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Guidelines for Prevention of Nosocomial Pneumonia

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Guidelines for Prevention of Nosocomial Pneumonia

Summary

*This document updates and replaces CDC's previously published "Guideline for Prevention of Nosocomial Pneumonia" (*Infect Control *1982;3:327–33,* Respir Care *1983;28:221–32, and* Am J Infect Control *1983;11:230–44). This revised guideline is designed to reduce the incidence of nosocomial pneumonia and is intended for use by personnel who are responsible for surveillance and control of infections in acute-care hospitals; the information may not be applicable in long-term–care facilities because of the unique characteristics of such settings. This revised guideline addresses common problems encountered by infection-control practitioners regarding the prevention and control of nosocomial pneumonia in U.S. hospitals. Sections on the prevention of bacterial pneumonia in mechanically ventilated and/or critically ill patients, care of respiratory-therapy devices, prevention of cross-contamination, and prevention of viral lower respiratory tract infections (e.g., respiratory syncytial virus [RSV] and influenza infections) have been expanded and updated. New sections on Legionnaires disease and pneumonia caused by* Aspergillus *sp. have been included. Lower respiratory tract infection caused by* Mycobacterium tuberculosis *is not addressed in this document. Part I, "An Overview of the Prevention of Nosocomial Pneumonia, 1994," provides the background information for the consensus recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC) in Part II, "Recommendations for Prevention of Nosocomial Pneumonia."*

Pneumonia is the second most common nosocomial infection in the United States and is associated with substantial morbidity and mortality. Most patients who have nosocomial pneumonia are infants, young children, and persons >65 years of age; persons who have severe underlying disease, immunosuppression, depressed sensorium, and/or cardiopulmonary disease; and persons who have had thoracoabdominal surgery. Although patients receiving mechanically assisted ventilation do not represent a major proportion of patients who have nosocomial pneumonia, they are at highest risk for acquiring the infection. Most bacterial nosocomial pneumonias occur by aspiration of bacteria colonizing the oropharynx or upper gastrointestinal tract of the patient. Because intubation and mechanical ventilation alter first-line patient defenses, they greatly increase the risk for nosocomial bacterial pneumonia. Pneumonias caused by Legionella *sp.,* Aspergillus *sp., and influenza virus are often caused by inhalation of contaminated aerosols. RSV infection usually occurs after viral inoculation of the conjunctivae or nasal mucosa by contaminated hands. Traditional preventive measures for nosocomial pneumonia include decreasing aspiration by the patient, preventing cross-contamination or colonization via hands of personnel, appropriate disinfection or sterilization of respiratory-therapy devices, use of available vaccines to protect against particular infections, and education of hospital staff and patients. New measures being investigated involve reducing oropharyngeal and gastric colonization by pathogenic microorganisms.*

Part 1. An Overview of the Prevention of Nosocomial Pneumonia, 1994

INTRODUCTION

This document updates and replaces CDC's previously published "Guideline for Prevention of Nosocomial Pneumonia" (*Infect Control* 1982;3:327–33, *Respir Care* 1983; 28:221–32, and *Am J Infect Control* 1983;11:230–44). This revised guideline is designed to reduce the incidence of nosocomial pneumonia and is intended for use by personnel who are responsible for surveillance and control of infections in acute-care hospitals; the information may not be applicable in long-term–care facilities because of the unique characteristics of such settings.

This revised guideline addresses common problems encountered by infection-control practitioners regarding the prevention and control of nosocomial pneumonia in U.S. hospitals. Sections concerning the prevention of bacterial pneumonia in mechanically ventilated and/or critically ill patients, care of respiratory-therapy devices, prevention of cross-contamination, and prevention of viral lower respiratory tract infections (e.g., respiratory syncytial virus [RSV] and influenza infections) have been expanded and updated. New sections on Legionnaires disease and pneumonia caused by *Aspergillus* sp. have been included. Lower respiratory tract infection caused by *Mycobacterium tuberculosis* is not addressed in this document; CDC published such recommendations previously (*1*).

Part I, "An Overview of the Prevention of Nosocomial Pneumonia, 1994," provides the background information for the consensus recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC) in Part II, "Recommendations for Prevention of Nosocomial Pneumonia." HICPAC was established in 1991 to provide advice and guidance to the Secretary and the Assistant Secretary for Health, U.S. Department of Health and Human Services; the Director, CDC; and the Director, National Center for Infectious Diseases (NCID), CDC, regarding the practice of hospital infection control and strategies for surveillance, prevention, and control of nosocomial infections in U.S. hospitals. HICPAC also advises CDC on periodic updating of guidelines and other policy statements regarding prevention of nosocomial infections. This guideline is the first of a series of CDC guidelines being revised by HICPAC and NCID.

This guideline can be an important resource for educating health-care workers (HCWs) regarding prevention and control of nosocomial respiratory tract infections. Because education of HCWs is the cornerstone of an effective infection-control program, hospitals should give high priority to continuing infection-control educational programs for these personnel.

BACKGROUND

Pneumonia is the second most common nosocomial infection in the United States and is associated with substantial morbidity and mortality. Most patients who have nosocomial pneumonia are infants, young children, and persons >65 years of age; persons who have severe underlying disease, immunosuppression, depressed sensorium,

and/or cardiopulmonary disease; and persons who have had thoracoabdominal surgery. Although patients receiving mechanically assisted ventilation do not represent a major proportion of patients who have nosocomial pneumonia, they are at highest risk for acquiring the infection.

Most bacterial nosocomial pneumonias occur by aspiration of bacteria colonizing the oropharynx or upper gastrointestinal tract of the patient. Because intubation and mechanical ventilation alter first-line patient defenses, they greatly increase the risk for nosocomial bacterial pneumonia. Pneumonias caused by *Legionella* sp., *Aspergillus* sp., and influenza virus are often caused by inhalation of contaminated aerosols. RSV infection usually occurs after viral inoculation of the conjunctivae or nasal mucosa by contaminated hands.

Traditional preventive measures for nosocomial pneumonia include decreasing aspiration by the patient, preventing cross-contamination or colonization via hands of HCWs, appropriate disinfection or sterilization of respiratory-therapy devices, use of available vaccines to protect against particular infections, and education of hospital staff and patients. New measures being investigated involve reducing oropharyngeal and gastric colonization by pathogenic microorganisms.

BACTERIAL PNEUMONIA

I. Etiologic Agents

The reported distribution of etiologic agents that cause nosocomial pneumonia differs between hospitals because of different patient populations and diagnostic methods employed (*2–10*). In general, however, bacteria have been the most frequently isolated pathogens (*2–6,9,11–13*). During 1986–1989, aerobic bacteria comprised at least 73%, and fungi 4%, of isolates from sputum and tracheal aspirates obtained from patients who had pneumonia at the University of Michigan Hospitals and at hospitals participating in the National Nosocomial Infection Surveillance (NNIS) System; only a few anaerobic bacteria and no viruses were reported, probably because anaerobic and viral cultures were not performed routinely in the reporting hospitals (Table 1) (*3*). Similarly, cultures of bronchoscopic specimens obtained from mechanically ventilated patients who had pneumonia have rarely yielded anaerobes (*5–7,9,11,14,15*). Only one study, which was based primarily on cultures of transtracheal aspirates obtained from patients not receiving mechanically assisted ventilation, reported a predominance of anaerobes (*4*).

Nosocomial bacterial pneumonias are frequently polymicrobial (*4,7,9,11,12,15–19*), and gram-negative bacilli are usually the predominant organisms (Table 1) (*2–6,9,11–13*). However, *Staphylococcus aureus* (especially methicillin-resistant *S. aureus*) (*5,7,10,15,20,21*) and other gram-positive cocci, including *Streptococcus pneumoniae* (*5,7*), have emerged recently as important isolates (*14*). In addition, *Haemophilus influenzae* has been isolated from mechanically ventilated patients who had pneumonia that occurred within 48–96 hours after intubation (*3–5,12,15,22*). In hospitals participating in the NNIS, *Pseudomonas aeruginosa,*

Enterobacter sp., *Klebsiella pneumoniae, Escherichia coli, Serratia marcescens*, and *Proteus* sp. comprised 50% of the isolates from cultures of respiratory tract specimens obtained from patients for whom nosocomial pneumonia was diagnosed by using clinical criteria; *S. aureus* accounted for 16%, and *H. influenzae*, for 6% (Table 1) (*3*). Another study reported that gram-negative bacilli were present in 75% of quantitative cultures of protected-specimen brushings (PSB) obtained from patients who had acquired nosocomial pneumonia after receiving mechanically assisted ventilation; 40% of these cultures were polymicrobial (*5*). In another published report, 20% of pathogens recovered from cultures of PSB, blood, pleural fluid, or percutaneous lung aspirate were gram-negative bacilli in pure culture, and 17% were polymicrobial; however, 54% of specimens did not yield any microorganism, probably because the patients from whom these cultures were obtained had been treated with antibiotics (*6*).

II. Diagnosis

Nosocomial bacterial pneumonia has been difficult to diagnose (*7,8,16,23–32*). Frequently, the criteria for diagnosis have been fever, cough, and development of purulent sputum, in conjunction with radiologic evidence of a new or progressive pulmonary infiltrate, a suggestive Gram stain, and positive cultures of sputum, tracheal aspirate, pleural fluid, or blood (*3,4,23,25,33–36*). Although clinical findings in conjunction with cultures of sputum or tracheal specimens may be sensitive for bacterial pathogens, they are highly nonspecific, especially in patients receiving mechanically assisted ventilation (*8,9,12–15,18,24–26,29,31, 37–42*); conversely, cultures of blood or pleural fluid have very low sensitivity (*8,18,19,43*).

Because of these problems, a group of investigators recently formulated consensus recommendations for standardizing methods used to diagnose pneumonia in clinical research studies of ventilator-associated pneumonia (*44–46*). These methods involve bronchoscopic techniques such as quantitative culture of PSB (*5,7–9,13,15,27,31,38,41,47,48*), bronchoalveolar lavage (BAL) (*7,12,41,47,49–54*), and protected BAL (pBAL) (*14*). The reported sensitivities of such methods have ranged, depending on the tests or diagnostic criteria with which they were compared, from 70% to 100%, and the reported specificities of these methods have ranged from 60% to 100%. These methods are invasive and might cause complications such as hypoxemia, bleeding, or arrhythmia (*8,13,42,44,52,55,56*). In addition, the sensitivity of the PSB procedure may be decreased for patients receiving antibiotic therapy (*9,13,27*). Nonbronchoscopic (NB) procedures (e.g., NB-pBAL [*12,27,57,58*] or NB-PSB [*13*], which utilize blind catheterization of the distal airways) and quantitative culture of endotracheal aspirate (*59,60*) have been developed recently. Of these procedures, endotracheal aspirate culture might be the most practical. The use of these bronchoscopic and nonbronchoscopic diagnostic tests could help to better define the epidemiology of nosocomial pneumonia, especially in patients receiving mechanically assisted ventilation; however, additional studies are needed to determine each test's applicability in daily clinical practice.

TABLE 1. Microorganisms isolated from respiratory tract specimens obtained by various representative methods from adult patients who had a diagnosis of nosocomial pneumonia, by epidemiologic investigation

Category	Schaberg (*3*)	Bartlett (*4*)	Fagon (*5*)	Torres (*6*)
Hospital type	NNIS and UMH*	Veterans	General	General
Patients studied				
Ventilated or nonventilated	Mixed	Mixed	Ventilated	Ventilated
No. of patients	N/A†	159	49	78
No. of episodes of pneumonia	N/A	159	52	78
Specimen(s) cultured	Sputum, tracheal aspirate	Transtracheal aspirate, pleural fluid, blood	Protected specimen brushing	Protected specimen brushing, lung aspirate, pleural fluid, blood
Culture results				
No organism isolated	N/A	0	0	54%§
Polymicrobial	N/A	54%§	40%§	13%§
No. of isolates	15,499	314	111	N/A
Aerobic bacteria				
Gram-negative bacilli	50%¶	46%**	75%**	16%††
Pseudomonas aeruginosa	17%¶	9%**	31%**	5%††
Enterobacter sp.	11	4	2	0
Klebsiella sp.	7	23	4	0
Escherichia coli	6	14	8	0
Serratia sp.	5	0	0	1
Proteus sp.	3	11	15	1
Citrobacter sp.	1	0	2	0
Acinetobacter calcoaceticus	N/A	0	15	9
Haemophilus influenzae	6%¶	17%**	10%**	0%††
Legionella sp.	N/A	N/A	2%**	2%††
Other	N/A	0	10	0
Gram-positive cocci	17%¶	56%**	52%**	4%††
Staphylococcus aureus	16%¶	25%**	33%**	2%††
Streptococcus sp.	1	31	21	2
Other	0	0	8	0

TABLE 1. Microorganisms isolated from respiratory tract specimens obtained by various representative methods from adult patients who had a diagnosis of nosocomial pneumonia, by epidemiologic investigation — Continued

Category	Schaberg (*3*)	Bartlett (*4*)	Fagon (*5*)	Torres (*6*)
Anaerobes	N/A	35%**	2%**	0
Peptostreptococcus	N/A	14%**	N/A	0
Fusobacterium sp.	N/A	10	N/A	0
Peptococcus sp.	N/A	11	N/A	0
Bacteroides melaninogenicus	N/A	9	N/A	0
Bacteroides fragilis	N/A	8	N/A	0
Fungi	4%¶	N/A	0	1%††
Aspergillus sp.	N/A	N/A	0	1%††
Candida sp.	4%¶	N/A	0	0
Viruses	N/A	N/A	N/A	N/A

* National Nosocomial Infection Surveillance System and University of Michigan Hospitals.
† Not applicable (i.e., not tested or not reported).
§ Percentage of episodes.
¶ Percentage of isolates.
** Percentage of episodes; percentages not additive because of polymicrobial etiology in some episodes.
†† Percentage of patients with pure culture.

III. Epidemiology

Results of the NNIS indicate that pneumonias (diagnosed on the basis of the CDC surveillance definition of nosocomial pneumonia) account for approximately 15% of all hospital-associated infections and are the second most common type of nosocomial infection after those of the urinary tract (*2,61*). In 1984, the overall incidence of lower respiratory tract infection was six cases per 1,000 discharged patients (*2*). The incidence per 1,000 discharged patients ranged from 4.2 cases in nonteaching hospitals to 7.7 in university-affiliated hospitals, probably reflecting institutional differences in the level of patients' risk for acquiring nosocomial pneumonia.

Nosocomial bacterial pneumonia often has been identified as a postoperative infection (*62,63*). In the Study of the Efficacy of Nosocomial Infection Control, which was conducted in the 1970s, 75% of reported cases of nosocomial bacterial pneumonia occurred in patients who had had a surgical operation; the risk was 38 times greater for patients who had thoracoabdominal procedures than for those who had procedures involving other body sites (*63*). More recent epidemiologic studies, including NNIS studies, have identified other subsets of patients at high risk for acquiring nosocomial bacterial pneumonia. Such patients include persons >70 years of age; persons who have endotracheal intubation and/or mechanically assisted ventilation, a depressed level of consciousness (particularly those with closed-head injury), or underlying chronic lung disease; and persons who have previously had an episode of a large-volume aspiration. Other risk factors include 24-hour ventilator-circuit changes, hospitalization during the fall or winter, stress-bleeding prophylaxis with cimetidine (either with or without antacid), administration of antimicrobials, presence of a nasogastric tube, severe trauma, and recent bronchoscopy (*6,34,35,64–74*).

The NNIS has stratified the incidence density of nosocomial pneumonia by patients' use of mechanical ventilation and type of intensive-care unit (ICU). From 1986 through 1990, the median rate of ventilator-associated pneumonia cases per 1,000 ventilator-days ranged from 4.7 cases in pediatric ICUs to 34.4 cases in burn ICUs (*66*). In comparison, the median rate of nonventilator-associated pneumonia cases per 1,000 ICU-days ranged from zero cases in pediatric and respiratory ICUs to 3.2 cases in trauma ICUs.

Nosocomial pneumonia has been associated with high fatality rates. Crude mortality rates of 20%–50% and attributable mortality rates of 30%–33% have been reported; in one study, the number of deaths attributed to pneumonia reflected 60% of all deaths resulting from nosocomial infections (*17,35,74–80*). Patients receiving mechanically assisted ventilation have higher mortality rates than do patients not receiving ventilation support; however, other factors (e.g., the patient's underlying disease[s] and organ failure) are stronger predictors of death in patients who have pneumonia (*34,74*).

Analyses of pneumonia-associated morbidity have indicated that pneumonia could prolong hospitalization by 4–9 days (*79–83*); in the United States, a conservative estimate of the direct cost of this prolonged hospitalization is $1.2 billion

per year (*83*). Nosocomial pneumonia is a major infection-control problem because of its reported frequency, associated high fatality rate, and attendant costs.

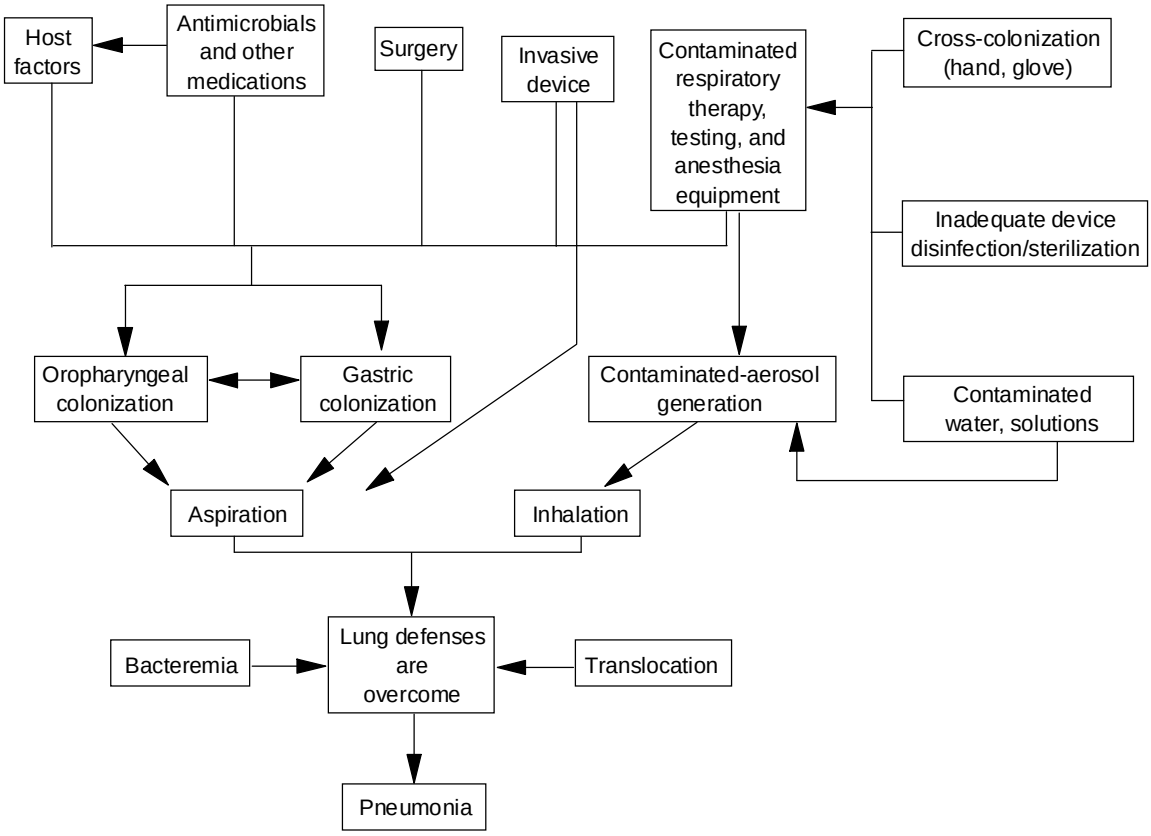
IV. Pathogenesis

Bacteria can invade the lower respiratory tract by aspiration of oropharyngeal organisms, inhalation of aerosols containing bacteria, or, less frequently, by hematogenous spread from a distant body site (Figure 1). In addition, bacterial translocation from the gastrointestinal tract has been hypothesized recently as a mechanism for infection. Of these routes, aspiration is believed to be the most important for both nosocomial and community-acquired pneumonia.

In radioisotope-tracer studies, 45% of healthy adults were found to aspirate during sleep (*84*). Persons who swallow abnormally (e.g., those who have depressed consciousness, respiratory tract instrumentation and/or mechanically assisted ventilation, or gastrointestinal tract instrumentation or diseases) or who have just undergone surgery are particularly likely to aspirate (*6,34,35,63,85–87*).

The high incidence of gram-negative bacillary pneumonia in hospitalized patients might result from factors that promote colonization of the pharynx by gram-negative bacilli and the subsequent entry of these organisms into the lower

FIGURE 1. Pathogenesis of nosocomial bacterial pneumonia



respiratory tract (*33,88–91*). Although aerobic gram-negative bacilli are recovered infrequently or are found in low numbers in pharyngeal cultures of healthy persons (*88,92*), the likelihood of colonization substantially increases in comatose patients, in patients treated with antimicrobial agents, and in patients who have hypotension, acidosis, azotemia, alcoholism, diabetes mellitus, leukocytosis, leukopenia, pulmonary disease, or nasogastric or endotracheal tubes in place (*33,91,93,94*).

Oropharyngeal or tracheobronchial colonization by gram-negative bacilli begins with the adherence of the microorganisms to the host's epithelial cells (*90,95–97*). Adherence may be affected by multiple factors associated with the bacteria (e.g., presence of pili, cilia, capsule, or production of elastase or mucinase), host cell (e.g., surface proteins and polysaccharides), and environment (e.g., pH and presence of mucin in respiratory secretions) (*89,90,95,98–107*). Although the exact interactions between these factors have not been fully elucidated, studies indicate that certain substances (e.g., fibronectin) can inhibit the adherence of gram-negative bacilli to host cells (*98,100,108*). Conversely, certain conditions (e.g., malnutrition, severe illness, or postoperative state) can increase adherence of gram-negative bacteria (*89,98,102,107,109*).

The stomach also might be an important reservoir of organisms that cause nosocomial pneumonia (*34,110–114*). The role of the stomach as such a reservoir might differ depending on the patient's underlying conditions and on prophylactic or therapeutic interventions (*22,111,115–118*). In healthy persons, few bacteria entering the stomach survive in the presence of hydrochloric acid at pH <2 (*119,120*). However, when gastric pH increases from the normal levels to ≥4, microorganisms are able to multiply to high concentrations in the stomach (*117,119,121–123*). This can occur in elderly patients (*121*); in patients who have achlorhydria (*119*), ileus, or upper gastrointestinal disease; and in patients receiving enteral feeding, antacids, or histamine-2 [H-2] antagonists (*111,117, 118,123–125*). Other factors (e.g., duodeno-gastric reflux and the presence of bile) may contribute to gastric colonization in patients who have impaired intestinal motility; these other factors need further investigation (*116*).

Bacteria also can enter the lower respiratory tract of hospitalized patients through inhalation of aerosols generated primarily by contaminated respiratory-therapy or anesthesia-breathing equipment (*126–129*). Outbreaks related to the use of respiratory-therapy equipment have been associated with contaminated nebulizers, which are humidification devices that produce large amounts of aerosol droplets <4 μm via ultrasound, spinning disk, or the Venturi mechanism (*126,129,130*). When the fluid in the reservoir of a nebulizer becomes contaminated with bacteria, the aerosol produced may contain high concentrations of bacteria that can be deposited deep in the patient's lower respiratory tract (*126,130,131*). Contaminated aerosol inhalation is particularly hazardous for intubated patients because endotracheal and tracheal tubes provide direct access to the lower respiratory tract. In contrast to nebulizers, bubble-through or wick humidifiers primarily increase the water-vapor (or molecular-water) content of inspired gases. Although heated bubble-through humidifiers generate aerosol

droplets, they do so in quantities that may not be clinically important (*127,132*); wick humidifiers do not generate aerosols.

Bacterial pneumonia has resulted, in rare instances, from hematogenous spread of infection to the lung from another infection site (e.g., pneumonia resulting from purulent phlebitis or right-sided endocarditis). Another mechanism, translocation of viable bacteria from the lumen of the gastrointestinal tract through epithelial mucosa to the mesenteric lymph nodes and to the lung, has been demonstrated in animal models (*133*). Translocation is postulated to occur in patients with immunosuppression, cancer, or burns (*133*); however, data are insufficient to describe this mechanism in humans (*134*).

V. Risk Factors and Control Measures

Several large studies have examined the potential risk factors for nosocomially acquired bacterial pneumonia (Table 2) (*6,34,35,135,136*). Although specific risk factors have differed between study populations, they can be grouped into the following general categories: a) host factors (e.g., extremes of age and severe underlying conditions, including immunosuppression); b) factors that enhance colonization of the oropharynx and/or stomach by microorganisms (e.g., administration of antimicrobials, admission to an ICU, underlying chronic lung disease, or coma); c) conditions favoring aspiration or reflux (e.g., endotracheal intubation, insertion of nasogastric tube, or supine position); d) conditions requiring prolonged use of mechanical ventilatory support with potential exposure to contaminated respiratory equipment and/or contact with contaminated or colonized hands of HCWs; and e) factors that impede adequate pulmonary toilet (e.g., undergoing surgical procedures that involve the head, neck, thorax, or upper abdomen or being immobilized as a result of trauma or illness) (*6,33–35,62,73, 74,135*).

A. Oropharyngeal, Tracheal, and Gastric Colonization

The association between colonization of the oropharynx (*88,137*), trachea (*138*), or stomach (*110,111,117,123*) and predisposition to gram-negative bacillary pneumonia prompted efforts to prevent infection by using either prophylactic local application of antimicrobial agent(s) (*139,140*) or local bacterial interference (*141,142*). Although early studies suggested that the first method (i.e., use of aerosolized antimicrobials) could eradicate common gram-negative pathogens from the upper respiratory tract (*138*), superinfection occurred in some patients receiving this therapy (*139–141,143,144*). The second method (i.e., bacterial interference [with alpha-hemolytic streptococci]) has been used successfully by some investigators to prevent oropharyngeal colonization by aerobic gram-negative bacilli (*141*). However, the efficacy of this method for general usage has not been evaluated.

In many studies, the administration of antacids and H-2 blockers for prevention of stress bleeding in critically ill, postoperative, and/or mechanically ventilated

TABLE 2. Risk factors and suggested infection-control measures for preventing nosocomial pneumonia

Disease/Risk factors	Suggested infection-control measures
Bacterial pneumonia	
Host-related (persons ages >65 yrs)	
• Underlying illness:	
— Chronic obstructive pulmonary disease	Perform incentive spirometry, positive end-expiratory pressure, or continuous positive airway pressure by face mask.
— Immunosuppression	Avoid exposure to potential nosocomial pathogens; decrease duration of immunosuppression (e.g., by administration of granulocyte macrophage colony stimulating factor [GMCSF]).
— Depressed consciousness	Administer central nervous system depressants cautiously.
— Surgery (thoracic/abdominal)	Properly position patients; promote early ambulation; appropriately control pain.
Device-related	Properly clean, sterilize or disinfect, and handle devices; remove devices as soon as the indication for their use ceases.
• Endotracheal intubation and mechanical ventilation	Gently suction secretions; place patient in semirecumbent position (i.e., 30^{o}–45^{o} head elevation); use nonalkalinizing gastric cytoprotective agent on patients at risk for stress bleeding; do not routinely change ventilator circuits more often than every 48 hours; drain and discard inspiratory-tubing condensate, or use heat-moisture exchanger if indicated.
• Nasogastric-tube (NGT) placement and enteral feeding	Routinely verify appropriate tube placement; promptly remove NGT when no longer needed; drain residual; place patient in semirecumbent position as described above.
Personnel- or procedure-related	
• Cross-contamination by hands	Educate and train personnel; wash hands adequately and wear gloves appropriately; conduct surveillance for cases of pneumonia and give feedback to personnel.
• Antibiotic administration	Use antibiotics prudently, especially in patients in intensive-care units who are at high risk.
Legionnaires disease	
Host-related	
• Immunosuppression	Decrease duration of immunosuppression.
Device-related	
• Contaminated aerosol from devices	Sterilize/disinfect aerosol-producing devices before use; use only sterile water for respiratory humidifying devices; do not use cool-mist room-air humidifiers without adequate sterilization or disinfection.

TABLE 2. Risk factors and suggested infection-control measures for preventing nosocomial pneumonia — Continued

Disease/Risk factors	Suggested infection-control measures
Environment-related	
• Aerosols from contaminated water supply	Hyperchlorinate or superheat hospital water system; routinely clean water-supply system; consider use of sterile water for drinking by immunosuppressed patients.
• Cooling-tower draft	Properly design, place, and maintain cooling towers.
Aspergillosis	
Host-related	
• Severe granulocytopenia	Decrease duration of immunosuppression (e.g., by administration of GMCSF); place patients who have severe and prolonged granulocytopenia in a protected environment.
Environment-related	
• Construction activity	Remove granulocytopenic patients from vicinity of construction; if not already done, place severely granulocytopenic patients in a protected environment; make severely granulocytopenic patients wear a mask when they leave the protected environment.
• Other environmental sources of aspergilli	Routinely maintain hospital air-handling systems and rooms of immunosuppressed patients.
Respiratory syncytial virus infection (RSV)	
Host-related	
• Persons ages <2 yrs; congenital pulmonary/cardiac disease; immunosuppression	Consider routine preadmission screening of high-risk patients for severe RSV infection, followed by cohorting of patients and nursing personnel during hospital outbreaks of RSV infection.
Personnel- or procedure-related	
• Cross-contamination by hands	Educate personnel; wash hands; wear gloves; wear a gown; during outbreaks, use private rooms or cohort patients and nursing personnel, and limit visitors.
Influenza	
Host-related	
• Persons ages >65 yrs; immunosuppression	Vaccinate patients who are at high risk before the influenza season begins each year; use amantadine or rimantadine for chemoprophylaxis during an outbreak.
Personnel-related	
• Infected personnel	Before the influenza season each year, vaccinate personnel who provide care for high-risk patients; use amantadine or rimantadine for prophylaxis and treatment during an outbreak.

patients has been associated with gastric bacterial overgrowth (*34,112,113, 118,122,123,145–147*). Sucralfate, a cytoprotective agent that has little effect on gastric pH and may have bactericidal properties of its own, has been suggested as a potential substitute for antacids and H-2 blockers (*148–150*). The results of clinical trials comparing the risk for pneumonia in patients receiving sucralfate with that in patients treated with antacids and/or H-2 blockers have been variable (*112,118,147,148,151–153*). In most randomized trials, ICU patients receiving mechanically assisted ventilation who were treated either with only antacids or with antacids and H-2 blockers had increased gastric pH, high bacterial counts in the gastric fluid, and increased risk for pneumonia in comparison with patients treated with sucralfate (*112,118,147,148,151*). In one study of a large number of patients, the incidence of early-onset pneumonia (i.e., onset occurring ≤4 days after intubation) did not differ between patient groups, but late-onset pneumonia occurred in 5% of 76 patients treated with sucralfate, 16% of 69 treated with antacids, and 21% of 68 treated with an H-2 blocker (*147*). Conversely, a meta-analysis of data from eight earlier studies (*154*) and a later study comparing sucralfate with ranitidine (*153*) did not indicate a strong association between nosocomial pneumonia and drugs that increase gastric pH. Additional studies, in which bronchoscopy with either PSB or BAL is used to more reliably diagnose pneumonia, are being conducted to compare the efficacy of sucralfate and ranitidine.

Selective decontamination of the digestive tract (SDD) is another strategy designed to prevent bacterial colonization and lower respiratory tract infection in mechanically ventilated patients (*155–179*). SDD is aimed at preventing oropharyngeal and gastric colonization with aerobic gram-negative bacilli and *Candida* sp. without altering the anaerobic flora (Table 3). Various SDD regimens use a combination of locally administered nonabsorbable antibiotic agents, such as polymyxin and an aminoglycoside (either tobramycin, gentamicin, or, rarely, neomycin) or a quinolone (either norfloxacin or ciprofloxacin) coupled with either amphotericin B or nystatin. The local antimicrobial preparation is applied as a paste to the oropharynx and administered either orally or via the nasogastric tube four times a day. In addition, in many studies, a systemic (intravenous) antimicrobial (e.g., cefotaxime or trimethoprim) is administered to the patient.

Although most studies (*155–158,160–167,169,170,175–177*), including two meta-analyses (*171,178*), have demonstrated a decrease in the rates of nosocomial respiratory infections after SDD, these studies have been difficult to assess because they have differed in design and study population and many have had short follow-up periods (Table 3). In most of these studies, the diagnosis of pneumonia was based on clinical criteria; bronchoscopy with BAL or PSB was used in only a few studies (*159,162,173,175–177,179*).

Two recently published reports of large, double-blind, placebo-controlled trials demonstrated no benefit from SDD (*173,174*). One of these studies, which was conducted in France, noted that the incidence of gram-negative bacillary pneumonia decreased significantly after SDD, but this decrease was not accom-

panied by a decrease in pneumonia from all causes (*173*). In the other study, no differences were noted between patients randomly assigned to SDD or placebo treatment conditions; however, both patient groups also received simultaneous treatment with intravenous cefotaxime (*174*).

Although an earlier meta-analysis indicated a trend toward decreased mortality in patients administered SDD (*171*), a more recent and more extensive analysis highlights the equivocal effect of SDD on patient mortality, as well as the high cost of using SDD to prevent nosocomial pneumonia or death resulting from nosocomial pneumonia (i.e., to prevent one case of nosocomial pneumonia, six patients [range: five to nine patients] would have to be administered SDD; to prevent one death, 23 patients [range: 13–39 patients]) (*178*). Furthermore, both the development of antimicrobial resistance and superinfection with gram-positive bacteria and other antibiotic-resistant nosocomial pathogens are public health concerns (*156,158,159,161,175,180*). Thus, currently available data do not justify the routine use of SDD for prevention of nosocomial pneumonia in ICU patients. SDD may be ultimately useful for specific subsets of ICU patients, such as patients with trauma or severe immunosuppression (e.g., bone-marrow–transplant recipients).

A new approach advocated to prevent oropharyngeal colonization in patients receiving enteral nutrition is to reduce bacterial colonization of the stomach by acidifying the enteral feed (*181*). Although the absence of bacteria from the stomach has been confirmed in patients given acidified enteral feeding, the effect on the incidence of nosocomial pneumonia has not been evaluated (*181*).

B. Aspiration of Oropharyngeal and Gastric Flora

Clinically important aspiration usually occurs in patients who a) have a depressed level of consciousness; b) have dysphagia resulting from neurologic or esophageal disorders; c) have an endotracheal (nasotracheal or orotracheal), tracheostomal, or enteral (nasogastric or orogastric) tube in place; and/or d) are receiving enteral feeding (*35,84,85,182–186*). Placement of an enteral tube may increase nasopharyngeal colonization, cause reflux of gastric contents, or allow bacterial migration via the tube from the stomach to the upper airway (*183,186–188*). When enteral feedings are administered, gross contamination of the enteral solution during preparation (*189–191*) and elevated gastric pH (*70,192,193*) may lead to gastric colonization with gram-negative bacilli. In addition, gastric reflux and aspiration might occur because of increased intragastric volume and pressure (*70,117,183*).

Although prevention of pneumonia in such patients may be difficult, methods that make regurgitation less likely (e.g., placing the patient in a semirecumbent position [i.e., by elevating the head of the bed] and withholding enteral feeding if the residual volume in the stomach is large or if bowel sounds are not heard upon auscultation of the abdomen) may be beneficial (*185,194–197*). Conversely, equivocal results have been obtained by a) administering enteral

TABLE 3. Controlled studies on nosocomial lower respiratory tract infections and other associated outcomes of selective decontamination of the digestive tract in adult patients with mechanically assisted ventilation

Author	Study patients	Diagnostic method	Lower respiratory tract infection: Infection rate SDD (%)	Lower respiratory tract infection: Infection rate Controls (%)	Colonization or infection with resistant microorganisms SDD (%)	Colonization or infection with resistant microorganisms Controls (%)	Overall mortality in hospital SDD (%)	Overall mortality in hospital Controls (%)	Mean total no. of days in ICU SDD	Mean total no. of days in ICU Controls
Stoutenbeek (1984) (*155*)	Trauma; SDD=63; Controls=59.	Clinical and radiologic;** TS culture.††	8	59	"No increase"		3	8	Not reported	
Unertl (1987) (*156*)	General ICU; SDD=19; Controls=20.	Clinical and radiologic.**	21	70	21§§	20§§	26	30	18¶¶	23¶¶
Kerver (1988) (*157*)	Surgical ICU; SDD=49; Controls=47.	Clinical and radiologic.**	12	85	"Not recorded"		29 IR***=4	32 IR=17	17	20
Ledingham (1988) (*158*)	General ICU; SDD=163; Controls=161.	Clinical and radiologic**	2	11	"No increase"		24	24	Not reported	
Brun-Buisson (1989) (*159*)	Medical ICU; SDD=36; Controls=50.	Clinical and radiologic;** TS and PSB culture††	20	22	3§§	16§§	22 IR=9	24 IR=10	14	15
Ulrich (1989) (*160*)	General ICU; SDD=48; Controls=52.	Clinical and radiologic;** TS culture††	15	50	GP=78††† GN=3†††	GP=44††† GN=2†††	31 IR=0	54 IR=15	17	13
Flaherty (1990) (*161*)	Cardiac surgery ICU; SDD=51; Controls=56.	Clinical and radiologic.**	2	9	GN=22§§	GN=21§§	0	2	Not reported	
Godard (1990) (*162*)	General ICU; SDD=97; Controls=84.	Clinical and radiologic;** TS and PSB culture.††	2	15	GN=15†††	GN=15†††	12	18	11	16
McClelland (1990) (*163*)	Renal and respiratory failure; SDD=15; Controls=12.	TS culture.††	7	50	Not reported		60 IR=27	58 IR=8	Not reported	
Rodriquez-Roldan (1990) (*164*)	General ICU; SDD=13; Controls=15.	Clinical and radiologic;** TS culture††	Pn=0§§§ TB=23§§§	Pn=73§§§ TB=20§§§	"None noticed"		30 IR=0	33 IR=13	Not reported	

TABLE 3. Controlled studies on nosocomial lower respiratory tract infections and other associated outcomes of selective decontamination of the digestive tract in adult patients with mechanically assisted ventilation — Continued

Author	Study patients	Diagnostic method	Lower respiratory tract infection: Infection rate SDD (%)	Lower respiratory tract infection: Infection rate Controls (%)	Colonization or infection with resistant microorganisms SDD (%)	Colonization or infection with resistant microorganisms Controls (%)	Overall mortality in hospital SDD (%)	Overall mortality in hospital Controls (%)	Mean total no. of days in ICU SDD	Mean total no. of days in ICU Controls
Tetteroo (1990) (*165*)	Esophageal resection; SDD=56; Controls=56.	Clinical and radiologic;** culture of bronchial aspirate	2	14	2§§	4§§	5 IR=4	4 IR=0	6	5
Aerdts (1991) (*166*)	General ICU; SDD=17; Controls-A=18;¶¶¶ Controls-B=21.¶¶¶	Clinical and radiologic;** TS culture.††	6	A=78 B=62	"Not observed"		12 IR=6	A=22 IR=11 B=10 IR= 0	23	A=30 B=25
Blair (1991) (*167*)	General ICU; SDD=126; Controls=130.	Clinical and radiologic.**	10	35	"No evidence of increased resistance"		14	19	8	8
Fox (1991) (*168*)	Cardiac bypass; SDD=12; Controls=12.	TS culture.††	66	50	Not reported		17	66	12	12
Hartenauer (1991) (*169*)	Surgical ICU; ICU-1: SDD=50, Controls=61; ICU-2: SDD=49, Controls=40.	Clinical and radiologic;** TS culture.††	ICU-1: 10 ICU-2: 10	46 45	S=34§§ GN=0§§ S=37§§ GN=0§§	S=33§§ GN=0§§ S=37§§ GN=0§§	38 IR=8 31 IR=6	48 IR=21 43 IR=25	12 13	13 17
Pugin (1991) (*170*)	Surgical ICU; SDD=25; Controls=27.	Clinical and radiologic;** TS culture.††	16	78	"No new antibiotic resistance"		28	26	13	15
Vandenbroucke-Grauls (1991) (*171*)	ICUs (pooled data);**** SDD-A=488, Controls-A (historical)=540; SDD-B=225, Controls-B (random)=266.	Clinical and radiologic;** TS culture††	A=7 B=8	A=28 B=45	"No increase in resistant micro-organisms in 10 of 11 studies"		A=25 B=21	A=26 B=26	Not reported	
Cockerill (1992) (*172*)	Surgical and medical ICUs; SDD=75; Controls=75.	Clinical and radiologic;** TS culture.††	Pn=5§§§ TB=4§§§	Pn=16§§§ TB=5§§§	16†††	11†††	15	21	10	12

TABLE 3. Controlled studies on nosocomial lower respiratory tract infections and other associated outcomes of selective decontamination of the digestive tract in adult patients with mechanically assisted ventilation — Continued

Author	Study patients	Diagnostic method	Lower respiratory tract infection: Infection rate SDD (%)	Lower respiratory tract infection: Infection rate Controls (%)	Colonization or infection with resistant microorganisms SDD (%)	Colonization or infection with resistant microorganisms Controls (%)	Overall mortality in hospital SDD (%)	Overall mortality in hospital Controls (%)	Mean total no. of days in ICU SDD	Mean total no. of days in ICU Controls
Gastinne (1992) (*173*)	Medical ICU; SDD=220; Controls=225.	Clinical and radiologic;** TS ± PSB culture.††	12	15	Not reported		40 34††††	36 34††††	18	19
Hammond (1992) (*174*)	General ICU; SDD=114; Controls=125.	Clinical and radiologic;** TS culture.††	Pn=15§§§ Br=6§§§	Pn=15§§§ Br=6§§§	Not reported§§§§		18 IR=6	17 IR=6	16	17
Rocha (1992) (*175*)	General ICU; SDD=47; Controls=54.	Clinical and radiologic;** TS ± BAL culture.††	26	63	GP=62††† GN=43†††	GP=38††† GN=30†††	21 IR=2	44 IR=20	19	18
Winter (1992) (*176*)	General ICU; SDD=91; Control-A=84, Control-B=92.	Clinical and radiologic;** BAL culture.††	3	A=11 B=23	1–8†††	A=1–7††† B=1–17†††	36	A=43 B=43	6	A=7 B=8
Korinek (1993) (*177*)	Neurosurgical ICU; SDD=63; Controls=60.	Clinical and radiologic;** TS and PSB culture.††	24	42	"No evidence of increased resistance"		8	7	24	29
SDD Trialists (1993) (*178*)	ICUs (pooled data);**** SDD=2,047; Controls=2,095.	Variable.	Odds ratio=0.37;¶¶¶ 95% CI*****=0.31–0.43		Not analyzed		27 Odds ratio=0.90; 95% CI=0.79–1.04	29	Not analyzed	
Ferrer (1994) (*179*)	Respiratory ICU; SDD=39; Controls=41.	Clinical and radiologic;** TS + PSB or BAL culture†† ± autopsy histology.	18	24	Not reported†††††		31	27	Not reported	

*Resistant to at least one antimicrobial in the SDD regimen.
†During the study period.
§ICU=intensive-care unit.
¶SDD=selective digestive-tract decontamination.
**Clinical criteria included temperature >38 C, purulent bronchorrhea, WBC >(12,000–15,000/mm^3). Radiologic criterion was evidence of new and progressive infiltrate(s).
††TS=tracheal secretions; PSB=protected-specimen brushing; BAL=bronchoalveolar lavage.
§§Percentage of patients infected or colonized with gram-positive (GP) and/or gram-negative (GN) bacillary organisms at any body site; GP=percentage of patients infected or colonized with gram-positive organisms at any body site; GN=percentage of patients infected or colonized with gram-negative bacillary organisms at any body site; S=percentage of patients with coagulase-negative staphylococcal infection or colonization.

TABLE 3. Controlled studies on nosocomial lower respiratory tract infections and other associated outcomes of selective decontamination of the digestive tract in adult patients with mechanically assisted ventilation — Continued

¶¶ Median.
*** Infection-related.
††† Percentage of isolates; GP=percentage of gram-positive isolates; GN=percentage of gram-negative bacillary isolates.
§§§ Pn=pneumonia; TB=tracheobronchial infection; Br=bronchial infection.
¶¶¶ Control-A=patients given penicillin (ampicillin, piperacillin, or flucloxacillin) for clinical infection(s); Control-B=patients given cephalosporin (cephadrine, cefuroxime, or cefotaxime) for clinical infection(s).
**** Meta-analysis.
†††† In ICU.
§§§§ However, at 4 weeks, the oropharyngeal cultures of 13% of SDD patients and 5% of control patients had methicillin-resistant *Staphylococcus aureus* (MRSA), and 41% of SDD patients and control patients were colonized with enterococci.
¶¶¶¶ Computed using data from 3,836 patients and 526 events, 260 in SDD patients and 366 in control patients.
***** CI=confidence interval.
††††† However, bronchial colonization with MRSA occurred in 45% of SDD patients and 21% of control patients.

nutrition intermittently in small boluses rather than continuously (*70,193*); b) using flexible, small-bore enteral tubes (*186,198*); or c) placing the enteral tube below the stomach (e.g., in the jejunum) (*199,200*).

C. Mechanically Assisted Ventilation and Endotracheal Intubation

Patients receiving continuous, mechanically assisted ventilation have 6–21 times the risk for acquiring nosocomial pneumonia compared with patients not receiving ventilatory support (*34,63,65,75*). One study indicated that the risk for developing ventilator-associated pneumonia increased by 1% per day (*5*). This increased risk was attributed partially to carriage of oropharyngeal organisms upon passage of the endotracheal tube into the trachea during intubation, as well as to depressed host defenses secondary to the patient's severe underlying illness (*6,34,35,201*). In addition, bacteria can aggregate on the surface of the tube over time and form a glycocalyx (i.e., a biofilm) that protects the bacteria from the action of antimicrobial agents or host defenses (*202*). Some researchers believe that these bacterial aggregates can become dislodged by ventilation flow, tube manipulation, or suctioning and subsequently embolize into the lower respiratory tract and cause focal pneumonia (*203,204*). Removing tracheal secretions by gentle suctioning and using aseptic techniques to reduce cross-contamination to patients from contaminated respiratory therapy equipment or contaminated or colonized hands of HCWs have been used traditionally to help prevent pneumonia in patients receiving mechanically assisted ventilation.

The risk for pneumonia also is increased by the direct access of bacteria to the lower respiratory tract, which often occurs because of leakage around the endotracheal cuff (*86,205*), thus enabling pooled secretions above the cuff to enter the trachea (*206*). In one study, the occurrence of nosocomial pneumonia was delayed and decreased in intubated patients whose endotracheal tubes had a separate dorsal lumen that allowed drainage (i.e., by suctioning) of secretions in the space above the endotracheal tube cuff and below the glottis (*206*). However, additional studies are needed to determine the cost-benefit ratio of using this device.

D. Cross-Colonization Via Hands of HCWs

Pathogens that cause nosocomial pneumonia (e.g., gram-negative bacilli and *S. aureus*) are ubiquitous in hospitals, especially in intensive- or critical-care areas (*207,208*). Transmission of these microorganisms to patients frequently occurs via an attending HCW's hands that have become contaminated or transiently colonized with the microorganisms (*209–215*). Procedures such as tracheal suctioning and manipulation of the ventilator circuit or endotracheal tubes increase the opportunity for cross-contamination (*215,216*). The risk for cross-contamination can be reduced by using aseptic techniques and sterile or disinfected equipment when appropriate (*65*) and by eliminating pathogens from the hands of HCWs (*65,215,217–219*).

In theory, adequate handwashing is an effective way of removing transient bacteria from the hands (*218,219*); however, personnel compliance with handwashing recommendations has been generally poor (*220–223*). For this reason, the routine use of gloves has been advocated to help prevent cross-contamination (*224,225*). The routine use of gloves, in addition to the use of gowns, was associated with a decrease in the incidence of nosocomial RSV infection (*226*) and other infections acquired in ICUs (*227*). However, nosocomial pathogens can colonize gloves (*228*), and outbreaks have been traced to HCWs who did not change gloves after having contact with one patient and before providing care to another (*229,230*). In addition, gloved hands can be contaminated through leaks in the gloves (*231*).

E. Contamination of Devices Used on the Respiratory Tract

Devices used on the respiratory tract for respiratory therapy (e.g., nebulizers), diagnostic examination (e.g., bronchoscopes and spirometers), and administration of anesthesia are potential reservoirs and vehicles for infectious microorganisms (*65,232–236*). Routes of transmission might be from device to patient (*127,129,234–244*), from one patient to another (*245,246*), or from one body site to the lower respiratory tract of the same patient via hand or device (*233,246–248*). Contaminated reservoirs of aerosol-producing devices (e.g., nebulizers) can allow the growth of hydrophilic bacteria that subsequently can be aerosolized during use of the device (*126,129,130,242*). Gram-negative bacilli (e.g., *Pseudomonas* sp., *Xanthomonas* sp., *Flavobacterium* sp., *Legionella* sp., and nontuberculous mycobacteria) can multiply to substantial concentrations in nebulizer fluid (*241,249–251*) and increase the risk for pneumonia in patients using such devices (*127–130,241,242,252,253*).

Proper cleaning and sterilization or disinfection of reusable equipment are important components of a program to reduce infections associated with respiratory therapy and anesthesia equipment (*234,235,237–240,242,254–259*). Many devices or parts of devices used on the respiratory tract have been categorized as semicritical in the Spaulding classification system for appropriate sterilization or disinfection of medical devices because they come into direct or indirect contact with mucous membranes but do not ordinarily penetrate body surfaces (Appendix A), and the associated risk for infection in patients after the use of such devices is less than that associated with devices that penetrate normally sterile tissues (*260*). Thus, if sterilization of these devices by steam autoclave or ethylene oxide is not possible or cost-effective (*261*), they can be subjected to high-level disinfection by pasteurization at 75 C for 30 minutes (*262–265*) or by use of liquid chemical disinfectants approved by the Environmental Protection Agency (EPA) as sterilants/disinfectants and approved for use on medical instruments by the Food and Drug Administration (*225, 266–268*).

If a respiratory device needs rinsing to remove a residual liquid chemical sterilant/disinfectant after chemical disinfection, sterile water is preferred because tap or locally prepared distilled water might contain microorganisms

that can cause pneumonia (*249,250,269–272*). In some hospitals, a tap-water rinse followed by air-drying with or without an alcohol rinse (i.e., to hasten drying) is used (*273*). In theory, if complete drying is achieved after a tap-water rinse, the risk for nosocomial pneumonia associated with the use of the device is probably low. Air drying reduces the level of microbial contamination of the hands of HCWs after washing, and air drying also reduces contamination of gastrointestinal endoscopes (*274–276*). However, many semicritical items used on the respiratory tract (e.g., corrugated tubing, jet or ultrasonic nebulizers, and bronchoscopes) are difficult to dry, and the degree of dryness of a device is difficult to assess (*265*). Data are insufficient regarding the safety of routinely using tap water for rinsing (followed by drying) reusable semicritical respiratory devices after their disinfection or between their uses on the same patient (*242,258,273,277*).

1. *Mechanical Ventilators, Breathing Circuits, Humidifiers, Heat-Moisture Exchangers, and In-Line Nebulizers*

   a. *Mechanical ventilators.* The internal machinery of mechanical ventilators used for respiratory therapy is not considered an important source of bacterial contamination of inhaled gas (*278*). Thus, routine sterilization or high-level disinfection of the internal machinery is considered unnecessary. Using high-efficiency bacterial filters at various positions in the ventilator breathing circuit had been advocated previously (*279,280*). Filters interposed between the machinery and the main breathing circuit can eliminate contaminants from the driving gas and prevent retrograde contamination of the machine by the patient; however, these filters also might alter the functional specifications of the breathing device by impeding high gas flows (*279–281*). Placement of a filter or condensate trap at the expiratory-phase tubing of the mechanical-ventilator circuit may help prevent cross-contamination of the ventilated patient's immediate environment (*247,282*), but the importance of such filters in preventing nosocomial pneumonia needs further evaluation.

   b. *Breathing circuits, humidifiers, and heat-moisture exchangers*. In the United States, most hospitals use ventilators with either bubble-through or wick humidifiers that produce either insignificant (*132,283*) or no aerosols, respectively, for humidification. Thus, these devices probably do not pose an important risk for pneumonia in patients. In addition, bubble-through humidifiers are usually heated to temperatures that reduce or eliminate bacterial pathogens (*283,284*). Sterile water, however, is still usually used to fill these humidifiers (*285*) because tap or distilled water might contain microorganisms, such as *Legionella* sp., that are more heat-resistant than other bacteria (*252,271*).

      The potential risk for pneumonia in patients using mechanical ventilators that have heated bubble-through humidifiers stems primarily from the condensate that forms in the inspiratory-phase tubing of the ventilator circuit as a result of the difference in the temperatures of the inspiratory-

phase gas and ambient air; condensate formation increases if the tubing is unheated (*286*). The tubing and condensate can rapidly become contaminated, usually with bacteria that originate in the patient's oropharynx (*286*). In one study, 33% of inspiratory circuits were colonized with bacteria via this route within 2 hours, and 80% within 24 hours, after initiation of mechanical ventilation (*286*). Spillage of the contaminated condensate into the patient's tracheobronchial tree, as can occur during procedures in which the tubing is moved (e.g., for suctioning, adjusting the ventilator setting, or feeding or caring for the patient), may increase the risk for pneumonia in the patient (*286*). Thus, in many hospitals, HCWs are trained to prevent such spillage and to drain the fluid periodically. Microorganisms contaminating ventilator-circuit condensate can be transmitted to other patients via the hands of HCWs handling the fluid, especially if the HCW neglects washing hands after handling the condensate.

The role of ventilator-tubing changes in preventing pneumonia in patients using mechanical ventilators with bubble-through humidifiers has been investigated. Initial studies of in-use contamination of mechanical ventilator circuits with humidifiers have indicated that neither the rate of bacterial contamination of inspiratory-phase gas nor the incidence of pneumonia was significantly increased when tubing was changed every 24 hours rather than every 8 or 16 hours (*287*). A later study indicated that changing the ventilator circuit every 48 hours rather than every 24 hours did not result in an increase in contamination of the inspiratory-phase gas or tubing of the ventilator circuits (*288*). In addition, the incidence of nosocomial pneumonia was not significantly higher when circuits were changed every 48 hours rather than every 24 hours (*288*). More recent reports suggest that the risk for pneumonia may not increase when the interval for circuit change is prolonged beyond 48 hours. Another study indicated that the risk for pneumonia was not significantly higher when the circuits were never changed for the duration of use by the patient (eight [29%] of 28 patients) rather than when the circuits were changed every 48 hours (11 [31%] of 35 patients) (*289*).

These findings indicate that the recommended daily change in ventilator circuits may be extended to ≥48 hours. This change in recommendation could result in substantial savings for U.S. hospitals by reducing the number of circuits used and the amount of personnel time required to change the circuits (*285,288*). The maximum time, however, that a circuit can be safely left unchanged on a patient has not been determined.

Condensate formation in the inspiratory-phase tubing of a ventilator breathing circuit can be decreased by elevating the temperature of the inspiratory-phase gas with a heated wire in the inspiratory-phase tubing. However, in one report, three cases of endotracheal- or tracheostomy-tube blockage by dried secretions of the patient were attributed to the decrease in the relative humidity of inspired gas that resulted from the

elevation of the gas temperature (*290*). Until additional information regarding the frequency of such cases is available, HCWs who provide care to patients requiring mechanical ventilation should be aware of the advantages and potential complications associated with using heated ventilator tubing.

Condensate formation can be eliminated by using a heat-moisture exchanger (HME) or a hygroscopic condenser humidifier (i.e., an "artificial nose") (*291–296*). An HME recycles heat and moisture exhaled by the patient and eliminates the need for a humidifier. In the absence of a humidifier, no condensate forms in the inspiratory-phase tubing of the ventilator circuit. Thus, bacterial colonization of the tubing is prevented, and the need to change the tubing on a periodic basis is obviated (*216*). Some models of HMEs are equipped with bacterial filters, but the advantage of using such filters is unknown. HMEs can increase the dead space (i.e., the area of the lung in which air is not exchanged) and resistance to breathing, might leak around the endotracheal tube, and might result in drying of sputum and blockage of the tracheobronchial tree (*297*). Although recently developed HMEs that have humidifiers increase airway humidity without increasing colonization of bacteria (*293,298*), additional studies are needed to determine whether the incidence of pneumonia is decreased (*299–302*).

c. *Small-volume ("in-line") medication nebulizers*. Small-volume medication nebulizers that are inserted in the inspiratory circuit of mechanical ventilators can produce bacterial aerosols (*242*). If such devices become contaminated by condensate in the inspiratory tubing of the breathing circuit, they can increase the patient's risk for pneumonia because the nebulizer aerosol is directed through the endotracheal tube and bypasses many of the normal host defenses against infection (*286*).

2. *Large-Volume Nebulizers*. Nebulizers with large-volume (>500 cc) reservoirs, including those used in intermittent positive-pressure breathing (IPPB) machines and ultrasonic or spinning-disk room-air humidifiers, pose the greatest risk for pneumonia to patients, probably because of the large amount of aerosols they generate (*237–241,252,303*). These reservoirs can become contaminated by the hands of HCWs, unsterile humidification fluid, or inadequate sterilization or disinfection between uses (*126*). Once introduced into the reservoir, various bacteria, including *Legionella* sp., can multiply to sufficiently large numbers within 24 hours to pose a risk for infection in patients who receive inhalation therapy (*128,129,241,253,303*). Sterilization or high-level disinfection of these nebulizers can eliminate vegetative bacteria from their reservoirs and make them safe for patient use (*260*). However, unlike nebulizers attached to IPPB machines, room-air humidifiers have a high cost-benefit ratio: evidence of clinical benefits from their use in hospitals is lacking, and the potential cost of daily sterilization or disinfection of, and use of sterile water to fill, such devices is substantial.

3. *Hand-Held Small-Volume Medication Nebulizers*. Small-volume medication nebulizers used to administer bronchodilators, including nebulizers that are hand-held, can produce bacterial aerosols. Hand-held nebulizers have been associated with nosocomial pneumonia, including Legionnaires disease, resulting from either contamination with medications from multidose vials (*304*) or *Legionella*-contaminated tap water used for rinsing and filling the reservoir (*258*).

4. *Suction Catheters, Resuscitation Bags, Oxygen Analyzers, and Ventilator Spirometers*. Tracheal suction catheters can introduce microorganisms into a patient's lower respiratory tract. Two types of suction-catheter systems are used in U.S. hospitals: the open single-use catheter system and the closed multi-use catheter system. Studies comparing the two systems have involved low numbers of patients; the results of these studies suggest that the risk for catheter contamination or pneumonia does not differ between patients on whom the single-use suction method is used and those on whom the closed multi-use catheter system is used (*305–307*). Although advantages of cost and decreased environmental contamination have been attributed to use of the closed-suction system (*308,309*), larger studies are needed to compare the advantages and disadvantages of both systems (*310*).

   Reusable resuscitation bags are particularly difficult to clean and dry between uses; microorganisms in secretions or fluid left in the bag may be aerosolized and/or sprayed into the lower respiratory tract of the patient on whom the bag is used; in addition, contaminating microorganisms might be transmitted from one patient to another via hands of HCWs (*311–313*). Oxygen analyzers and ventilator spirometers have been associated with outbreaks of gram-negative respiratory tract colonization and pneumonia resulting from patient-to-patient transmission of organisms via hands of HCWs (*233,245*). These devices require either sterilization or high-level disinfection between uses on different patients. Education of physicians, respiratory therapists, and nursing staff regarding the associated risks and appropriate care of these devices is essential.

5. *Anesthesia Equipment*. The contributory role of anesthesia equipment in outbreaks of nosocomial pneumonia was reported before hospitals implemented routine after-use cleaning and disinfection/sterilization of reusable anesthesia-equipment components that could become contaminated with pathogens during use (*314,315*).

   a. *Anesthesia machine*. The internal components of anesthesia machines, which include the gas sources and outlets, gas valves, pressure regulators, flowmeters, and vaporizers, are not considered an important source of bacterial contamination of inhaled gases (*316*). Thus, routine sterilization or high-level disinfection of the internal machinery is unnecessary.

b. *Breathing system or patient circuit.* The breathing system or patient circuit (including the tracheal tube or face mask, inspiratory and expiratory tubing, y-piece, CO_2 absorber and its chamber, anesthesia ventilator bellows and tubing, humidifier, adjustable pressure-limiting valve, and other devices and accessories), through which inhaled and/or exhaled gases flow to and from a patient, can become contaminated with microorganisms that might originate from the patient's oropharynx or trachea. Recommendations for in-use care, maintenance, and reprocessing (i.e., cleaning and disinfection or sterilization) of the components of the breathing system have been published (*317,318*). In general, reusable components of the breathing system that directly touch the patient's mucous membranes (e.g., face mask or tracheal tube) or become readily contaminated with the patient's respiratory secretions (e.g., y-piece, inspiratory and expiratory tubing, and attached sensors) are cleaned and subjected to high-level disinfection or sterilization between patients. The other parts of the breathing system (e.g., CO_2 absorber and its chamber), for which an appropriate and cost-effective schedule of reprocessing has not been firmly determined (*319*), are changed, cleaned, and sterilized or subjected to high-level disinfection periodically in accordance with published guidelines (*317,318*) and/or the manufacturers' instructions.

Using high-efficiency bacterial filters at various positions in the patient circuit (e.g., at the y-piece or on the inspiratory and expiratory sides of the patient circuit) has been advocated (*317,320,321*) and shown to decrease contamination of the circuit (*321–323*). However, the use of bacterial filters to prevent nosocomial pulmonary infections has not been proven to be effective and requires additional analysis (*324–326*).

6. *Pulmonary Function Testing Apparatus.*

a. *Internal parts of pulmonary function testing apparatus*. The internal parts of pulmonary function testing apparatus usually are not considered an important source of bacterial contamination of inhaled gas (*327*). However, because of concern about possible carry-over of bacterial aerosols from an infectious patient-user of the apparatus to the next patient (*246,328*), placement of bacterial filters (i.e., that remove exhaled bacteria) between the patient and the testing equipment has been advocated (*246,329*). More studies are needed to evaluate the need for and efficacy of these filters in preventing nosocomial pneumonia (*330*).

b. *Tubing, rebreathing valves, and mouthpieces*. Tubing, connectors, rebreathing valves, and mouthpieces could become contaminated with patient secretions during use of the pulmonary function testing apparatus. Thus, these items should be cleaned and subjected to high-level disinfection or sterilization between uses on different patients.

F. Thoracoabdominal Surgical Procedures

Certain patients are at high risk for developing postoperative pulmonary complications, including pneumonia. These persons include those who are obese or are >70 years of age or who have chronic obstructive pulmonary disease (*331–334*). Abnormal results from pulmonary function tests (especially decreased maximum expiration flow rate), a history of smoking, the presence of tracheostomy or prolonged intubation, or protein depletion that can cause respiratory-muscle weakness are also risk factors (*62,68,136*). Patients who undergo surgery of the head, neck, thorax, or abdomen might have impairment of normal swallowing and respiratory clearance mechanisms as a result of instrumentation of the respiratory tract, anesthesia, or increased use of narcotics and sedatives (*332,335,336*). Patients who undergo upper abdominal surgery usually have diaphragmatic dysfunction that results in decreased functional residual capacity of the lungs, closure of airways, and atelectasis (*337,338*).

Interventions aimed at reducing the postoperative patient's risk for pneumonia have been developed (*339*). These include deep breathing exercises, chest physiotherapy, use of incentive spirometry, IPPB, and continuous positive airway pressure by face mask (*339–349*). Studies evaluating the relative efficacy of these modalities reported variable results and were difficult to compare because of differences in outcome variables assessed, patient populations studied, and study design (*339,341,342,348–350*). Nevertheless, many studies have reported that deep breathing exercises, use of incentive spirometry, and IPPB are advantageous maneuvers, especially in patients who had preoperative pulmonary dysfunction (*342,343,345,346,348–350*). In addition, control of pain that interferes with cough and deep breathing during the immediate postoperative period decreases the incidence of pulmonary complications after surgery. Several methods of controlling pain have been used; these include both intramuscular or intravenous (including patient-controlled) administration of analgesia and regional (e.g., epidural) analgesia (*351–358*).

G. Other Prophylactic Measures

1. *Vaccination of Patients*. Although pneumococci are not a major cause of nosocomial pneumonia, these organisms have been identified as etiologic agents of serious nosocomial pulmonary infection and bacteremia (*359–361*). The following factors place patients at high risk for complications from pneumococcal infections: age ≥65 years of age, chronic cardiovascular or pulmonary disease, diabetes mellitus, alcoholism, cirrhosis, cerebrospinal fluid leaks, immunosuppression, functional or anatomic asplenia, or infection with human immunodeficiency virus (HIV). Pneumococcal vaccine is effective in preventing pneumococcal disease (*362,363*). Because two thirds or more of patients with serious pneumococcal disease have been hospitalized at least once within the 5 years preceding their pneumococcal illness, offering pneumococcal vaccine in hospitals (e.g., at the time of

patient discharge) should contribute substantially to preventing the disease (*362,364*).

2. *Prophylaxis with Systemic Antimicrobial Agents*. The systemic administration of antimicrobials is commonly used to prevent nosocomial pneumonia—especially for patients who are receiving mechanical ventilation, are postoperative, and/or are critically ill (*365–367*). However, the efficacy of this practice is questionable, and superinfection, which is possible as a result of any antimicrobial therapy, could occur (*74,91,366–371*).

3. *Use of "Kinetic Beds" or Continuous Lateral Rotational Therapy (CLRT) for Immobilized Patients*. Use of kinetic beds, or CLRT, is a maneuver for prevention of pulmonary and other complications resulting from prolonged immobilization or bed rest, such as in patients with acute stroke, critical illness, head injury or traction, blunt chest trauma, and/or mechanically assisted ventilation (*372–377*). This procedure involves the use of a bed that turns continuously and slowly (from ≤40° for CLRT to ≥40° for kinetic therapy) along its longitudinal axis. Among the hypothesized benefits are improved drainage of secretions within the lungs and lower airways, increased tidal volume, and reduction of venous thrombosis with resultant pulmonary embolization (*378–381*). However, the efficacy of CLRT in preventing pneumonia needs further evaluation because studies have yielded variable results (*372–376*). In addition, the studies either involved small numbers of patients (*373*), lacked adequate randomization (*372*), had no clear definition of pneumonia (*372*), did not distinguish between community-acquired and nosocomial pneumonia (*373,377*), or did not adjust for possible confounding factors (e.g., mechanical ventilation, endotracheal intubation, nasogastric intubation, and enteral feeding) (*372*).

LEGIONNAIRES DISEASE

I. Epidemiology

Legionnaires disease is a multisystem illness, with pneumonia, caused by *Legionella* sp. (*382*). Since the etiologic agent of Legionnaires disease was identified, numerous nosocomial outbreaks of the disease have been reported, thus enabling researchers to study the epidemiology of epidemic legionellosis. In contrast, the epidemiology of sporadic (i.e., nonoutbreak-related) nosocomial Legionnaires disease has not been well defined. However, when one case is identified, the presence of additional cases should be suspected. Of 196 cases of nosocomial Legionnaires disease reported in England and Wales during 1980–1992, 69% occurred during 22 nosocomial outbreaks (defined as two or more cases occurring at a hospital during a 6-month period) (*383*). Nine percent of cases occurred >6 months before or after a hospital outbreak, and another 13% occurred in hospitals in which other sporadic cases, but no outbreaks, were identified. Only 9%

occurred at institutions in which no outbreaks or additional sporadic cases were identified.

In North America, the overall proportion of nosocomial pneumonias caused by *Legionella* sp. has not been determined, although the reported proportions from individual hospitals have ranged from zero to 14% (*384–386*). Because diagnostic tests for *Legionella* sp. infection are not performed routinely on all patients who have hospital-acquired pneumonia in most U.S. hospitals, this range probably underestimates the incidence of Legionnaires disease.

Legionella sp. are commonly found in various natural and man-made aquatic environments (*387,388*) and may enter hospital water systems in low or undetectable numbers (*389,390*). Cooling towers, evaporative condensers, heated potable-water–distribution systems within hospitals, and locally produced distilled water can provide a suitable environment for legionellae to multiply. Factors known to enhance colonization and amplification of legionellae in man-made water environments include temperatures of 25–42 C (*391–395*), stagnation (*396*), scale and sediment (*392*), and the presence of certain free-living aquatic amoebae that are capable of supporting intracellular growth of legionellae (*397,398*).

A person's risk for acquiring legionellosis after exposure to contaminated water depends on a number of factors, including the type and intensity of exposure and the person's health status (*399–401*). Persons who are severely immunosuppressed or who have chronic underlying illnesses, such as hematologic malignancy or end-stage renal disease, are at a markedly increased risk for legionellosis (*401–404*). Persons in the later stages of acquired immunodeficiency syndrome (AIDS) also are probably at increased risk for legionellosis, but data are limited because of infrequent testing of patients (*401*). Persons who have diabetes mellitus, chronic lung disease, or nonhematologic malignancy; those who smoke cigarettes; and the elderly are at moderately increased risk (*382*). Nosocomial Legionnaires disease also has been reported among patients in pediatric hospitals (*405,406*).

Underlying disease and advanced age are risk factors not only for acquiring Legionnaires disease but also for dying as a result of the illness. In a multivariate analysis of 3,524 cases reported to CDC from 1980 through 1989, immunosuppression, advanced age, end-stage renal disease, cancer, and nosocomial acquisition of disease were each independently associated with a fatal outcome (*401*). The mortality rate was 40% among 803 persons who had nosocomially acquired cases, compared with 20% among 2,721 persons who had community-acquired cases (*401*); this difference probably reflected the increased severity of underlying disease in hospitalized patients.

II. Diagnosis

The clinical spectrum of disease caused by *Legionella* sp. is broad and ranges from asymptomatic infection to rapidly progressive pneumonia. Legionnaires

disease cannot be distinguished clinically or radiographically from pneumonia caused by other agents (*407,408*), and evidence of infection with other respiratory pathogens does not exclude the possibility of concomitant *Legionella* sp. infection (*409–411*).

The diagnosis of legionellosis may be confirmed by any one of the following: culture isolation of *Legionella* from respiratory secretions or tissues, microscopic visualization of the bacterium in respiratory secretions or tissue by immunofluorescent microscopy, or, for legionellosis caused by *Legionella pneumophila* serogroup 1, detection of *L. pneumophila* serogroup-1 antigens in urine by radioimmunoassay, or observation of a four-fold rise in *L. pneumophila* serogroup-1 antibody titer to ≥1:128 in paired acute and convalescent serum specimens by use of an indirect immunofluorescent antibody (IFA) test (*412,413*). A single elevated antibody titer does not confirm a case of Legionnaires disease because IFA titers ≥1:256 are found in 1%–16% of healthy adults (*410,414–417*).

Because the above tests complement each other, performing each test when Legionnaires disease is suspected increases the probability of confirming the diagnosis (*418*). However, because none of the laboratory tests is 100% sensitive, the diagnosis of legionellosis is not excluded even if one or more of the tests are negative (*413,418*). Of the available tests, the most specific is culture isolation of *Legionella* sp. from any respiratory tract specimen (*419,420*).

III. Modes of Transmission

Inhalation of aerosols of water contaminated with *Legionella* sp. might be the primary mechanism by which these organisms enter a patient's respiratory tract (*382*). In several hospital outbreaks, patients were considered to be infected through exposure to contaminated aerosols generated by cooling towers, showers, faucets, respiratory therapy equipment, and room-air humidifiers (*11,241,258, 421–427*). In other studies, aspiration of contaminated potable water or pharyngeal colonizers was proposed as the mode of transmission to certain patients (*425,428–430*). However, person-to-person transmission has not been observed.

IV. Definition of Nosocomial Legionnaires Disease

The incubation period for Legionnaires disease is usually 2–10 days (*431*); thus, for the purposes of this document and the accompanying HICPAC recommendations, laboratory-confirmed legionellosis that occurs in a patient who has been hospitalized continuously for ≥10 days before the onset of illness is considered a **definite** case of nosocomial Legionnaires disease, and laboratory-confirmed infection that occurs 2–9 days after hospital admission is a **possible** case of the disease.

V. Prevention and Control Measures

A. Prevention of Legionnaires Disease in Hospitals with No Identified Cases (Primary Prevention)

Prevention strategies in health-care facilities in which no cases of nosocomial legionellosis have been identified have differed depending on the immunologic status of the patients, the design and construction of the facility, the resources available for implementing prevention strategies, and state and local regulations.

At least two strategies are practiced with regard to the most appropriate and cost-effective means of preventing nosocomial legionellosis, especially in hospitals in which no cases or only sporadic cases of the illness have been detected. However, a study comparing the cost-benefit ratios of these strategies has not been conducted.

The first approach is based on periodic, routine culturing of water samples from the hospital's potable water system for the purpose of detecting *Legionella* sp. (*432,433*). When ≥30% of the samples obtained are culture-positive for *Legionella* sp., the hospital's potable water system is decontaminated (*433*), and diagnostic laboratory tests for legionellosis are made available to clinicians in the hospital's microbiology department so that active surveillance for cases can be implemented (*433,434*). This approach is based on the premise that no cases of nosocomial legionellosis can occur if *Legionella* sp. is not present in the potable water system, and, conversely, if *Legionella* sp. are cultured from the water, cases of nosocomial legionellosis could occur (*428,435*). Proponents of this strategy indicate that when physicians are informed that the potable water system of the hospital is culture-positive for *Legionella* sp., they are more inclined to conduct the necessary tests for legionellosis (*434*). A potential advantage of using this approach in hospitals in which no cases of nosocomial legionellosis have occurred is that routinely culturing a limited number of water samples is less costly than routinely performing laboratory diagnostic testing for all patients who have nosocomial pneumonia.

The main argument against this approach is that, in the absence of cases, the relationship between the results of water cultures and the risk for legionellosis remains undefined. The bacterium has been frequently present in water systems of buildings (*436*), often without being associated with known cases of disease (*271,385,437,438*). In a study of 84 hospitals in Quebec, 68% of the water systems were found to be colonized with *Legionella* sp., and 26% were colonized at >30% of sites sampled; however, cases of Legionnaires disease were reported rarely from these hospitals (*271*). Similarly, at one hospital in which active surveillance for legionellosis and environmental culturing for *Legionella* sp. were done, no cases of legionellosis occurred in a urology ward during a 3.5-month period when 70% of water samples from the ward were culture-positive for *L. pneumophila* serogroup 1 (*385*). Interpretation of the

results of routinely culturing the water might be confounded by differing results among the sites sampled within a single water system and by fluctuations in the concentration of *Legionella* sp. at the same site (*439,440*). In addition, the risk for illness after exposure to a given source might be influenced by a number of factors other than the presence or concentration of organisms; these factors include the degree to which contaminated water is aerosolized into respirable droplets, the proximity of the infectious aerosol to the potential host, the susceptibility of the host, and the virulence properties of the contaminating strain (*441–443*). Thus, data are insufficient to assign a level of risk for disease even on the basis of the number of colony-forming units detected in samples from the hospital environment. By routinely culturing water samples, many hospital administrators will have to initiate water-decontamination programs if *Legionella* sp. are identified. Because of this problem, routine monitoring of water from the hospital's potable water system and from aerosol-producing devices is not widely recommended (*444*).

The second approach to preventing and controlling nosocomial legionellosis involves a) maintaining a high index of suspicion for legionellosis and appropriately using diagnostic tests for legionellosis in patients who have nosocomial pneumonia and who are at high risk for developing the disease and dying from the infection (*385,445*), b) initiating an investigation for a hospital source of *Legionella* sp. upon identification of one case of definite or two cases of possible nosocomial Legionnaires disease, and c) routinely maintaining cooling towers and using only sterile water for the filling and terminal rinsing of nebulization devices.

Measures used in hospitals in which cases of nosocomial legionellosis have been identified include either a) routine maintenance of potable water at ≥50 C or <20 C at the tap or b) chlorination of heated water to achieve 1–2 mg/L of free residual chlorine at the tap, especially in areas where immunosuppressed and other high-risk patients are located (*385,428,439,446–449*). However, the cost-benefit ratio of such measures in hospitals in which no cases of legionellosis have been identified needs additional evaluation.

B. Prevention of Legionnaires Disease in Hospitals with Identified Cases (Secondary Prevention)

The indications for a full-scale environmental investigation to search for and subsequently decontaminate identified sources of *Legionella* sp. in hospital environments have not been clarified, and these indications probably differ depending on the hospital. In hospitals in which as few as one to three nosocomial cases are identified during a period of several months, intensified surveillance for Legionnaires disease has frequently identified numerous additional cases (*403,422,425,447*). This finding suggests the need for a low threshold for initiating an investigation after laboratory confirmation of cases of nosocomial legionellosis. However, when developing a strategy for responding to such an identification, infection-control personnel should consider

the level of risk for nosocomial acquisition of, and mortality from, *Legionella* sp. infection at their particular hospital.

An epidemiologic investigation conducted to determine the source of *Legionella* sp. involves several important steps. First, microbiologic and medical records should be reviewed. Second, active surveillance should be initiated to identify all recent or ongoing cases of legionellosis. Third, potential risk factors for infection (including environmental exposures such as showering or use of respiratory-therapy equipment) should be identified by creating a line listing of cases, analyzing the collected information (by time, place, and person), and comparing case-patients with appropriate controls. Fourth, water samples should be collected from environmental sources implicated by the epidemiologic investigation and from other potential sources of aerosolized water. Fifth, subtype-matching between legionellae isolated from patients and environmental samples should be conducted (*427,450–452*). This last step can be crucial in supporting epidemiologic evidence of a link between human illness and a specific source (*453*).

In some hospitals in which the heated-water system was identified as the source of the organism, the system was decontaminated by pulse (one-time) thermal disinfection or superheating (i.e., flushing each distal outlet of the hot-water system for at least 5 minutes with water at ≥65 C) and hyperchlorination (flushing all outlets of the hot-water system with water containing ≥10 mg/L of free residual chlorine) (*449,454–456*). After either of these procedures, most hospitals either a) maintain heated water at ≥50 C or <20 C at the tap or b) chlorinate heated water to achieve 1–2 mg/L of free residual chlorine at the tap (*385,428,439,446–449*). Additional measures (e.g., physical cleaning or replacement of hot-water storage tanks, water-heaters, faucets, and showerheads) may be required because scale and sediment might accumulate in this equipment and protect organisms from the biocidal effects of heat and chlorine (*392,449*). Alternative methods for controlling and eradicating legionellae in water systems (e.g., treating water with ozone, ultraviolet light, or heavy metal ions) have limited the growth of legionellae under laboratory and/or operating conditions (*457–462*). However, additional data are needed regarding the efficacy of these methods before they can be considered standard precautions. Measures for decontaminating hospital cooling towers have been published previously (*463*).

Additional preventive measures have been used to protect severely immunocompromised patients. At one hospital, immunosuppressed patients were restricted from taking showers, and, for these patients, only sterile water was used for drinking or flushing nasogastric tubes (*429*). In another hospital, a combined approach consisting of continuous heating, particulate filtration, ultraviolet treatment, and monthly pulse hyperchlorination of the water supply to the bone-marrow transplant unit was used to decrease the incidence of Legionnaires disease (*458*).

The decision to search for hospital environmental sources of *Legionella* sp. and the choice of procedures to use to eradicate such contamination should take into account the type of patient population served by the hospital. Furthermore, decision makers should consider a) the high cost of an environmental investigation and of instituting control measures to eradicate *Legionella* sp. from sources in the hospital (*464,465*) and b) the differential risk, based on host factors, for acquiring nosocomial legionellosis and of having severe and fatal infection with the microorganism.

ASPERGILLOSIS

I. Epidemiology

Aspergillus sp. are ubiquitous fungi that commonly occur in soil, water, and decaying vegetation. *Aspergillus* sp. have been cultured from unfiltered air, ventilation systems, contaminated dust dislodged during hospital renovation and construction, horizontal surfaces, food, and ornamental plants (*466*).

Aspergillus fumigatus and *Aspergillus flavus* are the most frequently isolated *Aspergillus* sp. in patients who have laboratory-confirmed aspergillosis (*467*). Nosocomial aspergillosis has been recognized increasingly as a cause of severe illness and mortality in highly immunocompromised patients (e.g., patients undergoing chemotherapy and/or organ transplantation, including bone-marrow transplantation for hematologic and other malignant neoplasms) (*468–472*).

The most important nosocomial infection caused by *Aspergillus* sp. is pneumonia (*473,474*). Hospital outbreaks of pulmonary aspergillosis have occurred primarily in granulocytopenic patients, especially those in bone-marrow transplant units (*473–480*). Although invasive aspergillosis has been reported in recipients of solid-organ (e.g., heart and kidney) transplants (*481–485*), the incidence of *Aspergillus* sp. infections in these patients has been lower than in recipients of bone-marrow transplants, probably because granulocytopenia is less severe in solid-organ transplant recipients and the use of corticosteroids, especially in kidney transplant recipients, has decreased with the introduction of cyclosporine (*483,486*). The efficacy of infection-control measures, such as provision of protected environments and prophylaxis with antifungal agents, in preventing aspergillosis in solid-organ transplant recipients has not been well evaluated (*483,484,486,487*). In one study of heart-transplant recipients, using only protective isolation of patients did not prevent fungal infections (*488*).

The reported attributable mortality from invasive pulmonary aspergillosis has differed depending on the patient population studied. Rates have been as high as 95% in recipients of allogeneic bone-marrow transplants and patients who have aplastic anemia, compared with rates of 13%–80% in leukemic patients (*489–491*).

II. Pathogenesis

In contrast to most bacterial pneumonias, the primary route of acquiring *Aspergillus* sp. infection is by inhalation of the fungal spores. In severely immunocompromised patients, primary *Aspergillus* sp. pneumonia results from invasion of local lung tissue (*467,474,492*). Subsequently, the fungus might disseminate via the bloodstream to involve multiple other deep organs (*467,474,493*). A role for nasopharyngeal colonization with *Aspergillus* sp., as an intermediate step before invasive pulmonary disease, has been proposed but remains to be elucidated (*494–496*). Conversely, colonization of the lower respiratory tract by *Aspergillus* sp. has predisposed patients, especially those with preexisting lung disease (e.g., chronic obstructive lung disease, cystic fibrosis, or inactive tuberculosis), to invasive pulmonary and/or disseminated infection (*467,474,497*).

III. Diagnosis

Diagnosing pneumonia caused by *Aspergillus* sp. is often difficult without performing invasive procedures. Although bronchoalveolar lavage has been a useful screening test (*498–500*), lung biopsy is still considered the most reliable technique (*501*). Histopathologic demonstration of tissue invasion by fungal hyphae has been required in addition to isolation of *Aspergillus* sp. from respiratory tract secretions because the latter, by itself, may indicate colonization (*502*). However, when *Aspergillus* sp. is grown from the sputum of a febrile, granulocytopenic patient who has a new pulmonary infiltrate, it is highly likely that the patient has pulmonary aspergillosis (*495,503*). Routine blood cultures are remarkably insensitive for detecting *Aspergillus* sp. (*504*), and systemic antibody responses in immunocompromised patients are probably unreliable indicators of infection (*505–507*). Antigen-based serologic assays are being developed in an attempt to allow for the rapid and specific diagnosis of *Aspergillus* sp. infections; however, the clinical usefulness of such assays has not been determined (*508,509*).

IV. Risk Factors and Control Measures

The primary risk factor for invasive aspergillosis is severe and prolonged granulocytopenia, both disease- and therapy-induced (*510*). Because bone-marrow–transplant recipients experience the most severe degree of granulocytopenia, they probably constitute the population at highest risk for developing invasive aspergillosis (*490,511*). The tendency of bone-marrow–transplant recipients to contract severe granulocytopenia (i.e., <1,000 polymorphonuclears/μL) is associated with the type of graft they receive. Although both autologous and allogeneic bone-marrow–transplant recipients are severely granulocytopenic for up to 4 weeks after the transplant procedure, acute or chronic graft-versus-host disease also could develop in allogeneic-transplant recipients. The latter might occur up to several months after the procedure, and the disease and/or its therapy (which often includes high doses of corticosteroids, cyclosporine, and other immunosuppressive agents) might result in severe granulocytopenia. Consequently, in

developing strategies to prevent invasive *Aspergillus* sp. infection in bone-marrow–transplant recipients, infection-control personnel should consider exposures of the patient to the fungus both during and subsequent to the immediate post-transplantation period. After hospital discharge, patients (especially allogeneic-transplant recipients) might continue to manifest severe granulocytopenia and, therefore, are susceptible to fungal exposures at home and in ambulatory-care settings. To help address the problem of invasive aspergillosis in bone-marrow–transplant recipients, various studies are in progress to evaluate newer methods of a) enhancing host resistance to invasive fungal (and other) infections and b) eliminating or suppressing respiratory fungal colonization of the upper respiratory tract. These methods include, respectively, the use of granulocyte-colony–stimulating factors and intranasal application of amphotericin B or oral or systemic antifungal drug prophylaxis (*466,512–515*). For solid-organ transplant recipients, risk factors for invasive aspergillosis have not been studied as extensively. In one study of liver-transplant recipients, risk factors for invasive infection with *Aspergillus* sp. included preoperative and postoperative receipt of steroids and antimicrobial agents and prolonged duration of transplant surgery (*516*).

The presence of aspergilli in the hospital environment is the most important extrinsic risk factor for opportunistic invasive *Aspergillus* sp. infection (*517,518*). Environmental disturbances caused by construction and/or renovation activities in and around hospitals markedly increase the airborne *Aspergillus* sp. spore counts in such hospitals and have been associated with nosocomial aspergillosis (*476,478,479,519–522*). Aspergillosis in immunosuppressed patients also has been associated with other hospital environmental reservoirs. Such reservoirs include contaminated fireproofing material, damp wood, and bird droppings in air ducts (*478,523,524*).

A single case of nosocomial *Aspergillus* sp. pneumonia is often difficult to link to a specific environmental exposure. However, additional cases may remain undetected without an active search that includes an intensive retrospective review of microbiologic, histopathologic, and postmortem records; notification of clinicians caring for high-risk patients; and establishment of a system for prospective surveillance for additional cases. When additional cases are detected, the likelihood is increased that a hospital environmental source of *Aspergillus* sp. can be identified (*476,478,519–524*). Previous investigations have demonstrated the importance of construction activities and/or fungal contamination of hospital air-handling systems as major sources for outbreaks (*473,476,478,519–523*). New molecular typing techniques (i.e., karyotyping [*525*] and DNA endonuclease profiling, which is now available for *A. fumigatus* [*526*]) may substantially aid in identifying the source of an outbreak.

Outbreaks of invasive aspergillosis reinforce the importance of maintaining an environment as free as possible of *Aspergillus* sp. spores for patients who have severe granulocytopenia. To achieve this goal, specialized services in many large hospitals—particularly bone-marrow transplant services—have installed "protected environments" for the care of their high-risk, severely granulocytopenic

patients and have increased their vigilance during hospital construction and routine maintenance of hospital air-filtration and ventilation systems to prevent exposing high-risk patients to bursts of fungal spores (*476,478,519–523,527–532*).

Although the exact configuration and specifications of the protected environments might differ between hospitals, such patient-care areas are built to minimize fungal spore counts in air by maintaining a) filtration of incoming air by using central or point-of-use high-efficiency particulate air (HEPA) filters that are capable of removing 99.97% of particles ≥0.3 μm in diameter; b) directed room airflow (i.e., from intake 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 relative to the corridor; d) well-sealed rooms; and e) high rates of room-air changes (range: 15 to >400 per hour), although air-change rates at the higher levels might pose problems of patient comfort (*473,528–530,532–534*). The oldest and most studied protected environment is a room with laminar airflow. Such an environment consists of a bank of HEPA filters along an entire wall of the room; air is pumped by blowers through these filters and into the room at a uniform velocity (90 ± 20 feet/minute), forcing the air to move in a laminar, or at least unidirectional, pattern (*535*). The air usually exits at the opposite end of the room, and ultra-high air-change rates (i.e., 100–400 air changes per hour) are achieved (*473,527*). The net effects are essentially sterile air in the room, minimal air turbulence, minimal opportunity for microorganism build-up, and a consistently clean environment (*473*).

The laminar-airflow system is effective in decreasing or eliminating the risk for nosocomial aspergillosis in high-risk patients (*473,528,532,534*). However, such a system is costly to install and maintain. Less expensive alternative systems with lower air-change rates (i.e., 10–15 air changes per hour) have been used in some hospitals (*529,530,536*). However, studies comparing the efficacy of these alternative systems with laminar-airflow rooms in eliminating *Aspergillus* sp. spores and preventing nosocomial aspergillosis are limited. One hospital that employed cross-flow ventilation, point-of-use HEPA filters, and 15 air changes per hour reported that cases of nosocomial aspergillosis had occurred in patients housed in these rooms, although this rate was low (i.e., 3.4%) (*530,536*). However, these infections had been caused by *A. flavus,* a species that was not cultured from the room air, suggesting that the patients were probably exposed to fungal spores when they were allowed outside their rooms (*530*).

Copper-8-quinolinolate was used on environmental surfaces contaminated with *Aspergillus* sp. to control one reported outbreak of aspergillosis (*537*), and it has been incorporated in the fireproofing material of a newly constructed hospital to help decrease the environmental spore burden (*530*); however, its general applicability has not been established.

VIRAL PNEUMONIAS

Viruses can be an important and often unappreciated cause of nosocomial pneumonia (*538–540*). In one prospective study of endemic nosocomial infections, approximately

20% of pneumonia cases resulted from viral infections (*539*). Although the early diagnosis and treatment of viral pneumonia infections have been possible in recent years (*541–544*), many hospitalized patients remain at high risk for developing severe and sometimes fatal viral pneumonia (*538,545–552*). These data and reports of well-documented outbreaks involving nosocomial viral transmission (*553–556*) indicate that measures to prevent viral transmission should be instituted.

Nosocomial respiratory viral infections a) usually follow community outbreaks that occur during a particular period every year (*555,557–560*), b) confer only short-term immunity (*561*), c) affect both healthy and ill persons (*547,548,554,562–564*), and d) have exogenous sources. A number of viruses—including adenoviruses, influenza virus, measles virus, parainfluenza viruses, RSV, rhinoviruses, and varicella-zoster virus—can cause nosocomial pneumonia (*548,555,556,565–571,572*); however, adenoviruses, influenza viruses, parainfluenza viruses, and RSV reportedly have accounted for most (70%) nosocomial pneumonias caused by viruses (*573*).

Influenza and RSV infections contribute substantially to the morbidity and mortality associated with viral pneumonia, and the epidemiology of both viral infections has been well researched; for these reasons, this section concerning viral pneumonias focuses on the principles of, and approaches to, the control of these two types of infection. Recommendations for preventing nosocomial pneumonia caused by infection with other viral pathogens were published previously (*224*).

RSV INFECTION

I. Epidemiology

RSV infection is most common during infancy and early childhood, but it can also occur in adults (*562,565,574,575*). Infection usually causes mild or moderately severe upper respiratory illness. However, both life-threatening pneumonia and bronchiolitis have occurred in immunocompromised patients, the elderly, and children who have chronic cardiac and pulmonary disease (*547,549,564,565, 576,577*).

Recent surveillance of 10 U.S. hospital laboratories in which cultures for RSV are performed suggests that community outbreaks occur on a seasonal basis from December through March; these outbreaks last 3–5 months and are associated with an increased number of hospitalizations and deaths among infants and young children (*578*). During community outbreaks of RSV, children who have respiratory symptoms at the time of hospital admission are often reservoirs for RSV (*553,555*).

II. Diagnosis

The clinical characteristics of RSV infection, especially in neonates, are often indistinguishable from those of other viral respiratory tract infections (*565,566*). Culture of RSV from respiratory secretions is the standard for diagnosis. Rapid

antigen-detection kits that use direct immunofluorescence or enzyme-linked immunosorbent assays can provide results within hours. The benefit of using these tests to identify infected patients depends on the sensitivity and specificity of the test. The reported sensitivity and specificity of RSV enzyme immunoassays vary between 80% and 95% and may be even lower in actual practice (*579–582*). In general, once laboratory-confirmed cases of RSV infection are identified in a hospital, a presumptive diagnosis of RSV infection in subsequent cases with manifestations suggestive of RSV infection may be acceptable for infection-control purposes.

III. Modes of Transmission

RSV is present in large numbers in the respiratory secretions of symptomatic persons infected with the virus, and it can be transmitted directly via large droplets during close contact with such persons or indirectly via RSV-contaminated hands or fomites (*553,583,584*). The portal of entry is usually the conjunctiva or the nasal mucosa (*585*). Inoculation by RSV-contaminated hands is the usual way of depositing the virus onto the eyes or nose (*553,583–585*). Hands can become contaminated by handling either the respiratory secretions of infected persons or contaminated fomites (*583,584*).

In nosocomial RSV outbreaks for which the viral isolates were typed, more than one strain of RSV often was identified (*554,563,586*), suggesting multiple sources of the virus. Potential sources include patients, HCWs, and visitors. Because infected infants shed large amounts of virus in their respiratory secretions and can easily contaminate their immediate surroundings, they are a major reservoir for RSV (*587*). HCWs might become infected after exposure in the community (*588*) or in the hospital and subsequently transmit infection to patients, other HCWs, or visitors (*566,589*).

IV. Control Measures

Different combinations of control measures, ranging from the simple to the complex, have been effective in varying degrees in preventing and controlling nosocomial RSV infection (*226,589–596*). Successful programs have shared two common elements: implementation of contact-isolation precautions and compliance with these precautions by HCWs. In theory, strict compliance with handwashing recommendations could prevent most nosocomial RSV infections; however, studies have indicated that such compliance among HCWs is poor (*221,222*). Thus, other preventive measures are usually necessary to prevent RSV infection.

The wearing of gloves and gowns has been associated with decreased incidence of nosocomial RSV (*226*). The wearing of gloves has helped decrease transmission of RSV, probably because the gloves remind HCWs to comply with handwashing and other precautions and deter them from touching their eyes or nose. However, the benefits derived from wearing gloves are offset if the gloves are not

changed after contact with an infected patient or with contaminated fomites and if hands are not washed adequately after glove removal (*229*). The wearing of both gloves and gowns during contact with RSV-infected infants or their immediate environment has been successful in preventing infection (*226*). In addition, the use of eye-nose goggles rather than masks has protected HCWs from infection; however, eye-nose goggles are not widely available and are inconvenient to wear (*593,597*).

Additional measures may be indicated to control ongoing nosocomial transmission of RSV or to prevent transmission to patients at high risk for serious complications resulting from the infection (e.g., patients whose cardiac, pulmonary, or immune systems are compromised). The following additional control measures have been used in various combinations: a) using private rooms for infected patients OR cohorting infected patients, with or without preadmission screening by rapid laboratory diagnostic tests; b) cohorting HCWs; c) excluding HCWs who have symptoms of upper respiratory tract infection from caring for uninfected patients at high risk for severe or fatal RSV infection (e.g., infants); d) limiting visitors; and e) postponing admission of patients at high risk for complications from RSV infection (*224,590,592,594,596*). Although the exact role of each of these measures has not been determined, their use for controlling RSV outbreaks seems prudent.

INFLUENZA

I. Epidemiology

Pneumonia that occurs in patients who have influenza can be caused by the influenza virus, a secondary bacterial infection, or a combination of both (*598–600*). Influenza-associated pneumonia can occur in any person but is more common in infants and young children, in persons >65 years of age, and in persons of any age who are immunosuppressed or have certain chronic medical conditions (e.g., severe underlying heart or lung disease) (*575,601–603*).

Influenza typically occurs on a seasonal basis during December–April; during this period, peak influenza activity in an affected community usually lasts 6–8 weeks (*604,605*). Nosocomial outbreaks can occur in a community affected by an influenza epidemic; these outbreaks are often characterized by abrupt onset and rapid transmission (*606–608*). Most reported institutional outbreaks of influenza have occurred in nursing homes; however, hospital outbreaks in pediatric and chronic-care wards and in medical and neonatal intensive-care units have been reported (*556,609–612*).

Influenza is believed to be spread from person to person by a) direct inhalation of droplet nuclei or small-particle aerosols or b) direct deposition of virus-laden large droplets onto the mucosal surfaces of the upper respiratory tract of a person during close contact with an infected person (*613–616*). The extent to which

transmission might occur by contact with virus-contaminated hands or fomites is unknown; however, such contact is not the primary mode of transmission (*617*).

The most important reservoirs of influenza virus are infected persons. Although the period of greatest communicability is during the first 3 days of illness, the virus can be shed both before the onset of symptoms and for ≥7 days afterward (*556,604,618*).

II. Diagnosis

Influenza is clinically indistinguishable from other febrile respiratory illnesses; however, during outbreaks with laboratory-confirmed cases, a presumptive diagnosis of the infection can be made for illnesses that have similar manifestations (*619*). Historically, diagnosis of influenza was made by virus isolation from nasopharyngeal secretions or by serologic conversion, but recently developed rapid diagnostic tests that are similar to culture in sensitivity and specificity now enable early diagnosis and treatment of cases and provide a basis for prompt initiation of antiviral prophylaxis as part of outbreak control (*620–625*).

III. Prevention and Control of Influenza

The most effective measure for reducing the impact of influenza is the vaccination of persons at high risk for complications of the infection before the influenza season begins each year. High-risk persons include persons 6 months–18 years of age who are receiving long-term aspirin therapy and persons who either a) are ≥65 years of age; b) are in long-term–care units; or c) have either chronic disorders of the pulmonary or cardiovascular systems, diabetes mellitus, renal dysfunction, hemoglobinopathies, or immunosuppression (*611,626–628*). Patients who have musculoskeletal disorders that impede adequate respiration also may be at high risk for complications resulting from influenza. When high vaccination rates are achieved in closed or semi-closed settings, the risk for outbreaks is reduced because of induction of herd immunity (*629,630*).

When an institutional outbreak is caused by influenza type A, antiviral agents can be used both for treatment of ill persons and as prophylaxis for others (*631*). Two related antiviral agents, amantadine hydrochloride and rimantadine hydrochloride, are effective against influenza type A but not against influenza type B (*543,632–634*). These agents can be used in the following ways to prevent illness caused by influenza A virus: a) as short-term prophylaxis for high-risk persons after late vaccination; b) as prophylaxis for persons for whom vaccination is contraindicated; c) as prophylaxis for immunocompromised persons who might not produce protective levels of antibody in response to vaccination; d) as prophylaxis for unvaccinated HCWs who provide care to patients at high risk for infection, either for the duration of influenza activity in the community or until immunity develops after vaccination; and e) as prophylaxis when vaccine strains do not closely match the epidemic virus strain (*631*).

Amantadine has been available in the United States for many years; rimantadine has been approved for use since 1993. Both drugs protect against all naturally occurring strains of influenza A virus; thus, antigenic changes in the virus that might reduce vaccine efficacy do not alter the effectiveness of amantadine or rimantadine. Both drugs are 70%–90% effective in preventing illness if administered before exposure to influenza A virus (*632,635*). In addition, they can reduce the severity and duration of illness caused by influenza A virus if administered within 24–48 hours after onset of symptoms (*636,637*). These drugs can limit nosocomial spread of influenza type A if they are administered to all or most patients when influenza type A illnesses begin in a facility (*609,638,639*).

Compared with rimantadine, amantadine has been associated with a higher incidence of adverse central nervous system (CNS) reactions (e.g., mild and transitory nervousness, insomnia, impaired concentration, mood changes, and lightheadedness). These symptoms have been reported in 5%–10% of healthy young adults receiving 200 mg of amantadine per day (*543,632*). In the elderly, CNS side effects may be more severe; in addition, dizziness and ataxia occur more frequently among persons in this age group than among younger persons (*640,641*). Dose reductions of both amantadine and rimantadine are recommended for certain patients, such as persons ≥65 years of age and/or those who have renal insufficiency. The drug package inserts for amantadine and rimantadine contain important information regarding administration of these drugs. Guidelines for the use of amantadine and rimantadine and considerations for the selection of these drugs were published previously by the Advisory Committee on Immunization Practices (ACIP) (*631*).

The emergence of amantadine- and rimantadine-resistant strains of influenza A virus has been observed in persons who have received these drugs for treatment of the infection (*642,643*). Because of the potential risk for transmitting resistant viral strains to contacts of persons receiving amantadine or rimantadine for treatment (*643,644*), infected persons taking either drug should avoid, as much as possible, contact with others during treatment and for 2 days after discontinuing treatment (*644,645*). This is particularly important if the contacts are uninfected high-risk persons (*644,646*).

The primary focus of efforts to prevent and control nosocomial influenza is the vaccination of high-risk patients and HCWs before the influenza season begins (*628,647,648*). The decision to use amantadine or rimantadine as an adjunct to vaccination in the prevention and control of nosocomial influenza is based partially on results of virologic and epidemiologic surveillance in the hospital and the community. When outbreaks of influenza type A occur in a hospital, and antiviral prophylaxis of high-risk persons and/or treatment of cases is undertaken, administration of amantadine or rimantadine should begin as early in the outbreak as possible to reduce transmission (*609,638,631*).

Measures other than vaccination and chemoprophylaxis have been recommended for controlling nosocomial influenza outbreaks. Because influenza can be transmitted during contact with an infected person, the following procedures have been recommended: observing contact-isolation precautions, placing

patients who have symptoms of influenza in private rooms, cohorting patients who have influenza-like illness, and wearing a mask when entering a room in which a person who has suspected or confirmed influenza is housed (*224*). Handwashing and the wearing of gloves and gowns by HCWs during the patient's symptomatic period also have been recommended; however, the exact role of these measures in preventing influenza transmission has not been determined (*224,608,649*). Although influenza can be transmitted via the airborne route, the efficacy of placing infected persons in rooms that have negative air pressure in relation to their immediate environment has not been assessed. In addition, this measure may be impractical during institutional outbreaks that occur during a community epidemic of influenza because many HCWs and newly admitted patients could be infected with the virus; thus, the hospital would face the logistical problem of accommodating all ill persons in rooms that have special ventilation. Although the effectiveness of the following measures has not been determined, their implementation could be considered during severe outbreaks: a) curtailment or elimination of elective admissions, both medical and surgical; b) restriction of cardiovascular and pulmonary surgery; c) restriction of hospital visitors, especially those who have acute respiratory illnesses; and d) restriction of HCWs who have an acute respiratory illness from the workplace (*649*).

Part II. Recommendations for Preventing Nosocomial Pneumonia

INTRODUCTION

These recommendations are presented in the following order based on the etiology of the infection: bacterial pneumonia, including Legionnaires disease; fungal pneumonia (i.e., aspergillosis); and virus-associated pneumonia (i.e., RSV and influenza infections). Each topic is subdivided according to the following general approaches for nosocomial infection control:

1. Staff education and infection surveillance;
2. Interruption of transmission of microorganisms by eradicating infecting microorganisms from their epidemiologically important reservoirs and/or preventing person-to-person transmission; and
3. Modifying host risk for infection.

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific evidence, theoretical rationale, applicability, and economic impact (*224,225,650–654*). However, the previous CDC system of categorizing recommendations has been modified as follows:

CATEGORY IA	Strongly recommended for all hospitals and strongly supported by well-designed experimental or epidemiologic studies.
CATEGORY IB	Strongly recommended for all hospitals and viewed as effective by experts in the field and a consensus of HICPAC. These recommendations are based on strong rationale and suggestive evidence, even though definitive scientific studies may not have been done.
CATEGORY II	Suggested for implementation in many hospitals. These recommendations may be supported by suggestive clinical or epidemiologic studies, a strong theoretical rationale, or definitive studies applicable to some but not all hospitals.
NO RECOMMENDATION; UNRESOLVED ISSUE	Practices for which insufficient evidence or consensus regarding efficacy exists.

BACTERIAL PNEUMONIA

I. Staff Education and Infection Surveillance

A. Staff education

Educate HCWs regarding nosocomial bacterial pneumonias and infection-control procedures used to prevent these pneumonias (*655–661*). *CATEGORY IA*

B. Surveillance

1. Conduct surveillance of bacterial pneumonia among ICU patients at high risk for nosocomial bacterial pneumonia (e.g., patients receiving mechanically assisted ventilation and selected postoperative patients) to determine trends and identify potential problems (*6,34,35,62,63,662–664*). Include data regarding the causative microorganisms and their antimicrobial susceptibility patterns (*2,3*). Express data as rates (e.g., number of infected patients or infections per 100 ICU days or per 1,000 ventilator-days) to facilitate intrahospital comparisons and determination of trends (*66,665–667*). *CATEGORY IA*

2. Do not **routinely** perform surveillance cultures of patients or of equipment or devices used for respiratory therapy, pulmonary-function testing, or delivery of inhalation anesthesia (*65,668,669*). *CATEGORY IA*

II. Interrupting Transmission of Microorganisms

A. Sterilization or disinfection and maintenance of equipment and devices

1. **General measures**

   a. Thoroughly clean all equipment and devices before sterilization or disinfection (*266,267,670*). *CATEGORY IA*

   b. Sterilize or use high-level disinfection for semicritical equipment or devices (i.e., items that come into direct or indirect contact with mucous membranes of the lower respiratory tract) (Appendix A). High-level disinfection can be achieved either by wet heat pasteurization at 76 C for 30 minutes or by using liquid chemical disinfectants approved as sterilants/ disinfectants by the Environmental Protection Agency and cleared for marketing for use on medical instruments by the Office of Device Evaluation, Center for Devices and Radiologic Health, Food and Drug Administration (*260,262,264,267,671*). Follow disinfection with appropriate rinsing, drying, and packaging, taking care not to contaminate the items in the process. *CATEGORY IB*

c. (1) Use sterile (not distilled, nonsterile) water for rinsing reusable semicritical equipment and devices used on the respiratory tract after they have been disinfected chemically (*241,249,250,258,269*). *CATEGORY IB*

(2) *No Recommendation* for using tap water (as an alternative to sterile water) to rinse reusable semicritical equipment and devices used on the respiratory tract after such items have been subjected to high-level disinfection, regardless of whether rinsing is followed by drying with or without the use of alcohol (*241,249,250,258,269,273,277*). *UNRESOLVED ISSUE*

d. Do not reprocess equipment or devices that are manufactured for a single use only, unless data indicate that reprocessing such items poses no threat to the patient, is cost-effective, and does not change the structural integrity or function of the equipment or device (*672,673*). *CATEGORY IB*

2. **Mechanical ventilators, breathing circuits, humidifiers, and nebulizers**

a. **Mechanical ventilators**

Do not routinely sterilize or disinfect the internal machinery of mechanical ventilators (*126,128,674*). *CATEGORY IA*

b. **Ventilator circuits with humidifiers**

(1) Do not routinely change more frequently than every 48 hours the breathing circuit, including tubing and exhalation valve, and the attached bubbling or wick humidifier of a ventilator that is being used on an individual patient (*34,283,288*). *CATEGORY IA*

(2) *No Recommendation* for the maximum length of time after which the breathing circuit and the attached bubbling or wick humidifier of a ventilator being used on a patient should be changed (*289*). *UNRESOLVED ISSUE*

(3) Sterilize reusable breathing circuits and bubbling or wick humidifiers or subject them to high-level disinfection between their uses on different patients (*259,260,262,264,267*). *CATEGORY IB*

(4) Periodically drain and discard any condensate that collects in the tubing of a mechanical ventilator, taking precautions not to allow condensate to drain toward the patient. Wash hands after performing the procedure or handling the fluid (*215,282,286*). *CATEGORY IB*

(5) *No Recommendation* for placing a filter or trap at the distal end of the expiratory-phase tubing of the breathing circuit to collect condensate (*247,282*). *UNRESOLVED ISSUE*

(6) Do not place bacterial filters between the humidifier reservoir and the inspiratory-phase tubing of the breathing circuit of a mechanical ventilator. *CATEGORY IB*

(7) **Humidifier fluids**

(a) Use sterile water to fill bubbling humidifiers (*132,241,249, 250, 286*). *CATEGORY II*

(b) Use sterile, distilled, or tap water to fill wick humidifiers (*249, 250,286*). *CATEGORY II*

(c) *No Recommendation* for preferential use of a closed, continuous-feed humidification system. *UNRESOLVED ISSUE*

c. **Ventilator breathing circuits with hygroscopic condenser-humidifiers or heat-moisture exchangers**

(1) *No Recommendation* for preferential use of hygroscopic condenser-humidifier or heat-moisture exchanger rather than a heated humidifier to prevent nosocomial pneumonia (*298–302*). *UNRESOLVED ISSUE*

(2) Change the hygroscopic condenser-humidifier or heat-moisture exchanger according to the manufacturer's recommendation and/or when evidence of gross contamination or mechanical dysfunction of the device is present (*298*). *CATEGORY IB*

(3) Do not routinely change the breathing circuit attached to a hygroscopic condenser-humidifier or heat-moisture exchanger while it is being used on a patient (*298,301*). *CATEGORY IB*

3. **Wall humidifiers**

a. Follow manufacturers' instructions for using and maintaining wall oxygen humidifiers unless data indicate that modifying the instructions poses no threat to the patient and is cost-effective (*675–679*). *CATEGORY IB*

b. Between uses on different patients, change the tubing, including any nasal prongs or mask, used to deliver oxygen from a wall outlet. *CATEGORY IB*

4. **Small-volume medication nebulizers: "in-line" and hand-held nebulizers**

a. (1) Between treatments on the same patient, disinfect, rinse with sterile water, or air-dry small-volume medication nebulizers (*242,258*). *CATEGORY IB*

(2) *No Recommendation* for using tap water as an alternative to sterile water when rinsing reusable small-volume medication nebulizers between treatments on the same patient (*242,258,273*). *UNRESOLVED ISSUE*

b. Between uses on different patients, replace nebulizers with those that have undergone sterilization or high-level disinfection (*126,128,129, 269,680*). *CATEGORY IB*

c. Use only sterile fluids for nebulization, and dispense these fluids aseptically (*238,241,249,250,258,269,304*). *CATEGORY IA*

d. If multi-dose medication vials are used, handle, dispense, and store them according to manufacturers' instructions (*238,304,680–682*). *CATEGORY IB*

5. **Large-volume nebulizers and mist tents**

a. Do not use large-volume room-air humidifiers that create aerosols (e.g., by Venturi principle, ultrasound, or spinning disk) and thus are actually nebulizers, unless they can be sterilized or subjected to high-level disinfection at least daily and filled only with sterile water (*239–241,252, 303,683*). *CATEGORY IA*

b. Sterilize large-volume nebulizers that are used for inhalation therapy (e.g., for tracheostomized patients) or subject them to high-level disinfection between uses on different patients and after every 24 hours of use on the same patient (*126,128,129*). *CATEGORY IB*

c. (1) Use mist-tent nebulizers and reservoirs that have undergone sterilization or high-level disinfection, and replace these items between uses on different patients (*684*). *CATEGORY IB*

(2) *No Recommendation* regarding the frequency of changing mist-tent nebulizers and reservoirs while such devices are being used on one patient. *UNRESOLVED ISSUE*

6. **Other devices used in association with respiratory therapy**

a. Between uses on different patients, sterilize or subject to high-level disinfection portable respirometers, oxygen sensors, and other respiratory devices used on multiple patients (*233,245*). *CATEGORY IB*

b. (1) Between uses on different patients, sterilize or subject to high-level disinfection reusable hand-powered resuscitation bags (e.g., Ambu bags) (*255,311–313*). *CATEGORY IA*

(2) *No Recommendation* regarding the frequency of changing hydrophobic filters placed on the connection port of resuscitation bags. *UNRESOLVED ISSUE*

7. **Anesthesia machines and breathing systems or patient circuits**

a. Do not routinely sterilize or disinfect the internal machinery of anesthesia equipment (*316*). *CATEGORY IA*

b. Clean and then sterilize or subject to high-level liquid chemical disinfection or pasteurization reusable components of the breathing system or patient circuit (e.g., tracheal tube or face mask, inspiratory and expiratory breathing tubing, y-piece, reservoir bag, humidifier, and humidifier tubing) between uses on different patients by following the device manufacturers' instructions for reprocessing such components (*260,264,267, 317,685*). *CATEGORY IB*

c. *No Recommendation* for the frequency of routinely cleaning and disinfecting unidirectional valves and carbon dioxide absorber chambers (*317–319*). *UNRESOLVED ISSUE*

d. Follow published guidelines and/or manufacturers' instructions regarding in-use maintenance, cleaning, and disinfection or sterilization of other components or attachments of the breathing system or patient circuit of anesthesia equipment (*317,318*). *CATEGORY IB*

e. Periodically drain and discard any condensate that collects in the tubing of a breathing circuit, taking precautions not to allow condensate to drain toward the patient. After performing the procedure or handling the fluid, wash hands with soap and water or with a waterless handwashing preparation (*218,219,686,687*). *CATEGORY IB*

f. *No Recommendation* for placing a bacterial filter in the breathing system or patient circuit of anesthesia equipment (*1,317,318,321–326,688*). *UNRESOLVED ISSUE*

8. **Pulmonary-function testing equipment**

a. Do not routinely sterilize or disinfect the internal machinery of pulmonary-function testing machines between uses on different patients (*327,328*). *CATEGORY II*

b. Sterilize or subject to high-level liquid-chemical disinfection or pasteurization reusable mouthpieces and tubing or connectors between uses on different patients, OR follow the device manufacturers' instructions for their reprocessing (*260,261,263–267*). *CATEGORY IB*

B. Interrupting person-to-person transmission of bacteria

1. **Handwashing**

Regardless of whether gloves are worn, wash hands after contact with mucous membranes, respiratory secretions, or objects contaminated with respiratory secretions. Regardless of whether gloves are worn, wash hands both before and after contact with a) a patient who has an endotracheal or tracheostomy tube in place and b) any respiratory device that is used on the patient (*210,212,218,219,231,689,690*). *CATEGORY IA*

2. **Barrier precautions**

   a. Wear gloves for handling respiratory secretions or objects contaminated with respiratory secretions of any patient (*226,227*). *CATEGORY IA*

   b. Change gloves and wash hands a) after contact with a patient; b) after handling respiratory secretions or objects contaminated with secretions from one patient and before contact with another patient, object, or environmental surface; and c) between contacts with a contaminated body site and the respiratory tract of, or respiratory device on, the same patient (*226,228–230*). *CATEGORY IA*

   c. Wear a gown if soiling with respiratory secretions from a patient is anticipated, and change the gown after such contact and before providing care to another patient (*226*). *CATEGORY IB*

3. **Care of patients who have a tracheostomy**

   a. Perform tracheostomy under sterile conditions. *CATEGORY IB*

   b. When changing a tracheostomy tube, use aseptic techniques and replace the tube with one that has undergone sterilization or high-level disinfection. *CATEGORY IB*

4. **Suctioning of respiratory tract secretions**

   a. *No Recommendation* for wearing sterile gloves rather than clean but nonsterile gloves when suctioning a patient's respiratory secretions. *UNRESOLVED ISSUE*

   b. If the open-suction system is employed, use a sterile single-use catheter. *CATEGORY II*

   c. Use only sterile fluid to remove secretions from the suction catheter if the catheter is to be used for re-entry into the patient's lower respiratory tract (*691*). *CATEGORY IB*

   d. *No Recommendation* for preferential use of the multiuse closed-system suction catheter or the single-use open-system catheter for prevention of pneumonia (*305–308,310*). *UNRESOLVED ISSUE*

   e. Change the entire length of suction-collection tubing between uses on different patients. *CATEGORY IB*

   f. Change suction-collection canisters between uses on different patients except when used in short-term–care units. *CATEGORY IB*

III. Modifying Host Risk for Infection

A. Precautions for preventing endogenous pneumonia

Discontinue enteral-tube feeding and remove devices such as endotracheal, tracheostomy, and/or enteral (i.e., orogastric, nasogastric, or jejunal) tubes from patients as soon as the clinical indications for these are resolved (*6,34,35,85–87,117,183,185,186,202,692*). *CATEGORY IB*

1. **Preventing aspiration associated with enteral feeding**

   a. If the maneuver is not contraindicated, elevate at an angle of 30°–45° the head of the bed of a patient at high risk for aspiration pneumonia (e.g., a patient receiving mechanically assisted ventilation and/or who has an enteral tube in place) (*74,185*). *CATEGORY IB*

   b. Routinely verify the appropriate placement of the feeding tube (*693–695*). *CATEGORY IB*

   c. Routinely assess the patient's intestinal motility (e.g., by auscultating for bowel sounds and measuring residual gastric volume or abdominal girth) and adjust the rate and volume of enteral feeding to avoid regurgitation (*692*). *CATEGORY IB*

   d. *No Recommendation* for the preferential use of small-bore tubes for enteral feeding (*694*). *UNRESOLVED ISSUE*

   e. *No Recommendation* for administering enteral feeding continuously or intermittently (*70,193,198*). *UNRESOLVED ISSUE*

   f. *No Recommendation* for preferentially placing the feeding tubes (e.g., jejunal tubes) distal to the pylorus (*199,200*). *UNRESOLVED ISSUE*

2. **Preventing aspiration associated with endotracheal intubation**

   a. *No Recommendation* for using orotracheal rather than nasotracheal tube to prevent nosocomial pneumonia (*696*). *UNRESOLVED ISSUE*

   b. *No Recommendation* for routinely using an endotracheal tube with a dorsal lumen above the endotracheal cuff to allow drainage (i.e., by suctioning) of tracheal secretions that accumulate in the patient's subglottic area (*206*). *UNRESOLVED ISSUE*

   c. Before deflating the cuff of an endotracheal tube in preparation for tube removal, or before moving the tube, ensure that secretions are cleared from above the tube cuff. *CATEGORY IB*

3. **Preventing gastric colonization**

   a. If stress-bleeding prophylaxis is needed for a patient receiving mechanically assisted ventilation, use an agent that does not raise the patient's gastric pH (*22,34,112,118,122,147–154*). *CATEGORY II*

b. *No Recommendation* for selective decontamination of a critically ill, mechanically ventilated, or ICU patient's digestive tract with oral and/or intravenous antimicrobials to prevent gram-negative bacillary (or *Candida* sp.) pneumonia (*155–180*). *UNRESOLVED ISSUE*

c. *No Recommendation* for routine acidification of gastric feedings to prevent nosocomial pneumonia (*181*). *UNRESOLVED ISSUE*

B. Preventing postoperative pneumonia

1. Instruct preoperative patients, especially those at high risk for contracting pneumonia, regarding frequent coughing, taking deep breaths, and ambulating as soon as medically indicated during the postoperative period (*346,348*). Patients at high risk include those who will receive anesthesia—especially those who will have an abdominal, thoracic, head, or neck operation—and those who have substantial pulmonary dysfunction (e.g., patients who have chronic obstructive lung disease, a musculoskeletal abnormality of the chest, or abnormal pulmonary function tests) (*331–334,337,338*). *CATEGORY IB*

2. Encourage postoperative patients to cough frequently, take deep breaths, move about the bed, and ambulate unless these actions are medically contraindicated (*345,346,348*). *CATEGORY IB*

3. Control pain that interferes with coughing and deep breathing during the immediate postoperative period by a) using systemic analgesia (*352,697*), including patient-controlled analgesia (*353–355*), with as little cough-suppressant effect as possible; b) providing appropriate support for abdominal wounds, such as tightly placing a pillow across the abdomen; or c) administering regional analgesia (e.g., epidural analgesia) (*356–358*). *CATEGORY IB*

4. Use an incentive spirometer or intermittent positive-pressure breathing equipment on patients at high risk for contracting postoperative pneumonia (*339,342,343,346,348,349*). (See Section III-B-1 above for definition of high-risk patients.) *CATEGORY II*

C. Other prophylactic procedures for pneumonia

1. **Vaccination of patients**

   Vaccinate patients at high risk for complications of pneumococcal infections with pneumococcal polysaccharide vaccine. Such patients include persons ages ≥65 years; adults who have chronic cardiovascular or pulmonary disease, diabetes mellitus, alcoholism, cirrhosis, or cerebrospinal fluid leaks; and children and adults who are immunosuppressed or who have functional or anatomic asplenia or HIV infection (*362–364*). *CATEGORY IA*

2. **Antimicrobial prophylaxis**

   Do not routinely administer systemic antimicrobial agents to prevent nosocomial pneumonia (*74,91,201,366–370,698*). *CATEGORY IA*

3. **Use of rotating "kinetic" beds or continuous lateral rotational therapy**

   No Recommendation for the routine use of kinetic beds or continuous lateral rotational therapy (i.e., placing the patient on a bed that turns intermittently or continuously on its longitudinal axis) for prevention of nosocomial pneumonia in patients in the ICU, critically ill patients, or patients immobilized by illness and/or trauma (*372–377,699*). *UNRESOLVED ISSUE*

PREVENTION AND CONTROL OF LEGIONNAIRES DISEASE

I. Staff Education and Infection Surveillance

A. Staff education

Educate a) physicians to heighten their suspicion for cases of nosocomial Legionnaires disease and to use appropriate methods for its diagnosis and b) other hospital personnel (i.e., patient-care, infection-control, and engineering personnel) about measures to control nosocomial legionellosis (*659–661*). *CATEGORY IA*

B. Surveillance

1. Establish mechanism(s) to provide clinicians with appropriate laboratory tests for the diagnosis of Legionnaires disease (*386,413-415,700*). *CATEGORY IA*

2. Maintain a high index of suspicion for the diagnosis of nosocomial Legionnaires disease, especially in patients who are at high risk for acquiring the disease. Such patients include those who are immunosuppressed (e.g., organ-transplant recipients, patients who have AIDS, and patients being treated with systemic steroids), those who are ≥65 years of age, and those who have a chronic underlying disease (e.g., diabetes mellitus, congestive heart failure, and chronic obstructive pulmonary disease) (*385,386,399,401–405,411*). *CATEGORY II*

3. *No Recommendation* for routinely culturing water systems for *Legionella* sp. (*271,385,428,432,434,435,437–439,454,701*). *UNRESOLVED ISSUE*

II. Interrupting Transmission of *Legionella* sp.

A. Primary prevention (preventing nosocomial Legionnaires disease when no cases have been documented)

1. Nebulization and other devices

a. (1) Use sterile (not distilled, nonsterile) water for rinsing nebulization devices and other semicritical respiratory-care equipment after such items have been cleaned and/or disinfected (*258,271,702*). *CATEGORY IB*

(2) *No Recommendation* for using tap water as an alternative to sterile water for rinsing reusable semicritical equipment and devices used on the respiratory tract after they have been subjected to high-level disinfection, regardless of whether rinsing is followed by drying with or without the use of alcohol. *UNRESOLVED ISSUE*

b. Use only sterile (not distilled, nonsterile) water to fill reservoirs of devices used for nebulization (*241,252,258,271,702*). *CATEGORY IA*

c. Do not use large-volume room-air humidifiers that create aerosols (e.g., by Venturi principle, ultrasound, or spinning disk), and thus are actually nebulizers, unless they can be sterilized or subjected to high-level disinfection daily and filled only with sterile water (*252,702*). *CATEGORY IA*

2. Cooling towers

a. When a new hospital building is constructed, place cooling tower(s) in such a way that the tower drift is directed away from the hospital's air-intake system and design the cooling towers such that the volume of aerosol drift is minimized (*421,703*). *CATEGORY IB*

b. For operational cooling towers, install drift eliminators, regularly use an effective biocide, maintain the tower according to the manufacturer's recommendations, and keep adequate maintenance records (Appendix D) (*421,463,704*). *CATEGORY IB*

3. Water-distribution system

a. *No Recommendation* for routinely maintaining potable water at the outlet at ≥50 C or <20 C, or chlorinating heated water to achieve 1–2 mg/L free residual chlorine at the tap (*385,428,439,446–449*). *UNRESOLVED ISSUE*

b. *No Recommendation* for treating water with ozone, ultraviolet light, or heavy-metal ions (*457,459–462,465*). *UNRESOLVED ISSUE*

B. Secondary prevention (response to identification of laboratory-confirmed nosocomial legionellosis)

When a single case of laboratory-confirmed, **definite** nosocomial Legionnaires disease is identified, OR if two or more cases of laboratory-confirmed, **possible** nosocomial Legionnaires disease occur during a 6-month period, the following procedures are recommended:

1. Contact the local or state health department or CDC if the disease is reportable in the state or if assistance is needed. *CATEGORY IB*

2. If a case is identified in a severely immunocompromised patient (e.g., an organ-transplant recipient) OR if severely immunocompromised patients are being treated in the hospital, conduct a combined epidemiologic and environmental investigation (as described in II-B-3-b-1 through II-B-5) to determine the source(s) of *Legionella* sp. *CATEGORY IB*

3. If severely immunocompromised patients are not being treated in the hopsital, conduct an epidemiologic investigation via a retrospective review of microbiologic, serologic, and postmortem data to identify previous cases, and begin an intensive prospective surveillance for additional cases of nosocomial Legionnaires disease. *CATEGORY IB*

   a. **If evidence of continued nosocomial transmission is not present,** continue the intensive prospective surveillance (as described in II-B-3) for at least 2 months after the date surveillance was initiated. *CATEGORY II*

   b. **If evidence of continued nosocomial transmission is present:**

      (1) Conduct an environmental investigation to determine the source(s) of *Legionella* sp. by collecting water samples from potential sources of aerosolized water, following the methods described in Appendix C, and saving and subtyping isolates of *Legionella* sp. obtained from patients and the environment (*241,258,421–427,450,452*). *CATEGORY IB*

      (2) If a source is not identified, continue surveillance for new cases for at least 2 months, and, depending on the scope of the outbreak, decide either to defer decontamination pending identification of the source(s) of *Legionella* sp. or proceed with decontamination of the hospital's water distribution system, with special attention to the specific hospital areas involved in the outbreak. *CATEGORY II*

      (3) If a source of infection is identified by epidemiologic and environmental investigation, promptly decontaminate it (*465*). *CATEGORY IB*

         (a) **If the heated-water system is implicated:**

            i. Decontaminate the heated-water system either by superheating (i.e., flushing for at least 5 minutes each distal outlet of the

system with water at ≥65 C) OR by hyperchlorination (i.e., flushing for at least 5 minutes all outlets of the system with water containing ≥10 mg/L of free residual chlorine) (*449,450,454,455*). Post warning signs at each outlet being flushed to prevent scald injury to patients, staff, or visitors. *CATEGORY IB*

ii. Depending on local and state regulations regarding potable water temperature in public buildings (*456*), in hospitals housing patients who are at high risk for acquiring nosocomial legionellosis (e.g., immunocompromised patients) either a) maintain potable water at the outlet at ≥50 C or <20 C or b) chlorinate heated water to achieve 1–2 mg/L of free residual chlorine at the tap (*385,428,439,446–449*) (Appendix B). *CATEGORY II*

iii. *No Recommendation* for treatment of water with ozone, ultraviolet light, or heavy-metal ions (*457,459,460,462*). *UNRESOLVED ISSUE*

iv. Clean hot-water storage tanks and water heaters to remove accumulated scale and sediment (*392*). *CATEGORY IB*

v. Restrict immunocompromised patients from taking showers, and use only sterile water for their oral consumption until *Legionella* sp. becomes undetectable by culture in the hospital water (*429*). *CATEGORY II*

(b) **If cooling towers or evaporative condensers are implicated,** decontaminate the cooling-tower system (Appendix D) (*463*). *CATEGORY IB*

(4) Assess the efficacy of implemented measures in reducing or eliminating *Legionella* sp. by collecting specimens for culture at 2-week intervals for 3 months. *CATEGORY II*

(a) If *Legionella* sp. are not detected in cultures during 3 months of monitoring at 2-week intervals, collect cultures monthly for another 3 months. *CATEGORY II*

(b) If *Legionella* sp. are detected in one or more cultures, reassess the implemented control measures, modify them accordingly, and repeat the decontamination procedures. Options for repeat decontamination include either the intensive use of the same technique used for initial decontamination or a combination of superheating and hyperchlorination. *CATEGORY II*

(5) Keep adequate records of all infection-control measures, including maintenance procedures, and of environmental test results for cooling towers and potable-water systems. *CATEGORY II*

PREVENTION AND CONTROL OF NOSOCOMIAL PULMONARY ASPERGILLOSIS

I. Staff Education and Infection Surveillance

A. Staff education

Educate HCWs about nosocomial pulmonary aspergillosis, especially with respect to immunocompromised patients, and about infection-control procedures used to reduce its occurrence (*659–661*). *CATEGORY IA*

B. Surveillance

1. Maintain a high index of suspicion for the diagnosis of nosocomial pulmonary aspergillosis in patients who are at high risk for the disease (i.e., patients who have prolonged, severe granulocytopenia [<1,000 polymorphonuclear cells/mm^3 for 2 weeks or <100 polymorphonuclear cells/mm^3 for 1 week], particularly bone-marrow–transplant recipients) (*510,511,705*). Patients who have received solid-organ transplants and patients who have hematologic malignancies and are receiving chemotherapy also are at high risk for acquiring the infection if they are severely granulocytopenic (*472,485,510,706*). *CATEGORY IB*

2. Maintain surveillance for cases of nosocomial pulmonary aspergillosis by periodically reviewing the hospital's microbiologic, histopathologic, and postmortem data. *CATEGORY IB*

3. *No Recommendation* for performing routine, periodic cultures of a) the nasopharynx of high-risk patients or b) devices, air samples, dust, ventilation ducts, and filters in rooms occupied by high-risk patients (*466,478,517,494,520–522*). *UNRESOLVED ISSUE*

II. Interrupting Transmission of *Aspergillus* sp. Spores

A. Planning new specialized-care units for patients at high risk for infection

1. When constructing new specialized-care units for patients at high risk for infection, ensure that patient rooms have adequate capacity to minimize fungal spore counts via maintenance of a) HEPA filtration, b) directed room airflow, c) positive air pressure in patients' rooms relative to the air pressure in the corridor, d) properly sealed rooms, and e) high rates of room-air changes (*473,528–530,533,537,707,708*). *CATEGORY IB*

   a. **Air filtration.** Install, either centrally or at the point of use (i.e., at the room-air intake site), HEPA filters that are 99.97% efficient in filtering particles ≥0.3 μm in diameter (*473,528–530,533,537,707,708*). *CATEGORY IB*

b. **Directed room airflow.** Place air-intake and exhaust ports such that room air comes in from one side of the room, flows across the patient's bed, and exits on the opposite side of the room (*529,530*). *CATEGORY IB*

c. **Well-sealed room.** Construct windows, doors, and intake and exhaust ports to achieve complete sealing of the room against air leaks (*529,530*). *CATEGORY IB*

d. **Room-air pressure.** Ensure that room-air pressure can be maintained continuously above that of the corridor (e.g., as can be demonstrated by performance of the smoke-tube test) unless contraindicated by clinical-care or infection-control considerations (*529,530*). *CATEGORY IB*

(1) To maintain positive room-air pressure in relation to the corridor, supply air to the room at a rate that is 10%–20% greater than the rate of air being exhausted from the room (*529,530*). *CATEGORY IB*

(2) For placement of patients who are at high risk for aspergillosis and who also have an infection (e.g., varicella or infectious tuberculosis) that necessitates negative room-air pressure in relation to the corridor, provide optimal conditions to prevent the spread of the airborne infection from and acquisition of aspergillosis by the patient (e.g., by providing anterooms with an independent exhaust) (*529*). *CATEGORY II*

e. **Room-air changes.** Ventilate the room to ensure ≥12 room-air changes per hour are maintained (*1,529,535,536*). *CATEGORY II*

2. *No Recommendation* for the preferential installation of a particular system, such as one with ultra-high air change rates (i.e., 100–400 air changes per hour) (e.g., laminar airflow), over other systems that meet the conditions in Sections II-A-1-a through II-A-1-e (*473,528–530,533,537,707,708*). *UNRESOLVED ISSUE*

3. Formulate hospital policies to minimize exposures of high-risk patients to potential sources of *Aspergillus* sp. (e.g., hospital construction and renovation, cleaning activities, carpets, food, potted plants, and flower arrangements) (*466,517,522,527,709–711*). *CATEGORY IB*

4. *No Recommendation* for prophylactic use of copper-8-quinolinolate biocide in fireproofing material (*466,477,530,537*). *UNRESOLVED ISSUE*

B. In existing facilities with no cases of nosocomial aspergillosis

1. Place patients who are at high risk for infection in a protected environment that meets the conditions described in Sections II-A-1-a through II-A-1-e (*473,517,528,537,707,708,712*). *CATEGORY IB*

2. Routinely inspect air-handling systems in hospital areas in which patients at high risk for infection are housed, maintain adequate air exchanges and pressure differentials, and eliminate air leakages. Coordinate repairs of the

system with the relocation of patients who are at high risk for infection to other hospital areas that have optimal air-handling capabilities (*466,478, 517*) . *CATEGORY IB*

3. Minimize the length of time that patients who are at high risk for infection are outside their rooms for diagnostic procedures and other activities; when such patients leave their rooms, require them to wear well-fitting masks capable of filtering *Aspergillus* sp. spores. *CATEGORY IB*

4. Prevent dust accumulation by damp-dusting horizontal surfaces on a daily basis, regularly cleaning ceiling tiles and air-duct grates when the rooms are not occupied by patients, and maintaining adequate seals on windows to prevent outside air from entering the room, especially in areas occupied by patients at high risk for aspergillosis (*517*). *CATEGORY IB*

5. Systematically review and coordinate infection-control strategies with personnel in charge of hospital engineering, maintenance, central supply and distribution, and catering (*466,522*). *CATEGORY IB*

6. When planning hospital construction and renovation activities, assess whether patients at high risk for aspergillosis are likely to be exposed to high ambient-air spore counts of *Aspergillus* sp. from construction and renovation sites, and, if so, develop a plan to prevent such exposures (*466,522*). *CATEGORY IB*

7. During construction or renovation activities:

   a. Construct barriers between patient-care and construction areas to prevent dust from entering patient-care areas; these barriers (e.g., plastic or drywall) should be impermeable to *Aspergillus* sp. (*67,478,521,522*). *CATEGORY IB*

   b. In construction/renovation areas inside the hospital, create and maintain negative air pressure relative to that in adjacent patient-care areas unless such a pressure differential is contraindicated (e.g., if patients in the adjacent patient-care areas have infectious tuberculosis) (*466,478,521,522, 537*). *CATEGORY II*

   c. Direct pedestrian traffic from construction areas away from patient-care areas to limit the opening and closing of doors or other barriers that might cause dust dispersion, entry of contaminated air, or tracking of dust into patient-care areas (*466,478,521,522*). *CATEGORY IB*

   d. Clean newly constructed areas before allowing patients to enter the areas (*466,522*). *CATEGORY IB*

8. Eliminate exposures of patients at high risk for aspergillosis to activities that might cause spores of *Aspergillus* sp. and other fungi to be aerosolized (e.g., floor or carpet vacuuming) (*466,517,522*). *CATEGORY IB*

9. Eliminate exposures of patients at high risk for aspergillosis to potential environmental sources of *Aspergillus* sp. (e.g., *Aspergillus*-contaminated food, potted plants, or flower arrangements) (*466,517,522,709–711*). *CATEGORY II*

10. Prevent birds from gaining access to hospital air-intake ducts (*523*). *CATEGORY IB*

C. The following procedures should be followed if a case of nosocomial aspergillosis occurs:

1. Begin a prospective search for additional cases in hospitalized patients and an intensified retrospective review of the hospital's microbiologic, histopathologic, and postmortem records. *CATEGORY IB*

2. If evidence of continuing transmission is not present, continue routine maintenance procedures to prevent nosocomial aspergillosis (see Sections II-B-1 through II-B-10). *CATEGORY IB*

3. If evidence of continuing *Aspergillus* sp. infection is present, conduct an environmental investigation to determine and eliminate the source. If assistance is needed, contact the local or state health department (*473,477,478, 521,533,537*). *CATEGORY IB*

   a. Collect environmental samples from potential sources of *Aspergillus* sp., especially those sources implicated in the epidemiologic investigation, by using appropriate methods (e.g., use of a high-volume air sampler rather than settle plates) (*473,477,478,521,533,537,713*). *CATEGORY IB*

   b. Depending on test availability, perform molecular subtyping of *Aspergillus* sp. obtained from patients and the environment to establish strain identity (*525,526*). *CATEGORY IB*

   c. If air-handling systems that supply air to areas in which high-risk patients are housed are not optimal, consider temporary deployment of portable HEPA filters until rooms with optimal air-handling systems are available for all patients at high risk for invasive aspergillosis. *CATEGORY II*

   d. If an environmental source of exposure to *Aspergillus* sp. is identified, perform corrective measures as needed to eliminate the source from the environment of patients at high risk for infection. *CATEGORY IB*

   e. If an environmental source of exposure to *Aspergillus* sp. is not identified, review existing infection-control measures, including engineering aspects, to identify potential areas that can be corrected or improved. *CATEGORY IB*

III. Modifying Host Risk for Infection

A. Administer cytokines, including granulocyte-colony–stimulating factor and granulocyte-macrophage–stimulating factor, to increase host resistance to aspergillosis by decreasing the duration and severity of chemotherapy-induced granulocytopenia (*512,513*). *CATEGORY II*

B. *No Recommendation* for administration of intranasal amphotericin B or oral antifungal agents (including amphotericin B and triazole compounds) to high-risk patients for prophylaxis against aspergillosis (*514,515,714*). *UNRESOLVED ISSUE*

PREVENTION AND CONTROL OF RSV INFECTION

I. Staff Education and Infection Surveillance

A. Staff education

Educate personnel regarding the epidemiology, modes of transmission, and means of preventing transmission of RSV (*226,659–661*). *CATEGORY IA*

B. Surveillance

1. Establish mechanism(s) by which the appropriate hospital personnel are promptly alerted to any increase in RSV activity in the local community. *CATEGORY IB*

2. During December–March and periods of increased prevalence of RSV in the community, attempt prompt diagnosis of RSV infection by using rapid diagnostic techniques as clinically indicated for pediatric patients, especially infants, and for immunocompromised adults who have a respiratory illness at the time of hospital admission (*592,596*). *CATEGORY IB*

II. Interrupting Transmission of RSV

A. Preventing person-to-person transmission

1. **Primary measures for contact isolation**

   a. *Handwashing.* Regardless of whether gloves have been worn, wash hands after contact with a patient or after touching respiratory secretions or fomites potentially contaminated with respiratory secretions (*218,231, 553,583–585,594*). *CATEGORY IA*

b. *Wearing gloves.*

(1) Wear gloves while handling patients or respiratory secretions of patients who have confirmed or suspected RSV infection and while handling fomites potentially contaminated with patient secretions (*226,553,583,584,590,596*). *CATEGORY IA*

(2) Change gloves a) between contact with different patients and b) after handling respiratory secretions or fomites contaminated with secretions from one patient before contact with another patient (*226,228*). Wash hands after removing gloves. (See II-A-1-a.) *CATEGORY IA*

c. *Wearing a gown.* Wear a gown if clothing could be soiled by the respiratory secretions of a patient (e.g., when handling infants who have RSV infection or other viral respiratory illness), and change the gown after such contact and before caring for another patient (*226,589,591,596*). *CATEGORY IB*

d. *Staffing.* Restrict HCWs who are in the acute stages of an upper respiratory illness (i.e., those who are sneezing and/or coughing) from providing care to infants and other patients at high risk for complications from RSV infection (e.g., children who have severe underlying cardiopulmonary conditions, children receiving chemotherapy for malignancy, premature infants, and patients who are otherwise immunocompromised) (*594, 596*). *CATEGORY IB*

e. *Limiting visitors.* Do not allow persons who have symptoms of respiratory infection to visit uninfected pediatric, immunosuppressed, or cardiac patients (*590*). *CATEGORY II*

2. **Controlling RSV outbreaks**

a. *Use of private rooms, cohorting, and patient-screening.* To control ongoing RSV transmission in the hospital, admit young children who have symptoms of viral respiratory illness to single rooms if possible, OR perform RSV-screening diagnostic tests on young children at the time of hospital admission and cohort them according to their RSV-infection status (*590,592,594,596*). *CATEGORY II*

b. *Personnel cohorting.* During an outbreak of nosocomial RSV, cohort personnel as much as practical (i.e., restrict personnel who provide care to infected patients from providing care to uninfected patients, and vice-versa) (*590,594,596*). *CATEGORY II*

c. *Postponing patient admission.* During outbreaks of nosocomial RSV, postpone elective admission of uninfected patients at high risk for complications from RSV infection. *CATEGORY II*

d. *Wearing eye-nose goggles. No Recommendation* for wearing eye-nose goggles during close contact with an RSV-infected patient (*593,597*). *UNRESOLVED ISSUE*

PREVENTION AND CONTROL OF INFLUENZA

I. Staff Education and Infection Surveillance

A. Staff education

Educate HCWs about the epidemiology, modes of transmission, and means of preventing transmission of influenza (*659–661,715,716*). *CATEGORY IA*

B. Surveillance

1. Establish mechanism(s) by which the appropriate hospital personnel are promptly alerted of any increase in influenza activity in the local community. *CATEGORY IB*

2. Arrange for laboratory tests to be available to clinicians, for use when clinically indicated, to promptly confirm the diagnosis of influenza and other acute viral respiratory illnesses, especially during November–April (*620–625*). *CATEGORY IB*

II. Modifying Host Risk for Infection

A. Vaccination

1. *Patients.* Offer vaccine to outpatients and inpatients at high risk for complications from influenza, beginning in September and continuing until influenza activity has begun to decline (*628,647,648,717–719*). Patients at high risk for complications from influenza include persons ≥65 years of age; persons who are in long-term–care units; or persons who have chronic disorders of the pulmonary or cardiovascular systems, diabetes mellitus, renal dysfunction, hemoglobinopathies, or immunosuppression; persons 6 months–18 years of age who are receiving long-term aspirin therapy (*628*); and persons who have musculoskeletal disorders that impede adequate respiration. *CATEGORY IA*

2. *Personnel.* Vaccinate HCWs before the influenza season begins each year, preferably between mid-October and mid-November. Until influenza activity declines, continue to make vaccine available to newly hired personnel and to those who initially refused vaccination. If vaccine supply is limited, give highest priority to vaccination of HCWs caring for patients at greatest risk for severe complications from influenza infection (see Section II-A-1) (*628*). *CATEGORY IB*

B. Use of antiviral agents. (See Section IV, Controlling Influenza Outbreaks.)

III. Interrupting Person-to-Person Transmission

A. Keep a patient who has suspected or confirmed influenza in a private room or, unless medically contraindicated, in a room with other patients who have confirmed influenza. *CATEGORY IB*

B. As much as feasible, maintain negative air pressure in rooms of patients for whom influenza is suspected or diagnosed, or place persons who have influenza-like illness together in a hospital area that has an independent air-supply and exhaust system (*613,614,616,720*). *CATEGORY II*

C. Institute the wearing of masks among persons—except those immune to the infecting virus strain—who enter the room of a patient who has influenza (*613,614,720*). *CATEGORY IB*

D. As much as possible during periods of influenza activity in the community, the hospital's employee health service should evaluate HCWs who have fever and symptoms of upper respiratory tract infection suggestive of influenza for possible removal from duties that involve direct patient contact. Use more stringent guidelines for HCWs working in certain patient-care areas (e.g., ICUs, nurseries, and units with severely immunosuppressed patients) (*649,721*). *CATEGORY II*

E. When community and/or nosocomial outbreaks occur, especially if they are characterized by high attack rates and severe illness, initiate the following:

1. Restrict hospital visitors who have a febrile respiratory illness. *CATEGORY IB*
2. Curtail or eliminate elective medical and surgical admissions as necessary. *CATEGORY IB*
3. Restrict cardiovascular and pulmonary surgery to emergency cases only. *CATEGORY IB*

IV. Controlling Influenza Outbreaks

A. Determining the outbreak strain

Early in the outbreak, obtain nasopharyngeal-swab or nasal-wash specimens from patients who recently had onset of symptoms suggestive of influenza for influenza virus culture or antigen detection. *CATEGORY IB*

B. Vaccinating patients and HCWs

Administer current influenza vaccine to unvaccinated patients and HCWs, especially if the outbreak occurs early in the influenza season (*609,628*). *CATEGORY IB*

C. Administering amantadine or rimantadine

1. When a nosocomial outbreak of influenza A is suspected or identified:

   a. Administer amantadine or rimantadine for prophylaxis to all uninfected patients in the involved unit who do not have contraindications to these drugs. Do not delay administration of amantadine or rimantadine unless the results of diagnostic tests to identify the infecting strain(s) can be obtained within 12–24 hours after specimen collection (*631,634*). *CATEGORY IB*

   b. Administer amantadine or rimantadine for prophylaxis to unvaccinated HCWs who do not have medical contraindications to these drugs and who are in the involved unit or providing care to patients at high risk for infection (*631*). *CATEGORY II*

2. Discontinue amantadine or rimantadine if laboratory tests confirm or strongly suggest that influenza type A is not the cause of the outbreak (*632*). *CATEGORY IA*

3. If the cause of the outbreak is confirmed or believed to be influenza type A AND vaccine has been administered only recently to susceptible patients and HCWs, continue amantadine or rimantadine prophylaxis until 2 weeks after the vaccination (*722*). *CATEGORY IB*

4. To the extent possible, do not allow contact between those at high risk for complications from influenza and patients or HCWs who are taking amantadine or rimantadine for treatment of acute respiratory illness; prevent contact during and for 2 days after the latter discontinue treatment (*633,642–646*). *CATEGORY IB*

D. Interrupting person-to-person transmission of microorganisms. (See Section III, A–E.)

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APPENDIX A
Examples of Semicritical Items* Used on the Respiratory Tract

- Anesthesia device or equipment, including:
  - — Face mask or tracheal tube,
  - — Inspiratory and expiratory tubing,
  - — Y-piece,
  - — Reservoir bag, and
  - — Humidifier;
- Breathing circuits of mechanical ventilators;
- Bronchoscopes and their accessories, except for biopsy forceps and specimen brush, which are considered critical items and are sterilized before reuse;
- Endotracheal and endobronchial tubes;
- Laryngoscope blades;
- Mouthpieces and tubing of pulmonary-function testing equipment;
- Nebulizers and their reservoirs;
- Oral and nasal airways;
- Probes of CO_2 analyzers, air-pressure monitors;
- Resuscitation bags;
- Stylets;
- Suction catheters;
- Temperature sensors.

*Items that directly or indirectly contact mucous membranes of the respiratory tract; these should be sterilized or subjected to high-level disinfection before reuse.

APPENDIX B
Maintenance Procedures Used to Decrease Survival and Multiplication of *Legionella sp.* in Potable-Water Distribution Systems

I. Providing water at ≥50 C at all points in the heated water system, including the taps.

This requires that water in calorifiers (water heaters) be maintained at ≥60 C. In the United Kingdom, where maintenance of water temperatures at ≥50 C in hospitals has been mandated, installation of blending or mixing valves at or near taps to reduce the water temperature to ≤43 C has been recommended in certain settings to reduce the risk for scald injury to patients, visitors, and HCWs (*446*). However, *Legionella* sp. 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 (*449,723*). Insulation of plumbing to ensure delivery of cold (<20 C) water to water heaters (and to cold-water outlets) may diminish the opportunity for bacterial multiplication (*391*). Both "dead legs" and "capped spurs"* within the plumbing system provide areas of stagnation and cooling to <50 C regardless of the circulating-water temperature; these segments may need to be removed to prevent colonization (*724*). Rubber fittings within plumbing systems have been associated with persistent colonization, and replacement of these fittings may be required for *Legionella* sp. eradication (*725*).

II. Continuous chlorination to maintain concentrations of free residual chlorine at 1–2 mg/L at the tap

This requires the placement of flow-adjusted, continuous injectors of chlorine throughout the water distribution system. The adverse effects of continuous chlorination include accelerated corrosion of plumbing, which results in system leaks and production of potentially carcinogenic trihalomethanes. However, when levels of free residual chlorine are below 3 mg/L, trihalomethane levels are kept below the maximum safety level recommended by the Environmental Protection Agency (*447,726,727*).

*A dead leg is a pipe, or spur, leading from the water recirculating system to an outlet that is used infrequently (i.e., the heat or chlorine in the recirculating system cannot adequately flow to the outlet). A capped spur is a pipe leading from the water recirculating system to an outlet that has been closed off (i.e., the spur has been "capped"). A capped spur cannot be flushed, and it might not be noticed unless the surrounding wall is removed.

APPENDIX C
Culturing Environmental Specimens for *Legionella* sp.

I. Recommended procedure for collecting and processing environmental specimens for *Legionella* sp. (*728*)

A. Collect water (if possible, 1-L samples) in sterile, screw-top bottles, preferably containing sodium thiosulfate at a concentration of 0.5 cc of 0.1 N solution of sample water. (Sodium thiosulfate inactivates any residual halogen biocide.)

B. Collect culture-swabs of the internal surfaces of faucets, aerators, and showerheads; in a sterile, screw-top container, such as a 50-cc plastic centrifuge tube, submerge each swab in 5–10 cc of sample water taken from the same device from which the sample was obtained.

C. As soon as possible after collection, water samples and swabs should be transported to and processed in a laboratory proficient at culturing water specimens for *Legionella* sp. Samples may be transported at room temperature but must be protected from temperature extremes.

D. Test samples for the presence of *Legionella* sp. by using semi-selective culture media. Use standard laboratory procedures. (Detection of *Legionella* sp. antigen by the direct fluorescent antibody technique is not suitable for environmental samples [*729–731*]. In addition, the use of polymerase chain reaction for identification of *Legionella* sp. is not recommended until more data regarding the sensitivity and specificity of this procedure are available [*732*].)

II. Possible samples and sampling sites for *Legionella* sp. in the hospital (*733*)

Water samples

- Potable water system
  - — Incoming water main
  - — Water softener
  - — Holding tanks/cisterns
  - — Water heater tanks (at the inflow and outflow sites)
- Potable water outlets (e.g., faucets or taps, showers), especially outlets located in or near case-patients' rooms
- Cooling tower/evaporative condenser
  - — Make-up water (i.e., water added to the system to replace water lost by evaporation, drift, and leakage)
  - — Basin (i.e., area under tower for collection of cooled water)
  - — Sump (i.e., section of basin from which cooled water returns to heat source)
  - — Heat source (e.g., chillers)

Water samples (cont'd.)

- Other sources
- Humidifiers (i.e., nebulizers)
  - Bubblers for oxygen
  - Water used for respiratory therapy equipment
  - Decorative fountains
  - Irrigation equipment
  - Fire sprinkler system (if recently used)
  - Whirlpools/spas

Swabs

- Potable water system
  - Faucets (proximal to aerators)
  - Faucet aerators
  - Shower heads
- Cooling towers
  - Internal components (e.g., splash bars and other fill surfaces)
  - Areas with visible biofilm accumulation

APPENDIX D
Procedure for Cleaning Cooling Towers and Related Equipment*

I. Before chemical disinfection and mechanical cleaning

A. Provide protective equipment to workers who perform the disinfection, to prevent their exposure to a) chemicals used for disinfection and b) aerosolized water containing *Legionella* sp. 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. If possible, shut off the heat source.
2. Shut off fans, if present, on the cooling tower/evaporative condenser (CT/EC).
3. Shut off the system blowdown (i.e., purge) valve. Shut off the automated blowdown controller, if present, and set the system controller to manual.
4. Keep make-up water valves open.
5. Close building air-intake vents within at least 30 m of the CT/EC until after the cleaning procedure is complete.
6. Continue operating pumps for water circulation through the CT/EC.

II. Chemical disinfection

A. Add fast-release, chlorine-containing disinfectant in pellet, granular, or liquid form, and follow safety instructions on the product label. 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 (i.e., 3.0 lbs [1.4 kg] industrial grade NaOCl [12%–15% available Cl] per 1,000 gal of CT/EC water; 10.5 lbs [4.8 kg] domestic grade NaOCl [3%–5% available Cl] per 1,000 gal of CT/EC water; or 0.6 lb [0.3 kg] $Ca[OCl]_2$ per 1,000 gal of CT/EC water. If significant biodeposits are present, additional chlorine may be required. If the volume of water in 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.

*Adapted from information published previously by the Wisconsin Department of Health and Social Services, 1987 (*463*).

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 gal 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 (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., Department of Natural Resources) 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 outlined in steps 2–6 in Section I-B above.

III. 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. After mechanical cleaning

A. Fill the system with water and add chlorine to achieve 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. CT/EC may be put back into service using an effective water-treatment program.

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