packing."

Finished water: treated, or potable water.

Fixed room-air HEPA recirculation systems: nonmobile devices or systems that remove airborne contaminants by recirculating air through a HEPA filter. These may be built into the room and permanently ducted or may be mounted to the wall or ceiling within the room. In either situation, they are fixed in place and are not easily movable.

Fomite: an inanimate object that may be contaminated with microorganisms and serves in their transmission.

Free and available chlorine: the term applied to the three forms of chlorine that may be found in solution (i.e., chlorine [Cl₂], hypochlorite [OCl⁻], and hypochlorous acid [HOCl]).

Germicide: a chemical that destroys microorganisms. Germicides may be used to inactivate microorganisms in or on living tissue (antiseptics) or on environmental surfaces (disinfectants).

Health-care-associated: an outcome, usually an infection, that occurs in any health-care facility as a result of medical care. The term "health-care-associated" replaces "nosocomial," the latter term being limited to adverse infectious outcomes occurring only in hospitals.

Hemodiafiltration: a form of renal replacement therapy in which waste solutes in the patient's blood are removed by both diffusion and convection through a high-flux membrane.

Hemodialysis: a treatment for renal replacement therapy in which waste solutes in the patient's blood are removed by diffusion and/or convection through the semipermeable membrane of an artificial kidney or dialyzer.

Hemofiltration: cleansing of waste products or other toxins from the blood by convection across a semipermeable, high-flux membrane where fluid balance is maintained by infusion of sterile, pyrogen-free substitution fluid pre- or post-hemodialyzer.

HEPA filter: High Efficiency Particulate Air filters capable of removing 99.97% of particles 0.3 μm in diameter and may assist in controlling the transmission of airborne disease agents. These filters may be used in ventilation systems to remove particles from the air or in personal respirators to filter air before it is inhaled by the person wearing the respirator. The use of HEPA filters in ventilation systems requires expertise in installation and maintenance. To test this type of filter, 0.3 μm particles of dioctylphthalate (DOP) are drawn through the filter. Efficiency is calculated by comparing the downstream and upstream particle counts. The optimal HEPA filter allows only three particles to pass through for every 10,000 particles that are fed to the filter.

Heterotrophic (heterotroph): that which requires some nutrient components from exogenous sources. Heterotrophic bacteria cannot synthesize all of their metabolites and therefore require certain nutrients from other sources.

High-efficiency filter: a filter with a particle-removal efficiency of 90%–95%.

High flux: a type of dialyzer or hemodialysis treatment in which large molecules (>8,000 daltons [e.g., β₂ microglobulin]) are removed from blood.

High-level disinfection: a disinfection process that inactivates vegetative bacteria, mycobacteria, fungi, and viruses, but not necessarily high numbers of bacterial spores.

Housekeeping surfaces: environmental surfaces (e.g., floors, walls, ceilings, and tabletops) that are not involved in direct delivery of patient care in health-care facilities.

Hoyer lift: an apparatus that facilitates the repositioning of the non-ambulatory patient from bed to wheelchair or gurney and subsequently to therapy equipment (immersion tanks).

Hubbard tank: a tank used in hydrotherapy that may accommodate whole-body immersion (e.g., as may be indicated for burn therapy). Use of a Hubbard tank has been replaced largely by bedside post-lavage therapy for wound care management.

HVAC: Heating, Ventilation, Air Conditioning.

Iatrogenic: induced in a patient by a physician's activity, manner, or therapy. The term is used especially in reference to an infectious complication or other adverse outcome of medical treatment.

Impactor: an air-sampling device in which particles and microorganisms are directed onto a solid surface and retained there for assay.

Impingement: an air-sampling method during which particles and microorganisms are directed into a liquid and retained there for assay.

Indirect transmission: involves contact of a susceptible host with a contaminated intermediate object, usually inanimate (a fomite).

Induction unit: the terminal unit of an in-room ventilation system. Induction units take centrally conditioned air and further moderate its temperature. Induction units are not appropriate for areas with high exhaust requirements (e.g., research laboratories).

Intermediate-level disinfection: a disinfection process that inactivates vegetative bacteria, most fungi, mycobacteria, and most viruses (particularly the enveloped viruses), but does not inactivate bacterial spores.

Isoform: a possible configuration (tertiary structure) of a protein molecule. With respect to prion proteins, the molecules with large amounts of α -conformation are the normal isoform of that particular protein, whereas those prions with large amounts of β -sheet conformation are the proteins associated with the development of spongiform encephalopathy (e.g., Creutzfeldt-Jakob disease [CJD]).

Laminar flow: HEPA-filtered air that is blown into a room at a rate of 90 ± 10 feet/min in a unidirectional pattern with 100 ACH–400 ACH.

Large enveloped virus: viruses whose particle diameter is >50 nm and whose outer surface is covered by a lipid-containing structure derived from the membranes of the host cells. Examples of large enveloped viruses include influenza viruses, herpes simplex viruses, and poxviruses.

Laser plume: the transfer of electromagnetic energy into tissues which results in a release of particles, gases, and tissue debris.

Lipid-containing viruses: viruses whose particle contains lipid components. The term is generally synonymous with enveloped viruses whose outer surface is derived from host cell membranes. Lipid-containing viruses are sensitive to the inactivating effects of liquid chemical germicides.

Lithotriptors: instruments used for crushing calculi (i.e., calcified stones, and sand) in the bladder or kidneys.

Low efficiency filter: the prefilter with a particle-removal efficiency of approximately 30% through which incoming air first passes. See also Prefilter.

Low-level disinfection: a disinfection process that will inactivate most vegetative bacteria, some fungi, and some viruses, but cannot be relied upon to inactivate resistant microorganisms (e.g., mycobacteria or bacterial spores).

Makeup air: outdoor air supplied to the ventilation system to replace exhaust air.

Makeup water: a cold water supply source for a cooling tower.

Manometer: a device that measures the pressure of liquids and gases. A manometer is used to verify air filter performance by measuring pressure differentials on either side of the filter.

Membrane filtration: an assay method suitable for recovery and enumeration of microorganisms from liquid samples. This method is used when sample volume is large and anticipated microbial contamination levels are low.

Mesophilic: that which favors a moderate temperature. For mesophilic bacteria, a temperature range of

68°F–131°F (20°C–55°C) is favorable for their growth and proliferation.

Mixing box: the site where the cold and hot air streams mix in the HVAC system, usually situated close to the air outlet for the room.

Mixing faucet: a faucet that mixes hot and cold water to produce water at a desired temperature.

MMAD: Mass Median Aerodynamic Diameter. This is the unit used by ACGIH to describe the size of particles when particulate air sampling is conducted.

Moniliaceous: hyaline or brightly colored. This is a laboratory term for the distinctive characteristics of certain opportunistic fungi in culture (e.g., *Aspergillus* spp. and *Fusarium* spp.).

Monochloramine: the result of the reaction between chlorine and ammonia that contains only one chlorine atom. Monochloramine is used by municipal water systems as a water treatment.

Natural ventilation: the movement of outdoor air into a space through intentionally provided openings (i.e., windows, doors, or nonpowered ventilators).

Negative pressure: air pressure differential between two adjacent airspaces such that air flow is directed into the room relative to the corridor ventilation (i.e., room air is prevented from flowing out of the room and into adjacent areas).

Neutropenia: a medical condition in which the patient's concentration of neutrophils is substantially less than that in the normal range. Severe neutropenia occurs when the concentration is <1,000 polymorphonuclear cells/ μ L for 2 weeks or <100 polymorphonuclear cells /mL for 1 week, particularly for hematopoietic stem cell transplant (HSCT) recipients.

Noncritical devices: medical devices or surfaces that come into contact with only intact skin. The risk of infection from use of these devices is low.

Non-enveloped virus: a virus whose particle is not covered by a structure derived from a membrane of the host cell. Non-enveloped viruses have little or no lipid compounds in their biochemical composition, a characteristic that is significant to their inherent resistance to the action of chemical germicides.

Nosocomial: an occurrence, usually an infection, that is acquired in a hospital as a result of medical care.

NTM: nontuberculous mycobacteria. These organisms are also known as atypical mycobacteria, or as "Mycobacteria other than tuberculosis" (MOTT). This descriptive term refers to any of the fast- or slow-growing *Mycobacterium* spp. found in primarily in natural or man-made waters, but it excludes *Mycobacterium tuberculosis* and its variants.

Nuisance dust: generally innocuous dust, not recognized as the direct cause of serious pathological conditions.

Oocysts: a cyst in which sporozoites are formed; a reproductive aspect of the life cycle of a number of parasitic agents (e.g., *Cryptosporidium* spp., and *Cyclospora* spp.).

Outdoor air: air taken from the external atmosphere and, therefore, not previously circulated through the ventilation system.

Parallel streamlines: a unidirectional airflow pattern achieved in a laminar flow setting, characterized by little or no mixing of air.

Particulate matter (particles): a state of matter in which solid or liquid substances exist in the form of aggregated molecules or particles. Airborne particulate matter is typically in the size range of 0.01–100 μ m diameter.

Pasteurization: a disinfecting method for liquids during which the liquids are heated to 140°F (60°C) for a short time (\geq 30 mins.) to significantly reduce the numbers of pathogenic or spoilage microorganisms.

Plinth: a treatment table or a piece of equipment used to reposition the patient for treatment.

Portable room-air HEPA recirculation units: free-standing portable devices that remove airborne contaminants by recirculating air through a HEPA filter.

Positive pressure: air pressure differential between two adjacent air spaces such that air flow is directed from the room relative to the corridor ventilation (i.e., air from corridors and adjacent areas is prevented from entering the room).

Potable (drinking) water: water that is fit to drink. The microbiological quality of this water as defined by EPA microbiological standards from the Surface Water Treatment Rule: a) *Giardia lamblia*: 99.9% killed/inactivated; b) viruses: 99.9% inactivated; c) *Legionella* spp.: no limit, but if *Giardia* and viruses are inactivated, *Legionella* will also be controlled; d) heterotrophic plate count [HPC]: ≤ 500 CFU/mL; and e) $>5\%$ of water samples total coliform-positive in a month.

PPE: Personal Protective Equipment.

ppm: parts per million. The term is a measure of concentration in solution. Chlorine bleaches (undiluted) that are available in the U.S. (5.25%–6.15% sodium hypochlorite) contain approximately 50,000–61,500 parts per million of free and available chlorine.

Prefilter: the first filter for incoming fresh air in a HVAC system. This filter is approximately 30% efficient in removing particles from the air. See also Low-Efficiency Filter.

Prion: a class of agent associated with the transmission of diseases known as transmissible spongiform encephalopathies (TSEs). Prions are considered to consist of protein only, and the abnormal isoform of this protein is thought to be the agent that causes diseases such as Creutzfeldt-Jakob disease (CJD), kuru, scrapie, bovine spongiform encephalopathy (BSE), and the human version of BSE which is variant CJD (vCJD).

Product water: water produced by a water treatment system or individual component of that system.

Protective environment: a special care area, usually in a hospital, designed to prevent transmission of opportunistic airborne pathogens to severely immunosuppressed patients.

Pseudoepidemic (pseudo-outbreak): a cluster of positive microbiologic cultures in the absence of clinical disease. A pseudoepidemic usually results from contamination of the laboratory apparatus and process used to recover microorganisms.

Pyrogenic: an endotoxin burden such that a patient would receive ≥ 5 endotoxin units (EU) per kilogram of body weight per hour, thereby causing a febrile response. In dialysis this usually refers to water or dialysate having endotoxin concentrations of ≥ 5 EU/mL.

Rank order: a strategy for assessing overall indoor air quality and filter performance by comparing airborne particle counts from lowest to highest (i.e., from the best filtered air spaces to those with the least filtration).

RAPD: a method of genotyping microorganisms by randomly amplified polymorphic DNA. This is one version of the polymerase chain reaction method.

Recirculated air: air removed from the conditioned space and intended for reuse as supply air.

Relative humidity: the ratio of the amount of water vapor in the atmosphere to the amount necessary for saturation at the same temperature. Relative humidity is expressed in terms of percent and measures the percentage of saturation. At 100% relative humidity, the air is saturated. The relative humidity decreases when the temperature is increased without changing the amount of moisture in the air.

Reprocessing (of medical instruments): the procedures or steps taken to make a medical instrument safe for use on the next patient. Reprocessing encompasses both cleaning and the final or terminal step (i.e., sterilization or disinfection) which is determined by the intended use of the instrument according to the Spaulding classification.

Residuals: the presence and concentration of a chemical in media (e.g., water) or on a surface after the chemical has been added.

Reservoir: a nonclinical source of infection.

Respirable particles: those particles that penetrate into and are deposited in the nonciliated portion of the lung. Particles >10 μm in diameter are not respirable.

Return air: air removed from a space to be then recirculated.

Reverse osmosis (RO): an advanced method of water or wastewater treatment that relies on a semi-permeable membrane to separate waters from pollutants. An external force is used to reverse the normal osmotic process resulting in the solvent moving from a solution of higher concentration to one of lower concentration.

Riser: water piping that connects the circulating water supply line, from the level of the base of the tower or supply header, to the tower's distribution system.

RODAC: Replicate Organism Direct Agar Contact. This term refers to a nutrient agar plate whose convex agar surface is directly pressed onto an environmental surface for the purpose of microbiologic sampling of that surface.

Room-air HEPA recirculation systems and units: devices (either fixed or portable) that remove airborne contaminants by recirculating air through a HEPA filter.

Routine sampling: environmental sampling conducted without a specific, intended purpose and with no action plan dependent on the results obtained.

Sanitizer: an agent that reduces microbial contamination to safe levels as judged by public health standards or requirements.

Saprophytic: a naturally-occurring microbial contaminant.

Sedimentation: the act or process of depositing sediment from suspension in water. The term also refers to the process whereby solids settle out of wastewater by gravity during treatment.

Semicritical devices: medical devices that come into contact with mucous membranes or non-intact skin.

Service animal: any animal individually trained to do work or perform tasks for the benefit of a person with a disability.

Shedding: the generation and dispersion of particles and spores by sources within the patient area, through activities such as patient movement and airflow over surfaces.

Single-pass ventilation: ventilation in which 100% of the air supplied to an area is exhausted to the outside.

Small, non-enveloped viruses: viruses whose particle diameter is <50 nm and whose outer surface is the protein of the particle itself and not that of host cell membrane components. Examples of small, non-enveloped viruses are polioviruses and hepatitis A virus.

Spaulding Classification: the categorization of inanimate medical device surfaces in the medical environment as proposed in 1972 by Dr. Earle Spaulding. Surfaces are divided into three general categories, based on the theoretical risk of infection if the surfaces are contaminated at time of use. The categories are “critical,” “semicritical,” and “noncritical.”

Specific humidity: the mass of water vapor per unit mass of moist air. It is expressed as grains of water per pound of dry air, or pounds of water per pound of dry air. The specific humidity changes as moisture is added or removed. However, temperature changes do not change the specific humidity unless the air is cooled below the dew point.

Splatter: visible drops of liquid or body fluid that are expelled forcibly into the air and settle out quickly, as distinguished from particles of an aerosol which remain airborne indefinitely.

Steady state: the usual state of an area.

Sterilization: the use of a physical or chemical procedure to destroy all microbial life, including large numbers of highly-resistant bacterial endospores.

Stop valve: a valve that regulates the flow of fluid through a pipe. The term may also refer to a faucet.

Substitution fluid: fluid that is used for fluid management of patients receiving hemodiafiltration. This fluid can be prepared on-line at the machine through a series of ultrafilters or with the use of sterile peritoneal dialysis fluid.

Supply air: air that is delivered to the conditioned space and used for ventilation, heating, cooling, humidification, or dehumidification.

Tensile strength: the resistance of a material to a force tending to tear it apart, measured as the maximum tension the material can withstand without tearing.

Therapy animal: an animal (usually a personal pet) that, with their owners or handlers, provide supervised, goal-directed intervention to clients in hospitals, nursing homes, special-population schools, and other treatment sites.

Thermophilic: capable of growing in environments warmer than body temperature.

Thermotolerant: capable of withstanding high temperature conditions.

TLV®: an exposure level under which most people can work consistently for 8 hours a day, day after day, without adverse effects. The term is used by the ACGIH to designate degree of exposure to

contaminants. TLV® can be expressed as approximate milligrams of particulate per cubic meter of air (mg/m^3). TLVs® are listed as either an 8-hour TWA (time weighted average) or a 15-minute STEL (short term exposure limit).

TLV-TWA: Threshold Limit Value-Time Weighted Average. The term refers to the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be exposed repeatedly, day after day, without adverse effects. The TLV-TWA for “particulates (insoluble) not otherwise classified” (PNOC) - (sometimes referred to as nuisance dust) - are those particulates containing no asbestos and <1% crystalline silica. A TLV-TWA of $10 \text{ mg}/\text{m}^3$ for inhalable particulates and a TLV-TWA of $3 \text{ mg}/\text{m}^3$ for respirable particulates (particulates $\leq 5 \mu\text{m}$ in aerodynamic diameter) have been established.

Total suspended particulate matter: the mass of particles suspended in a unit of volume of air when collected by a high-volume air sampler.

Transient: a change in the condition of the steady state that takes a very short time compared with the steady state. Opening a door, and shaking bed linens are examples of transient activities.

TWA: average exposure for an individual over a given working period, as determined by sampling at given times during the period. TWA is usually presented as the average concentration over an 8-hour workday for a 40-hour workweek.

Ultraclean air: air in laminar flow ventilation that has also passed through a bank of HEPA filters.

Ultrafilter: a membrane filter with a pore size in the range of $0.001\text{--}0.05 \mu\text{m}$, the performance of which is usually rated in terms of a nominal molecular weight cut-off (defined as the smallest molecular weight species for which the filter membrane has more than 90% rejection).

Ultrafiltered dialysate: the process by which dialysate is passed through a filter having a molecular weight cut-off of approximately 1 kilodalton for the purpose of removing bacteria and endotoxin from the bath.

Ultraviolet germicidal irradiation (UVGI): the use of ultraviolet radiation to kill or inactivate microorganisms.

Ultraviolet germicidal irradiation lamps: lamps that kill or inactivate microorganisms by emitting ultraviolet germicidal radiation, predominantly at a wavelength of 254 nm. UVGI lamps can be used in ceiling or wall fixtures or within air ducts of ventilation systems.

Vapor pressure: the pressure exerted by free molecules at the surface of a solid or liquid. Vapor pressure is a function of temperature, increasing as the temperature rises.

Vegetative bacteria: bacteria that are actively growing and metabolizing, as opposed to a bacterial state of quiescence that is achieved when certain bacteria (gram-positive bacilli) convert to spores when the environment can no longer support active growth.

Vehicle: any object, person, surface, fomite, or media that may carry and transfer infectious microorganisms from one site to another.

Ventilation: the process of supplying and removing air by natural or mechanical means to and from any space. Such air may or may not be conditioned.

Ventilation air: that portion of the supply air consisting of outdoor air plus any recirculated air that has been treated for the purpose of maintaining acceptable indoor air quality.

Ventilation, dilution: an engineering control technique to dilute and remove airborne contaminants by the flow of air into and out of an area. Air that contains droplet nuclei is removed and replaced by contaminant-free air. If the flow is sufficient, droplet nuclei become dispersed, and their concentration in the air is diminished.

Ventilation, local exhaust: ventilation used to capture and removed airborne contaminants by enclosing the contaminant source (the patient) or by placing an exhaust hood close to the contaminant source.

v/v: volume to volume. This term is an expression of concentration of a percentage solution when the principle component is added as a liquid to the diluent.

w/v: weight to volume. This term is an expression of concentration of a percentage solution when the principle component is added as a solid to the diluent.

Weight-arrestance: a measure of filter efficiency, used primarily when describing the performance of low- and medium-efficiency filters. The measurement of weight-arrestance is performed by feeding a standardized synthetic dust to the filter and weighing the fraction of the dust removed.

Appendix B. Air

1. Airborne Contaminant Removal

Table B.1. Air changes/hour (ACH) and time required for airborne-contaminant removal efficiencies of 99% and 99.9%*

ACH+ § ¶	Time (mins.) required for removal:	
	99% efficiency	99.9% efficiency
2	138	207
4	69	104
6	46	69
8	35	52
10	28	41
12	23	35
15	18	28
20	14	21
50	6	8

* This table is revised from Table S3-1 in reference 4 and has been adapted from the formula for the rate of purging airborne contaminants presented in reference 1435.

+ Shaded entries denote frequently cited ACH for patient-care areas.

§ Values were derived from the formula:

$$t_2 - t_1 = -[\ln(C_2 / C_1) / (Q / V)] \times 60, \text{ with } t_1 = 0 \text{ and where}$$

t_1 = initial timepoint in minutes

t_2 = final timepoint in minutes

C_1 = initial concentration of contaminant

C_2 = final concentration of contaminant

$C_2 / C_1 = 1 - (\text{removal efficiency} / 100)$

Q = air flow rate in cubic feet/hour

V = room volume in cubic feet

$Q / V = \text{ACH}$

¶ Values apply to an empty room with no aerosol-generating source. With a person present and generating aerosol, this table would not apply. Other equations are available that include a constant generating source. However, certain diseases (e.g., infectious tuberculosis) are not likely to be aerosolized at a constant rate. The times given assume perfect mixing of the air within the space (i.e., mixing factor = 1). However, perfect mixing usually does not occur. Removal times will be longer in rooms or areas with imperfect mixing or air stagnation.²¹³ Caution should be exercised in using this table in such situations. For booths or other local ventilation enclosures, manufacturers' instructions should be consulted.

2. Air Sampling for Aerosols Containing Legionellae

Air sampling is an insensitive means of detecting *Legionella pneumophila*, and is of limited practical value in environmental sampling for this pathogen. In certain instances, however, it can be used to a) demonstrate the presence of legionellae in aerosol droplets associated with suspected bacterial

reservoirs; b) define the role of certain devices [e.g., showers, faucets, decorative fountains, or evaporate condensers] in disease transmission; and c) quantitate and determine the size of the droplets containing legionellae.¹⁴³⁶ Stringent controls and calibration are necessary when sampling is used to determine particle size and numbers of viable bacteria.¹⁴³⁷ Samplers should be placed in locations where human exposure to aerosols is anticipated, and investigators should wear a NIOSH-approved respirator (e.g., N95 respirator) if sampling involves exposure to potentially infectious aerosols.

Methods used to sample air for legionellae include impingement in liquid, impaction on solid medium, and sedimentation using settle plates.¹⁴³⁶ The Chemical Corps.-type all-glass impingers (AGI) with the stem 30 mm from the bottom of the flask have been used successfully to sample for legionellae.¹⁴³⁶ Because of the velocity at which air samples are collected, clumps tend to become fragmented, leading to a more accurate count of bacteria present in the air. The disadvantages of this method are a) the velocity of collection tends to destroy some vegetative cells; b) the method does not differentiate particle sizes; and c) AGIs are easily broken in the field. Yeast extract broth (0.25%) is the recommended liquid medium for AGI sampling of legionellae;¹⁴³⁷ standard methods for water samples can be used to culture these samples.

Andersen samplers are viable particle samplers in which particles pass through jet orifices of decreasing size in cascade fashion until they impact on an agar surface.¹²¹⁸ The agar plates are then removed and incubated. The stage distribution of the legionellae should indicate the extent to which the bacteria would have penetrated the respiratory system. The advantages of this sampling method are a) the equipment is more durable during use; b) the sampler can determine the number and size of droplets containing legionellae; c) the agar plates can be placed directly in an incubator with no further manipulations; and d) both selective and nonselective BCYE agar can be used. If the samples must be shipped to a laboratory, they should be packed and shipped without refrigeration as soon as possible.

3. Calculation of Air Sampling Results

Assuming that each colony on the agar plate is the growth from a single bacteria-carrying particle, the contamination of the air being sampled is determined from the number of colonies counted. The airborne microorganisms may be reported in terms of the number per cubic foot of air sampled. The following formulas can be applied to convert colony counts to organisms per cubic foot of air sampled.¹²¹⁸

For solid agar impactor samplers:

$$C / (R \times P) = N \quad \text{where}$$

N = number of organisms collected per cubic foot of air sampled
 C = total plate count
 R = airflow rate in cubic feet per minute
 P = duration of sampling period in minutes

For liquid impingers:

$$(C \times V) / (Q \times P \times R) = N \quad \text{where}$$

C = total number of colonies from all aliquots plated
 V = final volume in mL of collecting media
 Q = total number of mL plated
 P, R, and N are defined as above

4. Ventilation Specifications for Health-Care Facilities

The following tables from the AIA *Guidelines for Design and Construction of Hospitals and Health-Care Facilities, 2001* are reprinted with permission of the American Institute of Architects and the publisher (The Facilities Guidelines Institute).¹²⁰

Table B.2. Ventilation requirements for areas affecting patient care in hospitals and outpatient facilities¹

Notes: This table is Table 7.2 in the AIA guidelines, 2001 edition. Superscripts used in this table refer to notes following the table.

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
<u>Surgeru and critical care</u>							
Operating/surgical cystoscopic rooms ^{10, 11}	Out	3	15	–	No	30–60	68–73 (20–23) ¹²
Delivery room ¹⁰	Out	3	15	–	No	30–60	68–73 (20–23)
Recovery room ¹⁰	–	2	6	–	No	30–60	70–75 (21–24)
Critical and intensive care	–	2	6	–	No	30–60	70–75 (21–24)
Newborn intensive care	–	2	6	–	No	30–60	72–78 (22–26)
Treatment room ¹³	–	–	6	–	–	–	75 (24)
Trauma room ¹³	Out	3	15	–	No	30–60	70–75 (21–24)
Anesthesia gas storage	In	–	8	Yes	–	–	–
Endoscopy	In	2	6	–	No	30–60	68–73 (20–23)
Bronchoscopy ¹¹	In	2	12	Yes	No	30–60	68–73 (20–23)
ER waiting rooms	In	2	12	Yes ^{14, 15}	–	–	70–75 (21–24)
Triage	In	2	12	Yes ¹⁴	–	–	70–75 (21–24)
Radiology waiting rooms	In	2	12	Yes ^{14, 15}	–	–	70–75 (21–24)
Procedure room	Out	3	15	–	No	30–60	70–75 (21–24)
<u>Nursing</u>							
Patient room	–	2	6 ¹⁶	–	–	–	70–75 (21–24)
Toilet room	In	–	10	Yes	–	–	–
Newborn nursery suite	–	2	6	–	No	30–60	72–78 (22–26)
Protective environment room ^{11, 17}	Out	2	12	–	No	–	75 (24)
Airborne infection isolation room ^{17, 18}	In	2	12	Yes ¹⁵	No	–	75 (24)
Isolation alcove or anteroom ^{17, 18}	In/Out	–	10	Yes	No	–	–
Labor/delivery/recovery	–	2	6 ¹⁶	–	–	–	70–75 (21–24)
Labor/delivery/recovery/postpartum	–	2	6 ¹⁶	–	–	–	70–75 (21–24)
Patient corridor	–	–	2	–	–	–	–

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
<u>Ancillary</u>							
Radiology¹⁹							
X-ray (surgical/critical care and catheterization)	Out	3	15	–	No	30-60	70–75 (21–24)
X-ray (diagnostic & treatment)	–	–	6	–	–	–	75 (24)
Darkroom	In	–	10	Yes	No	–	–
Laboratory							
General ¹⁹	–	–	6	–	–	–	75 (24)
Biochemistry ¹⁹	Out	–	6	–	No	–	75 (24)
Cytology	In	–	6	Yes	No	–	75 (24)
Glass washing	In	–	10	Yes	–	–	–
Histology	In	–	6	Yes	No	–	75 (24)
Microbiology ¹⁹	In	–	6	Yes	No	–	75 (24)
Nuclear medicine	In	–	6	Yes	No	–	75 (24)
Pathology	In	–	6	Yes	No	–	75 (24)
Serology	Out	–	6	–	No	–	75 (24)
Sterilizing	In	–	10	Yes	–	–	–
Autopsy room ¹¹	In	–	12	Yes	No	–	–
Nonrefrigerated body-holding room	In	–	10	Yes	–	–	70 (21)
Pharmacy	Out	–	4	–	–	–	–
<u>Diagnostic and treatment</u>							
Examination room	–	–	6	–	–	–	75 (24)
Medication room	Out	–	4	–	–	–	–
Treatment room	–	–	6	–	–	–	75 (24)
Physical therapy and hydrotherapy	In	–	6	–	–	–	75 (24)
Soiled workroom or soiled holding	In	–	10	Yes	No	–	–
Clean workroom or clean holding	Out	–	4	–	–	–	–
<u>Sterilizing and supply</u>							
ETO-sterilizer room	In	–	10	Yes	No	30-60	75 (24)
Sterilizer equipment room	In	–	10	Yes	–	–	–
Central medical and surgical supply							
Soiled or decontamination room	In	–	6	Yes	No	–	68–73 (20–23)
Clean workroom	Out	–	4	–	No	30-60	75 (24)
Sterile storage	Out	–	4	–	–	(Max.) 70	–

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ^{4, 5}	All air exhausted directly to outdoors ⁶	Recirculated by means of room units ⁷	Relative humidity ⁸ (%)	Design temperature ⁹ (degrees F [C])
Service							
Food preparation center ²⁰	–	–	10	–	No	–	–
Ware washing	In	–	10	Yes	No	–	–
Dietary day storage	In	–	2	–	–	–	–
Laundry, general	–	–	10	Yes	–	–	–
Soiled linen (sorting and storage)	In	–	10	Yes	No	–	–
Clean linen storage	Out	–	2	–	–	–	–
Soiled linen and trash chute room	In	–	10	Yes	No	–	–
Bedpan room	In	–	10	Yes	–	–	–
Bathroom	In	–	10	–	–	–	75 (24)
Janitor's closet	In	–	10	Yes	No	–	–

Notes:

1. The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of acute care hospitals that directly affect patient care and are determined based on health-care facilities being predominantly “No Smoking” facilities. Where smoking may be allowed, ventilation rates will need adjustment. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook - HVAC Applications*. Specialized patient care areas, including organ transplant units, burn units, specialty procedure rooms, etc., shall have additional ventilation provisions for air quality control as may be appropriate. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within health-care facilities.
2. Design of the ventilation system shall provide air movement which is generally from clean to less clean areas. If any form of variable air volume or load shedding system is used for energy conservation, it must not compromise the corridor-to-room pressure balancing relationships or the minimum air changes required by the table.
3. To satisfy exhaust needs, replacement air from the outside is necessary. Table B2 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice. Minimum outside air quantities shall remain constant while the system is in operation.
4. Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed, if the maximum infiltration or exfiltration permitted in Note 2 is not exceeded and if adjacent pressure balancing relationships are not compromised. Air quantity calculations must account for filter loading such that the indicated air change rates are provided up until the time of filter change-out.
5. Air change requirements indicated are minimum values. Higher values should be used when required to maintain indicated room conditions (temperature and humidity), based on the cooling load of the space (lights, equipment, people, exterior walls and windows, etc.).

6. Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to the outside, (e.g., in intensive care units in which patients with pulmonary infection are treated) and rooms for burn patients.
7. Recirculating room HVAC units refer to those local units that are used primarily for heating and cooling of air, and not disinfection of air. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked “No.” However, for airborne infection control, air may be recirculated within individual isolation rooms if HEPA filters are used. Isolation and intensive care unit rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in operating rooms and other special care areas. See this table’s Appendix I for a description of recirculation units to be used in isolation rooms (A7).
8. The ranges listed are the minimum and maximum limits where control is specifically needed. The maximum and minimum limits are not intended to be independent of a space’s associated temperature. The humidity is expected to be at the higher end of the range when the temperature is also at the higher end, and vice versa.
9. Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range during normal operation. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable when patients may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the patients' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.
10. National Institute for Occupational Safety and Health (NIOSH) criteria documents regarding “Occupational Exposure to Waste Anesthetic Gases and Vapors,” and “Control of Occupational Exposure to Nitrous Oxide” indicate a need for both local exhaust (scavenging) systems and general ventilation of the areas in which the respective gases are utilized.
11. Differential pressure shall be a minimum of 0.01" water gauge (2.5 Pa). If alarms are installed, allowances shall be made to prevent nuisance alarms of monitoring devices.
12. Some surgeons may require room temperatures which are outside of the indicated range. All operating room design conditions shall be developed in consultation with surgeons, anesthesiologists, and nursing staff.
13. The term “trauma room” as used here is the operating room space in the emergency department or other trauma reception area that is used for emergency surgery. The “first aid room” and/or “emergency room” used for initial treatment of accident victims may be ventilated as noted for the “treatment room.” Treatment rooms used for bronchoscopy shall be treated as Bronchoscopy rooms. Treatment rooms used for cryosurgery procedures with nitrous oxide shall contain provisions for exhausting waste gases.
14. In a ventilation system that recirculates air, HEPA filters can be used in lieu of exhausting the air from these spaces to the outside. In this application, the return air shall be passed through the HEPA filters before it is introduced into any other spaces.
15. If it is not practical to exhaust the air from the airborne infection isolation room to the outside, the air may be returned through HEPA filters to the air-handling system exclusively serving the isolation room.
16. Total air changes per room for patient rooms, labor/delivery/recovery rooms, and labor/delivery/recovery/postpartum rooms may be reduced to 4 when supplemental heating and/or cooling systems (radiant heating and cooling, baseboard heating, etc.) are used.
17. The protective environment airflow design specifications protect the patient from common environmental airborne infectious microbes (i.e., *Aspergillus* spores). These special ventilation areas shall be designed to provide directed airflow from the cleanest patient care area to less clean areas. These rooms shall be protected with HEPA filters at 99.97 percent efficiency for a 0.3 μm sized particle in the supply airstream. These interrupting filters protect patient rooms from maintenance-derived release of environmental microbes from the ventilation system components. Recirculation HEPA filters can be used to increase the equivalent room air exchanges. Constant volume airflow is required for consistent ventilation for the protected environment. If the facility determines that airborne infection isolation is necessary for protective environment patients, an anteroom should be

provided. Rooms with reversible airflow provisions for the purpose of switching between protective environment and airborne infection isolation functions are not acceptable.

18. The infectious disease isolation room described in these guidelines is to be used for isolating the airborne spread of infectious diseases, such as measles, varicella, or tuberculosis. The design of airborne infection isolation (AII) rooms should include the provision for normal patient care during periods not requiring isolation precautions. Supplemental recirculating devices may be used in the patient room to increase the equivalent room air exchanges; however, such recirculating devices do not provide the outside air requirements. Air may be recirculated within individual isolation rooms if HEPA filters are used. Rooms with reversible airflow provisions for the purpose of switching between protective environment and AII functions are not acceptable.

19. When required, appropriate hoods and exhaust devices for the removal of noxious gases or chemical vapors shall be provided (see Section 7.31.D14 and 7.31.D15 in the AIA guideline [reference 120] and NFPA 99).

20. Food preparation centers shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use. See Section 7.31.D1.p in the AIA guideline (reference 120).

Appendix I:

A7. Recirculating devices with HEPA filters may have potential uses in existing facilities as interim, supplemental environmental controls to meet requirements for the control of airborne infectious agents. Limitations in design must be recognized. The design of either portable or fixed systems should prevent stagnation and short circuiting of airflow. The supply and exhaust locations should direct clean air to areas where health-care workers are likely to work, across the infectious source, and then to the exhaust, so that the health-care worker is not in position between the infectious source and the exhaust location. The design of such systems should also allow for easy access for scheduled preventative maintenance and cleaning.

A11. The verification of airflow direction can include a simple visual method such as smoke trail, ball-in-tube, or flutterstrip. These devices will require a minimum differential air pressure to indicate airflow direction.

Table B.3. Pressure relationships and ventilation of certain areas of nursing facilities¹

Notes: This table is Table 8.1 in the AIA guidelines, 2001 edition. Superscripts used in this table refer to notes following the table.

Area designation	Air movement relationship to adjacent area ²	Minimum air changes of outdoor air per hour ³	Minimum total air changes per hour ⁴	All air exhausted directly to outdoors ⁵	Recirculated by means of room units ⁶	Relative humidity ⁷ (%)	Design temperature ⁸ (degrees F [C])
Resident room	–	2	2	–	–	⁹	70–75 (21–24)
Resident unit corridor	–	–	4	–	–	⁹	–
Resident gathering areas	–	4	4	–	–	–	–
Toilet room	In	–	10	Yes	No	–	–
Dining rooms	–	2	4	–	–	–	75 (24)
Activity rooms, if provided	–	4	4	–	–	–	–
Physical therapy	In	2	6	–	–	–	75 (24)
Occupational therapy	In	2	6	–	–	–	75.(24)
Soiled workroom or soiled holding	In	2	10	Yes	No	–	–
Clean workroom or clean holding	Out	2	4	–	–	(Max. 70)	75 (24)
Sterilizer exhaust room	In	–	10	Yes	No	–	–
Linen and trash chute room, if provided	In	–	10	Yes	No	–	–
Laundry, general, if provided	–	2	10	Yes	No	–	–
Soiled linen sorting and storage	In	–	10	Yes	No	–	–
Clean linen storage	Out	–	2	Yes	No	–	–
Food preparation facilities ¹⁰	–	2	10	Yes	No	–	–
Dietary warewashing	In	–	10	Yes	No	–	–
Dietary storage areas	–	–	2	Yes	No	–	–
Housekeeping rooms	In	–	10	Yes	No	–	–
Bathing rooms	In	–	10	Yes	No	–	75 (24)

Notes:

1. The ventilation rates in this table cover ventilation for comfort, as well as for asepsis and odor control in areas of nursing facilities that directly affect resident care and are determined based on nursing facilities being predominantly “No Smoking” facilities. Where smoking may be allowed, ventilation rates will need adjustment. Areas where specific ventilation rates are not given in the table shall be ventilated in accordance with ASHRAE Standard 62, *Ventilation for Acceptable Indoor Air Quality*, and ASHRAE *Handbook - HVAC Applications*. OSHA standards and/or NIOSH criteria require special ventilation requirements for employee health and safety within nursing facilities.

2. Design of the ventilation system shall, insofar as possible, provide that air movement is from clean to less clean areas. However, continuous compliance may be impractical with full utilization of some forms of variable air volume and load shedding systems that may be used for energy conservation. Areas that do require positive and continuous control are noted with “Out” or “In” to indicate the required direction of air movement in relation to the space named. Rate of air movement may, of course, be varied as needed

within the limits required for positive control. Where indication of air movement direction is enclosed in parentheses, continuous directional control is required only when the specialized equipment or device is in use or where room use may otherwise compromise the intent of movement from clean to less clean. Air movement for rooms with dashes and nonpatient areas may vary as necessary to satisfy the requirements of those spaces. Additional adjustments may be needed when space is unused or unoccupied and air systems are deenergized or reduced.

3. To satisfy exhaust needs, replacement air from outside is necessary. Table B.3 does not attempt to describe specific amounts of outside air to be supplied to individual spaces except for certain areas such as those listed. Distribution of the outside air, added to the system to balance required exhaust, shall be as required by good engineering practice.

4. Number of air changes may be reduced when the room is unoccupied if provisions are made to ensure that the number of air changes indicated is reestablished any time the space is being utilized. Adjustments shall include provisions so that the direction of air movement shall remain the same when the number of air changes is reduced. Areas not indicated as having continuous directional control may have ventilation systems shut down when space is unoccupied and ventilation is not otherwise needed.

5. Air from areas with contamination and/or odor problems shall be exhausted to the outside and not recirculated to other areas. Note that individual circumstances may require special consideration for air exhaust to outside.

6. Because of cleaning difficulty and potential for buildup of contamination, recirculating room units shall not be used in areas marked "No." Isolation rooms may be ventilated by reheat induction units in which only the primary air supplied from a central system passes through the reheat unit. Gravity-type heating or cooling units such as radiators or convectors shall not be used in special care areas.

7. The ranges listed are the minimum and maximum limits where control is specifically needed. See A8.31.D in the AIA guideline (reference 120) for additional information.

8. Where temperature ranges are indicated, the systems shall be capable of maintaining the rooms at any point within the range. A single figure indicates a heating or cooling capacity of at least the indicated temperature. This is usually applicable where residents may be undressed and require a warmer environment. Nothing in these guidelines shall be construed as precluding the use of temperatures lower than those noted when the residents' comfort and medical conditions make lower temperatures desirable. Unoccupied areas such as storage rooms shall have temperatures appropriate for the function intended.

9. See A8.31.D1 in the AIA guideline (reference 120).

10. Food preparation facilities shall have ventilation systems whose air supply mechanisms are interfaced appropriately with exhaust hood controls or relief vents so that exfiltration or infiltration to or from exit corridors does not compromise the exit corridor restrictions of NFPA 90A, the pressure requirements of NFPA 96, or the maximum defined in the table. The number of air changes may be reduced or varied to any extent required for odor control when the space is not in use.

Table B.4. Filter efficiencies for central ventilation and air conditioning systems in general hospitals*

Note: This table is Table 7.3 in the AIA guidelines, 2001 edition.

Area designation	Number of filter beds	Filter bed No.1 (%)	Filter bed No. 2 (%)
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct service or clean supplies, such as sterile and clean processing, etc.	2	30	90
Protective environment room	2	30	99.97
Laboratories	1	80	–
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	–

* Additional roughing or prefilters should be considered to reduce maintenance required for filters with efficiency higher than 75 percent. The filtration efficiency ratings are based on average dust sopt efficiency per ASHRAE 52.1–1992.

Table B.5. Filter efficiencies for central ventilation and air conditioning systems in outpatient facilities*

Note: This table is Table 9.1 in the AIA guidelines, 2001 edition.

Area designation	Number of filter beds	Filter bed No. 1 (%)	Filter bed No. 2+ (%)
All areas for patient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies such as sterile and clean processing, etc.	2	30	90
Laboratories	1	80	–
Administrative, bulk storage, soiled holding areas, food preparation areas, and laundries	1	30	–

* Additional roughing or prefilters should be considered to reduce maintenance required for main filters. The filtration efficiency ratings are based on dust spot efficiency per ASHRAE 52.1–1992.

+ These requirements do not apply to small primary (e.g., neighborhood) outpatient facilities or outpatient facilities that do not perform invasive applications or procedures.

Table B.6. Filter efficiencies for central ventilation and air conditioning systems in nursing facilities

Note: This table is Table 8.2 in the AIA guidelines, 2001 edition.

Area designation	Minimum number of filter beds	Filter bed No. 1 (%)*	Filter bed No. 2 (%)*
All areas for inpatient care, treatment, and/or diagnosis, and those areas providing direct service or clean supplies	2	30	80
Administrative, bulk storage, soiled holding, laundries, and food preparation areas	1	30	–

* The filtration efficiency ratings are based on average dust spot efficiency as per ASHRAE 52.1–1992.

Table B.7. Filter efficiencies for central ventilation and air conditioning systems in psychiatric hospitals

Note: This table is Table 11.1 in the AIA guidelines, 2001 edition.

Area designation	Minimum number of filter beds	Filter bed No. 1 (%)*	Filter bed No. 2 (%)*
All areas for inpatient care, treatment, and diagnosis, and those areas providing direct services	2	30	90
Administrative, bulk storage, soiled holding, laundries, and food preparation areas	1	30	–

* The filtration efficiency ratings are based on average dust spot efficiency as per ASHRAE 52.1–1992.

Appendix C. Water

1. Biofilms

Microorganisms have a tendency to associate with and stick to surfaces. These adherent organisms can initiate and develop biofilms, which are comprised of cells embedded in a matrix of extracellularly produced polymers and associated abiotic particles.¹⁴³⁸ It is inevitable that biofilms will form in most water systems. In the health-care facility environment, biofilms may be found in the potable water supply piping, hot water tanks, air conditioning cooling towers, or in sinks, sink traps, aerators, or shower heads. Biofilms, especially in water systems, are not present as a continuous slime or film, but

are more often scanty and heterogeneous in nature.¹⁴³⁹ Biofilms may form under stagnant as well as flowing conditions, so storage tanks, in addition to water system piping, may be vulnerable to the development of biofilm, especially if water temperatures are low enough to allow the growth of thermophilic bacteria (e.g., *Legionella* spp.). Favorable conditions for biofilm formation are present if these structures and equipment are not cleaned for extended periods of time.¹⁴⁴⁰

Algae, protozoa, and fungi may be present in biofilms, but the predominant microorganisms of water system biofilms are gram-negative bacteria. Although most of these organisms will not normally pose a problem for healthy individuals, certain biofilm bacteria (e.g., *Pseudomonas aeruginosa*, *Klebsiella* spp., *Pantoea agglomerans*, and *Enterobacter cloacae*) all may be agents for opportunistic infections for immunocompromised individuals.^{1441, 1442} These biofilm organisms may easily contaminate indwelling medical devices or intravenous (IV) fluids, and they could be transferred on the hands of health-care workers.^{1441–1444} Biofilms may potentially provide an environment for the survival of pathogenic organisms, such as *Legionella pneumophila* and *E. coli* O157:H7. Although the association of biofilms and medical devices provides a plausible explanation for a variety of health-care-associated infections, it is not clear how the presence of biofilms in the water system may influence the rates of health-care-associated waterborne infection.

Organisms within biofilms behave quite differently than their planktonic (i.e., free floating) counterparts. Research has shown that biofilm-associated organisms are more resistant to antibiotics and disinfectants than are planktonic organisms, either because the cells are protected by the polymer matrix, or because they are physiologically different.^{1445–1450} Nevertheless, municipal water utilities attempt to maintain a chlorine residual in the distribution system to discourage microbiological growth. Though chlorine in its various forms is a proven disinfectant, it has been shown to be less effective against biofilm bacteria.¹⁴⁴⁸ Higher levels of chlorine for longer contact times are necessary to eliminate biofilms.

Routine sampling of health-care facility water systems for biofilms is not warranted. If an epidemiologic investigation points to the water supply system as a possible source of infection, then water sampling for biofilm organisms should be considered so that prevention and control strategies can be developed. An established biofilm is difficult to remove totally in existing piping. Strategies to remediate biofilms in a water system would include flushing the system piping, hot water tank, dead legs, and those areas of the facility's water system subject to low or intermittent flow. The benefits of this treatment would include a) elimination of corrosion deposits and sludge from the bottom of hot water tanks, b) removal of biofilms from shower heads and sink aerators, and c) circulation of fresh water containing elevated chlorine residuals into the health-care facility water system.

The general strategy for evaluating water system biofilm depends on a comparison of the bacteriological quality of the incoming municipal water and that of water sampled from within facility's distribution system. Heterotrophic plate counts and coliform counts, both of which are routinely run by the municipal water utility, will at least provide an indication of the potential for biofilm formation. Heterotrophic plate count levels in potable water should be <500 CFU/mL. These levels may increase on occasion, but counts consistently >500 CFU/mL would indicate a general decrease in water quality. A direct correlation between heterotrophic plate count and biofilm levels has been demonstrated.¹⁴⁵⁰ Therefore, an increase in heterotrophic plate count would suggest a greater rate and extent of biofilm formation in a health-care facility water system. The water supplied to the facility should also contain <1 coliform bacteria/100 mL. Coliform bacteria are organisms whose presence in the distribution system could indicate fecal contamination. It has been shown that coliform bacteria can colonize biofilms within drinking water systems. Intermittant contamination of a water system with these organisms could lead to colonization of the system.

Water samples can be collected from throughout the health-care facility system, including both hot and cold water sources; samples should be cultured by standard methods.⁹⁴⁵ If heterotrophic plate counts in samples from the facility water system are higher than those from samples collected at the point of water entry to the building, it can be concluded that the facility water quality has diminished. If biofilms are detected in the facility water system and determined by an epidemiologic and environmental investigation to be a reservoir for health-care-associated pathogens, the municipal water supplier could be contacted with a request to provide higher chlorine residuals in the distribution system, or the health-care facility could consider installing a supplemental chlorination system.

Sample collection sites for biofilm in health-care facilities include a) hot water tanks; b) shower heads; and c) faucet aerators, especially in immunocompromised patient-care areas. Swabs should be placed into tubes containing phosphate buffered water, pH 7.2 or phosphate buffered saline, shipped to the laboratory under refrigeration and processed within 24 hrs. of collection. Samples are suspended by vortexing with sterile glass beads and plated onto a nonselective medium (e.g., Plate Count Agar or R2A medium) and selective media (e.g., media for *Legionella* spp. isolation) after serial dilution. If the plate counts are elevated above levels in the water (i.e. comparing the plate count per square centimeter of swabbed surface to the plate count per milliliter of water), then biofilm formation can be suspected. In the case of an outbreak, it would be advisable to isolate organisms from these plates to determine whether the suspect organisms are present in the biofilm or water samples and compare them to the organisms isolated from patient specimens.

2. Water and Dialysate Sampling Strategies in Dialysis

In order to detect the low, total viable heterotrophic plate counts outlined by the current AAMI standards for water and dialysate in dialysis settings, it is necessary to use standard quantitative culture techniques with appropriate sensitivity levels.^{792, 832, 833} The membrane filter technique is particularly suited for this application because it permits large volumes of water to be assayed.^{792, 834} Since the membrane filter technique may not be readily available in clinical laboratories, the spread plate assay can be used as an alternative.⁸³⁴ If the spread plate assay is used, however, the standard prohibits the use of a calibrated loop when applying sample to the plate.⁷⁹² The prohibition is based on the low sensitivity of the calibrated loop. A standard calibrated loop transfers 0.001 mL of sample to the culture medium, so that the minimum sensitivity of the assay is 1,000 CFU/mL. This level of sensitivity is unacceptable when the maximum allowable limit for microorganisms is 200 CFU/mL. Therefore, when the spread plate method is used, a pipette must be used to place 0.1–0.5 mL of water on the culture medium.

The current AAMI standard specifically prohibits the use of nutrient-rich media (e.g., blood agar, and chocolate agar) in dialysis water and dialysate assays because these culture media are too rich for growth of the naturally occurring organisms found in water.⁷⁹² Debate continues within AAMI, however, as to the most appropriate culture medium and incubation conditions to be used. The original clinical observations on which the microbiological requirements of this standard were based used Standard Methods Agar (SMA), a medium containing relatively few nutrients.⁶⁶⁶ The use of tryptic soy agar (TSA), a general purpose medium for isolating and cultivating microorganisms was recommended in later versions of the standard because it was thought to be more appropriate for culturing bicarbonate-containing dialysate.^{788, 789, 835} Moreover, culturing systems based on TSA are readily available from commercial sources. Several studies, however, have shown that the use of nutrient-poor media, such as R2A, results in an increased recovery of bacteria from water.^{1451, 1452} The original standard also specified incubation for 48 hours at 95°F–98.6°F (35°C–37°C) before enumeration of bacterial colonies. Extending the culturing time up to 168 hours, or 7 days and using incubation temperatures of 73.4°F–82.4°F (23°C–28°C) have also been shown to increase the recovery of bacteria.^{1451, 1452} Other

investigators, however, have not found such clear cut differences between culturing techniques.^{835, 1453} After considerable discussion, the AAMI Committee has not reached a consensus regarding changes in the assay technique, and the use of TSA or its equivalent for 48 hours at 95°F–98.6°F (35°C–37°C) remains the recommended method. It should be recognized, however, that these culturing conditions may underestimate the bacterial burden in the water and fail to identify the presence of some organisms. Specifically, the recommended method may not detect the presence of various NTM that have been associated with several outbreaks of infection in dialysis units.^{31, 32} In these instances, however, the high numbers of mycobacteria in the water were related to the total heterotrophic plate counts, each of which was significantly greater than that allowable by the AAMI standard. Additionally, the recommended method will not detect fungi and yeast, which have been shown to contaminate water used for hemodialysis applications.¹⁴⁵⁴ Biofilm on the surface of the pipes may hide viable bacterial colonies, even though no viable colonies are detected in the water using sensitive culturing techniques.¹⁴⁵⁵ Many disinfection processes remove biofilm poorly, and a rapid increase in the level of bacteria in the water following disinfection may indicate significant biofilm formation. Therefore, although the results of microbiological surveillance obtained using the test methods outlined above may be useful in guiding disinfection schedules and in demonstrating compliance with AAMI standards, they should not be taken as an indication of the absolute microbiological purity of the water.⁷⁹²

Endotoxin can be tested by one of two types of assays a) a kinetic test method [e.g., colorimetric or turbidimetric] or b) a gel-clot assay. Endotoxin units are assayed by the *Limulus* Amebocyte Lysate (LAL) method. Because endotoxins differ in their activity on a mass basis, their activity is referred to a standard *Escherichia coli* endotoxin. The current standard (EC-6) is prepared from *E. coli* O113:H10. The relationship between mass of endotoxin and its activity varies with both the lot of LAL and the lot of control standard endotoxin used. Since standards for endotoxin were harmonized in 1983 with the introduction of EC-5, the relationship between mass and activity of endotoxin has been approximately 5–10 EU/ng. Studies to harmonize standards have led to the measurement of endotoxin units (EU) where 5 EU is equivalent to 1 ng *E. coli* O55:B5 endotoxin.¹⁴⁵⁶

In summary, water used to prepare dialysate and to reprocess hemodialyzers should not contain a total microbial count >200 CFU/mL as determined by assay on TSA agar for 48 hrs. at 96.8°F (36°C), and ≤2 endotoxin units (EU) per mL. The dialysate at the end of a dialysis treatment should not contain >2,000 CFU/mL.^{31, 32, 668, 789, 792}

3. Water Sampling Strategies and Culture Techniques for Detecting Legionellae

Legionella spp. are ubiquitous and can be isolated from 20%–40% of freshwater environments, including man-made water systems.^{1457, 1458} In health-care facilities, where legionellae in potable water rarely result in disease among immunocompromised patients, courses of remedial action are unclear.

Scheduled microbiologic monitoring for legionellae remains controversial because the presence of legionellae is not necessarily evidence of a potential for causing disease.¹⁴⁵⁹ CDC recommends aggressive disinfection measures for cleaning and maintaining devices known to transmit legionellae, but does not recommend regularly scheduled microbiologic assays for the bacteria.³⁹⁶ However, scheduled monitoring of potable water within a hospital might be considered in certain settings where persons are highly susceptible to illness and mortality from *Legionella* infection (e.g., hematopoietic stem cell transplantation units and solid organ transplant units).⁹ Also, after an outbreak of

legionellosis, health officials agree monitoring is necessary to identify the source and to evaluate the efficacy of biocides or other prevention measures.

Examination of water samples is the most efficient microbiologic method for identifying sources of legionellae and is an integral part of an epidemiologic investigation into health-care–associated Legionnaires disease. Because of the diversity of plumbing and HVAC systems in health-care facilities, the number and types of sites to be tested must be determined before collection of water samples. One environmental sampling protocol that addresses sampling site selection in hospitals might serve as a prototype for sampling in other institutions.¹²⁰⁹ Any water source that might be aerosolized should be considered a potential source for transmission of legionellae. The bacteria are rarely found in municipal water supplies and tend to colonize plumbing systems and point-of-use devices. To colonize, legionellae usually require a temperature range of 77°F–108°F (25°C–42.2°C) and are most commonly located in hot water systems.¹⁴⁶⁰ Legionellae do not survive drying. Therefore, air-conditioning equipment condensate, which frequently evaporates, is not a likely source.¹⁴⁶¹

Water samples and swabs from point-of-use devices or system surfaces should be collected when sampling for legionellae (Box C.1).¹⁴³⁷ Swabs of system surfaces allow sampling of biofilms, which frequently contain legionellae. When culturing faucet aerators and shower heads, swabs of surface areas should be collected first; water samples are collected after aerators or shower heads are removed from their pipes. Collection and culture techniques are outlined (Box C.2). Swabs can be streaked directly onto buffered charcoal yeast extract agar (BCYE) plates if the plates are available at the collection site. If the swabs and water samples must be transported back to a laboratory for processing, immersing individual swabs in sample water minimizes drying during transit. Place swabs and water samples in insulated coolers to protect specimens from temperature extremes.

Box C.1. Potential sampling sites for *Legionella* spp. in health-care facilities*

-
- **Potable water systems**
incoming water main, water softener unit, holding tanks, cisterns, water heater tanks
(at the inflows and outflows)
 - **Potable water outlets, especially those in or near patient rooms**
faucets or taps, showers
 - **Cooling towers and evaporative condensers**
makeup water (e.g., added to replace water lost because of evaporation, drift, or leakage),
basin (i.e., area under the tower for collection of cooled water), sump (i.e., section of basin
from which cooled water returns to heat source), heat sources (e.g., chillers)
 - **Humidifiers (e.g., nebulizers)**
bubblers for oxygen, water used for respiratory therapy equipment
 - **Other sources**
decorative fountains, irrigation equipment, fire sprinkler system (if recently used), whirlpools,
spas
-

* Material in this box is adapted from reference 1209.

Box C.2. Procedures for collecting and processing environmental specimens for *Legionella* spp.*

1. Collect water (1-liter samples, if possible) in sterile, screw-top bottles.
2. Collect culture swabs of internal surfaces of faucets, aerators, and shower heads in a sterile, screw-top container (e.g., 50 mL plastic centrifuge tube). Submerge each swab in 5–10 mL of sample water taken from the same device from which the sample was obtained.
3. Transport samples and process in a laboratory proficient at culturing water specimens for *Legionella* spp. as soon as possible after collection.+
4. Test samples for the presence of *Legionella* spp. by using semiselective culture media using procedures specific to the cultivation and detection of *Legionella* spp.§¶

* Material in this table is compiled from references 1209, 1437, 1462–1465.

+ Samples may be transported at room temperature but must be protected from temperature extremes. Samples not processed within 24 hours of collection should be refrigerated.

§ Detection of *Legionella* spp. antigen by the direct fluorescent antibody technique is not suitable for environmental samples.

¶ Use of polymerase chain reaction for identification of *Legionella* spp. is not recommended until more data regarding the sensitivity and specificity of this procedure are available.

4. Procedure for Cleaning Cooling Towers and Related Equipment

- I. Perform these steps prior to chemical disinfection and mechanical cleaning.
 - A. Provide protective equipment to workers who perform the disinfection, to prevent their exposure to chemicals used for disinfection and aerosolized water containing *Legionella* spp. Protective equipment may include full-length protective clothing, boots, gloves, goggles, and a full- or half-face mask that combines a HEPA filter and chemical cartridges to protect against airborne chlorine levels of up to 10 mg/L.
 - B. Shut off cooling tower.
 1. Shut off the heat source, if possible.
 2. Shut off fans, if present, on the cooling tower/evaporative condenser (CT/EC).
 3. Shut off the system blowdown (i.e., purge) valve.
 4. Shut off the automated blowdown controller, if present, and set the system controller to manual.
 5. Keep make-up water valves open.
 6. Close building air-intake vents within at least 30 meters of the CT/EC until after the cleaning procedure is complete.
 7. Continue operating pumps for water circulation through the CT/EC.
- II. Perform these chemical disinfection procedures.
 - A. Add fast-release, chlorine-containing disinfectant in pellet, granular, or liquid form, and follow safety instructions on the product label. Use EPA-registered products, if available. Examples of disinfectants include sodium hypochlorite (NaOCl) or calcium hypochlorite (Ca[OCl]₂), calculated to achieve initial free residual chlorine (FRC) of 50 mg/L: either a) 3.0 lbs [1.4 kg] industrial grade NaOCl [12%–15% available Cl] per 1,000 gallons of CT/EC water; b) 10.5 lbs [4.8 kg] domestic grade NaOCl [3%–5% available Cl] per 1,000 gallons of CT/EC water; or c)

0.6 lb [0.3 kg] $\text{Ca}[\text{OCl}]_2$ per 1,000 gallons of CT/EC water. If significant biodeposits are present, additional chlorine may be required. If the volume of water in the CT/EC is unknown, it can be estimated (in gallons) by multiplying either the recirculation rate in gallons per minute by 10 or the refrigeration capacity in tons by 30. Other appropriate compounds may be suggested by a water-treatment specialist.

- B. Record the type and quality of all chemicals used for disinfection, the exact time the chemicals were added to the system, and the time and results of FRC and pH measurements.
- C. Add dispersant simultaneously with or within 15 minutes of adding disinfectant. The dispersant is best added by first dissolving it in water and adding the solution to a turbulent zone in the water system. Automatic-dishwasher compounds are examples of low- or nonfoaming, silicate-based dispersants. Dispersants are added at 10–25 lbs (4.5–11.25 kg) per 1,000 gallons of CT/EC water.
- D. After adding disinfectant and dispersant, continue circulating the water through the system. Monitor the FRC by using an FRC-measuring device with the DPD method (e.g., a swimming-pool test kit), and measure the pH with a pH meter every 15 minutes for 2 hours. Add chlorine as needed to maintain the FRC at ≥ 10 mg/L. Because the biocidal effect of chlorine is reduced at a higher pH, adjust the pH to 7.5–8.0. The pH may be lowered by using any acid (e.g., muriatic acid or sulfuric acid used for maintenance of swimming pools) that is compatible with the treatment chemicals.
- E. Two hours after adding disinfectant and dispersant or after the FRC level is stable at ≥ 10 mg/L, monitor at 2-hour intervals and maintain the FRC at ≥ 10 mg/L for 24 hours.
- F. After the FRC level has been maintained at ≥ 10 mg/L for 24 hours, drain the system. CT/EC water may be drained safely into the sanitary sewer. Municipal water and sewerage authorities should be contacted regarding local regulations. If a sanitary sewer is not available, consult local or state authorities (e.g., a department of natural resources or environmental protection) regarding disposal of water. If necessary, the drain-off may be dechlorinated by dissipation or chemical neutralization with sodium bisulfite.
- G. Refill the system with water and repeat the procedure outline in steps 2–7 in I-B above.

III. Perform mechanical cleaning.

- A. After water from the second chemical disinfection has been drained, shut down the CT/EC.
- B. Inspect all water-contact areas for sediment, sludge, and scale. Using brushes and/or a low-pressure water hose, thoroughly clean all CT/EC water-contact areas, including the basin, sump, fill, spray nozzles, and fittings. Replace components as needed.
- C. If possible, clean CT/EC water-contact areas within the chillers.

IV. Perform these procedures after mechanical cleaning.

- A. Fill the system with water and add chlorine to achieve an FRC level of 10 mg/L.
- B. Circulate the water for 1 hour, then open the blowdown valve and flush the entire system until the water is free of turbidity.
- C. Drain the system.
- D. Open any air-intake vents that were closed before cleaning.
- E. Fill the system with water. The CT/EC may be put back into service using an effective water-treatment program.

5. Maintenance Procedures Used to Decrease Survival and Multiplications of *Legionella* spp. in Potable-Water Distribution Systems

Wherever allowable by state code, provide water at $\geq 124^{\circ}\text{F}$ ($\geq 51^{\circ}\text{C}$) at all points in the heated water system, including the taps. This requires that water in calorifiers (e.g., water heaters) be maintained at $\geq 140^{\circ}\text{F}$ ($\geq 60^{\circ}\text{C}$). In the United Kingdom, where maintenance of water temperatures at $\geq 122^{\circ}\text{F}$ ($\geq 50^{\circ}\text{C}$) in hospitals has been mandated, installation of blending or mixing valves at or near taps to reduce the water temperature to $\leq 109.4^{\circ}\text{F}$ ($\leq 63^{\circ}\text{C}$) has been recommended in certain settings to reduce the risk for scald injury to patients, visitors, and health care workers.⁷²⁶ However, *Legionella* spp. can multiply even in short segments of pipe containing water at this temperature. Increasing the flow rate from the hot-water-circulation system may help lessen the likelihood of water stagnation and cooling.^{711, 1465} Insulation of plumbing to ensure delivery of cold ($< 68^{\circ}\text{F}$ [$< 20^{\circ}\text{C}$]) water to water heaters (and to cold-water outlets) may diminish the opportunity for bacterial multiplication.⁴⁵⁶ Both dead legs and capped spurs within the plumbing system provide areas of stagnation and cooling to $< 122^{\circ}\text{F}$ ($< 50^{\circ}\text{C}$) regardless of the circulating water temperature; these segments may need to be removed to prevent colonization.⁷⁰⁴ Rubber fittings within plumbing systems have been associated with persistent colonization, and replacement of these fittings may be required for *Legionella* spp. eradication.¹⁴⁶⁷

Continuous chlorination to maintain concentrations of free residual chlorine at 1–2 mg/L (1–2 ppm) at the tap is an alternative option for treatment. This requires the placement of flow-adjusted, continuous injectors of chlorine throughout the water distribution system. Adverse effects of continuous chlorination can include accelerated corrosion of plumbing (resulting in system leaks) and production of potentially carcinogenic trihalomethanes. However, when levels of free residual chlorine are below 3 mg/L (3 ppm), trihalomethane levels are kept below the maximum safety level recommended by the EPA.^{727, 1468, 1469}

Appendix D. Insects and Microorganisms

Table D.1. Microorganisms isolated from arthropods in health-care settings

Insect	Microorganism category	Microorganisms	References
Cockroaches	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp., <i>E. cloacae</i> ; <i>Escherichia coli</i> ; <i>Flavobacterium</i> spp.; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas</i> spp., <i>P. aeruginosa</i> , <i>P. fluorescens</i> , <i>P. putida</i> ; <i>Salmonella</i> spp.; <i>Serratia</i> spp., <i>S. marcescens</i> ; <i>Shigella boydii</i>	1048, 1051, 1056, 1058, 1059, 1062
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus aureus</i> , <i>S. epidermidis</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	1056, 1058, 1059
	Acid-fast bacteria	<i>Mycobacterium tuberculosis</i>	1065
	Fungi	<i>Aspergillus niger</i> ; <i>Mucor</i> spp.; <i>Rhizopus</i> spp.	1052, 1059
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	1059
Houseflies	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Campulobacter fetus</i> subsp. <i>Jejuni</i> ; <i>Chlamydia</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter</i> spp.; <i>Escherichia coli</i> ; <i>Helicobacter pylori</i> ; <i>Klebsiella</i> spp.; <i>Proteus</i> spp.; <i>Pseudomonas aeruginosa</i> ; <i>Serratia marcescens</i> ; <i>Shigella</i> spp.	1047, 1048, 1050, 1053–1055, 1060
	Gram-positive bacteria	<i>Bacillus</i> spp.; <i>Enterococcus faecalis</i> ; <i>Micrococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus</i> spp., <i>S. viridans</i>	1048, 1060
	Fungi / yeasts	<i>Candida</i> spp.; <i>Geotrichum</i> spp.	1060
	Parasites	<i>Endolimax nana</i> ; <i>Entamoeba coli</i>	1060
	Viruses	Rotaviruses	1049
Ants	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Escherichia coli</i> ; <i>Klebsiella</i> spp.; <i>Neisseria sicca</i> ; <i>Proteus</i> spp.; <i>Providencia</i> spp.; <i>Pseudomonas aeruginosa</i> , <i>P. fluorescens</i>	1057
	Gram-positive bacteria	<i>Bacillus</i> spp., <i>B. cereus</i> , <i>B. pumilis</i> ; <i>Clostridium cochlearium</i> , <i>C. welchii</i> ; <i>Enterococcus faecalis</i> ; <i>Staphylococcus</i> spp. (coagulase-negative), <i>S. aureus</i> ; <i>Streptococcus pyrogenes</i>	1057
Spiders	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Citrobacter freundii</i> ; <i>Enterobacter aerogenes</i> ; <i>Morganella morganii</i>	1048
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	1048
Mites, midges	Gram-negative bacteria	<i>Acinetobacter</i> spp.; <i>Burkholderia cepacia</i> ; <i>Enterobacter agglomerans</i> , <i>E. aerogenes</i> ; <i>Hafnia alvei</i> ; <i>Pseudomonas aeruginosa</i>	1048
	Gram-positive bacteria	<i>Staphylococcus</i> spp. (coagulase-negative)	1048
Mosquitoes	Gram-negative bacteria	<i>Acinetobacter calcoaceticus</i> ; <i>Enterobacter cloacae</i>	1048
	Gram-positive bacteria	<i>Enterococcus</i> spp.; <i>Staphylococcus</i> spp. (coagulase-negative)	1048

Appendix E. Information Resources

The following sources of information may be helpful to the reader. Some of these are available at no charge, while others are available for purchase from the publisher.

Air and Water

- Jensen PA, Schafer MP. Sampling and characterization of bioaerosols. NIOSH Manual of Analytical Methods; revised 6/99. www.cdc.gov/niosh/nmam/pdfs/chapter-j.pdf
- American Institutes of Architects. *Guidelines for Design and Construction of Hospital and Health Care Facilities*. Washington DC; American Institute of Architects Press; 2001. AIA, 1735 New York Avenue, NW, Washington DC 20006. 1-800-AIA-3837 or (202) 626-7541
- ASHRAE. Standard 62, and Standard 12-2000. These documents may be purchased from: American Society of Heating, Refrigerating, and Air-Conditioning Engineers, Inc. 1791 Tullie Circle, NE, Atlanta GA 30329 1-800-527-4723 or (404) 636-8400.
- University of Minnesota websites: www.dehs.umn.edu Indoor air quality site: www.dehs.umn.edu/resources.htm#indoor Water infiltration and use of the wet test (moisture) meter: www.dehs.umn.edu/remangi.html
- The CDC website for bioterrorism information contains the interim intervention plan for smallpox. The plan discusses infection control issues both for home-based care and hospital-based patient management. www.bt.cdc.gov/agent/smallpox/response-plan/index.asp

Environmental Sampling

- ISO. Sterilization of medical devices – microbiological methods, Part 1. ISO standard 11737-1. Paramus NJ; International Organization for Standardization; 1995.

Animals in Health-Care Facilities

- Service animal information with respect to the Americans with Disabilities Act. Contact the U.S. Department of Justice ADA Information Line at (800) 514-0301 (voice) or (800) 514-0383 (TDD), or visit the ADA website at: www.usdoj.gov/crt/ada/adahom1.htm

Regulated Medical Waste

- U.S. Environmental Protection Agency. This is the Internet address on their Internet web site that will link to any state for information about medical waste rules and regulations at the state level: www.epa.gov/epaoswer/other/medical/stregs.htm

General Resources

- APIC Text of Infection Control and Epidemiology. Association for Professionals in Infection Control and Epidemiology, Inc. Washington DC; 2000. (Two binder volumes, or CD-ROM)
- Abrutyn E, Goldmann DA, Scheckler WE. Saunders Infection Control Reference Service, 2nd Edition. Philadelphia PA; WB Saunders; 2000.
- ECRI publications are available on a variety of healthcare topics. Contact ECRI at (610) 825-6000. CRI, 5200 Butler Pike, Plymouth Meeting, PA 19462-1298.

Appendix F. Areas of Future Research

Air

- Standardize the methodology and interpretation of microbiologic air sampling (e.g., determine action levels or minimum infectious dose for aspergillosis, and evaluate the significance of airborne bacteria and fungi in the surgical field and the impact on postoperative SSI).
- Develop new molecular typing methods to better define the epidemiology of health-care–associated outbreaks of aspergillosis and to associate isolates recovered from both clinical and environmental sources.
- Develop new methods for the diagnosis of aspergillosis that can lead reliably to early recognition of infection.
- Assess the value of laminar flow technology for surgeries other than for joint replacement surgery.
- Determine if particulate sampling can be routinely performed in lieu of microbiologic sampling for purposes such as determining air quality of clean environments (e.g., operating rooms, HSCT units).

Water

- Evaluate new methods of water treatment, both in the facility and at the water utility (e.g., ozone, chlorine dioxide, copper/silver/monochloramine) and perform cost-benefit analyses of treatment in preventing health-care–associated legionellosis.
- Evaluate the role of biofilms in overall water quality and determine the impact of water treatments for the control of biofilm in distribution systems.
- Determine if the use of ultrapure fluids in dialysis is feasible and warranted, and determine the action level for the final bath.
- Develop quality assurance protocols and validated methods for sampling filtered rinse water used with AERs and determine acceptable microbiologic quality of AER rinse water.

Environmental Services

- Evaluate the innate resistance of microorganisms to the action of chemical germicides, and determine what, if any, linkage there may be between antibiotic resistance and resistance to disinfectants.

Laundry and Bedding

- Evaluate the microbial inactivation capabilities of new laundry detergents, bleach substitutes, other laundry additives, and new laundry technologies.

Animals in Health-Care Facilities

- Conduct surveillance to monitor incidence of infections among patients in facilities that use animal programs, and conduct investigations to determine new infection control strategies to prevent these infections.
- Evaluate the epidemiologic impact of performing procedures on animals (e.g., surgery or imaging) in human health-care facilities.

Regulated Medical Waste

- Determine the efficiency of current medical waste treatment technologies to inactivate emerging pathogens that may be present in medical waste (e.g., SARS-coV).
- Explore options to enable health-care facilities to reinstate the capacity to inactivate microbiological cultures and stocks on-site.

Index—Parts I and IV

A

AAMI standards 59, 60, 62, 222, 223
Acinetobacter spp. 11, 20, 43, 44, 99, 104
aerators 47, 48, 94, 220–222, 224, 225
aerosols... 12, 27, 41, 47, 56, 59, 67, 75, 76, 78, 80, 85, 89,
90, 98, 106, 111, 113, 114
AIA guidelines..... 17, 18, 19, 25, 37, 39, 99
AII rooms 35–37
air changes per hour (ACH)..... 6, 12, 16, 18, 31, 111, 210
air conditioners 8, 22
air conditioning systems 13, 20, 57, 59
air filtration..... 111
air intakes 31, 226
air sampling 26, 29, 89, 90, 91, 93, 210
airborne infection isolation (AII)..... 6, 19
airborne transmission..... 12
air-fluidized beds 104
alcohol-based hand rubs 53
alkaline glutaraldehyde..... 70
allergens..... 17, 80, 107
American Institute of Architects (AIA)..... 13
Americans with Disabilities Act..... 108, 110
amplified stocks and cultures..... 114, 115
Animal Assisted Activities 106, 107
Animal Assisted Therapy 106, 107
animal bites..... 107
animal handler 107
animal patient 110
Animal Welfare Act..... 112
anterooms 12, 25, 33, 36–38
ants 81
ASHRAE 13, 47, 49
aspergillosis 7, 8, 16, 19, 21, 35, 79, 80
Aspergillus fumigatus 7, 8, 29
Aspergillus spp. 5, 7, 20, 21, 28, 32, 34, 81
automated cyclers 65
automated endoscope reprocessor..... 50, 69, 70
autopsy suites/rooms..... 12, 87

B

bacterial spores 73, 84, 89
bank of filters..... 14
barrier 34
barrier precautions/protection..... 74, 109, 116
barriers..... 27, 31, 33
bassinets..... 76
biofilms..... 46, 54, 64, 71, 220–222, 224
biosafety level..... 114
bioterrorism 89, 114
bird droppings..... 9, 20, 22
birthing tanks..... 67, 69

blood.. 12, 64, 69, 75, 77–79, 86, 87, 98, 99, 102, 113, 116
bloodborne pathogens..... 73, 116
boil water advisory 51, 52

C

calibrated loop 222
carpet cleaning 79
carpet tiles..... 79
carpeting 22, 25, 52, 78, 79
cats..... 105, 106, 108, 109
chain of infection 4, 87
chemical germicides 73, 74, 77, 80, 84
chloramine/chloramine-T 68
chlorine..... 46, 50, 53, 69, 84, 221, 226
chlorine bleach..... 78, 101
chlorine residual 50, 68, 69, 94, 101, 221
cleaning .. 68, 70–72, 74, 78, 80, 83, 85, 86, 107, 109, 112,
225
cleaning cloths 75
cleaning solutions 75, 76
Clostridium difficile 5, 84
cloth chairs..... 79
cockroaches 81
coliform bacteria..... 221
colonization 42–44, 68, 70, 83, 99, 106, 227
colony counts..... 211
commissioning..... 29, 89
construction 7, 13, 14, 21, 23, 26, 27, 29, 31, 37, 76, 89
construction workers..... 24, 26, 31
contact precautions 85
contact time 74, 84, 88, 221
contaminants..... 14, 18, 19, 59, 210
contaminated fabrics..... 98, 101, 102
contingency plans 21, 50
continuous chlorination 227
cooling tower..... 41, 53, 55, 57–59, 220, 225
copper/silver ions..... 54
copper-8-quinolinolate..... 35
Creutzfeldt-Jakob disease 86, 116
Cryptosporidium parvum..... 46

D

dead legs 59, 64, 221
decorative fountains..... 47, 49, 224
demolition..... 23, 25, 26, 29
dental unit water lines..... 71
detergent/disinfectant..... 74–76
dialysate 59–62
dialysis machines 64
dialysis water 222
dialyzer 62
dialyzer membranes 61

dialyzer reprocessing 59
 dioctylphthalate (DOP) particle test 15
 direct contact 6, 41, 67, 85, 86, 98, 108, 111
 direct threat 109
 disinfectant fogging 75
 disinfectant residuals 96
 disinfectants 21, 76, 225
 disinfecting 71, 74, 80, 83, 85, 86, 112, 226
 disinfection 63, 64, 68, 70
 dispersant 226
 disposal (of medical waste) 113
 distribution system 94, 221
 dogs 105, 106, 108, 109
 drift eliminators 58
 drinking water 71
 droplet nuclei 6, 7, 10, 12, 89
 droplets 6, 55, 85, 86, 89
 dry cleaning 102
 drying 11
 dual-duct system 20
 duct cleaning 21
 ductwork 20, 22
 dust 8, 20, 24, 27, 30, 32, 74, 79, 93
 dust-spot test 15

E

education 24
 electrical generators 53
 emergency 53
 endotoxin 60–62, 64, 223
 engineering controls 36
 enteric viruses 85
 environmental cultures 83, 88
 Environmental Protection Agency (EPA) 21, 73–75, 77, 103, 227
 environmental sampling 88, 95
 environmental surfaces 11, 44, 71, 72, 74, 82–86, 88, 98, 107
 environmental surveillance 54, 55
 EPA registration 73, 76, 83
 EPA-registered germicides 75, 78, 85, 86
 evaporative condensers 41, 57–59
 exclusion (of a service animal) 109
 exotic animals 110

F

fan-coil units 18
 faucets 47, 54, 94, 222, 224, 225
 fecal contamination 84
 FIFRA 75, 103
 filter efficiency 27, 29
 filtration 15
 fire codes 31
 fish 105, 108
 fish tanks 108
 flies 81
 flooding 51

floors 25, 75, 82, 83
 flowers 80
 flush times 51
 flutter strips 20, 34, 36
 fomites 3, 4, 7, 85
 Food and Drug Administration (FDA) 69, 73, 103
 free residual chlorine 51, 54, 225–227
 fungal spores 8, 15, 16, 19–21, 26–28, 31, 34, 38, 79, 89, 93
 fungi 8
 furniture 52, 79, 82

G

gram-negative bacteria 11, 41, 42, 48, 50, 60, 63, 64, 221
 gram-positive bacteria 11, 84

H

hand hygiene 25, 71, 107, 109
 hand transferral 3, 44, 65, 82–84, 106, 221
 handwashing 25, 80, 84, 99, 107
 hantaviruses 12
 hematopoietic stem cell transplant 6
 hemodiafiltration 62
 hemodialysis 59, 60, 62, 64, 223
 hemodialysis patients 7
 hemofiltration 62
 HEPA filtration/filters 6, 12, 14, 15, 17, 31, 32, 36, 76
 hepatitis B virus 40, 73, 98
 heterotrophic plate counts 51, 62, 66, 95, 221–223
 high-flux membranes 61
 high-level disinfectants 73
 high-level disinfection 60, 69, 72
 high-temperature flushing 50
 high-touch surfaces 75, 83–85
 holding tank 47
 hospital disinfectant 73
 hot water system 51, 54
 hot water tanks 53, 220–222
 hot water temperature 49
 housekeeping surfaces 3, 64, 72, 74–77, 83
 HSCT patients 6, 37
 HSCT units 11, 26, 79, 80, 107
 Hubbard tanks 68
 human health-care facilities 110, 111
 human immunodeficiency virus 73
 humidifiers 17, 23, 41
 humidity 13, 14, 17, 20, 38, 90
 HVAC systems 13, 14, 16, 17, 19–21, 27, 30, 51
 hydrotherapy equipment 67–69
 hydrotherapy pools 68
 hydrotherapy tanks 67, 68, 82
 hygienically-clean laundry 98–100, 102
 hyperchlorination 50, 53, 54, 59

I, J

iatrogenic cases.....	87
ice machines and ice.....	25, 48, 65, 66
ice-storage chests.....	66
immunocompromised patients.....	6, 7, 9, 26, 29–31, 34, 42, 47, 56, 66, 80, 107, 108, 223
impaction.....	90, 211
impactors.....	28, 93
impingement.....	90, 93, 211
impingers.....	211
inactivation studies.....	87
incineration.....	113, 114
incubators (nursery).....	76
indirect transmission.....	6
indirect contact.....	41
indoor air.....	21, 24, 26, 27, 90
industrial-grade HEPA filter.....	16, 31, 38, 39
infection-control risk assessment (ICRA)....	26, 29, 31, 35, 108, 111
influenza viruses.....	6, 12, 73, 85
innate resistance.....	72, 73, 84
insects.....	67, 81
insulation material.....	20
intermediate-level disinfectants.....	73, 78, 83, 85, 86
intermediate-level disinfection.....	72
isolation/isolation areas.....	11, 36, 100
JCAHO.....	13, 14, 51, 59

L

laboratories.....	12, 13, 32, 47, 78, 79, 83, 105, 111, 112, 114, 222
laboratory confirmation.....	55
laminar airflow.....	18, 34, 38
laser plumes.....	40
laundry.....	49
laundry bags.....	100
laundry chutes.....	100
laundry cycles.....	101
laundry disinfection.....	101
laundry facility.....	99
laundry packaging.....	100, 101
laundry process.....	98, 99, 102
laundry services.....	99
laundry transport.....	100, 101
<i>Legionella pneumophila</i>	210, 221
<i>Legionella</i> spp.	41, 42, 50, 54–57, 59, 71, 222, 223, 225, 227
legionellae.....	41, 54, 211, 223
legionellosis.....	53–56, 224
Legionnaires disease.....	41, 47, 57, 58, 224
liquid chemical sterilant.....	70
low-level disinfectants.....	72, 73, 83, 86
low-level disinfection.....	60, 64

M

manufacturer's instructions.....	67, 69, 74, 86, 102, 116
material safety data sheets (MSDS).....	75, 87
mattress cover.....	77, 104
mattresses.....	77, 104
medical equipment.....	74, 83
medical equipment surfaces.....	72
medical gas piping.....	30
medical records.....	51
medical waste.....	112, 113, 117
medical waste management.....	112
membrane filtration.....	70, 95, 96, 222
methicillin-resistant <i>Staphylococcus aureus</i> (MRSA) ...	82, 83, 104, 105
microbial inactivation.....	72
microbial resistance.....	70
microbiologic air sampling.....	27
microbiologic cultures and stocks.....	112
microbiologic sampling.....	64
microbiologic sampling of laundry.....	102
microbiological wastes.....	112, 114
moisture.....	20, 24, 32, 51, 70, 96
moisture meters.....	51
molecular typing.....	28
monochloramine.....	54
mop heads.....	75
multidisciplinary team.....	23, 91
municipal water.....	47, 50, 224
municipal water systems/utilities.....	45, 221
<i>Mycobacterium tuberculosis</i>	5, 7, 10, 73, 114
myiasis.....	81

N

negative air pressure.....	6, 12, 18, 19, 21, 36, 99, 100, 104, 111
neutralizer chemicals.....	96
NIOSH.....	40
nontuberculous mycobacteria (NTM).....	5, 41, 44–46, 60, 63, 70, 71, 223

O

operating rooms.....	13, 15, 17, 34, 38, 76, 82, 87, 109, 111
opportunistic infections.....	4, 5
organic matter.....	78
OSHA.....	13, 73, 77, 79, 98, 100, 113
outdoor air.....	14, 15, 18, 25, 91
oxygen-based laundry detergents.....	101

P

particle sampling.....	27, 33, 89
performance measures.....	2
periodic culturing.....	57
peritoneal dialysis.....	64, 65

personal protective equipment	77, 98, 99, 112, 114, 225
persons with disabilities	108, 109
person-to-person transmission	12, 85
pest control	82
phenolics	76
pillows	104
pipes	64, 69, 221, 223, 224
planktonic organisms	221
plastic enclosures	31
plastic wrapping	74
<i>Pneumocystis carinii</i>	9
pneumonia	42, 55
point-of-use fixtures	47, 51, 224
polyvinylchloride (PVC)	46, 64
pools	67
positive air pressure	18, 38
potable water	220
potted plants	8, 80
pressure differentials	18, 19, 25, 30, 38
primates	105, 106, 111
prions	86, 116
privacy curtains	75
product water	64, 70
protective environment (PE)	6, 18, 19, 34, 56, 108
<i>Pseudomonas aeruginosa</i>	5, 11, 20, 42, 68, 70, 71, 73, 79, 80, 96, 104, 221
pseudo-outbreaks	44, 70
pyrogenic reactions	60, 61

Q

quality assurance	89, 94, 95
-------------------	------------

R

R2A media	222
rank order	27
recirculation	16, 18
recirculation loops	46
recreational equipment	69
reduced nutrient media	94
reducing agent	94
relative humidity	17
renovation	13, 14, 23, 37
repairs	31
reprocess hemodialyzers	61, 223
research animals	111
reservoirs	3, 6, 41, 42, 71, 79, 83, 95, 105, 211
resident animals	107
respirable particles	27, 28, 90
respirators	26, 40
respiratory protection	36, 78
respiratory syncytial virus (RSV)	6, 12, 85
respiratory therapy equipment	224
return air	14
return temperature	54
reverse osmosis (RO)	52, 54, 59, 60, 63
rewiring	25
rinse water monitoring	70

RODAC plates	102
rodents	67
rooftops	30

S

Sabouraud dextrose agar	96
sample/rinse methods	97
sanitary sewer	116, 117
SARS	86
SARS-CoV	86
scalding	49, 51
screens	82
scrub suits	98, 99
sealed windows	19, 26, 29, 89
sedimentation	90, 93, 211
select agents	114, 115
self-closing doors	19
semicritical device	70
service animal	105, 108–110
settle plates	28, 90, 93, 211
sewage spills	51
sharps containers	113
shock decontamination	51
shower heads	47, 49, 54, 220, 221, 222, 224, 225
showers	47, 48
skin antiseptics	73
smallpox	36
smallpox virus	7, 12
smoke tubes	20, 34, 36
sodium hydroxide	87
sodium hypochlorite	67, 69, 73, 77, 83, 84, 87, 88, 94, 225
solid-organ transplant program	56
sorting (laundry)	98, 100
Spaulding classification	71, 98
spills	75, 77, 79
standard precautions	100, 111
standards	2, 14, 71, 88, 90, 112, 223
<i>Staphylococcus aureus</i>	10, 11, 38, 64, 73, 99, 104
state codes/regulations	55, 69
steam jet	101
steam sterilization (of medical waste)	113, 114
sterile water	55
storage tanks	63, 64
streptococci	10, 38
supplemental treatment methods	53
surgical gowns and drapes	103
surgical site infections (SSI)	11, 38, 65
surgical smoke	40
surveillance	26, 51, 57, 99, 223
swabs	224

T

tacky mats	76
tap water	42, 44, 57, 65, 66, 70
TB patients	38
temperature (air)	13, 14, 17, 89
temperature (water)	40, 45, 49, 68, 101, 221, 227

thermostatic mixing valves	49
transport and storage (of medical waste)	113
treated items/products	79, 103
tryptic soy agar	94, 96, 222
tub liners	68
tuberculocidal claim	73
tuberculosis (TB)	35

U

ultrapure dialysate	61
ultraviolet germicidal irradiation (UVGI)	14, 16, 17, 36, 38
uniforms	98, 99

V

vacuum breakers	47, 50
vacuum cleaners	76, 79
vacuuming	79
vancomycin-resistant enterococci (VRE)	3, 5, 82, 83, 105
vancomycin-resistant <i>Staphylococcus aureus</i> (VRSA)	83
variable air ventilation	20, 38
varicella-zoster virus (VZV)	5, 7, 40
vase water	81
vegetative bacteria	73
ventilation rates	18
ventilation systems	8, 9, 111
viable particles	9, 91

viral hemorrhagic fever	12
viral particles	11
viruses	11, 85
visual monitoring device	34
volumetric air samplers	29
volumetric sampling methods	28

W

wallboard	8, 22, 52
walls	25
washing machines and dryers	102
water conditioning	68
water distribution systems	64, 221, 227
water droplets	58
water pipes	46
water pressure	50
water quality	71, 94
water sampling	54, 94, 221, 224
water stagnation	227
water treatment system	63
waterborne transmission	46
weight-arrestance test	15
wet cleaning	79
whirlpool spas	59, 67, 69
whirlpools	68, 69
window chutes	33
windows	22, 59
wood	8, 9, 15, 35