

SPECIAL ARTICLE

Guideline for infection control in health care personnel, 1998

Elizabeth A. Bolyard, RN, MPH,^a Ofelia C. Tablan, MD,^a Walter W. Williams, MD,^b Michele L. Pearson, MD,^a Craig N. Shapiro, MD,^a Scott D. Deitchman, MD,^c and The Hospital Infection Control Practices Advisory Committee

Centers for Disease Control and Prevention
Public Health Service
U.S. Department of Health and Human Services
Hospital Infection Control Practices Advisory Committee
Membership List, June 1997

Chairman

Walter J. Hierholzer, Jr., MD
Yale-New Haven Hospital
New Haven, Connecticut

Executive Secretary

Michele L. Pearson, MD
Centers for Disease Control and Prevention
Atlanta, Georgia

Personnel Health Guideline Sponsor

Susan W. Forlenza, MD
New York City Department of Health
New York, New York

Members

Audrey B. Adams, RN, MPH

Affiliations: National Center for Infectious Diseases,^a National Immunization Program,^b National Institute of Occupational Safety and Health.^c

Published simultaneously in *AJIC: American Journal of Infection Control* (1998;26:289-354) and *Infection Control and Hospital Epidemiology* (1998;19:407-63)

17/52/88841

Montefiore Medical Center
Bronx, New York

Mary J. Gilchrist, PhD
University of Iowa
Iowa City, Iowa

Elaine L. Larson, RN, PhD
Georgetown University
Washington, D.C.

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

Rita D. McCormick, RN
University of Wisconsin Hospital and Clinics
Madison, Wisconsin

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

Ronald L. Nichols, MD
Tulane University School of Medicine
New Orleans, Louisiana

Jane D. Siegel, MD
University of Texas Southwestern Medical Center
Dallas, Texas

Table of Contents

I. Infection control issues for health care personnel: An overview

A. EXECUTIVE SUMMARY	291
B. INTRODUCTION	292
C. INFECTION CONTROL OBJECTIVES FOR A PERSONNEL HEALTH SERVICE	292
D. ELEMENTS OF A PERSONNEL HEALTH SERVICE FOR INFECTION CONTROL	293
1. Coordination with other departments	293
2. Medical evaluations	293
3. Personnel health and safety education	293
4. Immunization programs	296
5. Management of job-related illnesses and exposures	298
6. Health counseling	301
7. Maintenance of records, data management, and confidentiality	301
E. EPIDEMIOLOGY AND CONTROL OF SELECTED INFECTIONS TRANSMITTED AMONG HEALTH CARE PERSONNEL AND PATIENTS	302
1. Bloodborne pathogens	302

a.	Overview	302
b.	Hepatitis B	302
c.	Hepatitis C	304
d.	Human immunodeficiency virus	305
2.	Conjunctivitis	305
3.	Cytomegalovirus	305
4.	Diphtheria	306
5.	Gastrointestinal infections, acute	307
6.	Hepatitis A	308
7.	Herpes simplex	309
8.	Measles	309
9.	Meningococcal disease	310
10.	Mumps	311
11.	Parvovirus	311
12.	Pertussis	312
13.	Poliomyelitis	313
14.	Rabies	313
15.	Rubella	314
16.	Scabies and pediculosis	315
17.	<i>Staphylococcus aureus</i> infection and carriage	316
18.	<i>Streptococcus</i> , group A infection	316
19.	Tuberculosis	316
20.	Vaccinia (smallpox)	320
21.	Varicella	320
22.	Viral respiratory infections	323
a.	Influenza	323
b.	Respiratory syncytial virus	323
c.	Work restrictions	324
F.	PREGNANT PERSONNEL	324
G.	LABORATORY PERSONNEL	324
H.	EMERGENCY-RESPONSE PERSONNEL	325
I.	LATEX HYPERSENSITIVITY	325
J.	THE AMERICANS WITH DISABILITIES ACT	327
II.	Recommendations for prevention of infections in health care personnel	328
A.	INTRODUCTION	328
B.	ELEMENTS OF A PERSONNEL HEALTH SERVICE FOR INFECTION CONTROL	328
1.	Coordinated planning and administration	328
2.	Placement evaluation	328
3.	Personnel health and safety education	329
4.	Job-related illnesses and exposures	329
5.	Record keeping, data management, and confidentiality	329
C.	PROTECTION OF PERSONNEL AND OTHER PATIENTS FROM PATIENTS WITH INFECTIONS	330
D.	IMMUNIZATION OF HEALTH CARE PERSONNEL, GENERAL RECOMMENDATIONS	330
E.	PROPHYLAXIS AND FOLLOW-UP AFTER EXPOSURE, GENERAL RECOMMENDATIONS	330
F.	PERSONNEL RESTRICTION BECAUSE OF INFECTIOUS ILLNESSES OR SPECIAL CONDITIONS, GENERAL RECOMMENDATIONS	330
G.	PREVENTION OF NOSOCOMIAL TRANSMISSION OF SELECTED INFECTIONS	330
1.	Bloodborne pathogens, general recommendation	330
a.	Hepatitis B	331
b.	Hepatitis C	331
c.	Human immunodeficiency virus	331
2.	Conjunctivitis	331
3.	Cytomegalovirus	331
4.	Diphtheria	331

5. Gastroenteritis	332
6. Hepatitis A	332
7. Herpes simplex	332
8. Measles	332
9. Meningococcal disease	333
10. Mumps	333
11. Parvovirus	333
12. Pertussis	333
13. Poliomyelitis	333
14. Rabies	334
15. Rubella	334
16. Scabies and pediculosis	334
17. Staphylococcal infection or carriage	334
18. Group A <i>Streptococcus</i> infections	334
19. Tuberculosis	335
20. Vaccinia	337
21. Varicella	337
22. Viral respiratory infections	337
H. SPECIAL ISSUES	338
1. Pregnancy	338
2. Emergency-response employees	338
3. Personnel linked to outbreaks of bacterial infection	338
4. Latex hypersensitivity	338
References	339
Table 1. Immunobiologics and schedules for health care personnel	294
Table 2. Summary of ACIP recommendations on immunization of health care workers with special conditions	298
Table 3. Summary of suggested work restrictions for health care personnel exposed to or infected with infectious diseases of importance in health care settings, in the absence of state and local regulations	299
Table 4. Recommendation for postexposure prophylaxis for percutaneous or permucosal exposure to hepatitis B virus, United States	303
Table 5. Selected reported etiologic agents causing community or nosocomially acquired gastrointestinal infections in developed countries	307
Table 6. Pregnant health care personnel: Pertinent facts to guide management of occupational exposures to infectious agents	322
Appendix A. Recommended readings for infection control in health care personnel	354

Part I. Infection control issues for health care personnel: An overview

A. EXECUTIVE SUMMARY

This guideline updates and replaces the previous edition of the Centers for Disease Control and Prevention (CDC) "Guideline for Infection Control in Hospital Personnel," published in 1983. The revised guideline, designed to provide methods for reducing the transmission of infections from patients to health care personnel and from personnel to patients, also provides an overview of the evidence for recommendations considered prudent by consensus of the Hospital Infection

Control Practices Advisory Committee members. A working draft of this guideline was also reviewed by experts in infection control, occupational health, and infectious diseases; however, all recommendations contained in the guideline may not reflect the opinion of all reviewers.

This document focuses on the epidemiology of and preventive strategies for infections known to be transmitted in health care settings and those for which there are adequate scientific data on which to base recommendations for prevention.

The prevention strategies addressed in this document include immunizations for vaccine-preventable diseases, isolation precautions to prevent exposures to infectious agents, management of health care personnel exposure to infected persons, including postexposure prophylaxis, and work restrictions for exposed or infected health care personnel. In addition, because latex barriers are frequently used to protect personnel against transmission of infectious agents, this guideline addresses issues related to latex hypersensitivity and provides recommendations to prevent sensitization and reactions among health care personnel.

B. INTRODUCTION

In the United States, there are an estimated 8.8 million persons who work in health care professions and about 6 million persons work in more than 6000 hospitals. However, health care is increasingly being provided outside hospitals in facilities such as nursing homes, freestanding surgical and outpatient centers, emergency care clinics, and in patients' homes or during prehospital emergency care. Hospital-based personnel and personnel who provide health care outside hospitals may acquire infections from or transmit infections to patients, other personnel, household members, or other community contacts.^{1,2}

In this document, the term *health care personnel* refers to all paid and unpaid persons working in health care settings who have the potential for exposure to infectious materials, including body substances, contaminated medical supplies and equipment, contaminated environmental surfaces, or contaminated air. These personnel may include but are not limited to emergency medical service personnel, dental personnel, laboratory personnel, autopsy personnel, nurses, nursing assistants, physicians, technicians, therapists, pharmacists, students and trainees, contractual staff not employed by the health care facility, and persons not directly involved in patient care but potentially exposed to infectious agents (e.g., clerical, dietary, housekeeping, maintenance, and volunteer personnel). In general, health care personnel in or outside hospitals who have contact with patients, body fluids, or specimens have a higher risk of acquiring or transmitting infections than do other health care personnel who have only brief casual contact with patients and their environment (e.g., beds, furniture, bathrooms, food trays, medical equipment).

Throughout this document, terms are used to describe routes of transmission of infections. These terms have been fully described in the "Guideline for Isolation Precautions in Hospitals."³ They are summarized as follows: *direct contact* refers to body surface-to-body surface contact and physical transfer of microorganisms between a susceptible host and an infected or colonized person (e.g., while performing oral care or procedures); *indirect contact* refers to contact of a susceptible host with a contaminated object (e.g., instruments, hands); *droplet contact* refers to conjunctival, nasal, or oral mucosa contact with droplets containing microorganisms generated from an infected person (by coughing, sneezing, and talking, or during certain procedures such as suctioning and bronchoscopy) that are propelled a short distance; *airborne transmission* refers to contact with droplet nuclei containing microorganisms that can remain suspended in the air for long periods or to contact with dust particles containing an infectious agent that can be widely disseminated by air currents; and, finally, *common vehicle transmission* refers to contact with contaminated items such as food, water, medications, devices, and equipment.

In 1983 the CDC published the "Guideline for Infection Control in Hospital Personnel."⁴ The document focused on the prevention of infections known to be transmitted to and from health care personnel. This revision of the guideline has been expanded to include (a) recommendations for non-patient care personnel, both in and outside hospitals, (b) management of exposures, (c) prevention of transmission of infections in microbiologic and biomedical laboratories, and, because of the common use of latex barriers to prevent infections, (d) prevention of latex hypersensitivity reactions. As in the 1983 guideline, readers are frequently referred to the "Guideline for Isolation Precautions in Hospitals"³ and other published guidelines and recommendations for precautions that health care personnel may use when caring for patients or handling patient equipment or specimens.^{5,6}

C. INFECTION CONTROL OBJECTIVES FOR A PERSONNEL HEALTH SERVICE

The infection control objectives of the personnel health service should be an integral part of a health care organization's general program for infection control. The objectives usually include the following: (a) educating personnel about the principles of infection control and stressing indi-

vidual responsibility for infection control, (b) collaborating with the infection control department in monitoring and investigating potentially harmful infectious exposures and outbreaks among personnel, (c) providing care to personnel for work-related illnesses or exposures, (d) identifying work-related infection risks and instituting appropriate preventive measures, and (e) containing costs by preventing infectious diseases that result in absenteeism and disability. These objectives cannot be met without the support of the health care organization's administration, medical staff, and other health care personnel. Documents that provide more detailed information regarding infection control issues for personnel health are listed in Appendix A.

D. ELEMENTS OF A PERSONNEL HEALTH SERVICE FOR INFECTION CONTROL

Certain elements are necessary to attain the infection control goals of a personnel health service: (a) coordination with other departments, (b) medical evaluations, (c) health and safety education, (d) immunization programs, (e) management of job-related illnesses and exposures to infectious diseases, including policies for work restrictions for infected or exposed personnel, (f) counseling services for personnel on infection risks related to employment or special conditions, and (g) maintenance and confidentiality of personnel health records.

The organization of a personnel health service may be influenced by the size of the institution, the number of personnel, and the services offered. To ensure that contractual personnel who are not paid by the health care facility receive appropriate personnel health services, contractual agreements with their employers should contain provisions consistent with the policies of the facility that uses those employees. Personnel with specialized training and qualifications in occupational health can facilitate the provision of effective services.

1. Coordination with other departments

For infection control objectives to be achieved, the activities of the personnel health service must be coordinated with infection control and other appropriate departmental personnel. This coordination will help ensure adequate surveillance of infections in personnel and provision of preventive services. Coordinating activities will also help to ensure that investigations of exposures and outbreaks are conducted efficiently and preventive measures implemented promptly.

2. Medical evaluations

Medical evaluations before placement can ensure that personnel are not placed in jobs that would pose undue risk of infection to them, other personnel, patients, or visitors. An important component of the placement evaluation is a health inventory. This usually includes determining immunization status and obtaining histories of any conditions that might predispose personnel to acquiring or transmitting communicable diseases. This information will assist in decisions about immunizations or postexposure management.

A physical examination, another component of the medical evaluation, can be used to screen personnel for conditions that might increase the risk of transmitting or acquiring work-related diseases and can serve as a baseline for determining whether future diseases are work related. However, the cost-effectiveness of routine physical examinations, including laboratory testing (such as complete blood cell counts, serologic tests for syphilis, urinalysis, and chest radiographs) and screening for enteric or other pathogens for infection control purposes, has not been demonstrated. Conversely, screening for some vaccine-preventable diseases, such as hepatitis B, measles, mumps, rubella, or varicella, may be cost-effective. In general, the health inventory can be used to guide decisions regarding physical examinations or laboratory tests. However, some local public health ordinances may mandate that certain screening procedures be used.

Periodic evaluations may be done as indicated for job reassignment, for ongoing programs (e.g., TB screening), or for evaluation of work-related problems.

3. Personnel health and safety education

Personnel are more likely to comply with an infection control program if they understand its rationale. Thus, personnel education is a cardinal element of an effective infection control program. Clearly written policies, guidelines, and procedures ensure uniformity, efficiency, and effective coordination of activities. However, because the risk of infection varies by job category, infection control education should be modified accordingly. In addition, some personnel may need specialized education on infection risks related to their employment and on preventive measures that will reduce those risks. Furthermore, educational materials need to be appropriate in content and vocabulary to the educational level, literacy, and

Table 1A. Immunobiologics and schedules for health care personnel (modified from ACIP recommendations⁹): Immunizing agents strongly recommended for health care personnel

Generic name	Primary booster dose schedule	Indications	Major precautions and contraindications	Special considerations
Hepatitis B recombinant vaccine	Two doses IM in the deltoid muscle 4 wk apart; 3rd dose 5 mo after 2nd; booster doses not necessary	Health care personnel at risk of exposure to blood and body fluids	No apparent adverse effects to developing fetuses, not contraindicated in pregnancy; history of anaphylactic reaction to common baker's yeast	No therapeutic or adverse effects on HBV-infected persons; cost-effectiveness of prevaccination screening for susceptibility to HBV depends on costs of vaccination and antibody testing and prevalence of immunity in the group of potential vaccinees; health care personnel who have ongoing contact with patients or blood should be tested 1-2 mo after completing the vaccination series to determine serologic response
Influenza vaccine (inactivated whole or split virus)	Annual single-dose vaccination IM with current (either whole- or split-virus) vaccine	Health care personnel with contact with high-risk patients or working in chronic care facilities; personnel with high-risk medical conditions and/or ≥ 65 yr	History of anaphylactic hypersensitivity after egg ingestion	No evidence of maternal or fetal risk when vaccine was given to pregnant women with underlying conditions that render them at high risk for serious influenza complications.
Measles live-virus vaccine	One dose SC; 2nd dose at least 1 mo later	Health care personnel born in or after 1957 without documentation of (a) receipt of two doses of live vaccine on or after their 1st birthday, (b) physician-diagnosed measles, or (c) laboratory evidence of immunity; vaccine should be considered for all personnel, including those born before 1957, who have no proof of immunity	Pregnancy; immunocompromised* state; (including HIV-infected persons with severe immunosuppression) history of anaphylactic reactions after gelatin ingestion or receipt of neomycin; or recent receipt of immune globulin	MMR is the vaccine of choice if recipients are also likely to be susceptible to rubella and/or mumps; persons vaccinated between 1963 and 1967 with (a) a killed measles vaccine alone, (b) killed vaccine followed by live vaccine, or (c) a vaccine of unknown type should be revaccinated with two doses of live measles vaccine
Mumps live-virus vaccine	One dose SC; no booster	Health care personnel believed to be susceptible can be vaccinated; adults born before 1957 can be considered immune	Pregnancy; immunocompromised* state; history of anaphylactic reaction after gelatin ingestion or receipt of neomycin	MMR is the vaccine of choice if recipients are also likely to be susceptible to measles and rubella
Rubella live-virus vaccine	One dose SC; no booster	Health care personnel, both male and female, who lack documentation of receipt of live vaccine on or after their 1st birthday, or of laboratory evidence of immunity; adults born before 1957 can be considered immune, except women of childbearing age	Pregnancy; immunocompromised* state; history of anaphylactic reaction after receipt of neomycin	Women pregnant when vaccinated or who become pregnant within 3 mo of vaccination should be counseled on the theoretic risks to the fetus, the risk of rubella vaccine-associated malformations in these women is negligible; MMR is the vaccine of choice if recipients are also likely to be susceptible to measles or mumps
Varicella-zoster live-virus vaccine	Two 0.5 ml doses SC, 4-8 wk apart if ≥ 13 yr	Health care personnel without reliable history of varicella or laboratory evidence of varicella immunity	Pregnancy, immunocompromised* state, history of anaphylactic reaction after receipt of neomycin or gelatin; salicylate use should be avoided for 6 wk after vaccination	Because 71%-93% of persons without a history of varicella are immune, serologic testing before vaccination may be cost-effective

IM, Intramuscularly; SC, subcutaneously.

*Persons immunocompromised because of immune deficiencies, HIV infection, leukemia, lymphoma, generalized malignancy, or immunosuppressive therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1B. Immunobiologics and schedules for health care personnel (modified from ACIP recommendations⁹): Other immunizing agents available for health care personnel in special circumstances

Generic name	Primary/booster dose schedule	Indications	Major precautions and contraindications	Special considerations
BCG vaccine (for tuberculosis)	One percutaneous dose of 0.3 ml; no booster dose recommended	Health care personnel in communities where (a) MDR-TB is prevalent, (b) strong likelihood of infection exists, and (c) full implementation of TB infection control precautions has been inadequate in controlling the spread of infection (<i>NOTE: BCG should be used after consultation with local and/or state health department</i>)	Immunocompromised* state and pregnancy	In the United States, TB control efforts are directed toward early identification and treatment of cases of active TB and toward preventive therapy with isoniazid for PPD converters
Hepatitis A vaccine	Two doses of vaccine IM, either (HAVRIX™) 6-12 mo apart or (VAQTA™) 6 mo apart	Not routinely indicated for U.S. health care personnel; persons who work with HAV-infected primates or with HAV in a laboratory setting should be vaccinated	History of anaphylactic reaction to alum or the preservative 2-phenoxy ethanol; vaccine safety in pregnant women has not been evaluated, risk to fetus is likely low and should be weighed against the risk of hepatitis A in women at high risk	Health care personnel who travel internationally to endemic areas should be evaluated for vaccination
Meningococcal polysaccharide (quadrivalent A, C, W135, and Y) vaccine	One dose in volume and by route specified by manufacturer; need for boosters is unknown	Not routinely indicated for health care workers in the United States	Vaccine safety in pregnant women has not been evaluated; vaccine should not be given during pregnancy unless risk of infection is high	May be useful in certain outbreak situations (see text)
Polio vaccine	IPV, two doses SC given 4-8 wk apart followed by 3rd dose 6-12 mo after 2nd dose; booster doses may be IPV or OPV	Health care personnel in close contact with persons who may be excreting wild virus and laboratory personnel handling specimens that may contain wild poliovirus	History of anaphylactic reaction after receipt of streptomycin or neomycin; because safety of vaccine has not been evaluated in pregnant women, it should not be given during pregnancy	Use only IPV for immunosuppressed persons or personnel who care for immunosuppressed patients; if immediate protection against poliomyelitis is needed, OPV should be used.
Rabies vaccine	Primary, HDCV or RVA, IM, 1.0 ml (deltoid area) one each on days 0, 7, 21, or 28, or HDCV, ID, 1.0 ml, one each on days 0, 7, 21, and 28; booster, HDCV or RVA, IM, 0.1 ml (deltoid area), day 0 only, or HDCV, ID, 0.1 ml, day 0 only	Personnel who work with rabies virus or infected animals in diagnostic or research activities		The frequency of booster doses should be based on frequency of exposure. See CDC reference for Rabies Prevention for postexposure recommendations. ²²
Tetanus and diphtheria (Td)	Two doses IM 4 wk apart; 3rd dose 6-12 mo after 2nd dose; booster every 10 yr	All adults; tetanus prophylaxis in wound management	First trimester of pregnancy; history of a neurologic reaction or immediate hypersensitivity reaction; individuals with severe local (Arthus-type) reaction after previous dose of Td vaccine should not be given further routine or emergency doses of Td for 10 yr	

Continued

HDCV, Human diploid cell rabies vaccine; RVA, rabies vaccine absorbed; IPV, inactivated poliovirus vaccine; OPV, oral poliovirus vaccine; ID, intradermally. *Persons immunocompromised because of immune deficiencies, HIV infection, leukemia, lymphoma, generalized malignancy, or immunosuppressive therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1B. Continued

Generic name	Primary/booster dose schedule	Indications	Major precautions and contraindications	Special considerations
Typhoid vaccines: IM, SC, and oral	One 0.5 ml dose IM; booster doses of 0.5 ml every 2 yr; (Vi capsular polysaccharide) or two 0.5 ml doses SC, 4 or more wk apart; boosters of 0.5 ml SC or 0.1 ml ID every 3 yr if exposure continues or four oral doses on alternate days; (Ty21a) vaccine manufacturer's recommendation is revaccination with the entire four-dose series every 5 yr	Personnel in laboratories who frequently work with <i>Salmonella typhi</i>	History of severe local or systemic reaction to a previous dose of typhoid vaccine; Ty21a vaccine should not be given to immunocompromised* personnel	Vaccination should not be considered as an alternative to the use of proper procedures when handling specimens and cultures in the laboratory
Vaccinia vaccine (smallpox)	One dose administered with a bifurcated needle; boosters every 10 yr	Personnel who directly handle cultures of or animals contaminated with recombinant vaccinia viruses or orthopox viruses (monkeypox, cowpox, vaccinia, etc.) that infect human beings	Pregnancy, presence or history of eczema, or immunocompromised* status in potential vaccinees or in their household contacts	Vaccination may be considered for health care personnel who have direct contact with contaminated dressings or other infectious material from volunteers in clinical studies involving recombinant vaccinia virus

language of the employee. The training should comply with existing federal, state, and local regulations regarding requirements for employee education and training. All health care personnel need to be educated about the organization's infection control policies and procedures.

4. Immunization programs

Ensuring that personnel are immune to vaccine-preventable diseases is an essential part of successful personnel health programs. Optimal use of vaccines can prevent transmission of vaccine-preventable diseases and eliminate unnecessary work restriction. Prevention of illness through comprehensive personnel immunization programs is far more cost-effective than case management and outbreak control. Mandatory immunization programs, which include both newly hired and currently employed persons, are more effective than voluntary programs in ensuring that susceptible persons are vaccinated.⁷

National guidelines for immunization of and postexposure prophylaxis for health care personnel are provided by the U.S. Public Health Service's Advisory Committee on Immunization Practices (ACIP; Table 1).^{8,9} ACIP guidelines also contain (a) detailed information on the epidemiology of vaccine-preventable diseases, (b) data on

the safety and efficacy of vaccines and immune globulin preparations,⁸⁻²² and (c) recommendations for immunization of immunocompromised persons* (Table 2).^{16,23} The recommendations in this guideline have been adapted from the ACIP recommendations.⁹ In addition, individual states and professional organizations have regulations or recommendations on the vaccination of health care personnel.²⁴

Decisions about which vaccines to include in immunization programs have been made by considering (a) the likelihood of personnel exposure to vaccine-preventable diseases and the potential consequences of not vaccinating personnel, (b) the nature of employment (type of contact with patients and their environment), and (c) the characteristics of the patient population within the health care organization. Immunization of personnel before they enter high-risk situations is the most efficient and effective use of vaccines in health care settings.

Screening tests are available to determine susceptibility to certain vaccine-preventable diseases

*The term immunocompromised includes persons who are immunocompromised from immune deficiency diseases, HIV infection, leukemia, lymphoma, or generalized malignancy, or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1C. Immunobiologics and schedules for health care personnel (modified from ACIP recommendations⁹): Diseases for which postexposure prophylaxis may be indicated for health care personnel

Disease	Prophylaxis	Indications	Major precautions and contraindications	Special considerations
Diphtheria	Benzathine penicillin, 1.2 mU IM, single dose, or erythromycin (1 gm/day) PO × 7 days	For health care personnel exposed to diphtheria or identified as carriers		Also administer one dose Td to previously immunized if no Td has been given in ≥5 yr
Hepatitis A	One IM dose IG 0.02 ml/kg given within 2 wk of exposure in large muscle mass (deltoid, gluteal)	May be indicated for health care personnel exposed to feces of infected persons during outbreaks	Persons with IgA deficiency; do not administer within 2 wk after MMR or within 3 wk after varicella vaccine	
Hepatitis B	HBIG 0.06 ml/kg IM as soon as possible (and within 7 days) after exposure (with dose 1 of hepatitis B vaccine given at different body site); if hepatitis B series has not been started, 2nd dose of HBIG should be given 1 mo after 1st	HBV-susceptible health care personnel with percutaneous or mucous-membrane exposure to blood known to be HBsAg seropositive (see Table 5)		
Meningococcal disease	Rifampin, 600 mg PO every 12 hours for 2 days, or ceftriaxone, 250 mg IM, single dose, or ciprofloxacin, 500 mg PO, single dose	Personnel with direct contact with respiratory secretions from infected persons without the use of proper precautions (e.g., mouth-to-mouth resuscitation, endotracheal intubation, endotracheal tube management, or close examination of oropharynx)	Rifampin and ciprofloxacin not recommended during pregnancy	
Pertussis	Erythromycin, 500 mg qid PO, or trimethoprim-sulfamethoxazole, 1 tablet bid PO, for 14 days after exposure	Personnel with direct contact with respiratory secretions or large aerosol droplets from respiratory tract of infected persons.		
Rabies	For those never vaccinated: HRIG 20 IU/kg, half infiltrated around wound, and HDCV or RVA vaccine, 1.0 ml, IM (deltoid area), 1 each on days 0, 3, 7, 14, and 28	Personnel who have been bitten by human being or animal with rabies or have had scratches, abrasions, open wounds, or mucous membranes contaminated with saliva or other potentially infective material (e.g., brain tissue)		Personnel who have previously been vaccinated, give HDCV or RVA vaccine, 1.0 ml, IM, on days 0 and 3; no HRIG is necessary
Varicella-zoster virus	VZIG for persons ≤50 kg: 125 U/10kg IM; for persons >50 kg: 625 U†	Personnel known or likely to be susceptible to varicella and who have close and prolonged exposure to an infectious health care worker or patient, particularly those at high risk for complications, such as pregnant or immunocompromised persons		Serologic testing may help in assessing whether to administer VZIG; if varicella is prevented by the use of VZIG, vaccine should be offered later

PO, Orally; Td, tetanus-diphtheria toxoid; IG, immune globulin; IgA, immunoglobulin A; qid, four times daily; bid, twice daily; HRIG, human rabies immunoglobulin; HDCV, human diploid cell rabies vaccine; RVA, rabies vaccine absorbed.

*Persons immunocompromised because of immune deficiencies, HIV infection, leukemia, lymphoma, generalized malignancy, or immunosuppressive therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

†Some persons have recommended 125 U/10 kg regardless of total body weight.

Table 2. Summary of ACIP recommendations on immunization of health care workers with special conditions (modified from ACIP recommendations⁹⁾)

Vaccine	Pregnancy	HIV infection	Severe immunosuppression*	Asplenia	Renal failure	Diabetes	Alcoholism & alcoholic cirrhosis
BCG	UI	C	C	UI	UI	UI	UI
Hepatitis A	UI	UI	UI	UI	UI	UI	R†
Hepatitis B	R	R	R	R	R	R	R
Influenza	R‡	R	R	R	R	R	R
Measles, mumps, rubella	C	R§	C	R	R	R	R
Meningococcus	UI	UI	UI	R†	UI	UI	UI
Polio, IPV II	UI	UI	UI	UI	UI	UI	UI
Polio, OPV II	UI	C	C	UI	UI	UI	UI
Pneumococcus†	UI	R	R	R	R	R	R
Rabies	UI	UI	UI	UI	UI	UI	UI
Tetanus/diphtheria†	R	R	R	R	R	R	R
Typhoid, inactivated & V _i	UI	UI	UI	UI	UI	UI	UI
Typhoid, Ty21a	UI	C	C	UI	UI	UI	UI
Varicella	C	C	C	R	R	R	R
Vaccinia	UI	C	C	UI	UI	UI	UI

UI, Use if indicated; C, contraindicated; R, recommended.

*Severe immunosuppression can be the result of congenital immunodeficiency, leukemia, lymphoma, generalized malignancy or therapy with alkylating agents, antimetabolites, radiation, or large amounts of corticosteroids.

†Recommendation is based on the person's underlying condition rather than occupation.

‡Women who will be in the second or third trimester of pregnancy during influenza season.

§Contraindicated in persons with HIV infection and severe immunosuppression; see text.

¶Vaccination is recommended for unvaccinated health care workers who have close contact with patients who may be excreting wild polioviruses. Primary vaccination with IPV is recommended because the risk for vaccine-associated paralysis after administration of OPV is higher among adults than among children. Health care workers who have had a primary series of OPV or IPV who are directly involved with the provision of care to patients who may be excreting poliovirus may receive another dose of either IPV or OPV. Any suspected case of poliomyelitis should be investigated immediately. If evidence suggests transmission of wild poliovirus, control measures to contain further transmission should be instituted immediately, including an OPV vaccination campaign.

(e.g., hepatitis B, measles, mumps, rubella, and varicella). Such screening programs need to be combined with tracking systems to ensure accurate maintenance of personnel immunization records. Accurate immunization records ensure that susceptible personnel are promptly identified and appropriately vaccinated.

5. Management of job-related illnesses and exposures

Primary functions of the personnel health service are to arrange for prompt diagnosis and management of job-related illnesses and to provide appropriate postexposure prophylaxis after job-related exposures.

It is the responsibility of the health care organization to implement measures to prevent further transmission of infection, which sometimes warrants exclusion of personnel from work or patient contact.²⁵ Decisions on work restrictions are based on the mode of transmission and the epidemiology of the disease (Table 3). The term *exclude from duty* in this document should be interpreted as exclusion from the health care facility and from health care activities outside the facility. Personnel who are

excluded should avoid contact with susceptible persons both in the facility and in the community. Exclusion policies should include a statement of authority defining who may exclude personnel. The policies also need to be designed to encourage personnel to report their illnesses or exposures and not to penalize them with loss of wages, benefits, or job status. Workers' compensation laws do not cover exclusion from duty for exposures to infectious diseases; policies therefore should include a method for providing wages during the period that personnel are not able to work. In addition, exclusion policies must be enforceable and all personnel, especially department heads, supervisors, and nurse managers, should know which infections may warrant exclusion and where to report the illnesses 24 hours a day. Health care personnel who have contact with infectious patients outside of hospitals also need to be included in the postexposure program and encouraged to report any suspected or known exposures promptly. Notification of emergency-response personnel possibly exposed to selected infectious disease is mandatory (1990 Ryan White Act, Subtitle B, 42 USC 300ff-80).

Table 3. Summary of suggested work restrictions for health care personnel exposed to or infected with infectious diseases of importance in health care settings, in the absence of state and local regulations (modified from ACIP recommendations^a)

Disease/problem	Work restriction	Duration	Category
Conjunctivitis	Restrict from patient contact and contact with the patient's environment	Until discharge ceases	II
Cytomegalovirus infections	No restriction		II
Diarrheal diseases			
Acute stage (diarrhea with other symptoms)	Restrict from patient contact, contact with the patient's environment, or food handling	Until symptoms resolve	IB
Convalescent stage, <i>Salmonella</i> spp.	Restrict from care of high-risk patients	Until symptoms resolve; consult with local and state health authorities regarding need for negative stool cultures	IB
Diphtheria	Exclude from duty	Until antimicrobial therapy completed and 2 cultures obtained ≥ 24 hours apart are negative	IB
Enteroviral infections	Restrict from care of infants, neonates, and immunocompromised patients and their environments	Until symptoms resolve	II
Hepatitis A	Restrict from patient contact, contact with patient's environment, and food handling	Until 7 days after onset of jaundice	IB
Hepatitis B			
Personnel with acute or chronic hepatitis B surface antigenemia who do not perform exposure-prone procedures	No restriction*; refer to state regulations; standard precautions should always be observed		II
Personnel with acute or chronic hepatitis B e antigenemia who perform exposure-prone procedures	Do not perform exposure-prone invasive procedures until counsel from an expert review panel has been sought; panel should review and recommend procedures the worker can perform, taking into account specific procedure as well as skill and technique of worker; refer to state regulations	Until hepatitis B e antigen is negative	II
Hepatitis C	No recommendation		Unresolved issue
Herpes simplex			
Genital	No restriction		II
Hands (herpetic whitlow)	Restrict from patient contact and contact with the patient's environment	Until lesions heal	IA
Orofacial	Evaluate for need to restrict from care of high-risk patients		II
Human immunodeficiency virus	Do not perform exposure-prone invasive procedures until counsel from an expert review panel has been sought; panel should review and recommend procedures the worker can perform, taking into account specific procedure as well as skill and technique of the worker; standard precautions should always be observed; refer to state regulations		II

Continued

*Unless epidemiologically linked to transmission of infection

†Those susceptible to varicella and who are at increased risk of complications of varicella, such as neonates and immunocompromised persons of any age.

‡ High-risk patients as defined by the ACIP for complications of influenza.

Table 3. Continued

Disease/problem	Work restriction	Duration	Category
Measles			
Active	Exclude from duty	Until 7 days after the rash appears	IA
Postexposure (susceptible personnel)	Exclude from duty	From 5th day after 1st exposure through 21st day after last exposure and/or 4 days after rash appears	IB
Meningococcal infections	Exclude from duty	Until 24 hours after start of effective therapy	IA
Mumps			
Active	Exclude from duty	Until 9 days after onset of parotitis	IB
Postexposure (susceptible personnel)	Exclude from duty	From 12th day after 1st exposure through 26th day after last exposure or until 9 days after onset of parotitis	II
Pediculosis	Restrict from patient contact	Until treated and observed to be free of adult and immature lice	IB
Pertussis			
Active	Exclude from duty	From beginning of catarrhal stage through 3rd wk after onset of paroxysms or until 5 days after start of effective antimicrobial therapy	IB
Postexposure (asymptomatic personnel)	No restriction, prophylaxis recommended		II
Postexposure (symptomatic personnel)	Exclude from duty	Until 5 days after start of effective antimicrobial therapy	IB
Rubella			
Active	Exclude from duty	Until 5 days after rash appears	IA
Postexposure (susceptible personnel)	Exclude from duty	From 7th day after 1st exposure through 21st day after last exposure	IB
Scabies			
<i>Staphylococcus aureus</i> infection			
Active, draining skin lesions	Restrict from contact with patients and patient's environment or food handling	Until lesions have resolved	IB
Carrier state	No restriction, unless personnel are epidemiologically linked to transmission of the organism		IB
Streptococcal infection, group A	Restrict from patient care, contact with patient's environment, or food handling	Until 24 hours after adequate treatment started	IB
Tuberculosis			
Active disease	Exclude from duty	Until proved noninfectious	IA
PPD converter	No restriction		IA

Continued

Table 3. Continued

Disease/problem	Work restriction	Duration	Category
Varicella			
Active	Exclude from duty	Until all lesions dry and crust	IA
Postexposure (susceptible personnel)	Exclude from duty	From 10th day after 1st exposure through 21st day (28th day if VZIG given) after last exposure	IA
Zoster			
Localized, in healthy person	Cover lesions; restrict from care of high-risk patients†	Until all lesions dry and crust	II
Generalized or localized in immunosuppressed person	Restrict from patient contact	Until all lesions dry and crust	IB
Postexposure (Susceptible personnel)	Restrict from patient contact	From 10th day after 1st exposure through 21st day (28th day if VZIG given) after last exposure or, if varicella occurs, until all lesions dry and crust	IA
Viral respiratory infections, acute febrile	Consider excluding from the care of high risk patients‡ or contact with their environment during community outbreak of RSV and influenza	Until acute symptoms resolve	IB

6. Health counseling

Access to adequate health counseling for personnel is another crucial element of an effective personnel health service. Health counseling allows personnel to receive individually targeted information regarding (a) the risk and prevention of occupationally acquired infections, (b) the risk of illness or other adverse outcome after exposures, (c) management of exposures, including the risks and benefits of postexposure prophylaxis regimens, and (d) the potential consequences of exposures or communicable diseases for family members, patients, or other personnel, both inside and outside the health care facility.

7. Maintenance of records, data management, and confidentiality

Maintenance of records on medical evaluations, immunizations, exposures, postexposure prophylaxis, and screening tests in a retrievable, preferably computerized, database allows efficient monitoring of the health status of personnel. Such record keeping also helps to ensure that the organization will provide consistent and appropriate services to health care personnel.

Individual records for all personnel should be maintained in accordance with the Occupational Safety and Health Administration

(OSHA) medical records standard, which requires the employer to retain records, maintain employee confidentiality, and provide records to employees when they ask to review them.²⁶ In addition, the 1991 OSHA "Occupational Exposure to Bloodborne Pathogens; Final Rule"²⁷ requires employers, including health care facilities, to establish and maintain an accurate record for each employee with occupational exposure to bloodborne pathogens. The standard also requires that each employer ensure that the employee medical records are (a) kept confidential, (b) not disclosed or reported without the employee's express written consent to any person within or outside the workplace, except as required by law, and (c) maintained by the employer for at least the duration of the worker's employment plus 30 years.

OSHA's record keeping regulation also requires employers to record work-related injuries and illnesses on the OSHA 200 log and the OSHA 101 form. The records include all occupational fatalities, all occupational illnesses, and occupational injuries that result in loss of consciousness, restriction of work or motion, transfer to another job, or medical treatment beyond first aid. Infectious diseases are recordable if they are work related and result in illness.²⁸

More recently, OSHA developed policies that require the recording of positive tuberculin skin-test results.²⁹ It would be beneficial to health care organizations and personnel if the principles of record keeping and confidentiality mandated by OSHA were to be expanded to other work-related exposures and incidents, immunizations, TB screening, and investigation and management of nosocomial outbreaks.

E. EPIDEMIOLOGY AND CONTROL OF SELECTED INFECTIONS TRANSMITTED AMONG HEALTH CARE PERSONNEL AND PATIENTS

Almost any transmissible infection may occur in the community at large or within health care organizations and can affect both personnel and patients. Only those infectious diseases that occur frequently in the health care setting or are most important to personnel are discussed here.

1. Bloodborne pathogens

a. Overview

Assessment of the risk and prevention of transmission of bloodborne pathogens, such as hepatitis B virus (HBV), hepatitis C virus (HCV), and human immunodeficiency virus (HIV), in health care settings are based on information from a variety of sources, including surveillance and investigation of suspected cases of transmission to health care personnel and patients, seroprevalence surveys of health care personnel and patients, and studies of the risk of seroconversion after exposure to blood or other body fluids from infected persons. In this document, the emphasis of the discussion of bloodborne pathogens will be on patient-to-personnel transmission.

The CDC has periodically issued and updated recommendations for prevention of transmission of bloodborne pathogens in health care settings; these provide detailed information and guidance.³⁰⁻⁴⁰ Also, in 1991 OSHA published a bloodborne pathogen standard that was based on the concept of universal precautions to prevent occupational exposure to bloodborne pathogens.²⁷ The use of standard precautions (which incorporates universal precautions), including appropriate handwashing and barrier precautions, will reduce contact with blood and body fluids.^{3,30,31,41} The use of engineering controls (e.g., safety devices) and changes in work practices (e.g., techniques to reduce handling of sharp instruments) can reduce the frequency of percutaneous injuries.^{41,42} In settings such as the operating room, changes in

instrument design and techniques for performing surgical procedures and modified personal barriers have been shown to reduce blood contacts.^{43,44} Despite adherence to standard precautions and implementation of some new techniques and devices, percutaneous injuries continue to occur. This is of concern because percutaneous injuries represent the greatest risk of transmission of bloodborne pathogens to health care personnel.⁴⁵ Only a few studies evaluating a limited number of safety devices have demonstrated a reduction in percutaneous injuries among health care workers.^{46,47} This document will not address the use of safety devices, because the Public Health Service is assessing the need for further guidance on selection, implementation, and evaluation of such devices in health care settings.

The risk posed to patients by health care personnel infected with bloodborne pathogens such as HBV and HIV has been the subject of much concern and debate. There are no data to indicate that infected workers who do not perform invasive procedures pose a risk to patients. Consequently, work restrictions for these workers are not appropriate. However, the extent to which infected workers who perform certain types of invasive procedures pose a risk to patients and the restrictions that should be imposed on these workers have been much more controversial. In 1991, CDC recommendations on this issue were published.⁴⁸ Subsequently, Congress mandated that each state implement the CDC guidelines or equivalent as a condition for continued federal public health funding to that state. Although all states have complied with this mandate, there is a fair degree of state-to-state variation regarding specific provisions. Local or state public health officials should be contacted to determine the regulations or recommendations applicable in a given area. CDC is currently in the process of reviewing relevant data regarding health care personnel-to-patient transmission of bloodborne pathogens.

b. Hepatitis B

Nosocomial transmission of HBV is a serious risk for health care personnel.⁴⁹⁻⁵³ Approximately 1000 health care personnel were estimated to have become infected with HBV in 1994. This 90% decline since 1985 is attributable to the use of vaccine and adherence to other preventive measures (e.g., standard precautions).⁵⁴ During the past decade, an estimated 100 to 200 health care personnel annually have died of occupationally acquired HBV infection.⁵⁴ The risk of acquiring

Table 4. Recommendation for postexposure prophylaxis for percutaneous or permucosal exposure to hepatitis B virus, United States

Vaccination and antibody status of exposed person	HBsAg seropositive	Treatment when source is HBsAg negative	Treatment when source is not tested or status is unknown
Unvaccinated	HBIG* × 1 and initiate HB vaccine series	Initiate HB vaccine series	Initiate HB
Previously vaccinated			
Known responder†	No treatment	No treatment	
Known nonresponder	HBIG* × 2 or HBIG* × 1 and initiate revaccination	No treatment	If known high-risk source, treat as if source were HBsAg positive
Antibody response unknown	Test exposed person for anti-HBs: (1) if adequate,† no treatment; (2) if inadequate,† HBIG × 1 and vaccine booster	No treatment	Test exposed person for anti-HBs: (1) if adequate,† no treatment; (2) if inadequate,† initiate revaccination

HBsAg, Hepatitis B surface antigen; HBIG, hepatitis B immune globulin; HB, hepatitis vaccine; anti-HBs, antibody to hepatitis B surface antigen.

*Dose 0.06 mg/kg IM.

†Responder is defined as a person with adequate serum levels of anti-HBs (≥10 mIU/ml); inadequate vaccination defined as serum anti-HBs <10 mIU/ml.

HBV infection from occupational exposure is dependent on the nature and frequency of exposure to blood or to body fluids containing blood.^{49,53} The risk of infection is at least 30% after a percutaneous exposure to blood from a hepatitis B e antigen–seropositive source.⁵⁴

HBV is transmitted by percutaneous or mucosal exposure to blood and serum-derived body fluids from persons who have either acute or chronic HBV infection. The incubation period is 45 to 180 days (average 60 to 90 days). Any person seropositive for hepatitis B surface antigen (HBsAg) is potentially infectious.

Hepatitis B vaccination of health care personnel who have contact with blood and body fluids can prevent transmission of HBV and is strongly recommended.^{9,10,40} The OSHA bloodborne pathogen standard mandates that hepatitis B vaccine be made available, at the employer's expense, to all health care personnel with occupational exposure to blood or other potentially infectious materials.²⁷ Provision of vaccine during training of health care professionals before such blood exposure occurs may both increase the vaccination rates among personnel and prevent infection among trainees, who are at increased risk for unintentional injuries while they are learning techniques.

Prevaccination serologic screening for susceptibility to HBV infection is not indicated for persons being vaccinated, unless the health care organization considers such screening to be cost-effective. Postvaccination screening for antibody to HBsAg (anti-HBs) is advised for personnel at ongoing risk for blood exposure to determine

whether response to vaccinations has occurred and to aid in determining the appropriate postexposure prophylaxis or the need for revaccination. Personnel who do not respond to or do not complete the primary vaccination series should be revaccinated with a second three-dose vaccine series or evaluated to determine whether they are HBsAg seropositive. Revaccinated persons should be tested for anti-HBs at the completion of the second vaccine series.⁹ If they do not respond, no further vaccination series should be given and they should be evaluated for the presence of HBsAg (possible chronic HBV infection). No specific work restrictions are recommended for nonresponders; in the event of percutaneous exposure to blood or body fluids, however, they should see their health care providers as soon as possible to evaluate the need for postexposure prophylaxis. Personnel in chronic dialysis centers who do not respond to vaccine need to be screened for HBsAg and anti-HBs every 6 months.⁵⁵

Vaccine-induced antibodies decline gradually with time, and as many as 60% of those who initially respond to vaccination will lose detectable anti-HBs by 8 years.⁵⁶ Booster doses of vaccine are not routinely recommended, because persons who respond to the initial vaccine series remain protected against clinical hepatitis and chronic infection even when their anti-HBs levels become low or undetectable.⁵⁷

The need for postexposure prophylaxis, vaccination, or both depends on the HBsAg status of the source of the exposure as well as the immunization status of the person exposed (Table 4).⁴⁰

Vaccine should be offered after any exposure in an unvaccinated person; if the source is known to be HBsAg seropositive, hepatitis B immune globulin (HBIG) should be given, preferably within 24 hours. The effectiveness of HBIG given later than 7 days after HBV exposure is unknown.^{8,10,40} If the source is HBsAg seropositive and the exposed person is known not to have responded to a three-dose vaccine series, a single dose of HBIG and a dose of hepatitis B vaccine need to be given as soon as possible after the exposure with subsequent vaccine doses given at 1 month and 6 months after the initial dose. If the exposed person is known not to have responded to a three-dose vaccine series and to revaccination, two doses of HBIG need to be given, one dose as soon as possible after exposure and the second dose 1 month later.

c. Hepatitis C

HCV is the etiologic agent in most cases of parenterally transmitted non-A, non-B hepatitis in the United States.^{58,59} During the past decade, the annual number of newly acquired HCV infections has ranged from an estimated 180,000 in 1984 to an estimated 28,000 in 1995. Of these, an estimated 2% to 4% occurred among health care personnel who were occupationally exposed to blood.⁵⁹

A case-control study of patients with acute non-A, non-B hepatitis, conducted before the identification of HCV, showed a significant association between acquisition of disease and health care employment, specifically patient care or laboratory work.⁶⁰ Seroprevalence studies among hospital-based health care personnel have shown seroprevalence rates of antibody to HCV (anti-HCV) ranging from 1% to 2%.⁶¹⁻⁶⁴ In a study that assessed risk factors for infection in health care personnel, a history of accidental needlesticks was independently associated with anti-HCV seropositivity.⁶¹

Several case reports have documented transmission of HCV infection from anti-HCV-seropositive patients to health care personnel as a result of accidental needlesticks or cuts with sharp instruments.^{65,66} In follow-up studies of health care personnel who sustained percutaneous exposures to blood from anti-HCV-seropositive patients, the rate of anti-HCV seroconversion averaged 1.8% (range 0% to 7%).⁶⁷⁻⁷⁰ In a study in which HCV RNA polymerase chain reaction methods were used to measure HCV infection, the rate of HCV transmission was 10%.⁷⁰

The incubation period for hepatitis C is 6 to 7 weeks, and nearly all persons with acute infec-

tion will have chronic HCV infection occur with persistent viremia and the potential for transmission of HCV to others.

Serologic assays to detect anti-HCV are commercially available. The interpretation of anti-HCV test results is limited by several factors: (a) these assays will not detect anti-HCV in approximately 5% of persons infected with HCV; (b) these assays do not distinguish between acute, chronic, and past infection; (c) there may be a prolonged interval between the onset of acute illness with HCV and seroconversion; and (d) when the assays are used in populations with a low prevalence of HCV infection, commercial screening assays for anti-HCV yield a high proportion (as great as 50%) of false-positive results.^{34,59} Although no true confirmatory test has been developed, supplemental tests for specificity are available and should be used to judge the validity of repeatedly reactive results by screening assays.

Although the value of immune globulin for postexposure prophylaxis after occupational exposure to HCV has been difficult to assess,⁷¹⁻⁷³ postexposure prophylaxis with immune globulin does not appear to be effective in preventing HCV infection. Current immune globulin preparations are manufactured from plasma that has been screened for HCV antibody; positive lots are excluded from use. An experimental study in chimpanzees found that administration 1 hour after exposure to HCV of immune globulin manufactured from anti-HCV-screened plasma did not prevent infection or disease.⁷⁴ Thus, available data do not support the use of immune globulin for postexposure prophylaxis against hepatitis C, and its use is not recommended. There is no information regarding the use of antiviral agents, such as interferon alfa, in the postexposure setting, and such prophylaxis is not recommended.³⁷

Health care institutions should consider implementing recommended policies and procedures for follow-up for HCV infection after percutaneous or mucosal exposures to blood. At a minimum, such policies can include (1) baseline testing of the source for anti-HCV, (2) baseline and follow-up testing (e.g., 6 months) for anti-HCV and alanine aminotransferase activity of the person exposed to an anti-HCV seropositive source, (3) confirmation by supplemental anti-HCV testing of all anti-HCV results reported as repeatedly active by enzyme immunoassay, (4) recommendation against postexposure prophylaxis with immune globulin or antiviral agents (e.g., interferon), and (5) education of health care personnel

about the risk for and prevention of bloodborne infections, including HCV, in occupational settings, with the information routinely updated to ensure accuracy.³⁷ Among health care personnel in the postexposure period, onset of HCV infection may be detected earlier by measuring HCV RNA with polymerase chain reaction rather than by measuring anti-HCV with enzyme immunoassay. However, polymerase chain reaction is not a licensed assay, and the accuracy of the results are highly variable.³⁷

d. Human immunodeficiency virus

Nosocomial transmission of human immunodeficiency virus (HIV) infection from patients to health care personnel may occur after percutaneous or, infrequently, mucocutaneous exposure to blood or body fluids containing blood. According to prospective studies of health care personnel percutaneously exposed to HIV-infected blood, the average risk for HIV infection has been estimated to be 0.3%.^{45,75-78} A retrospective case-control study to identify risk factors for HIV seroconversion among health care personnel after a percutaneous exposure to HIV-infected blood found that they were more likely to become infected if they were exposed to a larger quantity of blood, represented in the study as (1) presence of visible blood on the device before injury, (2) a procedure that involved a needle placed directly in the patient's vein or artery, or (3) deep injury.⁴⁵ Transmission of HIV infection also was associated with injuries in which the source patient was terminally ill with AIDS; this may be attributable to the increased titer of HIV in blood that is known to accompany late stages of illness or possibly to other factors, such as the presence of syncytia-inducing strains of HIV in these patients. In addition, the findings of this study suggested that the postexposure use of zidovudine may be protective for health care personnel.⁴⁵

Factors that determine health care personnel's risk of infection with HIV include the prevalence of infection among patients, the risk of infection transmission after an exposure, and the frequency and nature of exposures.⁷⁹ Most personnel who acquire infection after percutaneous exposure have HIV antibody develop within 6 months of exposure. HIV-infected persons are likely to transmit virus from the time of early infection throughout life.

In 1990, CDC published guidelines for postexposure management of occupational exposure to HIV,³³ and provisional recommendations for postexposure chemoprophylaxis were published

in 1996.⁸⁰ In 1998, both of these documents were updated and consolidated to reflect current scientific knowledge on the efficacy of postexposure prophylaxis and the use of antiretroviral therapies.⁸¹ The U.S. Public Health Service will periodically review scientific information on antiretroviral therapies and publish updated recommendations for their use as postexposure prophylaxis as necessary.

2. Conjunctivitis

Although conjunctivitis can be caused by a variety of bacteria and viruses, adenovirus has been the primary cause of nosocomial outbreaks of conjunctivitis. Nosocomial outbreaks of conjunctivitis caused by other pathogens are rare.

Adenoviruses, which can cause respiratory, ocular, genitourinary, and gastrointestinal infections, are a major cause of epidemic keratoconjunctivitis in the community and health care settings. Nosocomial outbreaks have primarily occurred in eye clinics or offices but have also been reported in neonatal intensive care units and long-term care facilities.⁸²⁻⁸⁶ Patients and health care personnel have acquired and transmitted epidemic keratoconjunctivitis during these outbreaks. The incubation period ranges from 5 to 12 days, and shedding of virus occurs from late in the incubation period to as long as 14 days after onset of disease.⁸³ Adenovirus survives for long periods on environmental surfaces; ophthalmologic instruments and equipment can become contaminated and transmit infection. Contaminated hands are also a major source of person-to-person transmission of adenovirus, both from patients to health care personnel and from health care personnel to patients. Handwashing, glove use, and disinfection of instruments can prevent the transmission of adenovirus.^{82,83}

Infected personnel should not provide patient care for the duration of symptoms after onset of epidemic keratoconjunctivitis^{82,83} or purulent conjunctivitis caused by other pathogens.

3. Cytomegalovirus

There are two principal reservoirs of cytomegalovirus (CMV) in health care institutions: (a) infants and young children infected with CMV and (b) immunocompromised patients, such as those undergoing solid-organ or bone-marrow transplantation or those with AIDS.⁸⁷⁻⁹⁴ However, personnel who provide care to such high-risk patients have a rate of primary CMV infection that

is no higher than that among personnel without such patient contact (3% vs 2%).⁹⁵⁻¹⁰¹ In areas where there are patient populations with a high prevalence of CMV, seroprevalence studies and epidemiologic investigations have also demonstrated that personnel who care for patients have no greater risk of acquiring CMV than do personnel who have no patient contact.^{92,95-98,100,102-107} In addition, epidemiologic studies that included DNA testing of viral strains have demonstrated that personnel who acquired CMV infections while providing care to CMV-infected infants had not acquired their infections from the CMV-infected patients.^{88,92,96,108-110}

CMV transmission appears to occur directly either through close, intimate contact with an excreter of CMV or through contact with contaminated secretions or excretions, especially saliva or urine.^{101,111-114} Transmission by the hands of personnel or infected persons has also been suggested.^{92,115} The incubation period for person-to-person transmission is not known. Although CMV can survive on environmental surfaces and other objects for short periods,¹¹⁶ there is no evidence that the environment plays a role in the transmission of infection.⁹²

Because infection with CMV during pregnancy may have adverse effects on the fetus, women of childbearing age need to be counseled regarding the risks and prevention of transmission of CMV in both nonoccupational and occupational settings.¹¹⁷ Although most fetal infections follow primary maternal infection, fetal infection may follow maternal reinfection or reactivation.^{118,119} There are no studies that clearly indicate that seronegative personnel may be protected from infection by transfer to areas with less contact with patients likely to be reservoirs for CMV infection.^{88,92,95-97,102,105,106,119,120}

Serologic or virologic screening programs to identify CMV-infected patients or seronegative female personnel of childbearing age are impractical and costly for the following reasons: (a) the virus can be intermittently shed,¹²¹ and repeated screening tests may be needed to identify shedders; (b) seropositivity for CMV does not offer complete protection against maternal reinfection or reactivation and subsequent fetal infection^{118,119}; and (c) no currently available vaccines¹²²⁻¹²⁵ or prophylactic therapy^{90,126-129} can provide protection against primary infection.

Work restrictions for personnel who contract CMV illnesses are not necessary. The risk of transmission of CMV can be reduced by careful adherence to handwashing and standard precautions.^{3,119,130}

4. Diphtheria

Nosocomial transmission of diphtheria among patients and personnel has been reported.¹³¹⁻¹³³ Diphtheria is currently a rare disease in the United States. During 1980 through 1994, only 41 diphtheria cases were reported¹³⁴; however, community outbreaks of diphtheria have occurred in the past,¹³⁵ and clusters of infection may occur in communities where diphtheria was previously endemic.¹³⁶ In addition, diphtheria epidemics have been occurring since 1990 in the new independent states of the former Soviet Union¹³⁷⁻¹³⁹ and in Thailand.¹⁴⁰ At least 20 imported cases of diphtheria have been reported in countries in Europe,^{139,141} and two cases occurred in U.S. citizens visiting or working in the Russian Federation and Ukraine.¹⁴² Health care personnel are not at substantially higher risk than the general adult population for acquiring diphtheria; however, there is a potential for sporadic or imported cases to require medical care in the United States.

Diphtheria, caused by *Corynebacterium diphtheriae*, is transmitted by contact with respiratory droplets or contact with skin lesions of infected patients. The incubation period is usually 2 to 5 days. Patients with diphtheria are usually infectious for 2 weeks or less, but communicability can persist for several months.¹⁴³ Droplet precautions are recommended for patients with pharyngeal symptoms, and contact precautions are recommended for patients with cutaneous lesions. Precautions need to be maintained until antibiotic therapy is completed and results of two cultures taken at least 24 hours apart are negative.³

Limited serosurveys conducted since 1977 in the United States indicate that 22% to 62% of adults 18 to 39 years old may lack protective diphtheria antibody levels.¹⁴⁴⁻¹⁴⁸ Prevention of diphtheria is best accomplished by maintaining high levels of diphtheria immunity among children and adults.^{19,137,138} Immunization with tetanus and diphtheria toxoid (Td) is recommended every 10 years for all adults who have completed the primary immunization series (Table 1).^{9,19} Health care personnel need to consider obtaining Td immunization from their health care providers.⁹

To determine whether health care personnel directly exposed to oral secretions of patients infected with toxigenic strains of *C. diphtheriae* are carriers, cultures of the nasopharynx may be obtained. Exposed personnel need to be evaluated for evidence of disease daily for 1 week.¹⁴⁹ Although the efficacy of antimicrobial prophylaxis in preventing secondary disease has not been proved, prophylaxis with either a single intramuscular

Table 5. Selected reported etiologic agents causing community-acquired or nosocomially acquired gastrointestinal infections in developed countries

Agent	Community-acquired, patients	Nosocomially acquired, patients	Nosocomially acquired, health care personnel
Bacterial			
<i>Bacillus cereus</i>	++	0	0
<i>Campylobacter</i> species	++++	+	0
<i>Clostridium difficile</i>	+	++++	+
<i>Clostridium perfringens</i>	+	+	0
Diarrheogenic <i>Escherichia coli</i>	++++	++	+
<i>Salmonella</i> species	+++	++	+
<i>Shigella</i> species	++	+	+
<i>S. aureus</i> , toxigenic	+++	+++	0
<i>Yersinia enterocolitica</i>	+	+	+
Viral			
Adenovirus	++	+	+
Astrovirus	*	*	?
Calicivirus (Norwalk and Norwalk-like viruses or SRSVs)	*	*	*
Coxsackievirus	++	+	+
Rotavirus	++++	++++	++
Fungal			
<i>Candida</i> species	+	+	0
<i>Cryptococcus neoformans</i>	++	+	0
Parasitic			
<i>Cryptosporidium</i>	++	+	+
<i>Cyclospora</i>	++	0	0
<i>Entamoeba histolytica</i>	++	+	0
<i>Giardia lamblia</i>	++	+	0
<i>Isospora belli</i>	+	0	0
<i>Strongyloides</i>	+	0	0

++++, Most frequently reported; +++, reported often; ++, occasionally reported; +, rarely reported; 0, never reported; *, common but rarely reported because of limited availability of diagnostic assays; ?, unknown; SRSV, small round-structured viruses.

injection of benzathine penicillin (1.2 mouse units) or oral erythromycin (1 gm/day) for 7 days has been recommended.¹⁹ Follow-up nasopharyngeal cultures for *C. diphtheriae* need to be obtained at least 2 weeks after antimicrobial therapy is completed. If the organism has not been eradicated, a 10-day course of erythromycin needs to be given.¹⁴⁹ In addition, previously immunized exposed personnel need to receive a dose of Td if they have not been vaccinated within the previous 5 years.¹⁹

Exclusion from duty is indicated for personnel with *C. diphtheriae* infection or those determined to be asymptomatic carriers until antimicrobial therapy is completed and nasopharyngeal culture results are negative.

5. Gastrointestinal infections, acute

Gastrointestinal infections may be caused by a variety of agents, including bacteria, viruses, and protozoa. However, only a few agents have been documented in nosocomial transmission (Table 5).¹⁵⁰⁻¹⁶⁸ Nosocomial transmission of agents that cause gastrointestinal infections usually results

from contact with infected individuals,^{150,161,163,169} from consumption of contaminated food, water, or other beverages,^{150,166,169,170} or from exposure to contaminated objects or environmental surfaces.^{152,153,171} Airborne transmission of small round-structured viruses (Norwalk-like viruses) has been postulated but not proved.^{164,165,172-175} Inadequate handwashing by health care personnel¹⁷⁶ and inadequate sterilization or disinfection of patient-care equipment and environmental surfaces increase the likelihood of transmission of agents that cause gastrointestinal infections. Generally, adherence to good personal hygiene by personnel before and after all contacts with patients or food and to either standard or contact precautions³ will minimize the risk of transmitting enteric pathogens.^{167,177}

Laboratory personnel who handle infectious materials also may be at risk for occupational acquisition of gastrointestinal infections, most commonly with *Salmonella typhi*. Although the incidence of laboratory-acquired *S. typhi* infection has decreased substantially since 1955,

infections continue to occur among laboratory workers, particularly those performing proficiency exercises or research tests.^{151,162} Several typhoid vaccines are available for use in laboratory workers who regularly work with cultures or clinical materials containing *S. typhi*.¹⁷⁸ The oral live-attenuated Ty21a vaccine, the intramuscular Vi capsular polysaccharide vaccine, or the subcutaneous inactivated vaccine may be given (Table 1).¹⁷⁸ Booster doses of vaccine are required at 2- to 5-year intervals, depending on the preparation used. The live-attenuated Ty21a vaccine should not be used for immunocompromised persons, including those known to be infected with HIV.¹⁷⁸

Personnel who acquire an acute gastrointestinal illness (defined as vomiting, diarrhea, or both, with or without associated symptoms such as fever, nausea, and abdominal pain) are likely to have high concentrations of the infecting agent in their feces (bacteria, viruses, and parasites) or vomitus (viruses and parasites).^{165,179,180} It is important to determine the etiology of gastrointestinal illness in health care personnel who care for patients at high risk for severe disease (e.g., neonates, elderly persons, and immunocompromised patients). The initial evaluation of personnel with gastroenteritis needs to include a thorough history and determination of the need for specific laboratory tests, such as stool or blood cultures, staining procedures, and serologic or antigen-antibody tests.^{162,171,181,182}

After resolution of some acute bacterial gastrointestinal illnesses, some personnel may have persistent carriage of the infectious agent. Once the person has clinically recovered and is having formed stools, however, the risk of transmission of enteric pathogens is minimized by adherence to standard precautions.^{3,167} In addition, appropriate antimicrobial therapy may eradicate fecal carriage of *Shigella*¹⁸³ or *Campylobacter*.¹⁸⁴ In contrast, antimicrobial or antiparasitic therapy may not eliminate carriage of *Salmonella*¹⁸⁵ or *Cryptosporidium*. Moreover, antimicrobials may prolong excretion of *Salmonella*¹⁸⁶ and lead to emergence of resistant strains.¹⁸⁷ However, transmission of *Salmonella* to patients from personnel who are asymptomatic carriers of *Salmonella* has not been well documented.¹⁶⁷ In general, antimicrobial therapy is not recommended, unless the person is at high risk for severe disease.¹⁸⁸ When antibiotics are given, stool cultures

should be obtained at least 48 hours after completion of antibiotic therapy.

Restriction from patient care and the patient's environment or from food handling is indicated for personnel with diarrhea or acute gastrointestinal symptoms, regardless of the causative agent.^{3,171} Some local and state agencies have regulations that require work exclusion for health care personnel, food handlers, or both who have gastrointestinal infections caused by *Salmonella* or *Shigella*. These regulations may require such personnel to be restricted from duty until results of at least two consecutive stool cultures obtained at least 24 hours apart are negative.

6. Hepatitis A

Nosocomial hepatitis A occurs infrequently, and transmission to personnel usually occurs when the source patient has unrecognized hepatitis and is fecally incontinent or has diarrhea.¹⁸⁹⁻¹⁹⁸ Other risk factors for hepatitis A virus (HAV) transmission to personnel include activities that increase the risk of fecal-oral contamination such as (a) eating or drinking in patient care areas,^{189,191,193,199} (b) not washing hands after handling an infected infant,^{191,199,200} and (c) sharing food, beverages, or cigarettes with patients, their families, or other staff members.^{189,191}

HAV is transmitted primarily by the fecal-oral route. It has not been reported to occur after inadvertent needlesticks or other contact with blood, but it has rarely been reported to be transmitted by transfusion of blood products.^{193,201,202} The incubation period for HAV is 15 to 50 days. Fecal excretion of HAV is greatest during the incubation period of disease before the onset of jaundice.²⁰³ Once disease is clinically obvious, the risk of transmitting infection is decreased. However, some patients admitted to the hospital with HAV, particularly immunocompromised patients, may still be shedding virus because of prolonged or relapsing disease, and such patients are potentially infective.^{190,203} Fecal shedding of HAV, formerly believed to continue only as long as 2 weeks after onset of dark urine,²⁰³ has been shown to occur as late as 6 months after diagnosis of infection in premature infants.¹⁸⁹ Anicteric infection is typical in young children and infants.²⁰⁴

Personnel can protect themselves and others from infection with HAV by adhering to standard precautions.³ Food-borne transmission of

hepatitis A is not discussed in this guideline, but it has occurred in health care settings.^{205,206}

Two inactivated hepatitis A vaccines are now available and provide long-term preexposure protection against clinical infection with greater than 94% efficacy.²⁰⁴ Serologic surveys among health care personnel have not shown greater prevalence of HAV infection than in control populations^{52,192,207,208}; therefore, routine administration of vaccine in health care personnel is not recommended. Vaccine may be useful for personnel working or living in areas where HAV is highly endemic and is indicated for personnel who handle HAV-infected primates or are exposed to HAV in a research laboratory. The role of hepatitis A vaccine in controlling outbreaks has not been adequately investigated.⁹ Immune globulin given within 2 weeks after an HAV exposure is more than 85% effective in preventing HAV infection²⁰⁴ and may be advisable in some outbreak situations.^{9,204}

Restriction from patient care areas or food handling is indicated for personnel with HAV infection. They may return to regular duties 1 week after onset of illness.⁹

7. Herpes simplex

Nosocomial transmission of herpes simplex virus (HSV) is rare. Nosocomial transmission has been reported in nurseries²⁰⁹⁻²¹¹ and intensive care units^{212,213} where high-risk patients (e.g., neonates, patients with severe malnutrition, patients with severe burns or eczema, and immunocompromised patients) are located. Nosocomial transmission of HSV occurs primarily through contact either with primary or recurrent lesions or with virus-containing secretions, such as saliva, vaginal secretions, or amniotic fluid.^{210,212,214} Exposed areas of skin are the most likely sites of nosocomial infection, particularly when minor cuts, abrasions, or other skin lesions are present.²¹³ The incubation period of HSV is 2 to 14 days.²¹⁵ The duration of viral shedding has not been well defined.²¹⁶

Personnel may acquire a herpetic infection of the fingers (herpetic whitlow or paronychia) from exposure to contaminated oral secretions.^{213,214} Such exposures are a distinct hazard for nurses, anesthesiologists, dentists, respiratory care personnel, and other personnel who have direct (usually hand) contact with either oral lesions or respiratory secretions from patients.²¹³ Less frequently, personnel may acquire mucocutaneous infection on other body sites from contact with infectious body secretions.²¹⁷

Personnel with active infection of the hands (herpetic whitlow) can potentially transmit HSV infection to patients with whom they have contact.²¹⁴ Transmission of HSV from personnel with orofacial HSV infection to patients has also been infrequently documented²⁰⁹; however, the magnitude of this risk is unknown.^{211,218} Although asymptomatic infected persons can shed the virus, they are less infectious than persons with active lesions.^{216,219}

Personnel can protect themselves from acquiring HSV by adhering to standard precautions.³ The risk of transmission of HSV from personnel with orofacial infections to patients can be reduced by handwashing before all patient care and by the use of appropriate barriers, such as a mask or gauze dressing, to prevent hand contact with the lesion.

Because personnel with orofacial lesions may touch their lesions and potentially transmit infections, they should be evaluated to determine their potential for transmitting herpes simplex to patients at high risk for serious disease (e.g., neonates, patients with severe malnutrition, patients with severe burns or eczema, and immunocompromised patients) and excluded from the care of such patients as indicated. The evaluation should consider the extent of the lesion and the severity of illness in the patient population that personnel will contact. Personnel with HSV infections of the fingers or hands can more easily transmit infection and therefore need to be excluded from patient care until their lesions have crusted. In addition, herpetic lesions may be secondarily infected by *Staphylococcus* and *Streptococcus*, and personnel with such infections should be evaluated to determine whether they need to be excluded from patient contact until the secondary infection has resolved. There have been no reports that personnel with genital HSV infections have transmitted HSV to patients; therefore, work restrictions for personnel with genital herpes are not indicated.

8. Measles

Nosocomial transmission of measles virus (sporadic and epidemic) has been well described.²²⁰⁻²²⁹ From 1985 through 1991, approximately 3000 (4%) of all reported episodes of measles in the United States were probably acquired in a medical facility; of these, more than 700 (25%) occurred in health care personnel, many of whom were not vaccinated.⁹ Data have suggested that health care personnel have a risk of measles 13-fold that of the general population.⁹

Of the 2765 episodes of measles reported during 1992 through 1995, 385 (13.9%) occurred in health care settings.^{221,230}

Measles is transmitted both by large droplets during close contact between infected and susceptible persons and by the airborne route.^{229,231} Measles is highly transmissible and frequently misdiagnosed during the prodromal stage. The incubation period for measles is 5 to 21 days. Immunocompetent persons with measles shed the virus from the nasopharynx, beginning with the prodrome until 3 to 4 days after rash onset; immunocompromised persons with measles may shed virus for extended periods.²³²

Strategies to prevent nosocomial transmission of measles include (a) documentation of measles immunity in health care personnel, (b) prompt identification and isolation of persons with fever and rash, and (c) adherence to airborne precautions for suspected and proven cases of measles.³

It is essential that all personnel have documentation of measles immunity, regardless of their length of employment or whether they are involved in patient care. Further, some states have regulations requiring measles immunity for health care personnel. Although persons born before 1957 are generally considered to be immune to measles, serologic studies indicate that 5% to 9% of health care personnel born before 1957 may not be immune.^{9,233,234} Furthermore, during 1985 through 1989, 29% of all measles cases in U.S. health care personnel occurred in those born before 1957.²²¹ Consideration should be given to recommending a dose of measles-mumps-rubella trivalent vaccine (MMR) to personnel born before 1957 who are unvaccinated and who lack (a) a history of previous measles disease, (b) documentation of receipt of one dose of live-measles vaccine, and (c) serologic evidence of measles immunity.⁹ Health care personnel born during or after 1957 should be considered immune to measles when they have (a) documentation of physician-diagnosed measles, (b) documentation of two doses of live measles vaccine on or after their first birthday, or (c) serologic evidence of measles immunity (persons with an "indeterminate" level of immunity on testing should be considered susceptible). Persons born between 1957 and 1984 who received childhood measles immunization were given only one dose of vaccine during infancy and may require a second dose of vaccine.⁸

Serologic screening for measles immunity is not necessary before administration of measles vaccine, unless the medical facility considers it cost-effective or the person to be vaccinated requests it.²³⁵⁻²³⁸ When serologic screening before vaccination is done, tracking systems are needed to ensure that those identified as susceptible are subsequently vaccinated in a timely manner.²³⁷ During measles outbreaks, serologic screening before vaccination is not necessary. In outbreak situations, prompt administration of vaccine is necessary to halt disease transmission.

Work restrictions are necessary for personnel who acquire measles; they need to be excluded from duty for 7 days after the rash appears. Likewise, personnel not immune to measles need to be excluded from duty from 5 days after the first exposure to 21 days after the last exposure to measles.

9. Meningococcal disease

Community-acquired meningococcal disease is typically caused by a variety of serogroups of *Neisseria meningitidis*; serogroups B and C cause 46% and 45% of the endemic cases, respectively. Serogroups A, Y, and W-135 account for nearly all the remaining endemic cases.¹⁵ In contrast, epidemic meningococcal disease has, since the early 1990s, been caused increasingly by serogroup C.^{15,239,240}

Nosocomial transmission of *N. meningitidis* is uncommon. In rare instances, when proper precautions were not used, *N. meningitidis* has been transmitted from patient to personnel, through contact with the respiratory secretions of patients with meningococcemia or meningococcal meningitis,²⁴¹⁻²⁴³ or through handling laboratory specimens.²⁴¹ Lower respiratory tract infections caused by *N. meningitidis* may present a greater risk of transmission than either meningococcemia or meningitis,^{243,244} especially if the patient has an active, productive cough.²⁴⁴ The risk of personnel acquisition of meningococcal disease from casual contact (e.g., cleaning rooms or delivering food trays) appears to be negligible.²⁴⁴

N. meningitidis infection is probably transmitted by large droplets; the incubation period is from 2 to 10 days, and patients infected with *N. meningitidis* are rendered noninfectious by 24 hours of effective therapy. Personnel who care for patients with suspected *N. meningitidis* infection can decrease their risk of infection by adhering to droplet precautions.³

Postexposure prophylaxis is advised for persons who have had intensive, unprotected contact (i.e., without wearing a mask) with infected patients (e.g., mouth-to-mouth resuscitation, endotracheal intubation, endotracheal tube management, or close examination of the oropharynx of patients).¹⁵ Antimicrobial prophylaxis can eradicate carriage of *N. meningitidis* and prevent infections in personnel who have unprotected exposure to patients with meningococcal infections.^{245,246}

Because secondary cases of *N. meningitidis* occur rapidly (within the first week) after exposure to persons with meningococcal disease,²⁴⁷ it is important to begin prophylactic therapy immediately after an intensive, unprotected exposure, often before results of antimicrobial testing are available. Prophylaxis administered later than 14 days after exposure is probably of limited or no value.¹⁵ Rifampin (600 mg orally every 12 hours for 2 days) is effective in eradicating nasopharyngeal carriage of *N. meningitidis*.²⁴⁵ Ciprofloxacin (500 mg orally) and ceftriaxone (250 mg intramuscularly) in single-dose regimens are also effective in reducing nasopharyngeal carriage of *N. meningitidis* and are reasonable alternatives to the multidose rifampin regimen.^{15,246} These antimicrobials may be useful when infections are caused by rifampin-resistant meningococci or rifampin is contraindicated. Rifampin and ciprofloxacin are not recommended for pregnant women.^{15,106,248,249}

The quadrivalent A,C,Y,W-135 polysaccharide vaccine has been used successfully to control community outbreaks caused by serogroup C,^{15,239,240,248} but its use is not recommended for postexposure prophylaxis in health care settings.¹⁵ However, preexposure vaccination may be considered for laboratory personnel who routinely handle soluble preparations of *N. meningitidis*.^{15,241}

Healthy persons may have nasopharyngeal carriage of *N. meningitidis*.^{245,250-252} Nosocomial transmission from carriers to personnel has not been reported. In the absence of exposures to patients with *N. meningitidis* infection, personnel who are asymptomatic carriers need not be identified, treated, or removed from patient care activities. However, personnel with meningococcal infection need to be excluded from duty until 24 hours after the start of effective therapy.

10. Mumps

Mumps transmission has occurred in hospitals and long-term care facilities housing adolescents and young adults.^{253,254} Most cases of

mumps in health care personnel have been community acquired.

Mumps is transmitted by contact with virus-containing respiratory secretions, including saliva; the portals of entry are the nose and mouth. The incubation period varies from 12 to 25 days and is usually 16 to 18 days. The virus may be present in saliva for 6 to 7 days before parotitis and may persist for as long as 9 days after onset of disease. Exposed personnel may be infectious for 12 to 25 days after their exposure, and many infected persons remain asymptomatic.²⁵⁵ Droplet precautions are recommended for patients with mumps; such precautions should be continued for 9 days after the onset of parotitis.³

An effective vaccination program is the best approach to prevention of nosocomial mumps transmission.¹² Vaccination with mumps virus vaccine is recommended, unless otherwise contraindicated, for all those who are susceptible to mumps;^{12,256} combined MMR is the vaccine of choice,²⁵⁷ especially when the recipient also is likely to be susceptible to measles, rubella, or both.

Personnel should be considered immune to mumps if they have (a) documentation of physician-diagnosed mumps, (b) documentation of receipt of one dose of live mumps vaccine on or after their first birthday, or (c) serologic evidence of immunity (individuals who have an “indeterminate” antibody level should be considered susceptible).¹² Most persons born before 1957 are likely to have been infected naturally and may be considered to be immune, even though they may not have had clinically recognized mumps. Outbreaks among highly vaccinated populations have occurred and have been attributed to primary vaccine failure.²⁵⁸

Work restrictions are necessary for personnel who acquire mumps; such restrictions should be imposed for 9 days after the onset of parotitis. Likewise, susceptible personnel who are exposed to mumps need to be excluded from duty from the 12th day after the first exposure until the 26th day after the last exposure.^{9,255}

11. Parvovirus

Human parvovirus B19 (B19) is the cause of erythema infectiosum (fifth disease), a common rash illness that is usually acquired in childhood. Immunocompetent persons infected with B19 may have an acute, self-limited arthropathy, with or without a rash or anemia of short duration. However, patients with preexisting anemia (e.g., patients with sickle-cell anemia or thalassemia)

may have aplastic crisis occur. Immunodeficient patients (e.g., patients with leukemia or AIDS) may become chronically infected with B19 and have chronic anemia.^{259,260}

Transmission of B19 to health care personnel from infected patients appears to be rare but has been reported.²⁶¹⁻²⁶⁵ In two investigations of health care personnel exposures to B19, the rate of infection among exposed nurses was not higher than the rate among unexposed control subjects.^{266,267} In another investigation of health care personnel exposed to a patient with undetected chronic B19 infection, none of the susceptible employees became infected.²⁶⁸ Personnel have acquired infection while working in laboratories or during the care of patients with B19-associated sickle-cell aplastic crises.^{263-265,269-271}

B19 may be transmitted through contact with infected persons, fomites, or large droplets.^{266,272,273} The incubation period is variable, depending on the clinical manifestation of disease, and ranges from 6 to 10 days.²⁶⁰ The period of infectivity also varies, depending on the clinical presentation or stage of disease. Persons with erythema infectiosum are infectious before the appearance of the rash, those with infection and aplastic crises for as long as 7 days after onset of illness, and persons with chronic infection for years.

Pregnant personnel are at no greater risk of acquiring B19 infection than are nonpregnant personnel; however, if a pregnant woman does acquire B19 infection during the first half of pregnancy, the risk of fetal death (fetal hydrops, spontaneous abortion, and stillbirth) is increased.^{274,275} Because of the serious nature of the consequences for the fetus, female personnel of childbearing age need to be counseled regarding the risk of transmission of B19 and appropriate infection control precautions.³

Isolation precautions are not indicated for most patients with erythema infectiosum because they are past their period of infectiousness at the time of clinical illness.^{271,274} However, patients in aplastic crisis from B19 or patients with chronic B19 infection may transmit the virus to susceptible health care personnel or other patients; therefore, patients with preexisting anemia who are admitted to the hospital with febrile illness and transient aplastic crises should remain on droplet precautions for 7 days and patients with known or suspected chronic infection with B19 should be placed on droplet precautions on admission and for the duration of hospitalization.^{3,263} Work restrictions are not necessary for personnel exposed to B19.

12. Pertussis

Nosocomial transmission of *Bordetella pertussis* has involved both patients and personnel; nonimmunized children are at greatest risk.²⁷⁶⁻²⁸⁰ Serologic studies of health care personnel indicate that personnel may be exposed to and infected with pertussis much more frequently than indicated by the occurrence of recognized clinical illness.^{277,279,281,282} In one such study, the level of pertussis agglutination antibodies was found to correlate with the degree of patient contact; the prevalence of such antibody was highest in pediatric house staff (82%) and ward nurses (71%) and lowest in nurses with administrative responsibilities (35%).²⁷⁷

Pertussis is highly contagious; secondary attack rates exceed 80% in susceptible household contacts.²⁸³⁻²⁸⁵ *B. pertussis* transmission occurs by contact with respiratory secretions or large aerosol droplets from the respiratory tracts of infected persons. The incubation period is usually 7 to 10 days. The period of communicability starts at the onset of the catarrhal stage and extends into the paroxysmal stage up to 3 weeks after onset of symptoms. Prevention of secondary transmission of pertussis is especially difficult during the early stages of the disease because pertussis is highly communicable in the catarrhal stage, when the symptoms are non-specific and the diagnosis is uncertain.

During nosocomial pertussis outbreaks, the risk of acquiring infection among patients or personnel is often difficult to quantify because exposure is not easily determined. Furthermore, clinical symptoms in adults are less severe than in children and may not be recognized as pertussis. Pertussis should be considered for any person seeking treatment with an acute cough lasting at least 7 days, particularly if accompanied by paroxysms of coughing, inspiratory whoop, or posttussive vomiting.^{280,281}

Prevention of transmission of *B. pertussis* in health care settings involves (a) early diagnosis and treatment of patients with clinical infection, (b) implementation of droplet precautions for infectious patients,³ (c) exclusion of infectious personnel from work, and (d) administration of postexposure prophylaxis to persons exposed to infectious patients.²⁷⁹ Patients with suspected or confirmed pertussis who are admitted to the hospital need to be placed on droplet precautions until they have clinical improvement and have received antimicrobial therapy for at least 5 days.

Vaccination of adolescents and adults with whole-cell *B. pertussis* vaccine is not recommended¹⁹ because local and systemic reactions have

been observed more frequently in these groups than in children. Acellular pertussis vaccine is immunogenic in adults and carries a lower risk of adverse events than does whole-cell vaccine.^{280,286} However, the acellular vaccine has not been licensed for use in persons 7 years old or older. Because immunity among vaccine recipients wanes 5 to 10 years after the last vaccine dose (usually given at 4 to 6 years of age), personnel may play an important role in transmitting pertussis to susceptible infants. However, additional studies are needed to assess whether booster doses of acellular vaccines are indicated for adults.

Postexposure prophylaxis is indicated for personnel exposed to pertussis; a 14-day course of either erythromycin (500 mg orally four times daily) or trimethoprim-sulfamethoxazole (one tablet twice daily) has been used for this purpose. The efficacy of such prophylaxis has not been well documented, but studies suggest that it may minimize transmission.^{19,279,287,288} There are no data on the efficacy of newer macrolides (clarithromycin or azithromycin) for prophylaxis in persons exposed to pertussis.

Restriction from duty is indicated for personnel with pertussis from the beginning of the catarrhal stage through the third week after onset of paroxysms, or until 5 days after the start of effective antimicrobial therapy. Exposed personnel do not need to be excluded from duty.

13. Poliomyelitis

The last cases of indigenously acquired wild-virus poliomyelitis occurred in the United States in 1979.²⁸⁹ Since then, all cases of endemic poliomyelitis reported in the United States (5 to 10 endemic cases/year) have been related to the administration of oral polio vaccine (OPV).²¹ Although the risk of transmission of poliovirus in the United States is very low, wild poliovirus may potentially be introduced into susceptible populations with low immunization levels.

Poliovirus is transmitted through contact with feces or urine of infected persons but can be spread by contact with respiratory secretions and, in rare instances, through items contaminated with feces. The incubation period for nonparalytic poliomyelitis is 3 to 6 days, but is usually 7 to 21 days for paralytic polio.²⁹⁰ Communicability is greatest immediately before and after the onset of symptoms, when the virus is in the throat and excreted in high concentration in feces. The virus can be recovered from the throat for 1 week and from feces for several weeks to months after onset of symptoms.

Vaccine-associated poliomyelitis may occur in the recipient (7 to 21 days after vaccine administration) or susceptible contacts of the vaccine recipient (20 to 29 days after vaccine administration).²⁸⁹ Adults have a slightly increased risk of vaccine-associated paralytic poliomyelitis after receipt of OPV; therefore, inactivated poliovirus vaccine (IPV) should be used when adult immunization is warranted.^{8,16,21} Also, because immunocompromised persons may be at greater risk for development of poliomyelitis after exposure to vaccine virus, IPV rather than OPV is recommended when vaccinating pregnant or immunocompromised personnel, or personnel who may have contact with immunocompromised patients.^{8,16,21,290}

Health care personnel who may have contact with patients excreting wild virus (e.g., imported poliomyelitis case) and laboratory personnel handling specimens containing poliovirus or performing cultures to amplify virus should receive a complete series of polio vaccine; if previously vaccinated, they may require a booster dose of either IPV or OPV.^{8,21} For situations where immediate protection is necessary (e.g., an imported case of wild-virus poliomyelitis requiring care), additional doses of OPV should be given to adults who have previously completed a polio vaccine series.²¹

14. Rabies

Human rabies cases occur primarily from exposure to rabid animals. Cases of human rabies have increased in the United States during the 1990s.²⁹¹ Laboratory and animal care personnel who are exposed to infected animals, their tissues, and their excretions are at risk for the disease. Also, rabies transmission to laboratory personnel has been reported in vaccine production and research facilities after exposure to high-titered infectious aerosols.^{292,293} Theoretically, rabies may be transmitted to health care personnel from exposures (bite and nonbite) to saliva from infected patients, but no cases have been documented after these types of exposures.²⁹⁴

It is also possible for rabies to be transmitted when other potentially infectious material (such as brain tissue) comes into contact with nonintact skin or mucous membranes.^{22,294} Bites that penetrate the skin, especially bites to the face and hands, pose the greatest risk of transmission of rabies virus from animals to human beings.²² The incubation period for rabies is usually 1 to 3 months, but longer periods have been reported.²⁹⁵

Exposures to rabies can be minimized by adhering to standard precautions when caring for persons with suspected or confirmed rabies³ and by

using proper biosafety precautions in laboratories.⁵ Preexposure vaccination has been recommended for all personnel who (a) work with rabies virus or infected animals or (b) engage in diagnostic, production, or research activities with rabies virus.^{5,22} Consideration also may be given to providing preexposure vaccination to animal handlers when research animals are obtained from the wild, rather than from a known supplier that breeds the animals.

Postexposure prophylaxis has been administered to health care personnel after exposures to patients with rabies (Table 1),²⁹⁵⁻²⁹⁷ but decisions regarding postexposure prophylaxis should be made on a case-by-case basis after discussion with public health authorities.²²

15. Rubella

Nosocomial transmission of rubella has occurred from both male and female personnel to other susceptible personnel and patients, as well as from patients to susceptible personnel and other patients.²⁹⁸⁻³⁰⁵

Rubella is transmitted by contact with nasopharyngeal droplets from infected persons. The incubation period is variable but may range from 12 to 23 days; most persons have the rash 14 to 16 days after exposure. The disease is most contagious when the rash is erupting, but virus may be shed from 1 week before to 5 to 7 days after the onset of the rash.³⁰⁶ Rubella in adults is usually a mild disease, lasting only a few days; 30% to 50% of cases may be subclinical or inapparent.

Droplet precautions are used to prevent transmission of rubella. Infants with congenital rubella may excrete virus for months to years; when caring for such patients, it is therefore advisable to use contact precautions for the first year of life, unless nasopharyngeal and urine culture results are negative for rubella virus after 3 months of age.³

Ensuring immunity among all health care personnel (male and female) is the most effective way to eliminate nosocomial transmission of rubella.^{8,9,14,256,307} Persons should be considered susceptible to rubella if they lack (a) documentation of one dose of live rubella vaccine on or after their first birthday and (b) laboratory evidence of immunity (persons with indeterminate levels are considered susceptible). A history of previous rubella infection is unreliable and should not be considered indicative of immunity to rubella. Although birth before 1957 is generally considered acceptable evidence of rubella immunity, a dose of MMR has been recommend-

ed for those health care personnel that do not have laboratory evidence of immunity.⁹ In addition, birth before 1957 is not considered acceptable evidence of rubella immunity for women of childbearing age; history of vaccination or laboratory evidence of rubella immunity is particularly important for women who may become pregnant.⁹ Voluntary immunization programs are usually inadequate to ensure personnel protection.^{7,308} Because many health departments mandate rubella immunity for health care personnel, personnel health programs should consult with their local or state health departments before establishing policies for their facilities.

Serologic screening of personnel for immunity to rubella need not be done before vaccinating against rubella, unless the medical facility considers it cost-effective or the person getting vaccinated requests it.^{7,235-237} When serologic screening before vaccination is done, tracking systems are needed to ensure that those identified as susceptible are subsequently vaccinated in a timely manner.²³⁷ Likewise, during rubella outbreaks, serologic screening is not necessary. Pregnant women who are already immune to rubella are not at increased risk for adverse events.³⁰⁹ However, for theoretic reasons, a risk to the fetus from administration of live-virus vaccines cannot be excluded. Women should be counseled to avoid pregnancy for 30 days after administration of MMR or other rubella-containing vaccines. Routine precautions for vaccinating postpubertal women include (a) asking whether they are or may be pregnant, (b) not vaccinating those who say they are or may be pregnant, and (c) vaccinating those who state they are not pregnant after the potential risk to the fetus has been explained. If a pregnant woman is vaccinated or a woman becomes pregnant within 3 months after vaccination, she should be counseled about the theoretic basis of concern for the fetus, but MMR vaccination during pregnancy should not ordinarily be a reason to consider termination of pregnancy. Rubella-susceptible women who are not vaccinated because of pregnancy should be counseled about the importance of being vaccinated as soon as they are no longer pregnant.⁹ MMR is the vaccine of choice for rubella, especially when the recipient also is likely to be susceptible to measles, mumps, or both (Table 2).

Work restrictions are necessary for personnel who acquire rubella; ill personnel need to be excluded from duty for 5 days after the rash appears. Likewise, personnel susceptible to rubella require exclusion from duty from the seventh

day after the first exposure through the 21st day after the last exposure (Table 3).

16. Scabies and pediculosis

a. Scabies

Scabies is caused by infestation with the mite *Sarcoptes scabiei*. The conventional (typical) clinical presentation of scabies includes intense pruritus and cutaneous tracks, where mites have burrowed into the skin. Crusted or “Norwegian” scabies may develop among immunocompromised and elderly individuals in which their skin may become hyperkeratotic; pruritus may not be present, which also makes diagnosis difficult. In conventional scabies, 10 to 15 mites are present, whereas in crusted scabies, thousands of mites are harbored in the skin, increasing the potential for transmission.^{310,311}

Nosocomial outbreaks of scabies have occurred in a variety of health care settings, including intensive care units,³¹² rehabilitation centers,³¹³ long-term care facilities,^{314,315} hospital wards,³¹⁶ a dialysis unit,³¹⁷ and a health care laundry.³¹⁸ In recent years there has been an increase in the occurrence of crusted scabies among immunocompromised patients, particularly persons with HIV, which has led to the transmission of scabies among personnel, patients, and their families.^{310,312-316,319-321}

Nosocomial transmission of scabies occurs primarily through prolonged skin-to-skin contact with an infested person who has conventional scabies.^{310,322} Shorter periods of skin-to-skin contact with persons who have crusted scabies may result in transmission of scabies.³²³ Personnel have acquired scabies while performing patient care duties such as sponge bathing, lifting, or applying body lotions.^{310,311,319,324} Transmission by casual contact, such as by holding hands, or through inanimate objects, such as infested bedding, clothes, or other fomites, has been reported infrequently.^{317,318}

The use of contact precautions when taking care of infested patients before application of scabicides can decrease the risk of transmission to personnel.^{3,311} Routine cleaning of the environment of patients with typical scabies, especially bed linens and upholstered furniture, will aid in eliminating the mites. Additional environmental cleaning procedures may be warranted for crusted scabies.^{310,311,325-327}

Recommendations for treatment and control of scabies in health care institutions have been published previously.^{310,311,327-331} The recommend-

ed topical scabicides include permethrin cream (5%), crotamiton (10%), and lindane (1%) lotion; resistance to and adverse effects from lindane have been reported.³²⁹ Single-dose oral ivermectin has recently been shown to be an effective therapy for scabies^{323,330,332} but has not received Food and Drug Administration (FDA) approval for this purpose.

Most infested health care workers have typical scabies with low mite loads³³³; a single correct application of a scabicide is adequate and immediately decreases the risk of transmission.^{25,315-317,319,322,324,334} There are no controlled evaluations of the efficacy of prophylactic scabicide therapy among health care personnel, and some experts recommend two applications of scabicide for all infested personnel.^{311,315,321} If personnel continue to have symptoms after initial treatment, another application of scabicide may be needed. Persistent symptoms likely represent newly hatched mites rather than new infestation; however, pruritus after scabies infestation and treatment may persist for as long as 2 weeks, even without infestation.²⁵ Patients with crusted scabies may require repeated treatments and should be observed for recurrence of the mite infestation.^{310,311,314,326} Personnel who are exposed to scabies but lack signs of infestation do not usually require prophylactic treatment with scabicides. In outbreak situations where transmission continues to occur, prophylaxis may be warranted for both patients and exposed health care personnel.^{311,313}

Restrictions from patient care are indicated for personnel infested with scabies until after they receive initial treatment and have been medically evaluated and determined to be free of infestation. They should be advised to report for further evaluation if symptoms do not subside.

b. Pediculosis

Pediculosis is caused by infestation with any of three species of lice: *Pediculus humanus capitus* (human head louse), *Pediculus humanus corporis* (human body louse), and *Phthirus pubis* (pubic or crab louse).

Head lice are transmitted by head-to-head contact or by contact with infested fomites such as hats, combs, or brushes. Nosocomial transmission, although not common, has occurred.³¹⁰

Body lice are usually associated with poor hygiene and overcrowded conditions. Transmission occurs by contact with the skin or clothing of an infested person. Nosocomial transmission is unlikely.

Pubic lice are primarily found in the pubic hair but can be found in the axilla, eyelashes, or eyebrows. Transmission occurs primarily through intimate physical or sexual contact. Transmission by fomites, such as toilet seats or bedding, is uncommon. Nosocomial transmission is very unlikely.

Recommendations for control of pediculosis have been published previously.^{310,327,335} The drugs recommended for treatment include permethrin cream 1%, pyrethrins with piperonyl butoxide, malathion 0.5%, and lindane 1%.^{328-330,335} Resistance to various drugs has been reported. Local health departments may have information about drugs that are effective in their areas. Health care personnel exposed to patients with pediculosis do not require treatment, unless they show evidence of infestation.

Restriction from patient care is indicated for personnel with pediculosis until after they receive initial treatment and are found to be free of adult and immature lice. If symptoms do not subside after initial treatment, they should be advised to report for further evaluation.

17. *Staphylococcus aureus* infection and carriage

Staphylococcal infection and carriage occur frequently in human beings. In hospitals, the most important sources of *S. aureus* are infected and colonized patients. Previously, methicillin-susceptible (but penicillin-resistant) *S. aureus* accounted for most staphylococcal infections. In recent years, however, methicillin-resistant *S. aureus* has accounted for approximately 80% of all *S. aureus* isolates reported to the National Nosocomial Infections Surveillance System.^{336,337} The epidemiology of methicillin-resistant *S. aureus* does not appear to differ from that of methicillin-susceptible, penicillin-resistant *S. aureus*, except that outbreaks of methicillin-resistant *S. aureus* tend to occur more frequently among elderly or immunocompromised patients or among patients with severe underlying conditions.^{338,339}

Nosocomial transmission of *S. aureus* occurs primarily by the hands of personnel, which can become contaminated by contact with the colonized or infected body sites of patients.^{339,340} Hospital personnel who are infected or colonized with *S. aureus* also can serve as reservoirs and disseminators of *S. aureus*,³⁴¹⁻³⁴⁴ and infected dietary personnel have been implicated in staphylococcal food poisoning.³⁴⁵ The role of contaminated environmental surfaces in transmission of *S. aureus*

has rarely been well documented³⁴⁶ and remains controversial, although heavy contamination of fomites may facilitate transmission to patients by hands of personnel.³³⁹

The incubation period for *S. aureus* infections varies by type of disease. For food-borne illness it is 30 minutes to 6 hours, for bullous impetigo it is 1 to 10 days, for toxic shock syndrome it is usually 2 days, and for other types of infection it is variable.³⁴⁷

Carriage of *S. aureus* is most common in the anterior nares, but other sites, such as the hands, axilla, perineum, nasopharynx, and oropharynx, may also be involved.³³⁹ The frequency of nasal carriage of *S. aureus* among health care personnel ranges between 20% and 90%, but fewer than 10% of healthy nasal carriers disperse the organisms into the air.³⁴² Nasal carriers with upper respiratory symptoms can disseminate the organism more effectively.³⁴² Carriage of *S. aureus* in the nares has been shown to correspond to hand carriage,³³⁶ and persons with skin lesions caused by *S. aureus* are more likely than asymptomatic nasal carriers to disseminate the organism.

Culture surveys of personnel can detect carriers of *S. aureus* but do not indicate which carriers are likely to disseminate organisms. Thus, such surveys are not cost-effective and may subject personnel with positive culture results to unnecessary treatment and removal from duty. A more reasonable approach is to conduct active surveillance for nosocomial *S. aureus* infections. Culture surveys may be indicated if, after a thorough epidemiologic investigation, personnel are linked to infections. Such implicated personnel can then be removed from clinical duties until carriage has been eradicated.^{339,341,348-350}

Several antimicrobial regimens have been used successfully to eradicate staphylococcal carriage in health care personnel. These regimens include orally administered antimicrobial agents (e.g., rifampin, clindamycin, or ciprofloxacin) alone or in combination with another oral (e.g., trimethoprim-sulfamethoxazole) or topical (mupirocin) antimicrobial.^{349,351-363} Resistant *S. aureus* strains have emerged after the use of these oral or topical antimicrobial agents for eradication of *S. aureus* colonization.^{18,210,349,353,364-366} Thus, antimicrobial treatment to eradicate carriage may be best if limited to personnel who are carriers epidemiologically linked to disease transmission. Nosocomial transmission of *S. aureus* can be prevented by adherence to standard precautions and other forms of transmission-based precautions as needed.³

Restriction from patient-care activities or food handling is indicated for personnel who have draining skin lesions that are infected with *S. aureus* until they have received appropriate therapy and the infection has resolved. No work restrictions are necessary for personnel who are colonized with *S. aureus*, unless they have been epidemiologically implicated in *S. aureus* transmission within the facility.

18. *Streptococcus*, group A infection

Group A *Streptococcus* (GAS) has been transmitted from infected patients to health care personnel after contact with infected secretions,³⁶⁷⁻³⁶⁹ and the infected personnel have subsequently acquired a variety of GAS-related illnesses (e.g., toxic shock-like syndrome, cellulitis, lymphangitis, and pharyngitis). Health care personnel who were GAS carriers have infrequently been linked to sporadic outbreaks of surgical site, postpartum, or burn wound infections³⁷⁰⁻³⁷⁶ and to food-borne transmission of GAS causing pharyngitis.³⁷⁷ In these outbreaks, GAS carriage was documented in the pharynx,^{369,372,378} the skin,^{369,370} the rectum,^{369,375} and the female genital tract of the infected personnel.^{369,374,379}

The incubation period for GAS pharyngitis is 2 to 5 days, but for impetigo is 7 to 10 days. The incubation period is variable for other GAS infections.³⁸⁰

Culture surveys to detect GAS carriage among personnel are not warranted, unless personnel are epidemiologically linked to cases of nosocomial infection.³⁷⁸ In instances where thorough epidemiologic investigation has implicated personnel in nosocomial transmission, cultures may be obtained from skin lesions, pharynx, rectum, and vagina; GAS isolates obtained from personnel and patients can be serotyped to determine strain relatedness.³⁷³ Treatment of personnel carriers needs to be individually determined because (a) experience is limited regarding the treatment of personnel carriers implicated in GAS outbreaks and (b) carriage of the organism by personnel may be recurrent through long periods.^{369-371,374} Contact is the major mode of transmission of GAS in these health care settings. Airborne transmission during outbreaks has been suggested by several investigators, and some have demonstrated that exercising and changing of clothing can lead to airborne dissemination of GAS from rectal and vaginal carriage.^{369,374,375,379} Nosocomial transmission of GAS to personnel can be prevented by adherence to standard precautions or other transmission-based precautions as needed.³

Restriction from patient care activities and food handling is indicated for personnel with GAS infections until 24 hours after they have received appropriate therapy. However, no work restrictions are necessary for personnel who are colonized with GAS, unless they have been epidemiologically linked to transmission of infection within the facility.

19. Tuberculosis

Nosocomial transmission of tuberculosis (TB) is well documented, but such transmission in the United States is generally low. However, the risk may be increased in health care facilities located in communities with (a) high rates of HIV, (b) high numbers of persons from TB-endemic countries, and (c) communities with a high prevalence of TB infection.^{381,382} In some areas in the United States, the incidence and prevalence of multidrug-resistant *Mycobacterium tuberculosis* (MDR-TB) have also increased, and nosocomial MDR-TB outbreaks have occurred.³⁸³⁻³⁹¹ The increased risk of occupational acquisition of TB by health care personnel has been reported for decades, and it dramatically decreased after the introduction of effective antituberculous drugs.^{392,393} Skin-test conversion rates among health care personnel after routine skin testing have ranged from 0.11% to 10%.³⁹⁴ Among health care personnel with known exposure to an infectious patient with TB or involved in prolonged nosocomial outbreaks of TB, the skin-test conversion rates have ranged from 18% to 55%.^{383-385,388,389,393,395-401} Health care personnel with severely compromised immune systems, especially those infected with HIV^{381,402} and including those with malignancies or receiving immunosuppressive therapy, are at high risk for development of active disease after acquisition of tuberculous infection. It has been estimated that persons infected with *M. tuberculosis* and coinfecting with HIV have an 8% to 10% risk per year for development of active TB, whereas immunocompetent persons infected with TB have a 10% lifetime risk for active disease.⁴⁰³

The transmission of TB in health care facilities has been primarily caused by incomplete implementation of recommended TB infection control measures.³⁹⁶ In 1994, the CDC published detailed recommendations for the prevention of transmission of TB in health care settings, "Guidelines for Preventing the Transmission of *M. tuberculosis* in Health Care Facilities, 1994."³⁸² A summary of the recommendations pertaining to personnel health follows.

a. Strategies for prevention of transmission of TB

The risk of transmission of TB to or from personnel in a health care facility varies according to the type and size of the facility, the prevalence of TB in the community, the patient population served by the facility, the occupational group the person represents, the area of the facility where the person works, and the effectiveness of the facility's TB control program. A detailed risk assessment is essential in identifying the nature of TB control measures that are appropriate for a particular facility, as well as for specific areas and occupational groups within a facility.^{382,404} A risk assessment should include the following: (a) review of the community TB profile, (b) review of the number of patients with TB who were treated in each area of the facility, (c) review of the drug-susceptibility patterns of TB isolates from patients treated in the facility, (d) an analysis of purified protein derivative (PPD) skin-test results of health care personnel by work area or occupational group, (e) an evaluation of infection control parameters, including isolation policies, laboratory diagnostic capabilities, and antituberculous therapy regimens, (f) an observational review of TB infection control practices, and (g) evaluation of the function and maintenance of environmental controls.³⁸²

Transmission of TB can be minimized by developing and implementing an effective TB control program that is based on a hierarchy of controls: (a) administrative controls, (b) engineering controls, and (c) personal respiratory protection.^{382,384,386,393,396,404,405}

b. TB screening program

A TB screening program for personnel is an integral part of a health care facility's comprehensive TB control program. The screening program should be based on the facility-specific risk assessment. It may be advisable to screen immunocompromised personnel every 6 months.³⁸²

Baseline PPD testing of all personnel (including personnel with a history of bacille Calmette-Guérin [BCG] vaccination) during their preemployment physical examination or their application for hospital privileges will identify personnel who have been previously infected. For the baseline testing, a two-step procedure for personnel without a PPD test in the past 12 months can be used to minimize the likelihood of confusing reactivity from an old infection (boosting) with reactivity from a recent infection (conversion). Decisions concerning the use of the two-step pro-

cedure for baseline testing in a particular facility should be based on the frequency of boosting in that facility. Criteria used for interpretation of a PPD-test reaction may vary depending on (a) the purpose (diagnostic or epidemiologic) of the test, (b) the prevalence of TB infection in the population being tested, (c) the immune status of the host, and (d) any previous receipt of BCG immunization. Detailed recommendations for performing and interpreting skin tests have been published.^{382,406-408}

c. Follow-up evaluation

The risk assessment will show which health care personnel have the potential for exposure to *M. tuberculosis* and determine how frequently they should receive PPD testing. At a minimum, annual PPD testing is indicated for personnel with the potential for exposure to TB.

It is also important to obtain an initial chest radiograph for personnel with positive PPD-test reactions, documented PPD-test conversions, or pulmonary symptoms suggestive of TB. There are no data to support the use of routine chest radiographic examinations for asymptomatic PPD-negative personnel. In addition, personnel who have positive PPD-test reactions but also received adequate preventive treatment do not need repeat chest films, unless they have pulmonary symptoms suggestive of TB. Repeat chest radiographic examinations of such persons have not been shown to be beneficial or cost-effective in monitoring persons for development of disease. However, more frequent monitoring for symptoms of TB may be considered for personnel who had recent conversion of their PPD test and those persons who, if infected, are at increased risk for development of active TB (e.g., HIV-infected or otherwise severely immunocompromised persons).³⁸² Routine anergy testing of HIV-seropositive individuals is limited in its usefulness; however, anergy testing may be useful in guiding individual decisions regarding preventive therapy in selected situations.⁴⁰⁸

d. Management of personnel after exposure to TB

It is important to administer PPD tests to personnel as soon as possible after TB exposures are recognized. Such immediate PPD testing establishes a baseline with which subsequent PPD tests can be compared. A PPD test performed 12 weeks after the last exposure will indicate whether infection has occurred. Persons already known to have reactive PPD tests need not be retested. Personnel with evidence of new infection (i.e., PPD-test conversions) need to be evalu-

ated for active TB. If active TB is not diagnosed, preventive therapy should be considered.³⁸²

e. Preventive therapy

For workers with positive PPD-test results who were probably exposed to drug-susceptible TB, preventive therapy with isoniazid is indicated, unless there are contraindications to such therapy.^{382,407} Alternative preventive regimens have been proposed for persons who have positive PPD-test results after exposure to drug-resistant TB.⁴⁰⁹

f. Work restrictions

Personnel with active pulmonary or laryngeal TB may be highly infectious; exclusion from duty is indicated until they are noninfectious. If personnel are excluded from duty because of active TB, the facility should have documentation from their health care providers that personnel are noninfectious before they are allowed to return to duty. The documentation needs to include evidence that (a) adequate therapy is being received, (b) the cough has resolved, and (c) results of three consecutive sputum acid-fast bacilli (AFB) smears collected on different days are negative. After personnel resume duty and while they remain on anti-TB therapy, periodic documentation from their health care providers is needed to show that effective drug therapy is being maintained for the recommended period and that their sputum AFB smear results continue to be negative. If personnel discontinue their treatment, they need to be evaluated for active TB; directly observed therapy may be considered.

Work restrictions are not necessary for personnel receiving preventive treatment for latent TB (positive PPD-test result without active disease) or for personnel with latent TB who do not accept preventive therapy. However, these personnel should be instructed to seek evaluation promptly if symptoms suggestive of TB develop.

g. Considerations for BCG vaccine

BCG has not been routinely used in the United States to protect health care personnel. Nevertheless, because of the resurgence of TB in the United States and new information about the protective effect of BCG,^{410,411} the role of BCG vaccination in the prevention and control of TB in the country has been reevaluated.⁴¹² The following is a summary of the joint statement by the Advisory Council for the Elimination of Tuberculosis and ACIP regarding the use of BCG in health care personnel.

Two recent metaanalyses of 18⁴¹⁰ and 26⁴¹¹ BCG studies, respectively, indicate that the efficacy of BCG vaccine in preventing serious TB is high (>80%) in children and suggest 50% efficacy in adults. However, the protective efficacy of the vac-

cine in adolescents and adults, including health care personnel and HIV-infected children and adults, has not been determined.⁴¹²

BCG vaccination should not be used as a primary TB control strategy because (a) the protective efficacy of the vaccine in health care personnel is uncertain and (b) even if vaccination is effective in an individual, other persons in the health care facility are not protected against possible exposure to and infection with drug-resistant strains of *M. tuberculosis*. However, BCG vaccination may be indicated for health care personnel in a few geographic areas where the prevalence of MDR-TB is high, transmission of TB is likely, and TB infection control measures have been implemented but have not been successful in controlling nosocomial transmission.⁴¹² Consultation with local and state health departments is advisable when determining whether to provide BCG vaccination to health care personnel.

BCG vaccination often results in local adverse effects (such as muscular soreness, erythema, purulent drainage, and axillary or cervical lymphadenopathy) for as long as 3 months after vaccination; serious long-term complications (such as musculoskeletal lesions, multiple lymphadenitis, and disseminated BCG disease) are infrequent.⁴¹³⁻⁴¹⁵ The safety of BCG vaccination in immunocompromised populations (i.e., immunocompromised from immune deficiency diseases, HIV infection, leukemia, lymphoma, or generalized malignancy, or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation) has not been determined by adequate epidemiologic studies. However, because of the possibility of disseminated BCG infection in such persons,⁴¹⁶⁻⁴¹⁹ BCG vaccination is not recommended for immunocompromised personnel.⁴¹² The safety of BCG vaccination in pregnant women has also not been evaluated; therefore, it is not recommended for pregnant personnel.⁴¹²

PPD testing is not contraindicated for persons who have received BCG vaccine and can be used to support or exclude the diagnosis of infection with *M. tuberculosis*.⁴¹² PPD-test reactivity caused by BCG vaccination wanes with time⁴²⁰⁻⁴²² and is unlikely to persist longer than 10 years after vaccination in the absence of infection with *M. tuberculosis*.^{420,421} After a person has been vaccinated with BCG, the presence or size of a PPD-test reaction cannot be used to predict whether BCG will provide any protection against TB disease^{423,424} or to determine whether the reaction is caused by *M. tuberculosis* infection or the previous BCG

vaccination.⁴²⁵ However, a BCG-vaccinated person who has a PPD-test reaction of ≥ 10 mm induration should be considered infected with TB, especially if the vaccinee (a) is a contact of a person with infectious TB, particularly if the infectious person has transmitted *M. tuberculosis* to others, (b) is from a country with high prevalence of TB, or (c) is continually exposed to populations in which the prevalence of TB is high.⁴¹²

20. Vaccinia (smallpox)

Through aggressive surveillance for smallpox combined with the effective use of smallpox vaccine (vaccinia virus vaccine), the World Health Organization was able to declare the world free of smallpox in 1980. The smallpox vaccine licensed for use in the United States is derived from infectious vaccinia virus. After vaccination, the virus can be cultured from the vaccination site until the scab has separated from the skin (2 to 21 days after vaccination); thus, susceptible persons may acquire vaccinia from a recently vaccinated person.⁴²⁶⁻⁴²⁹ Covering the vaccination site and washing hands after contact with the vaccination site (including bandages) will prevent transmission. Recently, recombinant vaccinia viruses have been engineered to express immunizing agents of several viruses (e.g., herpesvirus, HBV, influenza). There is a theoretic risk that transmission could occur from contact with contaminated dressings or by contact with recombinant vaccine, but no such transmission has been reported among personnel who provide care to recipients of the recombinant vaccine. Infections also have been reported among laboratory personnel who handle viral cultures or materials contaminated with vaccinia or recombinant viruses.^{18,162}

Smallpox vaccination (every 10 years) is indicated for personnel who work directly with orthopox viruses (e.g., monkeypox, vaccinia, variola) or in animal care areas where orthopox viruses are studied. In selected instances, vaccination may be considered for personnel who provide care to recipients of recombinant vaccinia vaccine.^{9,18} Personnel who receive the vaccine may continue to have contact with patients if the vaccination site is covered and handwashing is strictly observed.¹⁸ Vaccine is not recommended for personnel with immunosuppression or eczema or for personnel who are pregnant.

21. Varicella

Nosocomial transmission of varicella-zoster virus (VZV) is well recognized.⁴³⁰⁻⁴⁴¹ Sources for nosocomial exposures have included patients, health care

personnel, and visitors (including the children of personnel) with either varicella or herpes zoster.

All susceptible adults in health care settings are at risk for varicella and its complications. During 1990 through 1994, fewer than 5% of varicella cases occurred among adults older than 20 years, but they accounted for 55% of varicella-related deaths. Certain persons are at higher risk for severe disease and secondary complications: pregnant women, premature infants born to varicella-susceptible mothers, infants born at less than 28 weeks' gestation or weighing ≤ 1000 gm (regardless of maternal immune status), and immunocompromised patients.¹³

The incubation period for varicella is usually 14 to 16 days but may be from 10 to 21 days after exposure, although the incubation period may be shorter in immunocompromised persons.⁴⁴² In persons who receive postexposure VZV immune globulin, the incubation period may be as long as 28 days after exposure. Transmission of infection may occur from 2 days before onset of rash and usually as long as 5 days after rash onset.⁴⁴²

VZV is transmitted by the contact with infected lesions and, in hospitals, airborne transmission has occurred from patients with varicella or zoster to susceptible persons who had no direct contact with the infected patient.⁴⁴³⁻⁴⁴⁷ Adherence to airborne and contact precautions when caring for patients with known or suspected VZV infection can reduce the risk of transmission to personnel.³

It is generally advisable to allow only personnel who are immune to varicella to take care of patients with VZV. Because of the possibility of transmission to and development of severe illness in high-risk patients, personnel with localized zoster should not take care of such patients until all lesions are dry and crusted.^{13,447} Personnel with localized zoster are not likely to transmit infection to immunocompetent patients if their lesions can be covered. However, some institutions may exclude personnel with zoster from work until their lesions dry and crust.⁴³⁹

a. Varicella screening and vaccination

Serologic tests have been used to assess the accuracy of reported histories of chickenpox.^{440,448-450} In adults, a history of varicella is highly predictive of serologic immunity (97% to 99% seropositive). Most adults who have negative or uncertain histories of varicella are also seropositive (71% to 93%). In health care institutions, serologic screening of personnel who have negative or uncertain histories is

likely to be cost-effective, depending on the relative costs of the test and vaccine.^{9,13}

A variety of methods have been used for detecting varicella antibody, but a commercially available latex agglutination test provides prompt, sensitive, and specific serologic results at a reasonable cost. The latex agglutination test may not detect low levels of protective antibody that can occur after vaccination; however, a test with increased sensitivity and specificity is currently under development. Routine testing for varicella immunity after vaccination is not necessary, because 99% of persons are seropositive after the second dose. Moreover, seroconversion does not always result in full protection against disease. However, testing vaccinees after exposures may be warranted. In addition, vaccinated persons who are exposed to varicella but lack antibody may be retested in 5 to 6 days to determine whether they are antibody seropositive after the second test and therefore unlikely to acquire varicella.¹³

In March 1995, a live-attenuated varicella vaccine was licensed for use in the United States. Administration of varicella vaccine is recommended for all susceptible health care personnel, especially those who will have close contact with persons at high risk for serious complications.^{9,13,451,452} Effective varicella vaccination programs require two doses of vaccine to achieve high seroconversion rates in adults;⁴⁵¹ the need for and response to booster doses of vaccine are unknown. Vaccination provides approximately 70% protection against infection and 95% protection against severe disease in follow-up from 7 to 10 years after vaccination.¹³ Cases of varicella have occurred among vaccinees after exposure to wild-type virus ("breakthrough infection"). Data from vaccine trials in which vaccinees of all ages were followed up for as long as 9 years indicate that 1% to 4% of vaccine recipients per year acquire varicella, depending on the vaccine lot and interval after vaccination.^{9,13} However, vaccinated persons have milder disease (e.g., afebrile, a mean of 50 skin lesions that are often not vesicular, and shorter duration of illness) than do unvaccinated individuals (e.g., febrile with several hundred vesicular lesions)^{453,454} and are less likely to transmit disease than unvaccinated persons.

The rate of transmission of disease from vaccinees who contract varicella is low for vaccinated children but has not been studied in adults. Active surveillance for 1 to 8 years after vaccination of 2141 children between 1981 and

1989 in 10 different trials⁹ resulted in reports of breakthrough infections in 78 children, which further resulted in secondary cases in 12.2% (11/90) of vaccinated siblings. Illness was mild in both index and secondary cases. There also has been a report of transmission from a vaccinated child in whom breakthrough disease occurred to a susceptible mother.⁹

All information currently available on vaccine efficacy and the persistence of antibody in vaccinees is based on research conducted in settings where infection is highly prevalent and not affected by the wide use of vaccine. Thus, the extent to which the protection provided by vaccination has been increased by boosting from exposure to natural virus and whether longer term immunity may wane as the prevalence of natural VZV decreases are unknown.

b. Transmission of vaccine virus

In clinical trials, 3.8% of children and 5.5% of adolescents and adults acquired a nonlocalized rash (median five lesions) after the first injection, and 0.9% of adolescents and adults acquired a nonlocalized rash after the second injection. Available data suggest that healthy children have limited potential to transmit vaccine virus to susceptible contacts (estimated to be <1%) but that the risk of transmission from immunocompromised vaccinees is higher.^{13,455,456} Tertiary transmission of vaccine virus to a second healthy sibling of a vaccinated leukemic child has also occurred.⁴⁵⁶ These data suggest that healthy, vaccinated individuals have a very small risk of transmitting vaccine virus to their contacts; this risk may be higher in those who acquire a varicella-like rash after vaccination.

Although the risk of transmission of vaccine virus from vaccinees is not known, the risk if any appears to be very low, and the benefits of vaccinating susceptible health care personnel clearly outweigh this potential risk. As a safeguard, institutions may wish to consider precautions for vaccinated personnel who acquire a rash or who will have contact with susceptible persons at high risk for serious complications.

c. Management of health care personnel exposed to varicella

When unvaccinated susceptible personnel are exposed to varicella, they are potentially infectious 10 to 21 days after exposure, and exclusion from duty is indicated from the tenth day after the first exposure through the 21st day after the last exposure, or until all lesions are dry and crusted if varicella occurs (Table 3).²⁵⁶

Table 6. Pregnant health care personnel: Pertinent facts to guide management of occupational exposures to infectious agents

Agent	Potential effect on fetus	Rate of perinatal transmission	Maternal Screening	Prevention
1. Cytomegalovirus	Hearing loss; congenital syndrome*	15% after primary maternal infection; symptomatic 5%	Antibody provides some but not complete protection against clinical disease; routine screening not recommended	Standard precautions
2. Hepatitis B	Hepatitis; development of chronic infection in infant	HBeAg seropositive 90%; HBeAg negative 0-25%	HBsAg routine screening recommended	Vaccine and HBIG to infant; standard precautions
3. Hepatitis C	Hepatitis	2%-5%	Anti-HCV; HCV RNA in reference labs; routine screening not recommended	Standard precautions
4. Herpes simplex	Mucocutaneous lesions, sepsis, encephalitis; congenital malformations (rare)	Unlikely from nosocomial exposure; primary 33%-50%, recurrent 4%	Antibody testing not useful; inspection for lesions at delivery	Standard precautions
5. Human immunodeficiency virus	AIDS by 2-3 yr	8%-30%	Antibody by enzyme immunoassay, Western blot	Avoid high-risk behaviors; consider postexposure prophylaxis after high-risk needlestick exposure; intrapartum and postnatal zidovudine for HIV-seropositive mothers and their babies; standard precautions
6. Influenza	Inconsistent	Rare	None	Vaccine (safe during pregnancy); droplet precautions
7. Measles	Prematurity; abortion	Rare	History, antibody	Vaccine†; airborne precautions
8. Parvovirus B19	Hydrops, stillbirth	Rare, 3%-9% maximum adverse outcome	IgM and IgG antibody prepregnancy; antibody protective	Droplet precautions
9. Rubella	Congenital syndrome*	45%-50% overall; 90% in 1st 12 wk	Antibody	Vaccine†; droplet precautions for acute infection; contact precautions for congenital rubella
10. Tuberculosis	Hepatomegaly, pulmonary, CNS	Rare	Skin test	Isoniazid ± ethambutol for disease; airborne precautions
11. Varicella-zoster	Malformations (skin, limb, CNS, eye); chickenpox	Total 25%; congenital syndrome (0-4%)	Antibody	Vaccine†; VZIG within 96 hours of exposure if susceptible; airborne and contact precautions

Modified from Siegel JD. Risk and exposure for the pregnant health-care worker. In: Olmstead RN, editor. APIC infection control and applied epidemiology: principles and practices. St Louis: Mosby; 1996. p. 22-2-22-3 (table 22-1). *HBeAg*, Hepatitis B e antigen; *CNS*, central nervous system.

*Congenital syndrome: varying combinations of jaundice, hepatosplenomegaly, microcephaly, CNS abnormalities, thrombocytopenia, anemia, retinopathy, and skin and bone lesions.

†Live-virus vaccines are given routinely before pregnancy.

If vaccinated health care personnel are exposed to varicella, they may be serotested immediately after exposure to assess the presence of antibody.⁴⁵² If they are seronegative, they may be excluded from duty or monitored daily for development of symptoms. Exclusion from duty is indicated if symptoms (fever, upper respiratory tract symptoms, or rash) develop.

Vaccination should be considered for exposed unvaccinated health care personnel without documented immunity.^{441,452} Because the efficacy of post-exposure vaccination is unknown, however, persons vaccinated after an exposure should be managed as previously recommended for unvaccinated persons.

The routine postexposure use of VZV immune globulin (VZIG) is not recommended among immunocompetent health care personnel.¹³ VZIG can be costly, does not necessarily prevent varicella, and may prolong the incubation period by a week or more, thus extending the time that personnel will be restricted from duty. The use of VZIG may be considered for immunocompromised (e.g., HIV infected) or pregnant health care personnel.^{13,457} Postexposure use of acyclovir may be effective and less costly than the use of VZIG in some susceptible persons.⁴⁵⁷ However, additional data concerning the efficacy of acyclovir for post-exposure prophylaxis are needed before such use can be recommended.^{9,13,441,458}

22. Viral respiratory infections

Viral respiratory infections are common problems in health care settings. Nosocomial respiratory infections can be caused by a number of viruses, including adenoviruses, influenza virus, parainfluenza viruses, respiratory syncytial virus (RSV), and rhinoviruses. Because influenza and RSV substantially contribute to the morbidity and mortality associated with viral pneumonia and both have been well studied epidemiologically, this section focuses on prevention of these two viral infections among personnel. Additional information on influenza and RSV can be found in the "Guideline for Prevention of Nosocomial Pneumonia."⁴⁵⁹

a. Influenza

Nosocomial transmission of influenza has been reported in acute and long-term care facilities.⁴⁶⁰⁻⁴⁶⁵ Transmission has occurred from patients to health care personnel,^{462,464} from health care personnel to patients,⁴⁶⁶ and among health care personnel.^{465,467-472}

Influenza is believed to be transmitted from person to person by direct deposition of virus-

laden large droplets onto the mucosal surfaces of the upper respiratory tract of an individual during close contact with an infected person, as well as by droplet nuclei or small-particle aerosols.^{21,290,473} Although the extent of transmission by virus-contaminated hands or fomites is not known, it is not the primary mode of transmission.⁴⁷³

The incubation period of influenza is usually 1 to 5 days, and the period of greatest communicability is during the first 3 days of illness. However, virus can be shed before the onset of symptoms and as long as 7 days after illness onset.⁴⁷⁴⁻⁴⁷⁶ Persons at greatest risk for influenza-related complications include (a) persons older than 65 years, (b) residents of nursing homes and other chronic care facilities, (c) persons with chronic pulmonary or cardiovascular conditions, and (d) persons with diabetes mellitus.¹⁷ Adherence to droplet precautions may prevent nosocomial transmission.³

Administration of influenza vaccine to health care personnel, including pregnant women,⁹ before the beginning of each influenza season can help to (a) reduce the risk to health care personnel of influenza infection, (b) prevent transmission of influenza from personnel to persons at high risk for complications, and (c) reduce personnel absenteeism during community outbreaks. Innovative methods may be needed to increase influenza immunization rates among health care personnel.⁴⁷⁷ Immunization rates may also be increased by providing data to health care personnel on the low rates of systemic reactions to influenza vaccine among healthy adults.⁴⁷⁸

During institutional outbreaks of influenza, prophylactic antiviral agents (e.g., amantadine and rimantadine) may be used in conjunction with influenza vaccine to reduce the severity and duration of illness among unvaccinated health care personnel. Amantadine and rimantadine may be administered for 2 weeks after personnel vaccination or, in unvaccinated personnel, for the duration of influenza activity in the community.^{17,459,465,479}

b. Respiratory syncytial virus

Nosocomial transmission of respiratory syncytial virus (RSV) is greatest during the early winter when community RSV outbreaks occur; patients, visitors, and health care personnel may transmit the virus in the health care setting. RSV infection is most common among infants and children, who are likely to acquire more severe disease. Because RSV infection can also occur simultaneously with other respiratory viruses, it may go unrecog-

nized.^{480,481} Nosocomial transmission has been reported most frequently among newborn and pediatric patients,^{482,483} but outbreaks associated with substantial morbidity and mortality have been reported among adults in bone-marrow transplant centers,⁴⁸⁴ intensive care units,⁴⁸⁵ and long-term care facilities.^{486,487}

RSV is present in large numbers in the respiratory secretions of persons symptomatically infected with the virus and can be transmitted directly through large droplets during close contact with such persons or indirectly by hands or fomites that are contaminated with RSV. Hands can become contaminated through handling of infected persons' respiratory secretions or contaminated fomites and can transmit RSV by touching the eyes or nose.⁴⁵⁹ The incubation period ranges from 2 to 8 days; 4 to 6 days is most common. In general, infected persons shed the virus for 3 to 8 days, but young infants may shed virus for as long as 3 to 4 weeks. Adherence to contact precautions effectively prevents nosocomial transmission.

c. Work restrictions

Because large numbers of personnel may have viral respiratory illnesses during the winter, it may not be possible to restrict infected personnel from all patient care duties. Nevertheless, it may be prudent to restrict personnel with acute viral respiratory infections from the care of high-risk patients during community outbreaks of RSV and influenza.⁴⁸⁸

F. PREGNANT PERSONNEL

Immunologic changes occur during pregnancy, primarily depression of certain aspects of cell-mediated immunity such as decreased levels of helper T cells. These changes permit fetal development without rejection but generally do not increase maternal susceptibility to infectious diseases. Occupational acquisition of infections is of special concern to female health care personnel of childbearing age for several reasons. Some infections, such as varicella, may be more severe during pregnancy. Transplacental infections with viruses such as parvovirus, varicella, and rubella have been associated with abortion, congenital anomaly, and mental retardation. Other diseases in which the infectious agent may be transmitted to the fetus include CMV, hepatitis B, herpes simplex, influenza, and measles. In addition, certain drugs used to treat or prevent some infections, for example tuberculosis, may be contraindicated during pregnancy.

In general, pregnant health care personnel do not have an increased risk for acquiring infections

in the workplace. The risks to pregnant personnel and methods for prevention are discussed in the various sections of this document and are summarized in Table 6. Female personnel of childbearing age should be strongly encouraged to receive immunizations for vaccine-preventable diseases before pregnancy. Such personnel may also decrease their risk of acquiring infection by adhering to appropriate infection control practices, including standard precautions when caring for all patients. Additional information on occupational risks for pregnant health care personnel has been published elsewhere.⁴⁸⁹⁻⁴⁹¹

G. LABORATORY PERSONNEL

Despite the availability of improved engineering controls, work practices, and personal protective equipment, laboratory personnel remain at risk for occupational acquisition of infectious agents.^{5,18,53,151,162,241,492,493} Furthermore, newer technologies that require the use of large or concentrated specimens may further increase the risk of occupationally acquired infections among laboratory personnel.⁴⁹⁴

In a review of laboratory-acquired infections from 1950 through 1974, more than 4000 laboratory-associated infections were documented in the United States⁴⁹²; the 10 most commonly reported infections were brucellosis, Q fever, hepatitis (especially hepatitis B), typhoid fever, tularemia, tuberculosis, dermatomycosis, Venezuelan equine encephalitis, psittacosis, and coccidioidomycosis. However, laboratory-associated infections also have been caused by a wide variety of other pathogens.^{162,492,493} More recently, viral agents have accounted for a larger proportion of laboratory-associated infections than have bacterial agents.⁴⁹³⁻⁴⁹⁸

Laboratory personnel may acquire infection by aerosolization of specimens, mouth pipetting, or percutaneous injury. Information on the risks of laboratory-associated infections and appropriate biosafety procedures and precautions for laboratories have been published.^{5, 6, 494, 499, 500}

In addition to biosafety precautions, preventive measures (e.g., immunizations and postexposure prophylaxis) may also be indicated for laboratory personnel who handle infectious agents. In this document, disease-specific information and guidance are provided for prevention of laboratory-associated infections and for management of laboratory personnel exposed to infectious agents. Health care institutions need to ensure that laboratory personnel who may be exposed to infectious

agents are well informed about the risks of acquiring infections and about biosafety procedures to prevent transmission of infectious agents.

H. EMERGENCY-RESPONSE PERSONNEL

Emergency medical technicians, firefighters, policemen, and others who attend to and transport patients to the hospital may be exposed to recognized or undiagnosed transmissible infectious diseases in the patients with whom they come in contact. Subtitle B (42 USC 300ff-80) of the 1990 Ryan White Comprehensive AIDS Resources Emergency Act requires the establishment of notification systems in each state to ensure that emergency-response employees (including emergency medical technicians, firefighters, and the like) are informed when they have been exposed to an emergency medical patient with an infectious, potentially fatal disease such as HIV or meningococemia. CDC published a list of diseases for which emergency-response employees must be informed of an exposure.⁵⁰¹

I. LATEX HYPERSENSITIVITY

Since the introduction of universal precautions, the use of latex gloves has become commonplace in health care settings.^{31,502} The increased use of latex gloves has been accompanied by increasing reports of allergic reactions to natural rubber latex among health care personnel.⁵⁰³⁻⁵⁰⁸

Natural rubber latex is a combination of heat- and water-soluble proteins derived from the tree *Hevea brasiliensis*. Reactions to latex gloves may be localized or systemic and include dermatitis, conjunctivitis, rhinitis, urticaria, angioedema, asthma, and anaphylaxis.⁵⁰⁹⁻⁵¹² Most local reactions associated with latex glove use are not immunologically mediated and result from chemicals (e.g., thiurams, carbamates, mercaptobenzothiazole, phenylenediamine), accelerants or antioxidants added to gloves during manufacturing.^{502, 507, 513-515} It may be clinically difficult to differentiate irritant reactions from allergic contact dermatitis reactions; both may be manifested by itching, dryness, erythema, bleeding, or scaling of the hands. Nevertheless, neither of the types of local reactions to latex gloves are good predictors of latex allergy^{503, 516}; only a subset of health care personnel reporting glove-associated skin irritation will have immunoglobulin E (IgE) antibodies specific for latex.^{513, 517-519}

In contrast, systemic reactions to natural rubber latex, including urticaria, are mediated by antilatax IgE antibodies^{509,520,521} and may result

from direct skin contact or from exposure to airborne latex allergen adsorbed to glove powder. Occupational asthma from latex is becoming increasingly recognized.^{520,522-524} Asthmatic responses to latex may occur early (<8 hours) or late (>8 hours) after exposure.⁵²⁵⁻⁵²⁷

Local reactions (i.e., irritant or allergic contact dermatitis) to latex gloves account for most reported reactions among health care personnel.^{503,506} The risk of progression from localized to systemic reactions is unknown.

Latex gloves may vary considerably in total protein content from brand to brand and from lot to lot within brands.^{528,529} However, the total protein concentrations and allergenicity of latex gloves are not always directly correlated,⁵²⁸ suggesting that total protein concentrations are not necessarily a measure of the allergenic properties of latex gloves. Currently, the amount of latex allergen exposure required to produce sensitization or to elicit reactions in previously sensitized persons is unknown. The FDA has mandated labeling of all medical devices that contain natural rubber latex.⁵³⁰

Another recognized contributor to latex sensitization and reactions is the powder or cornstarch used as a lubricant for gloves. Levels of extractable protein and allergen in a given glove have been shown to be correlated with the presence of powder. Also, investigators have demonstrated that latex proteins adhere to the powder on gloves and that aerosolized latex protein-powder particles can provoke allergic respiratory symptoms if inhaled by a latex-sensitive individual⁵³¹; similar adherence has not been detected with powdered vinyl gloves. In one study, personnel wearing powdered latex gloves had a significantly higher rate of reaction than did workers who wore washed latex gloves, from which the powder had been removed (60% vs 28%); none of these workers had positive skin-test reactions to industrial or commercial cornstarch or powder.⁵⁰⁴ Although many health care personnel or clinicians may implicate the powder or cornstarch on gloves as the cause of their reactions, documented reactions to cornstarch powder are rare.

a. Prevalence and risk factors

In studies of health care personnel, the reported prevalence of IgE-mediated allergy to latex varies considerably, ranging from 2.9% to 17%. The broad range of prevalence rates reported likely represent differences in the personnel groups studied and the methods used for estimating sensitization or allergy.^{518,519,522,532,533} The prevalence

detected in some studies also has been biased by enrollment or testing of only personnel with symptoms.^{504,508} However, it is estimated that a minority of health care personnel seek medical evaluation or treatment for latex-allergic conditions, even if they have symptoms. Thus, the true prevalence of these reactions among health care personnel is unknown.

The prevalence of sensitization to latex among health care personnel has been shown to vary by job category and by location within a facility.^{506,533} In one study of 224 health care personnel, the overall prevalence of skin-prick reactivity to latex was 17% but ranged from 0% (0/17) among housekeepers and clerical workers to 38% (5/13) among dental residents and assistants.⁵⁰⁶ In another survey of 512 health care personnel, the prevalence among physicians (6.5%, 7/108) was greater than that among nurses (2.2%, 7/325) or other hospital personnel (1.3%, 1/79). Also, operating room personnel (6.2%, 9/145) were significantly more likely to be sensitized than were personnel assigned to general wards or laboratories (1.6%, 6/367); operating room nurses had fourfold the prevalence of general ward nurses (5.6% vs 1.2%).⁵³³ Measurable levels of latex aeroallergen have been detected in the breathing zones of operating room personnel and may vary as much as 100-fold, depending on the invasiveness of the procedure and frequency of glove changes.⁵³⁴

Several factors have been linked with latex sensitization among health care personnel, including the presence of other allergic conditions (e.g., asthma, eczema, hay fever),^{503,516,518,519,522,532,533} nonwhite ethnicity,^{519,532} elevated total IgE levels,⁵¹⁹ allergy to cosmetic powders or foods,⁵³⁵ years or status (full-time vs part-time) of employment, and frequency or duration of glove use.^{503,516,522,533} Coexistent allergy to certain fruits (e.g., bananas,^{536,537} avocados,^{538,539} and chestnuts⁵⁴⁰) also has been described in latex-allergic health care personnel.

Skin irritation and eczematous dermatitis^{516,533} (conditions that may allow passage of latex proteins through the skin) and use of other latex products (e.g., condoms, diaphragms) have not been consistently linked to latex sensitization in health care personnel.

b. Diagnosis and identification

Diagnosis of latex allergy in personnel relies largely on a clinical history of symptoms elicited by exposure to latex products (e.g., balloons, gloves). Clinical symptoms, such as urticaria, may be good predictors of IgE-mediated allergy.^{516,519}

A variety of methods have been used to aid in the identification of latex-allergic persons; most are experimental and have not been approved for clinical use. Skin-prick testing may be the most sensitive method for diagnosis of IgE-mediated allergy, but no standardized FDA-approved antigen is currently available in the United States for detection of latex-specific IgE antibodies. Moreover, the use of some skin-test reagents in highly sensitized persons has been associated with adverse outcomes,⁵⁴¹ suggesting that these nonstandardized reagents may not be safe for routine use. In Europe, where a standardized testing antigen has been developed, skin-prick testing has been used successfully.

FDA-approved immunoassays are available for detection of latex-specific IgE antibodies in blood. The FDA has recommended that these assays be used as confirmatory tests, rather than screening tests, for persons in whom latex allergy is suspected on the basis of clinical history and findings. Levels of detectable antibody appear to be associated with symptoms,^{504,519} but, as with other allergens, the correlation between serum concentrations of latex-specific IgE antibodies and symptom severity may not be predictable.^{312,504,516}

c. Prevention strategies

Avoiding latex products remains the cornerstone of preventing sensitization (primary prevention) and reactions (secondary prevention) to natural rubber latex products. Proposed strategies to reduce the risk of reactions to natural rubber latex have included the use of the following: (a) nonlatex (e.g., vinyl) products alone or in combination with latex gloves, (b) powder-free latex gloves, (c) powdered latex gloves washed to remove powder, and (d) "low-protein" latex gloves. However, none of these interventions has been prospectively studied in controlled trials to assess cost-effectiveness or efficacy in preventing sensitization or reactions.

Because latex proteins can be aerosolized when powdered gloves are donned or removed, systemic symptoms caused by latex aeroallergens may not be alleviated by simply avoiding latex products, particularly if coworkers of the affected worker continue to use powdered latex gloves. Although the risk of a worker's exposure is greatest when gloves are donned or removed, allergenic proteins also may settle on environmental surfaces, surgical gowns, or other clothing and become resuspended. The use of powder-free or low-protein gloves appears more effective and less costly than either laminar-flow or high-efficiency particulate air-filtered glove-changing stations in reducing

latex aeroallergens.⁵³⁴ For personnel with systemic manifestations of latex allergy, workplace restriction or reassignment may be necessary.

J. THE AMERICANS WITH DISABILITIES ACT

The Americans With Disabilities Act provides guidelines for hiring and placing employees with disabilities, as defined in the Act.⁵⁴²⁻⁵⁴⁵ In general, employers must assess applicants for their qualifications to perform the tasks inherent to the job for which the employee is being considered. Applicants may be asked about their ability to perform specific job functions but may not be asked about the existence, nature, or severity of a disability. Employers must make a “reasonable accommodation” to allow an individual to perform the essential functions of a job, unless the employer can prove that this would create undue hardship because of significant difficulty or expense.

The provisions of the Americans With Disabilities Act need to be incorporated into infec-

tion control policies for health care personnel. For example, applicants with a communicable disease spread by aerosol could justifiably be denied employment (until they are no longer infectious) because they could pose a direct threat to others. On the other hand, applicants who are immunocompromised may not necessarily be excluded because of an increased risk for acquiring an infection in the hospital if the employer can make reasonable accommodations that prevent exposure. Health care personnel who are known to be immunocompromised need to be referred to personnel health professionals who can individually counsel the employees on their risk for infection. At the request of the immunocompromised health care personnel, employers should offer but not compel a work setting in which health care personnel would have the lowest possible risk for occupational exposure to infectious agents. Evaluation of individual situations also needs to include consideration of the provisions of other applicable federal, state, and local laws.

Part II. Recommendations for prevention of infections in health care personnel

The Hospital Infection Control Practices Advisory Committee
Centers for Disease Control and Prevention
Public Health Service
U.S. Department of Health and Human Services

A. INTRODUCTION

In this document, the term *health care personnel* refers to all paid and unpaid persons working in health care settings who have the potential for exposure to infectious materials including body substances, contaminated medical supplies and equipment, contaminated environmental surfaces, or contaminated air. These personnel may include but are not limited to physicians, nurses, technicians, therapists, pharmacists, nursing assistants, laboratory personnel, autopsy personnel, emergency medical service personnel, dental personnel, students and trainees, contractual staff not employed by the health care facility, and persons not directly involved in patient care but potentially exposed to infectious agents (e.g., volunteer, dietary, housekeeping, maintenance, and clerical personnel).

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretic rationale, applicability, and potential economic impact. The system for categorizing recommendations is 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 reviewed as effective by experts in the field and a consensus of Hospital Infection Control Practices Advisory Committee members on the basis of strong rationale and suggestive evidence, even though definitive scientific studies have not been done.

Category II

Suggested for implementation in many hospitals. Recommendations may be supported by suggestive clinical or epidemiologic studies, a strong theoretic 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.

B. ELEMENTS OF A PERSONNEL HEALTH SERVICE FOR INFECTION CONTROL

1. Coordinated planning and administration

- a. Coordinate policy making and planning for the personnel health service among the hospital administration, personnel health service, infection control personnel clinical services, pharmacy personnel, various other hospital departments, and relevant external agencies. Include paid and nonpaid personnel (e.g., volunteers, trainees, physicians, out-of-hospital and contractual personnel, and emergency responders) in the plan. **Category IB**
- b. Establish an active system and develop a written policy for notifying infection control personnel of (1) infections in personnel (including volunteers, trainees, contractual personnel, and out-of-hospital personnel) that require work restrictions or exclusion from work, (2) clearance for work after an infectious illness that required work restrictions or exclusion, (3) work-related infections and exposures, and when appropriate (4) results of epidemiologic investigations. **Category IB**
- c. Develop protocols to ensure coordination between the personnel health program, the infection control program, and other relevant departments of the facility. **Category IB**

2. Placement evaluation

- a. Before personnel begin duty or are given a new work assignment, conduct health inventories. The inventories should include the following: (1) immunization status or history of vaccine-preventable diseases (e.g., chickenpox, measles, mumps, rubella, hepatitis B) and (2) history of any conditions that may predispose personnel toward acquiring or transmitting infectious diseases. **Category IB**
- b. Perform directed physical and laboratory examinations on personnel, as indicated by the results of the health inventory. Include examinations to detect conditions that might

increase the likelihood of transmitting disease to patients or cause unusual susceptibility to infection, and examinations to serve as a baseline for determining whether any future problems are work related. **Category IB**

- c. Conduct personnel health assessments other than placement evaluations on an as-needed basis, for example, as required to evaluate work-related illness or exposures to infectious diseases. **Category IB**
- d. Do not perform routine cultures on personnel (e.g., cultures of the nose, throat, or stool) as part of the placement evaluation. **Category IB**
- e. Conduct routine screening for TB by using the intradermal (Mantoux), intermediate-strength (5 tuberculin units) PPD test on personnel who have potential for exposure to TB. **Category II**
- f. Conduct routine serologic screening for some vaccine-preventable diseases, such as hepatitis B, measles, mumps, rubella, or varicella, if deemed to be cost-effective to the hospital and beneficial to the health care personnel. **Category II**

3. Personnel health and safety education

- a. Provide personnel, annually and whenever the need arises, with in-service training and education on infection control appropriate and specific for their work assignments, so that personnel can maintain accurate and up-to-date knowledge about the essential elements of infection control. Ensure that the following topics are included in the initial training on infection control: (1) handwashing; (2) modes of transmission of infection and importance of complying with standard and transmission-based precautions; (3) importance of reporting certain illnesses or conditions (whether work related or acquired outside the hospital), such as generalized rash or skin lesions that are vesicular, pustular, or weeping, jaundice, illnesses that do not resolve within a designated period (e.g., a cough that persists for >2 weeks, gastrointestinal illness, or febrile illness with fever of >103° F lasting >2 days), and hospitalizations resulting from febrile or other contagious diseases; (4) tuberculosis control; (5) importance of complying with standard precautions and reporting exposure to blood and body fluids to prevent transmission of bloodborne pathogens; (6) importance of cooperating with infection control personnel during outbreak investigations; and (7) impor-

tance of personnel screening and immunization programs. **Category IB**

- b. Ensure that all personnel know whether they have medical conditions or receive medical treatment that renders them more susceptible to or more likely to transmit infections, so that they can follow recommendations to greatly reduce their risk of transmitting or acquiring infections (e.g., request for work reassignment). **Category IB**
- c. Make specific written policies and procedures for control of infections in health care personnel readily available to all personnel. **Category IB**
- d. Provide educational information appropriate, in content and vocabulary, to the educational level, literacy, and language of the employee. **Category IB**

4. Job-related illnesses and exposures

- a. Maintain a record on health care personnel that includes information obtained during the medical evaluation, immunization records, results of tests obtained in any screening or control programs, and reports of work-related illnesses or exposures in accordance with state and federal regulatory requirements.
- b. Establish a readily available mechanism for personnel to obtain advice about illnesses they may acquire from or transmit to patients. **Category IB**
- c. Develop written protocols for handling job-related and community-acquired infectious diseases or important exposures. Record the occurrences of job-related infectious diseases or important exposures in the person's record and when applicable notify appropriate infection control personnel and members of the personnel health service. **Category IB**

5. Record keeping, data management, and confidentiality

- a. Establish and keep an updated record for all personnel and maintain the confidentiality of their records while ensuring that they receive appropriate management for occupational illnesses or exposures. Ensure that individual records for volunteers, trainees, contractual personnel, and personnel who provide care outside of hospitals are similarly kept and maintained. **Category IB**
- b. Ensure that when data on personnel health are made public, the individual's confidentiality is maintained, for example, by releasing only aggregate numbers. **Category IB**

- c. Maintain a personnel database, preferably computerized, that allows tracking of personnel immunizations, screening tests, and assessment of trends of infections and diseases in personnel. Copies of their individual records are to be available to personnel. **Category IB**
- d. Periodically review and assess aggregate data gathered on personnel health (e.g., rates of PPD-test conversion) to determine the need for action. **Category IB**
- e. Ensure that all federal, state, local, and community standards on medical record keeping and confidentiality are met.^{26,27} **Category IB**

C. PROTECTION OF PERSONNEL AND OTHER PATIENTS FROM PATIENTS WITH INFECTIONS

Apply precautions described in the current "Guideline for Isolation Precautions in Hospitals"³ and other guidelines.³⁸² **Category IB**

D. IMMUNIZATION OF HEALTH CARE PERSONNEL, GENERAL RECOMMENDATIONS

1. Formulate a written comprehensive policy on immunizing health care personnel. **Category IB**
2. Ensure that persons administering immunizing agents are (a) familiar with ACIP recommendations,^{8,9} (b) well informed about indications, storage, dosage, preparation, side effects, and contraindications for each of the vaccines, toxoids, and immune globulins used,^{8,9,24} and (c) kept updated on national and local recommendations regarding vaccination of health care personnel (Tables 1 and 2). **Category IB**
3. Ensure that immunization product information is available at all times and that a pertinent health history, especially a history of allergy and potential vaccine contraindications, is obtained from each person before an agent is given (Table 2). **Category IB**
4. Develop a list of needed immunizations for each employee during screening and an individual plan to provide the necessary vaccines. **Category IB**
5. In the absence of a known occupational exposure, provide personnel with on-site immunizations or refer personnel to their own health care providers for routine non-occupation-related immunizations against diphtheria, pneumococcal disease, hepatitis A, or tetanus (Table 1). **Category IB**
6. Provide vaccine to personnel who may have occupational exposure to uncommon dis-

eases such as plague, typhus, or yellow fever, or refer them to their own health care providers. **Category IB**

E. PROPHYLAXIS AND FOLLOW-UP AFTER EXPOSURE, GENERAL RECOMMENDATIONS

1. Ensure that when personnel are offered necessary prophylactic treatment with drugs, vaccines, or immune globulins, they are informed of (a) options for prophylaxis, (b) the risk (if known) of infection when treatment is not accepted, (c) the degree of protection provided by the therapy, and (d) the potential side effects of the therapy. **Category IB**
2. Ensure that when personnel are exposed to particular infectious agents, they are informed of (a) the recommended postexposure management that is based on current knowledge about the epidemiology of the infection, (b) the risk (if known) of transmitting the infection to patients, other personnel, or other contacts, and (c) the methods of preventing transmission of the infection to other persons. **Category IB**

F. PERSONNEL RESTRICTION BECAUSE OF INFECTIOUS ILLNESSES OR SPECIAL CONDITIONS, GENERAL RECOMMENDATIONS

1. Develop well-defined policies concerning contact of personnel with patients when personnel have potentially transmissible conditions. These policies should govern (a) personnel responsibility in using the health service and reporting illness, (b) work restrictions, and (c) clearance for work after an illness that required work restriction. **Category IB**
2. Identify the persons with authority to relieve personnel of duties. **Category IB**
3. Develop work-exclusion policies that encourage personnel to report their illnesses or exposures and that do not penalize them with loss of wages, benefits, or job status. **Category IB**
4. Educate and encourage personnel who have signs and symptoms of a transmissible infectious disease to report their condition promptly to their supervisor and occupational health. **Category IB**
5. Provide appropriate education for personnel on the importance of good hygienic practices, especially handwashing and covering the nose and mouth when coughing and sneezing. **Category IB**

G. PREVENTION OF NOSOCOMIAL TRANSMISSION OF SELECTED INFECTIONS

1. Bloodborne pathogens, general recommendation

Ensure that health care personnel are familiar with precautions to prevent occupational transmission of bloodborne pathogens.^{3,6,30,31,39}

Category IA

Follow state and federal guidelines and strategies for determining the need for work restrictions for health care personnel infected with bloodborne pathogens.⁴⁸ **Category IB**

a. Hepatitis B

- 1) Administer hepatitis B vaccine to personnel who perform tasks involving routine and inadvertent (e.g., as with housekeepers) contact with blood, other body fluids (including blood-contaminated fluids), and sharp medical instruments or other sharp objects.^{9,10,40} **Category IA**
- 2) Before vaccinating personnel, do not routinely perform serologic screening for hepatitis B, unless the health care organization considers screening cost-effective or the potential vaccinee requests it.⁹ **Category IA**
- 3) Conduct postvaccination screening for immunity to hepatitis B within 1 to 2 months after the administration of the third vaccine dose to personnel who perform tasks involving contact with blood, other body fluids (including blood-contaminated fluids), and sharp medical instruments or other sharp objects. **Category IA**
- 4) Revaccinate persons not found to have an antibody response after the initial hepatitis B vaccine series with a second three-dose vaccine series. If persons still do not respond after revaccination, refer them for evaluation for lack of response, (e.g., possible chronic HBV infection; Tables 1 and 4).⁹ **Category IB**
- 5) Semiannually test for HBsAg and anti-HBs staff in chronic dialysis centers who do not respond to the hepatitis B vaccine.⁵⁵ **Category IA**
- 6) Use both passive immunization with hepatitis B immune globulin and active immunization with hepatitis B vaccine for postexposure prophylaxis in susceptible personnel who have had a needlestick, percutaneous, or mucous membrane exposure to blood known or suspected to

be at high risk for being HBsAg seropositive (Table 6). **Category IA**

- 7) Follow current recommendations for post-exposure prophylaxis after percutaneous or mucous membrane exposure to blood and body fluids that is known or suspected to be at high risk for being HBsAg seropositive (Table 4).⁴⁰ **Category IA**

b. Hepatitis C

- 1) Do not administer immune globulin to personnel who have exposure to blood or body fluids positive for antibody to HCV.³⁷ **Category IB**
- 2) Consider implementing policies for post-exposure follow-up at baseline and 6 months for health care personnel who have had a percutaneous or mucosal exposure to blood containing antibody to HCV.³⁷ **Category IB**

c. Human immunodeficiency virus

Follow current recommendations for postexposure prophylaxis after percutaneous or mucocutaneous exposure to blood or body fluids containing blood from a source suspected or known to be HIV-infected.^{33,80} **Category IB**

2. Conjunctivitis

Restrict personnel with epidemic keratoconjunctivitis or purulent conjunctivitis caused by other microorganisms from patient care and the patient's environment for the duration of symptoms. If symptoms persist longer than 5 to 7 days, refer personnel to an ophthalmologist for evaluation of continued infectiousness. **Category IB**

3. Cytomegalovirus

- a. Do not restrict personnel from work who contract CMV-related illnesses.¹¹⁹ **Category IB**
- b. Ensure that pregnant personnel are aware of the risks associated with CMV infection and infection control procedures to prevent transmission when working with high-risk patient groups (Table 6).^{3,117} **Category IA**
- c. Do not routinely use workplace reassignment as a method to reduce CMV exposures among seronegative pregnant personnel.^{88,92,95-97,102,105,106,119,120} **Category IA**

4. Diphtheria

- a. Encourage vaccination with Td every 10 years for health care personnel (Table 1).^{9,19} **Category IB**

- b. Obtain nasopharyngeal cultures from exposed personnel and monitor for signs and symptoms of diphtheria for 7 days after exposure.¹⁴⁹ **Category IB**
- c. Administer antimicrobial prophylaxis to personnel who have contact with respiratory droplets or cutaneous lesions of patients infected with diphtheria. Also administer a dose of Td to previously immunized exposed personnel who have not been vaccinated within the previous 5 years (Table 1).^{19,149} **Category IB**
- d. Repeat nasopharyngeal cultures of personnel found to have positive cultures at least 2 weeks after completion of antimicrobial therapy. Repeat antimicrobial therapy if personnel remain culture positive.¹⁴⁹ **Category IB**
- e. Exclude exposed personnel and those identified as asymptomatic carriers from duty until antimicrobial therapy is completed and results of two nasopharyngeal cultures obtained at least 24 hours apart are negative (Table 3).¹⁴⁹ **Category IB**

5. Gastroenteritis

- a. Vaccinate microbiology laboratory personnel who work with *S. typhi* on a regular basis, according to published guidelines.^{151,162} **Category II**
- b. Pending their evaluation, exclude personnel with acute gastrointestinal illnesses (vomiting or diarrhea, with or without other symptoms such as nausea, fever, or abdominal pain) from contact with patients and their environment or from food handling (Table 3).^{3,171} **Category IB**
- c. Consult local and state health authorities regarding work restrictions for patient care personnel or food handlers with enteric infections. **Category IB**
- d. Determine the etiology of gastrointestinal illness among personnel who care for patients at high risk for severe disease. **Category IB**
- e. Allow personnel infected with enteric pathogens to return to work after their symptoms resolve, unless local regulations require exclusion from duty. **Category II**
- f. Ensure that personnel returning to work after a gastrointestinal illness practice good hygienic practices, especially handwashing, to reduce or eliminate the risk of transmission of the infecting agents.¹⁶⁷ **Category IB**
- g. Do not routinely perform follow-up cultures or examinations of stool for enteric pathogens other than *Salmonella* to determine when the stool is free of the infecting organism, unless local regulations require such procedures. **Category IB**
- h. Do not perform routine stool cultures on asymptomatic health care personnel, unless required by state and local regulations. **Category IB**

6. Hepatitis A virus

- a. Do not routinely administer inactivated hepatitis A vaccine to health care personnel. Susceptible personnel living in areas where hepatitis A is highly endemic should be vaccinated to prevent acquisition of community-acquired infection.^{9,204} **Category IB**
- b. Do not routinely administer immune globulin as prophylaxis for personnel providing care or who are exposed to a patient with hepatitis A.²⁰⁴ **Category IB**
- c. Administer immune globulin (0.02 ml/kg) to personnel who have had oral exposure to fecal excretions from a person acutely infected with HAV (Table 1).²⁰⁴ **Category IA**
- d. In documented outbreaks involving transmission of HAV from patient to patient or from patient to health care worker, use of immune globulin may be indicated in persons with close contact with infected persons. Contact the local health department regarding control measures (Table 1). **Category IB**
- e. Exclude personnel who have acute hepatitis A from duty until 1 week after the onset of jaundice (Table 3). **Category IA**

7. Herpes simplex virus

- a. Evaluate personnel with primary or recurrent orofacial herpes simplex infections on a case-by-case basis to assess the potential for transmission to high-risk patients (e.g., neonates, intensive care unit patients, patients with severe burns or eczema, and severely immunocompromised patients) and the need for exclusion from the care of such patients (Table 3).^{209,218} **Category IB**
- b. Counsel personnel with orofacial herpes simplex to cover and not touch the infected lesions, to observe handwashing policies, and not to allow the lesions to touch patients with dermatitis.²¹⁵ **Category IB**
- c. Exclude personnel with herpes simplex infections of the fingers or hands (herpetic

whitlow) from contact with patients until their lesions are healed.^{213,214} **Category IB**

8. Measles

- a. Ensure that all personnel have documented immunity to measles.
 - 1) Administer measles vaccine* to persons born in 1957 or later, unless they have evidence of measles immunity.⁹ **Category IA**
 - 2) Administer measles vaccine* to personnel born before 1957 if they do not have evidence of measles immunity and are at risk for occupational exposure to measles (Table 1).^{8,221,233,234} **Category IA**
 - 3) Do not routinely perform serologic screening for measles before administering measles vaccine* to personnel, unless the health care employer considers screening cost-effective or the potential vaccinee requests it.^{8,11,235-238} **Category IA**
 - 4) Administer postexposure measles vaccine* to measles-susceptible personnel who have contact with persons with measles within 72 hours after the exposure (Tables 1 through 3).⁸ **Category IA**
- b. Exclude exposed personnel who do not have documented immunity to measles from duty from the fifth day after the first exposure until the 21st day after the last exposure to measles, regardless of whether they receive postexposure vaccine (Table 3).^{11,237} **Category IB**
- c. Exclude personnel who acquire measles from duty for 7 days after rash develops or for the duration of their acute illness, whichever is longer (Table 3).⁹ **Category IB**

9. Meningococcal disease

- a. Do not routinely administer meningococcal vaccine to health care personnel.¹⁵ **Category IB**
- b. Consider vaccination of laboratory personnel who are routinely exposed to *N. meningitidis* in solutions that may be aerosolized (Table 1).¹⁵ **Category IB**
- c. Immediately offer antimicrobial prophylaxis to personnel who have had intensive close contact (e.g., mouth-to-mouth resuscitation, endotracheal intubation, endotracheal tube management) with a patient with meningococcal disease before administration of

antibiotics without the use of proper precautions (Table 1).¹⁵ **Category IB**

- d. Do not routinely give quadrivalent A,C,Y,W-135 meningococcal vaccines for postexposure prophylaxis (Table 1).¹⁵ **Category II**
- e. Administer meningococcal vaccine to personnel (and other persons likely to have contact with infected persons) to control serogroup C outbreaks after consultation with public health authorities.¹⁵ **Category IB**
- f. Consider preexposure vaccination of laboratory personnel who routinely handle soluble preparations of *N. meningitidis*.¹⁵ **Category II**
- g. Exclude personnel with *N. meningitidis* infections from duty until 24 hours after the start of effective therapy. Do not routinely exclude personnel from duty who only have nasopharyngeal carriage of *N. meningitidis*. **Category IA**

10. Mumps

- a. Administer mumps vaccine* to all personnel without documented evidence of mumps immunity, unless otherwise contraindicated (Table 1).^{9,258} **Category IA**
- b. Before vaccinating personnel with mumps vaccine,* do not routinely perform serologic screening for mumps, unless the health care employer considers screening cost-effective or it is requested by the potential vaccinee.¹² **Category IB**
- c. Exclude susceptible personnel who are exposed to mumps from duty from the 12th day after the first exposure through the 26th day after the last exposure or, if symptoms develop, until 9 days after the onset of parotitis (Table 3).^{9,255} **Category IB**

11. Parvovirus

- a. Ensure that pregnant personnel are aware of the risks associated with parvovirus infection and of infection control procedures to prevent transmission when working with high-risk patient groups (Table 6).^{274,275} **Category IB**
- b. Do not routinely exclude pregnant personnel from caring for patients with B19. **Category IB**

12. Pertussis

- a. Do not administer whole-cell pertussis vaccine to personnel (Table 1).⁹ **Category IB**
- b. NO RECOMMENDATION for routine administration of an acellular pertussis vaccine to health care personnel. UNRESOLVED ISSUE

*MMR is the vaccine of choice. If the recipient is known to be immune to one or more of the components, monovalent or bivalent vaccines may be used.

- c. Immediately offer antimicrobial prophylaxis against pertussis to personnel who have had unprotected (i.e., without the use of proper precautions), intensive (i.e., close, face-to-face) contact with a patient who has a clinical syndrome highly suggestive of pertussis and whose cultures are pending; discontinue prophylaxis if results of cultures or other tests are negative for pertussis and the clinical course is suggestive of an alternate diagnosis (Table 1).^{287,288} **Category II**
- d. Exclude personnel in whom symptoms develop (e.g., cough ≥ 7 days, particularly if accompanied by paroxysms of coughing, inspiratory whoop, or posttussive vomiting) after known exposure to pertussis from patient care areas until 5 days after the start of appropriate therapy (Table 3).⁹ **Category IB**

13. Poliomyelitis

- a. Determine whether the following personnel have completed a primary vaccination series: (1) persons who may have contact with patients or the secretions of patients who may be excreting wild polioviruses and (2) laboratory personnel who handle specimens that might contain wild polioviruses or who do cultures to amplify virus (Table 1).²¹ **Category IA**
- b. For above personnel, including pregnant personnel or personnel with an immunodeficiency, who have no proof of having completed a primary series of polio immunization, administer the enhanced inactivated poliovirus vaccine rather than oral poliovirus vaccine for completion of the series (Table 1).²¹ **Category IB**
- c. When a case of wild-type poliomyelitis infection is detected or an outbreak of poliomyelitis occurs, contact the CDC through the state health department. **Category IB**

14. Rabies

- a. Provide preexposure vaccination to personnel who work with rabies virus or infected animals in rabies diagnostic or research activities (Table 1).^{5,22} **Category IA**
- b. After consultation with public health authorities, give a full course of antirabies treatment to personnel who either have

been bitten by a human being with rabies or have scratches, abrasions, open wounds, or mucous membranes contaminated with saliva or other potentially infective material from a human being with rabies. In previously vaccinated individuals, postexposure therapy is abbreviated to include only a single dose of vaccine on day 0 and one on day 3 (Table 1).²⁹⁵⁻²⁹⁷

Category IB

15. Rubella

- a. Vaccinate all personnel without documented immunity to rubella with rubella vaccine* (Table 1).^{9,309} **Category IA**
- b. Consult local and state health departments regarding regulations for rubella immunity in health care personnel. **Category IA**
- c. Do not perform serologic screening for rubella before vaccinating personnel with rubella vaccine,* unless the health care employer considers it cost-effective or the potential vaccinee requests it.²³⁷ **Category IB**
- d. Do not administer rubella vaccine* to susceptible personnel who are pregnant or might become pregnant within 3 months of vaccination (Table 1).⁹ **Category IA**
- e. Administer rubella vaccine* in the postpartum period to female personnel not known to be immune. **Category IA**
- f. Exclude susceptible personnel who are exposed to rubella from duty from the seventh day after the first exposure through the 21st day after the last exposure (Table 3).⁹ **Category IB**
- g. Exclude personnel who acquire rubella from duty until 7 days after the beginning of the rash (Table 3).⁹ **Category IB**

16. Scabies and pediculosis

- a. Evaluate exposed personnel for signs and symptoms of mite infestation and provide appropriate therapy for confirmed or suspected scabies.³¹¹ **Category IA**
- b. Evaluate exposed personnel for louse infestation and provide appropriate therapy for confirmed pediculosis.³³⁰ **Category IA**
- c. Do not routinely provide prophylactic scabicide treatment to personnel who have had skin-to-skin contact with patients or other persons with scabies (Table 1).^{310,311,316,326} **Category II**
- d. Consider providing prophylactic scabicide treatment to personnel who have skin-to-

*MMR is the vaccine of choice. If the recipient is known to be immune to one or more of the components, monovalent or bivalent vaccines may be used.

skin contact with patients or other persons with scabies in situations where transmission has occurred.^{311,331} **Category II**

- e. Do not routinely provide prophylactic pediculicide treatment to personnel who have had contact with patients or other persons with pediculosis, unless they have evidence of infestation. **Category II**
- f. Exclude personnel with confirmed scabies from the care of patients until they have received appropriate treatment and have been shown, by medical evaluation, to have been effectively treated.³¹¹ **Category II**
- g. Exclude personnel with confirmed or suspected louse infestation from contact with patients until after they receive appropriate initial treatment and are found to be free of adult and immature lice (Table 3).³³⁵ **Category IB**

17. Staphylococcal infection or carriage

- a. Obtain appropriate cultures and exclude personnel from patient care or food handling if they have a draining lesion suspected to be caused by *S. aureus*, until the infections have been ruled out or personnel have received adequate therapy and their infections have resolved (Table 3).³⁴⁰ **Category IB**
- b. Do not routinely exclude personnel with suspected or confirmed carriage of *S. aureus* (on nose, hand, or other body site) from patient care or food handling unless it is shown epidemiologically that they are responsible for disseminating the organism in the health care setting (Table 3).^{340,342,343,350} **Category IB**

18. Group A Streptococcus infections

- a. Obtain appropriate cultures and exclude personnel from patient care or food handling if they have draining lesions that are suspected to be caused by Streptococcus. Work restrictions should be maintained until streptococcal infection has been ruled out or personnel have received adequate therapy for 24 hours (Table 3).^{369-371,374} **Category IB**
- b. Do not routinely exclude personnel with suspected or confirmed carriage of group A Streptococcus from patient care or food handling unless it is shown epidemiologically that they are responsible for disseminating the organism in the health care setting (Table 3).^{369,373,378} **Category IB**

19. Tuberculosis

a. General recommendations

- 1) Educate all health care personnel regarding the recognition, transmission, and prevention of TB. **Category IB**
- 2) Follow current recommendations outlined in the "Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities, 1994."³⁸² **Category IB**

b. TB screening program

- 1) Include all health care personnel who have potential for exposure to *M. tuberculosis* in a PPD skin-test program.³⁸² **Category IA**
- 2) Administer PPD tests by using the intracutaneous (Mantoux) method of administration of 5 tuberculin units (0.1 ml) PPD.^{382,406-408} **Category IB**
- 3) Do not routinely test personnel known to have conditions that cause severe suppression of cell-mediated immunity (such as HIV-infected persons with lowered CD4+ counts and organ-transplant recipients receiving immunosuppressive therapy) for cutaneous anergy at the time of PPD testing.⁴⁰⁸ **Category IB**
- 4) Ensure that the administration, reading, and interpretation of PPD tests are performed by specified, trained personnel.³⁸² **Category IA**

c. Baseline PPD

- 1) Perform baseline PPD tests on health care personnel who are new to a facility and who have potential for exposure to *M. tuberculosis*, including those with a history of BCG vaccination.³⁸² **Category IB**
- 2) Perform two-step, baseline PPD tests on newly employed health care personnel who have negative results of initial PPD testing and have not had a documented negative PPD-test result during the preceding 12 months, unless the institution has determined that two-step testing is not warranted in its facility.³⁸² **Category II**
- 3) Interpret baseline PPD-test results as outlined in the "Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities, 1994."³⁸² **Category IB**

d. Follow-up (repeat) PPD

- 1) Perform periodic follow-up PPD tests on all health care personnel with negative baseline PPD-test results who have the

potential for exposure to *M. tuberculosis*.³⁸² **Category IA**

- 2) Base the frequency of repeat PPD testing on the hospital's risk assessment, as described in the "Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities, 1994" and as provided by federal, state, and local regulations.³⁸² **Category IB**
- 3) Exempt from follow-up PPD tests personnel with documented history of positive baseline PPD-test result or adequate treatment for TB.³⁸² **Category IB**
- 4) Consider retesting immunocompromised health care personnel who have potential for exposure to *M. tuberculosis* at least every 6 months.³⁸² **Category II**
- 5) Interpret follow-up-PPD test results as outlined in the "Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities, 1994."³⁸² **Category IB**
- 6) Management of PPD-positive personnel
 - a) Promptly evaluate personnel with positive PPD-test results for active disease and obtain an adequate history on TB exposure to help determine whether the infection is occupational or community acquired.³⁸² **Category IB**
 - b) Perform chest radiographic examinations on personnel with a positive PPD-test result as part of the evaluation for active TB. If results of the initial chest radiographic examination are negative, do not repeat chest radiograph unless symptoms suggestive of TB develop.³⁸² **Category IB**
 - c) Periodically remind all personnel, especially those with positive PPD-test results, about the symptoms of TB and the need for prompt evaluation of any pulmonary symptoms suggestive of TB.³⁸² **Category IB**
 - d) Do not require routine chest radiographs for asymptomatic, PPD-negative workers.³⁸² **Category IB**

e. Preventive therapy

- 1) Offer preventive therapy to the following personnel, regardless of age, who have conversion of their PPD test: (a) recent converters, (b) close contacts of persons with active TB, (c) those with medical conditions that increase their risk for active TB, (d) those with HIV infection, and (e) injecting-drug users.^{382,407} **Category IB**

- 2) Offer preventive therapy to all other personnel (i.e., who do not have the above risk factors) with positive PPD reactions if they are younger than 35 years.⁴⁰⁷ **Category IA**
- 3) Provide preventive therapy to personnel through the occupational health program or refer them to the health department or their health care provider, as appropriate. **Category IB**

f. Postexposure management of personnel

- 1) As soon as possible after an exposure to TB (i.e., exposure to a person with pulmonary or laryngeal TB for whom proper isolation precautions were not implemented), conduct PPD testing on personnel who are known to have negative PPD-test results. If the initial postexposure PPD-test result is negative, repeat the PPD test 12 weeks after the exposure.³⁸² **Category IB**
- 2) Do not perform PPD tests or chest radiographs on personnel with previous positive PPD-test results, unless they have symptoms suggestive of active TB.³⁸² **Category IB**

g. Workplace restrictions

- 1) Exclude personnel with infectious pulmonary or laryngeal TB from the workplace until the facility has documentation from their health care provider that they are receiving adequate therapy, their coughs have resolved, and that they have had three consecutive sputum smears collected on different days with negative results for AFB. After personnel return to work, obtain periodic documentation from their health care provider that effective drug therapy has been maintained for the recommended period and that sputum smear results remain negative for AFB (Table 3).³⁸² **Category IB**
- 2) Promptly evaluate for infectiousness those personnel with active TB who discontinue treatment before they are cured. Exclude from duty those who are found to remain infectious until (a) treatment is resumed, (b) an adequate response to therapy is documented, and (c) sputum smear results are negative for AFB.³⁸² **Category IB**
- 3) Consider directly observed therapy for personnel with active TB who have not been compliant with drug regimens. **Category IB**
- 4) Do not exclude personnel from the workplace who have TB only at sites other than the lung or larynx.³⁸² **Category IB**

- 5) Do not restrict personnel from their usual work activities if they are receiving preventive therapy because of positive PPD-test results, even if they are unable or unwilling to accept or complete a full course of preventive therapy. Instruct them to seek prompt evaluation if symptoms suggestive of TB develop.³⁸² **Category IB**

h. Immunocompromised personnel

- 1) Refer personnel who are known to be immunocompromised to personnel health professionals who can individually counsel them regarding their risk for TB.³⁸² **Category II**
- 2) At the request of immunocompromised personnel, offer but do not compel reasonable accommodations for work settings in which they would have the lowest possible risk for occupational exposure to *M. tuberculosis*. Consider the provisions of the Americans With Disabilities Act of 1990 and other federal, state, and local regulations in evaluating these situations.³⁸² **Category II**

i. Bacille Calmette-Guérin vaccination

- 1) In settings associated with high risk for *M. tuberculosis* transmission:
 - a) Consider BCG vaccination of personnel on an individual basis, and only in settings where (1) a high proportion of isolates of *M. tuberculosis* are resistant to isoniazid and rifampin, (2) there is a strong likelihood of transmission and infection with such drug-resistant organisms, and (3) comprehensive infection control precautions have been implemented and have failed to halt nosocomial transmission of TB.⁴¹² Consult with the local and state health departments in making this determination. **Category II**
 - b) Do not require BCG vaccination for employment or for assignment of personnel in specific work areas.⁴¹² **Category II**
- 2) Counsel health care personnel who are being considered for receipt of BCG vaccination about the risks and benefits of both BCG vaccination and preventive therapy, including (a) the variable data on the efficacy of BCG vaccination, (b) the potentially serious complications of BCG vaccine in immunocompromised individuals, such as those with HIV infection, (c) the lack of information on chemoprophylaxis for

MDR-TB infections, (d) the risks of drug toxicity with multidrug prophylactic regimens, and (e) the fact that BCG vaccination interferes with the diagnosis of newly acquired TB infection.⁴¹² **Category IB**

- 3) Do not administer BCG vaccine to personnel in settings associated with a low risk for *M. tuberculosis* transmission. **Category IB**
- 4) Do not administer BCG vaccine to pregnant or immunocompromised persons with negative baseline PPD-test results. **Category II**

20. Vaccinia

- a. Ensure that personnel who directly handle cultures of or animals contaminated or infected with vaccinia, recombinant vaccinia viruses, or other orthopox viruses (e.g., monkeypox, cowpox) that infect human beings receive smallpox vaccination every 10 years (Table 1).^{9,18} **Category IB**
- b. Consider administering vaccinia vaccine to personnel who provide clinical care to recipients of recombinant vaccinia virus vaccines (Table 1).^{9,18} **Category II**
- c. Do not administer vaccinia vaccine to pregnant personnel or personnel with immunosuppression or eczema (Tables 1 and 2). **Category IB**
- d. Do not exclude from duty personnel who receive the vaccine, if they keep the vaccination site covered and adhere to handwashing practices.¹⁸ **Category IB**

21. Varicella

- a. Administer varicella vaccine to susceptible personnel, especially those that will have contact with patients at high risk for serious complications (Table 1).^{9,13} **Category IA**
- b. Do not perform serologic screening of persons with negative or uncertain history of varicella before administering varicella vaccine to personnel, unless the institution considers it cost-effective.^{9,13} **Category IB**
- c. Do not routinely perform postvaccination testing of personnel for antibodies to varicella.⁹ **Category IB**
- d. NO RECOMMENDATION for administering postexposure varicella vaccination for the protection of exposed, susceptible personnel.⁹ **UNRESOLVED ISSUE**
- e. Develop guidelines for managing health care personnel who receive varicella vaccine; for example, consider precautions for personnel who acquire a rash after receipt of varicella vaccine and for other health care personnel who

- receive varicella vaccine and will have contact with susceptible persons at high risk for serious complications from varicella.⁹ **Category IB**
- f. Develop written guidelines for postexposure management of vaccinated or susceptible personnel who are exposed to wild-type varicella.⁹ **Category IB**
 - g. Exclude personnel from work who have onset of varicella until all lesions have dried and crusted (Table 3).³ **Category IB**
 - h. Exclude from duty after exposure to varicella personnel who are not known to be immune to varicella (by history or serology), beginning on the tenth day after the first exposure until the 21st day after the last exposure (28th day if VZIG was given; Table 3).⁹ **Category IB**
 - i. Restrict immunocompetent personnel with localized zoster from the care of high-risk patients until lesions are crusted; allow them to care for other patients with lesions covered.⁹ **Category IB**
 - j. Restrict immunocompromised personnel with zoster from contact with patients until their lesions are crusted (Table 3).⁹ **Category IB**
 - k. Restrict susceptible personnel exposed to zoster from patient contact from the tenth day after the first exposure through the 21st day after the last exposure (28th day if VZIG was given; Table 3).⁹ **Category IB**
 - l. Perform serologic screening for immunity to varicella on exposed personnel who have not had varicella or are unvaccinated against varicella.^{9,13} **Category IB**
 - m. Consider performing serologic screening for immunity to varicella on exposed, vaccinated personnel whose antibody status is not known. If the initial test result is negative, retest 5 to 6 days after exposure to determine whether an immune response occurred. **Category IB**
 - n. Consider excluding vaccinated personnel from work beginning on the 10th day after the first exposure through the 21st day after the last exposure if they do not have detectable antibodies to varicella, or screen daily for symptoms of varicella (Table 3).⁹ **Category IB**
 - o. Do not routinely give VZIG to exposed susceptible personnel, unless immunosuppressed, HIV infected, or pregnant. If VZIG is given, exclude personnel from duty from the 10th day after the first exposure through the 28th day after the last exposure (Tables 1 and 3).^{9,13} **Category IB**

22. Viral respiratory infections

- a. Administer influenza vaccine annually to all personnel, including pregnant women, before the influenza season, unless otherwise contraindicated (Table 1).^{9,17} **Category IB**
- b. Consider the use of antiviral postexposure prophylaxis for unvaccinated health care personnel during institutional or community outbreaks of influenza for the duration of influenza activity, or consider giving vaccine to unvaccinated personnel and providing them with antiviral postexposure prophylaxis for 2 weeks after vaccination (Table 1).^{3,17,459} **Category IB**
- c. Consider excluding personnel with acute febrile respiratory infections or with laboratory evidence of epidemiologically significant viruses from the care of high-risk patients (e.g., neonates, young infants, patients with chronic obstructive lung disease, and immunocompromised patients) during community outbreaks of influenza or RSV infections (Table 3).³ **Category IB**

H. SPECIAL ISSUES

1. Pregnancy

- a. Counsel pregnant women and women of childbearing age regarding the risk of transmission of particular infectious diseases (e.g., CMV, hepatitis, herpes simplex, HIV, parvovirus, rubella) that, if acquired during pregnancy, may have adverse effects on the fetus, whether the infection is acquired in nonoccupational or occupational environments. Provide such women with information on standard and transmission-based precautions appropriate for each infection (Table 6).^{3,489-491} **Category IB**
- b. Do not routinely exclude women only on the basis of their pregnancy or intent to be pregnant from the care of patients with particular infections that have potential to harm the fetus (e.g., CMV, HIV, hepatitis, herpes simplex, parvovirus, rubella, and varicella; Table 6).⁴⁸⁹⁻⁴⁹¹ **Category IB**

2. Emergency-response employees

Ensure that emergency-response employees are routinely notified of infectious diseases in patients they have cared for or transported, in accordance with the mandates of the 1990 Ryan White Comprehensive AIDS Resources Emergency Act (Subtitle B 42 USC 300ff-80). **Category IA**

3. Personnel linked to outbreaks of bacterial infection

- a. Perform cultures and organism typing only on personnel who are linked epidemiologically to an increase in bacterial infections caused by a pathogen associated with a carrier state; if culture results are positive, exclude personnel from patient contact until carriage is eradicated or the risk of disease transmission is eliminated. **Category IB**
- b. Do not perform routine surveillance cultures of health care personnel for bacteria or multidrug-resistant organisms in the absence of a cluster or epidemic of bacterial infections in which personnel are implicated. **Category IA**
- c. Do not exclude personnel from duty who are colonized with bacteria, including multidrug-resistant bacteria, who are not epidemiologically linked to an increase in infections. **Category IB**

4. Latex hypersensitivity

- a. Develop an institutional protocol for (1) evaluating and managing personnel with suspected or known latex allergy, (2) establishing surveillance for latex reactions within the facility, (3) purchasing gloves, and (4) measuring the impact of preventive measures. Educational materials and activities should be provided to inform personnel about appropriate glove use and the manifestations and potential risk of latex allergy.^{31,546} **Category IB**
- b. Glove purchasers should review information on the barrier effectiveness of gloves and consider worker acceptance (e.g., comfort and fit) when selecting gloves for use in the health care organization.^{31,547-549} **Category IB**
- c. To facilitate the appropriate selection of gloves, the occupational health service should maintain a list of all gloves used the institution according to whether they do or do not contain latex. **Category II**
- d. Evaluate personnel with symptoms suggestive of latex allergy (e.g., localized dermatitis and workplace-related asthma).⁵²² Use serologic tests only for those who, on the basis of this evaluation, have suspected latex allergy.^{504,516} **Category IB**
- e. Avoid the use of all latex products by personnel with a history of systemic reactions to latex.^{509-512,520,522-524} **Category IB**
- f. Use nonlatex gloves for personnel with localized reactions to latex.^{502,507,513-515} **Category IB**

- g. Target interventions (e.g., substitution of nonlatex gloves and powder-free latex gloves) to areas of the facility where personnel have acquired systemic allergic reaction to latex.^{506,533,534} **Category IB**
- h. NO RECOMMENDATION for institution-wide substitution of nonlatex products in health care facilities to prevent sensitization to latex among health care personnel. **UNRESOLVED ISSUE**
- i. NO RECOMMENDATION for the routine use of environmental abatement interventions (such as laminar-flow or high-efficiency particulate air filtration) to reduce latex aeroallergens.⁵³⁴ **UNRESOLVED ISSUE**

The Hospital Infection Practices Advisory Committee (HICPAC) thanks the following subject-matter experts for reviewing a preliminary draft of this guideline: Bradley N. Doebbeling, MD, MSc, University of Iowa, Iowa City, Iowa; Victoria J. Fraser, MD, Washington University School of Medicine, St. Louis, Missouri; Kent A. Sepkowitz, MD, Memorial Sloan-Kettering Cancer Center, New York, New York; David J. Weber, MD, MPH, University of North Carolina, Chapel Hill, North Carolina. The opinions of all the reviewers might not be reflected in all the recommendations contained in this document.

References

1. Sepkowitz KA. Occupationally acquired infections in health care workers. Part I. *Ann Intern Med* 1996; 125:826-34.
2. Sepkowitz KA. Occupationally acquired infections in health care workers Part II. *Ann Intern Med* 1996; 125:917-28.
3. Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53-80.
4. Williams WW. CDC guideline for infection control in hospital personnel. *Infect Control* 1983;4(suppl):326-49.
5. Centers for Disease Control and Prevention, National Institutes for Health. Biosafety in microbiological and biomedical laboratories. 3rd ed. Atlanta: US Department of Health and Human Services, Public Health Service; 1993.
6. National Committee for Clinical Laboratory Standards. Protection of laboratory workers from infectious disease transmitted by blood, body fluids, and tissue: tentative guideline. NCCLS Document M29-T2. Villanova (PA): NCCLS; 1991;11(14):1-214.
7. Heseltine PNR, Ripper M, Wohlford P. Nosocomial rubella—consequences of an outbreak and efficacy of a mandatory immunization program. *Infect Control* 1985;6:371-4.
8. Centers for Disease Control. Update on adult immunization: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1991;40(RR-12):1-94.
9. Centers for Disease Control and Prevention. Immunization of health-care workers: recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Hospital Infection Control Practices Advisory Committee (HICPAC). *MMWR Morb Mortal Wkly Rep* 1997;46(RR-18):1-42.

10. Centers for Disease Control. Protection against viral hepatitis: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1990;39 (RR-2):1-27.
11. Centers for Disease Control. Measles prevention: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1989;38(S-9):1-18.
12. Centers for Disease Control. Mumps prevention: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1989;38:388-92, 397-400.
13. Centers for Disease Control and Prevention. Prevention of varicella: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1996;45(RR-11):1-36.
14. Centers for Disease Control. Rubella prevention: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1990;39(RR-15):1-18.
15. Centers for Disease Control and Prevention. Control and prevention of meningococcal disease and control and prevention of serogroup C meningococcal disease: evaluation and management of suspected outbreaks: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1997;46(RR-5):1-21.
16. Centers for Disease Control and Prevention. Update: vaccine side effects, adverse reactions, contraindications, and precautions: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1996;45(RR-12):1-35.
17. Centers for Disease Control and Prevention. Prevention and control of influenza: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1997;46(RR-9):1-25.
18. Centers for Disease Control. Vaccinia (smallpox) vaccine: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1991;40(RR-14):1-10.
19. Centers for Disease Control. Diphtheria, tetanus, pertussis: recommendations for vaccine use and other preventive measures—recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1991;40(RR-10):1-28.
20. Centers for Disease Control and Prevention. Prevention of pneumococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1997;46(RR-8):1-24.
21. Centers for Disease Control and Prevention. Poliomyelitis prevention in the United States: introduction of a sequential vaccination schedule of inactivated poliovirus vaccine followed by oral poliovirus vaccine: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1997;46(RR-3):1-25.
22. Centers for Disease Control. Rabies prevention—United States, 1991: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1991;40(RR-3):1-19.
23. Centers for Disease Control and Prevention. Recommendations of the Advisory Committee on Immunization Practices (ACIP): use of vaccines and immune globulins in persons with altered immunocompetence. *MMWR Morb Mortal Wkly Rep* 1993;42(RR-4):1-18.
24. American College of Physicians Task Force on Adult Immunization and Infectious Diseases Society of America. Guide for adult immunization. 3rd ed. Philadelphia: American College of Physicians; 1994.
25. Herwaldt LA, Pottinger JM, Carter CD, Barr BA, Miller ED. Exposure workups. *Infect Control Hosp Epidemiol* 1997;18:850-71.
26. US Department of Labor, Occupational Health and Safety Administration. Record keeping guidelines for occupational injuries and illnesses: the occupational safety and health act of 1970 and 29 CFR 1904. OMB no. 120-0029. Washington, DC: US Department of Labor; 1986.
27. US Department of Labor, Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens; final rule. CFR part 1910.1030. *Federal Register* 1991;56:64004-182.
28. US Department of Labor, Occupational Health and Safety Administration. Criteria for recording on OSHA form 200. OSHA instruction 1993; standard 1904. Washington, DC: US Department of Labor; 1993.
29. US Department of Labor, Occupational Safety and Health Administration. Enforcement procedures and scheduling for occupational exposure to tuberculosis. OSHA instruction 1996; CPL 2.106. Washington, DC: US Department of Labor; 1996.
30. Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. *MMWR Morb Mortal Wkly Rep* 1987;36(2S):1S-18S.
31. Centers for Disease Control. Update: universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *MMWR Morb Mortal Wkly Rep* 1988;37:377-82, 387-8.
32. Centers for Disease Control. Guidelines for prevention of transmission of human immunodeficiency virus and hepatitis B virus to health-care and public-safety workers. *MMWR Morb Mortal Wkly Rep* 1989;38(S-6):1-36.
33. Centers for Disease Control. Public Health Service statement on management of occupational exposure to human immunodeficiency virus, including considerations regarding zidovudine postexposure use. *MMWR Morb Mortal Wkly Rep* 1990;39(RR-1):1-14.
34. Centers for Disease Control. Public Health Service interagency guidelines for screening donors of blood, plasma, organs, tissues, and semen for evidence of hepatitis B and hepatitis C. *MMWR Morb Mortal Wkly Rep* 1991;40(RR-4):1-17.
35. Centers for Disease Control and Prevention. Recommended infection-control practices for dentistry, 1993. *MMWR Morb Mortal Wkly Rep* 1993;41(RR-8):1-12.
36. Centers for Disease Control and Prevention. Human immunodeficiency virus transmission in household settings—United States. *MMWR Morb Mortal Wkly Rep* 1994;43:347, 353-6.
37. Centers for Disease Control. Recommendations for follow-up of health-care workers after occupational exposure to hepatitis C virus. *MMWR Morb Mortal Wkly Rep* 1997;46:603-6.
38. Centers for Disease Control and Prevention, National Institutes for Health. Agent summary statement: retroviruses, including human and simian immunodeficiency viruses. In: Richmond JY, McKinney RW, editors. *Biosafety in microbiological and biomedical laboratories*. 3rd ed. Washington, DC: US Government Printing Office; 1993. p. 116-21.
39. Centers for Disease Control and Prevention. Occupationally acquired human immunodeficiency virus infections in laboratories producing virus concentrates in large quantities: conclusions and recommendations of an

- expert team convened by the director of the National Institutes of Health (NIH). *MMWR Morb Mortal Wkly Rep* 1988;37(S-4):19-22.
40. Centers for Disease Control. Hepatitis B virus: a comprehensive strategy for eliminating transmission in the United States through universal childhood vaccination recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1991;40(RR-13):1-25.
 41. Bolyard EA, Bell DM. Universal precautions in the health care setting. In: Devita VT, Hellman S, Rosenberg SA, editors. *AIDS: etiology, diagnosis, treatment and prevention*. 4th ed. Philadelphia: Lippincott-Raven; 1997. p. 655-64.
 42. Benson JS. FDA safety alert: needlestick and other risks from hypodermic needles on secondary IV administration sets—piggyback and intermittent IV. Rockville (MD): US Department of Health and Human Services, Food and Drug Administration; 1992 April 16.
 43. Rhodes RS, Bell DM, editors. *Prevention of transmission of bloodborne pathogens*. *Surg Clin North Am* 1995;75:1047-217.
 44. Short LJ, Benson DR. Special considerations for surgeons. In: Devita VT, Hellman H, Rosenberg SA, editors. *AIDS: etiology, diagnosis, treatment, and prevention*. Philadelphia: Lippincott-Raven; 1997. p. 665-73.
 45. Cardo DM, Culver DH, Ciesielski C, Srivastava PU, Marcus R, Abiteboul D, et al. A case-control study of HIV seroconversion in health care workers after percutaneous exposure. *N Engl J Med* 1997;337:1485-90.
 46. Centers for Disease Control and Prevention. Evaluation of safety devices for preventing percutaneous injuries among health-care workers during phlebotomy procedures—Minneapolis—St. Paul, New York City, and San Francisco, 1993-1995. *MMWR Morb Mortal Wkly Rep* 1997;46:21-5.
 47. Centers for Disease Control and Prevention. Evaluation of blunt suture needles in preventing percutaneous injuries among health-care workers during gynecologic surgical procedures—New York City, March 1993-June 1994. *MMWR Morb Mortal Wkly Rep* 1997;46:25-9.
 48. Centers for Disease Control. Recommendations for preventing transmission of human immunodeficiency virus and hepatitis B virus to patients during exposure-prone invasive procedures. *MMWR Morb Mortal Wkly Rep* 1991;40(RR-8):1-9.
 49. Thomas DL, Factor SH, Kelen GD, Washington AS, Taylor E Jr, Quinn TC. Viral hepatitis in health care personnel at the Johns Hopkins Hospital: the seroprevalence of and risk factors for hepatitis B virus and hepatitis C virus infection. *Arch Intern Med* 1993;153:1705-12.
 50. Dienstag JL, Ryan DM. Occupational exposure to hepatitis B virus in hospital personnel: infection or immunization? *Am J Epidemiol* 1982;115:26-39.
 51. Shapiro CN, Tokars JI, Chamberland ME, American Academy of Orthopaedic Surgeons Serosurvey Study Committee. Use of the hepatitis B vaccine and infection with hepatitis B and C among orthopaedic surgeons. *J Bone Joint Surg* 1996;78A:1791-800.
 52. Gibas A, Blewett DR, Schoenfield DA, Dienstag JL. Prevalence and incidence of viral hepatitis in health workers in the pre-hepatitis B vaccination era. *Am J Epidemiol* 1992;136:603-10.
 53. Hadler SC, Doto IL, Maynard JE, Smith J, Clark B, Mosley J, et al. Occupational risk of hepatitis B infection in hospital workers. *Infect Control* 1985;6:24-31.
 54. Shapiro CN. Occupational risk of infection with hepatitis B and hepatitis C virus. *Surg Clin North Am* 1995;75:1047-56.
 55. Moyer LA, Alter MJ, Favero MS. Hemodialysis-associated hepatitis B: revised recommendations for serologic screening. *Semin Dialysis* 1990;3:201-4.
 56. Hadler SC, Margolis HS. Hepatitis B immunization: vaccine types, efficacy, and indications for immunization. *Curr Clin Top Infect Dis* 1992;12:282-308.
 57. Wainwright RB, Bulkow LR, Parkinson AJ, Zanis C, McMahon BJ. Protection provided by hepatitis B vaccine in a Yupik Eskimo population—results of a 10-year study. *J Infect Dis* 1997;175:674-7.
 58. Alter MJ, Coleman PJ, Alexander WJ, Kramer E, Miller JK, Mandel E, et al. Importance of heterosexual activity in the transmission of hepatitis B and non-A, non-B hepatitis. *JAMA* 1989;262:1201-5.
 59. Alter MJ. The detection, transmission, and outcome of hepatitis C virus infection. *Infect Agents Dis* 1993;2:155-66.
 60. Alter MJ, Gerety RJ, Smallwood LA, Sampliner RE, Tabor E, Deinhardt F, et al. Sporadic non-A, non-B hepatitis: frequency and epidemiology in an urban United States population. *J Infect Dis* 1982;145:886-93.
 61. Polish LB, Tong MJ, Co RL, Coleman PJ, Alter MJ. Risk factors for hepatitis C virus infection among health care personnel in a community hospital. *AJIC Am J Infect Control* 1993;21:196-200.
 62. Cooper BW, Krusell A, Tilton RC, Goodwin R, Levitz RE. Seroprevalence of antibodies to hepatitis C virus in high-risk hospital personnel. *Infect Control Hosp Epidemiol* 1992;13:82-5.
 63. Panlilio AL, Shapiro CN, Schable CA, Mendelson MH, Montecalvo MA, Kunches LM, et al. Serosurvey of human immunodeficiency virus, hepatitis B virus, and hepatitis C virus infection among hospital-based surgeons. *J Am Coll Surg* 1995;180:16-24.
 64. Nishimura Y, Yamaguchi K, Williams NP, Takatsuki K. Antibodies to hepatitis C virus in Japanese blood donors and in hospital personnel. *Transfusion* 1990;30:667-8.
 65. Herbert AM, Walker DM, Kavies KJ, Bagg J. Occupationally acquired hepatitis C virus infection [letter]. *Lancet* 1992;339:305.
 66. Tsude K, Fujiyama S, Sato S, Kawano S, Taura Y, Yoshida K, et al. Two cases of accidental transmission of hepatitis C to medical staff. *Hepatogastroenterology* 1992;39:73-5.
 67. Zuckerman J, Clewley G, Griffiths P, Cockcroft A. Prevalence of hepatitis C antibodies in clinical health-care workers. *Lancet* 1994;343:1618-20.
 68. Petrosilla N, Puro V, Ipolito G, and the Italian Study Group on bloodborne Occupational Risk in Dialysis. Prevalence of hepatitis C antibodies in health-care workers. *Lancet* 1994;344:339-40.
 69. Lanphear BP, Linneman CC, Cannon CG, DeRonde MM, Pandy L, Kerley LM. Hepatitis C virus infection in health care workers: risk of exposure and infection. *Infect Control Hosp Epidemiol* 1994;15:745-50.
 70. Mitsui T, Iwano K, Masuko K, Yamazaki C, Okamoto H, Tsuda F, et al. Hepatitis C virus infection in medical personnel after needlestick accident. *Hepatology* 1992;16:1109-14.
 71. Knodell RG, Conrad ME, Ginsberg AL, Bell CJ. Efficacy of prophylactic gamma-globulin in preventing non-A, non-B post-transfusion hepatitis. *Lancet* 1976;1:557-61.
 72. Seeff LB, Zimmerman HJ, Wright EC, Finkelstein JD, Garcia-Pont P, Greenlee HB, et al. A randomized, double blind controlled trial of the efficacy of immune serum globulin for the prevention of post-transfusion hepatitis:

- a Veterans Administration cooperative study. *Gastroenterology* 1977;72:111-21.
73. Sanchez-Quijano A, Pineda JA, Lissen E, Leal M, Diaz-Torres MA, Garcia DePescuera F, et al. Prevention of post-transfusion non-A, non-B hepatitis by non-specific immunoglobulin in heart surgery patients. *Lancet* 1988;1:1245-9.
 74. Krawczynski K, Alter MJ, Govindarajan S, Tankersley DL, Lambert S, Meeks E, et al. Studies on protective efficacy of hepatitis C immunoglobulins (HCIG) in experimental hepatitis C virus infection [abstract]. *Hepatology* 1993;18:110A.
 75. Tokars JI, Marcus R, Culver DH, Schable CA, McKibben PS, Bandea CL, et al. Surveillance of HIV infection and zidovudine use among health care workers after occupational exposure to HIV-infected blood. *Ann Intern Med* 1993;118:913-9.
 76. Henderson D. HIV-1 in the health care setting. In: Mandel G, Bennett J, Dolan R, editors. *Principles and practices of infectious diseases*. 4th ed. New York: Churchill Livingstone; 1995. p. 2632-56.
 77. Puro V, Ippolito G, Guzzanti E, Serafini I, Pagano G, Suter F, et al. Zidovudine prophylaxis after accidental exposure to HIV: the Italian experience. *AIDS* 1992;6:963-9.
 78. Chamberland ME, Ciesielski CA, Howard RJ, Fry DE, Bell DM. Occupational risk of infection with human immunodeficiency virus. *Surg Clin North Am* 1995;75:1057-70.
 79. Marcus R, Bell DM. Occupational risk of human immunodeficiency virus. In: Devita VT, Hellman S, Rosenberg SA, editors. *AIDS: etiology, diagnosis, treatment and prevention*. 4th ed. Philadelphia: Lippincott-Raven; 1997. p. 645-54.
 80. Centers for Disease Control and Prevention. Update: provisional Public Health Service recommendations for chemoprophylaxis after occupational exposure to HIV. *MMWR Morb Mortal Wkly Rep* 1996;45:468-72.
 81. Centers for Disease Control and Prevention. Public Health Service (PHS) guidelines for the management of health care workers exposures to HIV and recommendations for postexposure prophylaxis. *MMWR Morb Mortal Wkly Rep*. In press 1998.
 82. Centers for Disease Control. Epidemic keratoconjunctivitis in an ophthalmology clinic—California. *MMWR Morb Mortal Wkly Rep* 1990;39:598-601.
 83. Ford E, Nelson KE, Warren D. Epidemiology of epidemic keratoconjunctivitis. *Epidemiol Rev* 1987;9:244-61.
 84. Birenbaum E, Linder N, Varsano N, Azar R, Kuint J, Spierer A, et al. Adenovirus type 8 conjunctivitis outbreak in a neonatal intensive care unit. *Arch Dis Child* 1993;68:610-1.
 85. Warren D, Nelson KE, Farrar JA, Hurwitz E, Hierholzer J, Ford E, et al. A large outbreak of epidemic keratoconjunctivitis: problems in controlling nosocomial spread. *J Infect Dis* 1989;160:938-43.
 86. Jernigan JA, Lowry BS, Hayden FG, Kyger SA, Conway BP, Gröschel DHM, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. *J Infect Dis* 1993;167:1307-13.
 87. Adler SP. Molecular epidemiology of cytomegalovirus: a study of factors affecting transmission among children at three day-care centers. *Pediatr Infect Dis J* 1991;10:584-90.
 88. Adler SP, Bagget J, Wilson M, Lawrence L, McVoy M. Molecular epidemiology of cytomegalovirus in a nursery: lack of evidence for nosocomial transmission. *J Pediatr* 1986;108:117-23.
 89. Meyers JD, Fluornoy N, Thomas ED. Nonbacterial pneumonia after allogeneic marrow transplantation: a review of ten years' experience. *Rev Infect Dis* 1982;3:1119-32.
 90. Bowden RA, Fisher LD, Rogers K, Cays M, Meyers JD. Cytomegalovirus (CMV)-specific intravenous immunoglobulin for the prevention of primary CMV infection and disease after marrow transplant. *J Infect Dis* 1991;164:483-7.
 91. Brady MT, Demmler GJ, Reis S. Factors associated with cytomegalovirus excretion in hospitalized children. *Am J Infect Control* 1988;16:41-5.
 92. Demmler GJ, Yow MD, Spector SA, Reis SC, Brady MT, Anderson DC, et al. Nosocomial cytomegalovirus infections within two hospitals caring for infants and children. *J Infect Dis* 1987;156:9-16.
 93. Rubin RH, Wolfson JS, Cosimi AB, Tolkoff-Rubin NE. Infection in the renal transplant recipient. *Am J Med* 1981;70:405-11.
 94. Pomeroy C, Englund JA. Cytomegalovirus: epidemiology and infection control. *Am J Infect Control* 1987;15:107-19.
 95. Ahlfors K, Ivarsson SA, Johnson T, Renmarker K. Risk of cytomegalovirus infection in nurses and congenital infection in their offspring. *Acta Paediatr Scand* 1981;70:819-23.
 96. Dworsky ME, Welch K, Cassidy G, Stango S. Occupational risk for primary cytomegalovirus infection among pediatric health-care workers. *N Engl J Med* 1983;309:950-3.
 97. Yeager AS. Longitudinal, serological study of cytomegalovirus infections in nurses and in personnel without patient contact. *J Clin Microbiol* 1975;2:448-52.
 98. Gerberding JL, Bryant-LeBlanc CE, Nelson K, Moss AR, Osmond D, Chambers HF, et al. Risk of transmitting the human immunodeficiency virus, cytomegalovirus, and hepatitis B virus to health care workers exposed to patients with AIDS and AIDS-related conditions. *J Infect Dis* 1987;156:1-8.
 99. Blackman JA, Murph JR, Bale JF. Risk of cytomegalovirus infection among educators and health care personnel serving disabled children. *Pediatr Infect Dis J* 1987;6:725-9.
 100. Tolkoff-Rubin NE, Rubin RH, Keller EE, Baker GP, Stewart JA, Hirsh MS. Cytomegalovirus infection in dialysis patients and personnel. *Ann Intern Med* 1978;89:625-8.
 101. Adler SP. Hospital transmission of cytomegalovirus. *Infect Agents Dis* 1992;1:43-9.
 102. Balfour CL, Balfour HH. Cytomegalovirus is not an occupational risk for nurses in renal transplant and neonatal units. *JAMA* 1986;256:1909-14.
 103. Brady MT, Demmler GJ, Anderson DC. Cytomegalovirus infection in pediatric house officers: susceptibility to and new rate of primary infection. *Infect Control* 1987;8:329-32.
 104. Lipscomb JA, Linneman CC, Hurst PF, Myers MG, Stringer W, Moore P, et al. Prevalence of cytomegalovirus antibody in nursing personnel. *Infect Control* 1984;5:513-8.
 105. Friedman HM, Lewis MR, Nemerovsky DM, Plotkin SA. Acquisition of cytomegalovirus infection among female employees at a pediatric hospital. *Pediatr Infect Dis J* 1984;3:233-5.
 106. Balcarek KB, Bagley R, Cloud GA, Pass RF. Cytomegalovirus infection among employees of a children's hospital: no evidence for increased risk associated with patient care. *JAMA* 1990;263:840-4.
 107. Tookey P, Peckham CS. Does cytomegalovirus present an occupational risk? *Arch Dis Child* 1991;66:1009-10.
 108. Hokeberg I, Grillner L, Reisenfeld T, Diderholm H. No evidence of hospital-acquired cytomegalovirus on environmental surfaces. *Pediatr Infect Dis J* 1988;7:812-4.
 109. Yow MD, Lakeman AD, Stagno S, Reynolds RB, Plavidal FJ. Use of restriction enzymes to investigate the source of a primary cytomegalovirus infection in a pediatric nurse. *Pediatrics* 1982;70:713-6.

110. Wilfert CM, Huang E, Stagno S. Restriction endonuclease analysis of cytomegalovirus deoxyribonucleic acid as an epidemiologic tool. *Pediatrics* 1982;70:717-21.
111. Spector SA. Transmission of cytomegalovirus among infants in hospital documented by restriction-endonuclease-digestion analyses. *Lancet* 1983;2:378-81.
112. Pass RF, Hutto C, Lyon MD, Cloud G. Increased rate of cytomegalovirus infection among day care center workers. *Pediatr Infect Dis J* 1990;9:465-70.
113. Pass RF, Hutto C, Ricks R, Cloud GA. Increased rate of cytomegalovirus infection among parents of children attending day-care centers. *N Engl J Med* 1986;314:1414-8.
114. Adler SP. Cytomegalovirus and child day care: evidence for an increased infection rate among day-care workers. *N Engl J Med* 1989;321:1290-6.
115. Hutto C, Little EA, Ricks R. Isolation of cytomegalovirus from toys and hands in a day care center. *J Infect Dis* 1986;154:527-30.
116. Faix RG. Survival of cytomegalovirus on environmental surfaces. *J Pediatr* 1985;106:649-52.
117. Finney JW, Miller KM, Adler SP. Changing protective and risky behaviors to prevent child-to-parent transmission of cytomegalovirus. *J Appl Behav Anal* 1993;26:471-2.
118. Stagno S, Pass RF, Dworsky ME, Alford CA Jr. Maternal cytomegalovirus infection and perinatal transmission. *Clin Obstet Gynecol* 1982;25:563-76.
119. Onorato IM, Morens DM, Martone WJ, Stansfield SK. Epidemiology of cytomegalovirus infections: recommendations for prevention and control. *Rev Infect Dis* 1985;7:479-97.
120. Balcarek KB, Bagley R, Cloud GA. Nosocomial cytomegalovirus infections within two hospitals caring for infants and children. *J Infect Dis* 1987;145:9-16.
121. American Academy of Pediatrics. Summaries of infectious diseases: cytomegalovirus infection. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove (IL): American Academy of Pediatrics; 1997. p. 187-91.
122. Plotkin SA, Starr SE, Friedman HM, Gonczole E, Brayman K. Vaccines for the prevention of human cytomegalovirus infection. *Rev Infect Dis* 1990;12(suppl 7):S827-38.
123. Adler SP, Starr SE, Plotkin SA, Hempfling SH, Buis J, Manning ML, et al. Immunity induced by primary human cytomegalovirus infection protects against secondary infection among women of childbearing age. *J Infect Dis* 1994;171:26-32.
124. Plotkin SA, Starr SE, Friedman HM, Brayman K, Harris S, Jackson S, et al. Effect of Towne live vaccine on cytomegalovirus disease after renal transplants: a controlled trial. *Ann Intern Med* 1991;114:525-31.
125. Fleisher GR, Starr SE, Friedman HM, Plotkin SA. Vaccination of pediatric nurses with live attenuated cytomegalovirus. *Am J Dis Child* 1982;136:294-6.
126. Snyderman DR, Werner BG, Heinz-Lacy B, Berardi VP, Tilney NL, Kirkman RL, et al. Use of cytomegalovirus immune globulin to prevent cytomegalovirus disease in renal-transplant recipients. *N Engl J Med* 1987;317:1049-54.
127. Meyers JD, Reed EC, Shepp DH, Thornquist M, Dandliker PS, Vicary CA, et al. Acyclovir for prevention of cytomegalovirus infection and disease after allogeneic marrow transplantation. *N Engl J Med* 1988;318:70-5.
128. Goodrich JM, Mori M, Gleaves CA, DuMond C, Cays M, Ebeling DF, et al. Early treatment with ganciclovir to prevent cytomegalovirus disease after allogeneic bone marrow transplantation. *N Engl J Med* 1991;325:1601-7.
129. Bailey TC, Trulock EP, Ettinger NA, Storch GA, Cooper JD, Powderly WG. Failure of prophylactic ganciclovir to prevent cytomegalovirus disease in recipients of lung transplants. *J Infect Dis* 1992;165:548-52.
130. Hatherly LI. Is primary cytomegalovirus infection an occupational hazard for obstetric nurses? A serological study. *Infect Control* 1986;7:452-5.
131. Anderson GS, Penfold JB. An outbreak of diphtheria in a hospital for the mentally subnormal. *J Clin Pathol* 1973;26:606-15.
132. Gray RD, James SM. Occult diphtheria infection in a hospital for the mentally subnormal. *Lancet* 1973;1:1105-6.
133. Palmer SR, Balfour AH, Jephcott AE. Immunisation of adults during an outbreak of diphtheria. *BMJ* 1983;286:624-6.
134. Bisgard KM, Hardy IRB, Popvic T, Strebel PM, Wharton M, Chen T, et al. Respiratory diphtheria in the United States, 1980-1995. *Am J Public Health*. In press 1997.
135. Harnisch JP, Tronca E, Nolan CM, Turck M, Holmes KK. Diphtheria among alcoholic urban adults: a decade of experience in Seattle. *Ann Intern Med* 1989;111:71-82.
136. Centers for Disease Control and Prevention. Toxigenic *Corynebacterium diphtheriae*—northern plains Indian community, August-October 1996. *MMWR Morb Mortal Wkly Rep* 1997;46:506-10.
137. Centers for Disease Control and Prevention. Update: diphtheria epidemic—new independent states of the former Soviet Union, January 1995-March 1996. *MMWR Morb Mortal Wkly Rep* 1996;45:693-7.
138. Centers for Disease Control and Prevention. Diphtheria epidemic—new independent states of the former Soviet Union, 1990-1994. *MMWR Morb Mortal Wkly Rep* 1995;44:177-81.
139. Hardy IRB, Dittmann S, Sutter RW. Current situation and control strategies for resurgence of diphtheria in newly independent states of the former Soviet Union. *Lancet* 1996;347:1739-44.
140. Centers for Disease Control and Prevention. Diphtheria outbreak—Saraburi Province, Thailand, 1994. *MMWR Morb Mortal Wkly Rep* 1996;45:271-3.
141. Lumio J, Jahkola M, Vuento R, Haikala O, Eskila J. Diphtheria after visit to Russia. *Lancet* 1993;342:53-4.
142. Centers for Disease Control and Prevention. Diphtheria acquired by U.S. citizens in the Russian Federation and Ukraine—1994. *MMWR Morb Mortal Wkly Rep* 1995;44:237, 243-4.
143. American Academy of Pediatrics. Summaries of infectious diseases: diphtheria. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 191-5.
144. Sargent RK, Rossing TH, Downton SB, Breyer MD, Levine L, Weinstein L. Diphtheria immunity in Massachusetts—a study of three urban patient populations. *Am J Med Sci* 1984;287:37-9.
145. Weiss BP, Strassburg MA, Feeley JC. Tetanus and diphtheria immunity in an elderly population in Los Angeles County. *Am J Public Health* 1983;73:802-4.
146. Crossley K, Irvine P, Warren JB, Lee BK, Mead K. Tetanus and diphtheria immunity in urban Minnesota adults. *JAMA* 1979;242:2298-3000.

147. Ruben FL, Nagel J, Fireman P. Antitoxin responses in the elderly to tetanus-diphtheria (TD) immunization. *Am J Epidemiol* 1978;108:145-9.
148. Koblin BA, Townsend TR. Immunity to diphtheria and tetanus in inner-city women of childbearing age. *Am J Public Health* 1989;79:1297-8.
149. Farizo KM, Strebel PM, Chen RT, Kimbler A, Cleary TJ, Cochi SL. Fatal respiratory disease due to *Corynebacterium diphtheriae*: case report and review of guidelines for management, investigation, and control. *Clin Infect Dis* 1993;16:59-68.
150. Steere AC, Craven PJ, Hall WJ 3rd, Leotsukis N, Wells JG, Farmer JJ 3rd, et al. Person-to-person spread of *Salmonella typhimurium* after a hospital common-source outbreak. *Lancet* 1975;1:319-22.
151. Blaser MJ, Hickman FW, Farmer JJ 3rd, Brenner DJ, Balow SA, Feldman RA. *Salmonella typhi*: the laboratory as a reservoir of infection. *J Infect Dis* 1980;142:934-8.
152. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of *Salmonella* gastroenteritis to laundry workers in a nursing home. *Infect Control Hosp Epidemiol* 1994;15:22-6.
153. Toivanen P, Toivanen A, Olkkonen L, Aantaa S. Hospital outbreak of *Yersinia enterocolitica* infection. *Lancet* 1973;1:801-3.
154. Ratnam S, Mercer E, Picco B, Parsons S, Butler R. A nosocomial outbreak of diarrheal disease due to *Yersinia enterocolitica* serotype 0:5, biotype 1. *J Infect Dis* 1982;145:242-7.
155. Anglim AM, Farr BM. Nosocomial gastrointestinal tract infections. In: Mayhall CG, editor. *Hospital epidemiology and infection control*. Baltimore: Williams & Wilkins; 1996. p. 196-219.
156. Mitchell DK, Pickering LK. Nosocomial gastrointestinal tract infections in pediatric patients. In: Mayhall CG, editor. *Hospital epidemiology and infection control*. Baltimore: Williams & Wilkins; 1996. p. 506-23.
157. McGowan JE Jr. Nosocomial infections in diagnostic laboratories. In: Mayhall CG, editor. *Hospital epidemiology and infection control*. Baltimore: Williams & Wilkins; 1996. p. 883-92.
158. Kurtz JB, Lee TW, Pickering D. Astrovirus associated gastroenteritis in a children's ward. *J Clin Pathol* 1977;30:948-52.
159. Dryjanski J, Gold JWM, Ritchie MT, Kurtz RC, Lim SL, Armstrong D. Cryptosporidiosis: case report in a health team worker. *Am J Med* 1986;80:751-2.
160. Lewis DC, Lightfoot NF, Cubitt WD, Wilson SA. Outbreaks of astrovirus type 1 and rotavirus gastroenteritis in a geriatric in-patient population. *J Hosp Infect* 1989;14:9-14.
161. Koch KL, Phillips DJ, Aber RC, Current WL. Cryptosporidiosis in hospital personnel: evidence for person-to-person transmission. *Ann Intern Med* 1985;102:593-6.
162. Pike RM. Laboratory-associated infections: summary and analysis of 3921 cases. *Health Lab Sci* 1976;13:105-14.
163. Rodriguez EM, Parrott C, Rolka H, Monroe SS, Dwyer DM. An outbreak of viral gastroenteritis in a nursing home: importance of excluding ill employees. *Infect Control Hosp Epidemiol* 1996;17:587-92.
164. Gellert GA, Waterman SH, Ewert D, Oshiro L, Giles MP, Monroe SS, et al. An outbreak of acute gastroenteritis caused by a small round structured virus in a geriatric convalescent facility. *Infect Control Hosp Epidemiol* 1990;11:459-64.
165. Chadwick PR, McCann R. Transmission of a small round structured virus by vomiting during a hospital outbreak of gastroenteritis. *J Hosp Infect* 1994;26:251-9.
166. Linneman CC Jr, Cannon CG, Staneck JL, McNeely BL. Prolonged hospital epidemic of salmonellosis: use of trimethoprim-sulfamethoxazole for control. *Infect Control* 1985;6:221-5.
167. Tauxe RV, Hassan LF, Findeisen KO, Sharrar RG, Blake PA. Salmonellosis in nurses: lack of transmission to patients. *J Infect Dis* 1988;157:370-3.
168. Guerrant RL. Cryptosporidiosis: an emerging, highly infectious threat. *Emerging Infect Dis* 1997;3:51-7.
169. Schroeder SA, Aserkoff B, Brachman PS. Epidemic salmonellosis in hospitals and institutions: public health importance and outbreak management. *N Engl J Med* 1968;279:674-8.
170. Khuri-Bulos NA, Abu Khalaf M, Shehabi A, Shami K. Foodhandler-associated *Salmonella* outbreak in a university hospital despite routine surveillance cultures of kitchen employees. *Infect Control Hosp Epidemiol* 1994;15:311-4.
171. Centers for Disease Control. Viral agents of gastroenteritis. *MMWR Morb Mortal Wkly Rep* 1990;39:(RR-5):1-24.
172. Caul EO. Small round structured viruses: airborne transmission and hospital control. *Lancet* 1994;343:1240-2.
173. Noah ND. Airborne transmission of a small round structured virus. *Lancet* 1994;343:608-9.
174. Sawyer LA, Murphy JJ, Kaplan JE, Pinsky PF, Chacon D, Walmsley S, et al. 25-to 30-nm virus particle associated with a hospital outbreak of acute gastroenteritis with evidence for airborne transmission. *Am J Epidemiol* 1988;127:1261-71.
175. Sharp TW, Hyams KC, Watts D, Trofa AF, Martin GJ, Kapikian AZ, et al. Epidemiology of Norwalk virus during an outbreak of acute gastroenteritis aboard a US aircraft carrier. *J Med Virol* 1995;45:61-7.
176. Doebbeling BN, Stanley GL, Sheetz CT, Pfaller MA, Houston AK, Annis L, et al. Comparative efficacy of alternative handwashing agents in reducing nosocomial infections in intensive care units. *N Engl J Med* 1992;327:88-93.
177. Black DE, Dykes AC, Anderson KE, Wells JG, Sinclair SP, Gary GW, et al. Handwashing to prevent diarrhea in day care centers. *Am J Epidemiol* 1982;113:445-51.
178. Centers for Disease Control and Prevention. Typhoid immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1994;43(RR14):1-7.
179. Ho MS, Glass RI, Monroe SS, Madore HP, Stine S, Pinsky PF, et al. Viral gastroenteritis aboard a cruise ship. *Lancet* 1989;2:961-5.
180. Kilgore PE, Belay ED, Hamlin DM, Noel JS, Humphrey CD, Gary HE Jr, et al. A university outbreak of gastroenteritis due to a small round-structured virus: application of molecular diagnostics to identify the etiologic agent and patterns of transmission. *J Infect Dis* 1996;173:787-93.
181. Grohmann GS, Glass RI, Pereira HG, Monroe SS, Hightower AW, Weber R, et al. Enteric viruses and diarrhea in HIV-infected patients. *N Engl J Med* 1993;329:14-20.
182. Centers for Disease Control. Recommendations for collection of laboratory specimens associated with outbreaks of gastroenteritis. *MMWR Morb Mortal Wkly Rep* 1990;39(RR-14):1-13.
183. Salam MA, Bennish ML. Antimicrobial therapy for shigellosis. *Rev Infect Dis* 1991;13(suppl 4):S332-41.
184. Allos BM, Blaser MJ. *Campylobacter jejuni* and the expanding spectrum of related infections. *Clin Infect Dis* 1995;20:1092-9.
185. Buchwald DS, Blaser MJ. A review of human salmonellosis: II. duration of excretion following infection with nontyphi *Salmonella*. *Rev Infect Dis* 1984;6:345-56.

186. Aserkoff B, Bennett JV. Effect of antibiotic therapy in acute salmonellosis in the fecal excretion of *Salmonellae*. *N Engl J Med* 1969;281:636-40.
187. Pavia AT, Shipman LD, Wells JG, Puhr ND, Smith JD, McKinney TW, et al. Epidemiologic evidence that prior antimicrobial exposure decreases resistance to infection by antimicrobial-sensitive *Salmonella*. *J Infect Dis* 1990; 161:255-60.
188. Miller SI, Hohmann EL, Pegues DA. *Salmonella* (including *Salmonella typhi*). In: Mandell GL, Bennett JE, Dolin R, editors. Principles and practice of infectious diseases. 4th ed. New York: Churchill Livingstone; 1995. p. 2013-33.
189. Rosenblum LS, Villarino ME, Nainan OV, Melish ME, Hadler SC, Pinsley PP, et al. Hepatitis A outbreak in a neonatal intensive care unit: risk factors for transmission and evidence of prolonged viral excretion among preterm infants. *J Infect Dis* 1991;164:476-82.
190. Carl M, Kantor RJ, Webster HM, Fields MA, Maynard JE. Excretion of hepatitis A virus in the stools of hospitalized patients. *J Med Virol* 1982;9:125-9.
191. Drusin LM, Sohmer M, Groshen SL, Spiritos MD, Senterfit LB, Christenson WN. Nosocomial hepatitis A infection in a paediatric intensive care unit. *Arch Dis Child* 1987;62:690-5.
192. Baptiste R, Koziol D, Henderson DK. Nosocomial transmission of hepatitis A in an adult population. *Infect Control* 1987;8:364-70.
193. Azimi PH, Roberto RR, Guralnik J, Livermore T, Hoag S, Hagens S, et al. Transfusion-acquired hepatitis A in a premature infant with secondary nosocomial spread in an intensive care nursery. *Am J Dis Child* 1986;140:23-7.
194. Goodman RA, Carder CC, Allen JR, Orenstein WA, Finton RJ. Nosocomial hepatitis A transmission by an adult patient with diarrhea. *Am J Med* 1982;73:220-6.
195. Skidmore SJ, Gully PR, Middleton JD, Hassam ZA, Singal GM. An outbreak of hepatitis A on a hospital ward. *J Med Virol* 1985;17:175-7.
196. Klein BS, Michaels JA, Rytel MW, Berg KG, Davis JP. Nosocomial hepatitis A: a multinursery outbreak in Wisconsin. *JAMA* 1984;252:2716-21.
197. Krober MS, Bass JW, Brown JD, Lemon SM, Rupert KJ. Hospital outbreak of hepatitis A: risk factors for spread. *Pediatr Infect Dis J* 1984;3:296-9.
198. Reed CM, Gustafson TL, Siegel J, Duer P. Nosocomial transmission of hepatitis A from a hospital-acquired case. *Pediatr Infect Dis J* 1984;3:300-3.
199. Doebbeling BN, Li N, Wenzel RP. An outbreak of hepatitis A among health care workers: risk factors for transmission. *Am J Public Health* 1993;83:1679-84.
200. Watson JC, Fleming DC, Borella AJ, Olcott ES, Conrad RE, Baron RC. Vertical transmission of hepatitis A resulting in an outbreak in a neonatal intensive care unit. *J Infect Dis* 1993;167:567-71.
201. Noble RC, Kane MA, Reeves SA, Roeckel I. Posttransfusion hepatitis A in a neonatal intensive care unit. *JAMA* 1984;252:2711-5.
202. Lee KK, Vargo LR, Le CT, Fernando L. Transfusion-acquired hepatitis A outbreak from fresh frozen plasma in a neonatal intensive care unit. *Pediatr Infect Dis J* 1992;11:122-3.
203. Coulepis AG, Locarnini SA, Lehman NI, Gust ID. Detection of hepatitis A virus in the feces of patients with naturally acquired infections. *J Infect Dis* 1980;141:151-6.
204. Centers for Disease Control and Prevention. Prevention of hepatitis A through active or passive immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1996;45(RR-15):1-30.
205. Meyers JD, Romm FJ, Tihen WS, Bryan JA. Food-borne hepatitis A in a general hospital: epidemiologic study of an outbreak attributed to sandwiches. *JAMA* 1975;231:1049-53.
206. Eisenstein AB, Aach RD, Jacobssohn W, Goldman A. An epidemic of infectious hepatitis in a general hospital: probable transmission by contaminated orange juice. *JAMA* 1963;185:171-4.
207. Papaevangelou GJ, Roumeliotou-Karayannis AJ, Contoyannis PC. The risk of nosocomial hepatitis A and B virus infections from patients under care without isolation precaution. *J Med Virol* 1981;7:143-8.
208. Kashiwagi S, Hayashi J, Ikematsu H, Nomura H, Kajiyama W, Ikematsu W, et al. Prevalence of immunologic markers of hepatitis A and B infection in hospital personnel in Miyazaki Prefecture, Japan. *Am J Epidemiol* 1985;122:960-9.
209. Van Dyke RB, Spector SA. Transmission of herpes simplex virus type 1 to a newborn infant during endotracheal suctioning for meconium aspiration. *Pediatr Infect Dis J* 1984;3:153-6.
210. Linneman CC, Buchman TG, Light IJ, Ballard JL. Transmission of herpes-simplex virus type 1 in a nursery for the newborn: identification of isolates by D.N.A. "fingerprinting." *Lancet* 1978;1:964-6.
211. Kleiman MB, Schreiner RL, Eitzen H, Lemons JA, Jansen RD. Oral herpesvirus infection in nursery personnel: infection control policy. *Pediatrics* 1982;70:609-12.
212. Buchman TG, Roizman B, Adams G, Stover BH. Restriction endonuclease fingerprinting of herpes simplex virus DNA: a novel epidemiological tool applied to a nosocomial outbreak. *J Infect Dis* 1978;138:488-98.
213. Greaves WL, Kaiser AB, Alford RH, Schaffner W. The problem of herpetic whitlow among hospital personnel. *Infect Control* 1980;1:381-5.
214. Adams G, Stover BH, Keenlyside RA, Hooton TM, Buchman TG, Roizman B, et al. Nosocomial herpetic infections in a pediatric intensive care unit. *Am J Epidemiol* 1981;113:126-32.
215. American Academy of Pediatrics. Summaries of infectious diseases: herpes simplex. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 266-76.
216. Pereira FA. Herpes simplex: evolving concepts. *J Am Acad Dermatol* 1996;35:503-20.
217. Perl TM, Haugen TH, Pfaller MA, Hollis R, Lakeman AD, Whitley RJ, et al. Transmission of herpes simplex virus type 1 infection in an intensive care unit. *Ann Intern Med* 1992;117:584-6.
218. Turner R, Shehab Z, Osborne K, Hendley JO. Shedding and survival of herpes simplex virus from "fever blisters." *Pediatrics* 1982;70:547-9.
219. Spruance SL, Overall JC Jr, Kern ER, Krueger GG, Pliam V, Miller W. The natural history of recurrent herpes simplex labialis: implications for antiviral therapy. *N Engl J Med* 1977;297:69-75.
220. Davis RM, Orenstein WA, Frank JA Jr, Sacks JJ, Dales LG, Preblud SR, et al. Transmission of measles in medical settings, 1980 through 1984. *JAMA* 1986;255:1295-8.
221. Atkinson WL, Markowitz LE, Adams NC, Seastrom GR. Transmission of measles in medical settings—United States, 1985-1989. *Am J Med* 1991;91(suppl 3B):320S-4S.

222. Raad II, Sheretz RJ, Rains CS, Cusick JL, Fauerbach LL, Reuman PD, et al. The importance of nosocomial transmission of measles in the propagation of a community outbreak. *Infect Control Hosp Epidemiol* 1989;10:161-6.
223. Istre GR, McKee PA, West GR, O'Mara DJ, Rettig PJ, Stuenkel J, et al. Measles spread in hospital settings: an important focus of disease transmission? *Pediatrics* 1987;79:356-8.
224. Dales LG, Kizer KW. Measles transmission in medical facilities. *West J Med* 1985;142:415-6.
225. Sienko DG, Friedman C, McGee HB, Allen MJ, Simeson WF, Wentworth BB, et al. A measles outbreak at university medical setting involving medical health care providers. *Am J Public Health* 1987;77:1222-4.
226. Rivera ME, Mason WH, Ross LA, Wright HT Jr. Nosocomial measles infection in a pediatric hospital during a community-wide epidemic. *J Pediatr* 1991;119:183-6.
227. Rank EL, Brettman L, Katz-Pollack H, DeHertogh D, Neville D. Chronology of a hospital-wide measles outbreak: lessons learned and shared from an extraordinary week in late March 1989. *AJIC Am J Infect Control* 1992;209:315-8.
228. Watkins NM, Smith RP Jr, St. Germain DL, MacKay DN. Measles (rubeola) infection in a hospital setting. *AJIC Am J Infect Control* 1987;15:201-6.
229. Remington PL, Hall WN, Davis IH, Herald A, Gunn RA. Airborne transmission of measles in a physician's office. *JAMA* 1985;253:1574-7.
230. Atkinson WL. Measles and health care workers. *Infect Control Hosp Epidemiol* 1994;15:5-7.
231. Bloch AB, Orenstein WA, Ewing WM. Measles outbreak in a pediatric practice: airborne transmission in an office setting. *Pediatrics* 1985;75:676-83.
232. American Academy of Pediatrics. Summaries of infectious diseases: measles. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 334-57.
233. Braunstein H, Thomas S, Ito R. Immunity to measles in a large population of varying age. *Am J Dis Child* 1990;144:296-8.
234. Smith E, Welch W, Berhow M, Wong VK. Measles susceptibility of hospital employees as determined by ELISA [abstract]. *Clin Res* 1990;38:183A.
235. Subbarao EK, Amin S, Kumar ML. Prevaccination serologic screening for measles in health care workers. *J Infect Dis* 1991;163:876-78.
236. Sellick J Jr, Longbine D, Schiffling R, Mylotte JM. Screening hospital employees for measles immunity is more cost effective than blind immunization. *Ann Intern Med* 1992;116:982-4.
237. Grabowsky M, Markowitz LE. Serologic screening, mass immunization, and implications for immunization programs. *J Infect Dis* 1991;164:1237-8.
238. Stover BH, Adams G, Kuebler CA, Cost KM, Rabalais GP. Measles-mumps-rubella immunization of susceptible hospital employees during a community measles outbreak: cost-effectiveness and protective efficacy. *Infect Control Hosp Epidemiol* 1994;15:18-21.
239. Jackson LA, Schuchat A, Reeves NW, Wenger JD. Serogroup C meningococcal outbreaks in the United States: an emerging threat. *JAMA* 1995;273:383-9.
240. Houck P, Patnode M, Atwood R, Powell K. Epidemiologic characteristics of an outbreak of serogroup C meningococcal disease and the public health response. *Public Health Rep* 1995;110:343-9.
241. Centers for Disease Control. Laboratory-acquired meningococemia—California and Massachusetts. *MMWR Morb Mortal Wkly Rep* 1991;40:46-7, 55.
242. Centers for Disease Control. Nosocomial meningococemia—Wisconsin. *MMWR Morb Mortal Wkly Rep* 1978;27:358-63.
243. Rose HD, Lenz IE, Sheth NK. Meningococcal pneumonia: a source of nosocomial infection. *Arch Intern Med* 1981;141:575-7.
244. Cohen MS, Steere AC, Baltimore R, von Graevenitz A, Pantelick E, Camp B, et al. Possible nosocomial transmission of group Y *Neisseria meningitidis* among oncology patients. *Ann Intern Med* 1979;91:7-12.
245. Broome CV. The carrier state: *Neisseria meningitidis*. *J Antimicrob Chemother* 1986;18(suppl. A):25-34.
246. Gaunt PN, Lambert BE. Single dose ciprofloxacin for the eradication of pharyngeal carriage of *Neisseria meningitidis*. *J Antimicrob Chemother* 1988;21:489-96.
247. Munford RS, Taunay A, de Morais JS, Fraser DW, Feldman RA. Spread of meningococcal infection within households. *Lancet* 1974;1:1275-8.
248. American Academy of Pediatrics. Meningococcal disease prevention and control strategies for practice-based physicians. *Pediatrics* 1996;97:404-11.
249. Riedo FX, Plikaytis BD, Broome CV. Epidemiology and prevention of meningococcal disease. *Pediatr Infect Dis J* 1995;14:643-57.
250. Griffis JM. Epidemic meningococcal disease: synthesis of a hypothetical immunoepidemiologic model. *Rev Infect Dis* 1982;4:159-72.
251. Caugant DA, Hoiby EA, Magnus P, Scheel O, Hoel T, Bjune G, et al. Asymptomatic carriage of *Neisseria meningitidis* in a randomly sampled population. *J Clin Microbiol* 1994;32:323-30.
252. Caugant DA, Hoiby EA, Rosenqvist E, Froholm LO, Selander RK. Transmission of *Neisseria meningitidis* among asymptomatic military recruits and antibody analysis. *Epidemiol Infect* 1992;109:241-53.
253. Wharton M, Cochi SL, Hutcheson RH, Schaffner W. Mumps transmission in hospitals. *Arch Intern Med* 1990;150:47-9.
254. Fischer PR, Brunetti C, Welch V, Christenson JC. Nosocomial mumps: report of an outbreak and its control. *AJIC Am J Infect Control* 1996;24:13-8.
255. American Academy of Pediatrics. Summaries of infectious diseases: mumps. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997:366-9.
256. Williams WW, Preblud SR, Reichelderfer PS, Hadler SC. Vaccines of importance in the hospital setting: problems and developments. *Infect Dis Clin North Am* 1989;3:701-22.
257. Koplan JP, Preblud SR. A benefit-cost analysis of mumps vaccine. *Am J Dis Child* 1982;136:362-4.
258. Hersh BS, Fine PEM, Kent WK, Cochi SL, Kahn LH, Zell ER, et al. Mumps outbreak in a highly vaccinated population. *J Pediatr* 1991;119:187-93.
259. Anderson LJ, Török TJ. The clinical spectrum of human parvovirus B19 infections. *Curr Clin Top Infect Dis* 1991;11:267-80.
260. Török TJ. Parvovirus B19 and human disease. *Adv Intern Med* 1992;37:431-55.
261. Shishiba T, Matsunaga Y. An outbreak of erythema infectiosum among hospital staff members including a patient with pleural fluid and pericardial effusion. *J Am Acad Dermatol* 1993;29:265-7.

262. Seng C, Watkins P, Morse D, Barrett SP, Zambon M, Andrews N, et al. Parvovirus B19 outbreak on an adult ward. *Epidemiol Infect* 1994;113:345-53.
263. Bell LM, Naides J, Stoffman P, Hodinka RL, Plotkin SA. Human parvovirus B19 infection among hospital staff members after contact with infected patients. *N Engl J Med* 1989;321:485-91.
264. Harrison J, Jones DE. Human parvovirus B19 in health care workers. *Occup Med* 1995;45:93-6.
265. Pillay D, Patou G, Hurt S, Kibbler CC, Griffiths PD. Parvovirus B19 outbreak in a children's ward. *Lancet* 1992;339:107-9.
266. Dowell SF, Török TJ, Thorp JA, Hedrick J, Erdman DD, Zaki SR, et al. Parvovirus B19 infection in hospital workers: community or hospital acquisition. *J Infect Dis* 1995;172:1076-9.
267. Ray SM, Erdman DD, Berschling JD, Cooper JE, Török TJ, Blumberg HM, et al. Nosocomial exposure to parvovirus B19: low risk of transmission to healthcare workers. *Infect Control Hosp Epidemiol* 1997;18:109-14.
268. Koziol DE, Kurtzman G, Ayub J, Young NS, Henderson DK. Nosocomial human parvovirus B19 infection: lack of transmission from a chronically infected patient to hospital staff. *Infect Control Hosp Epidemiol* 1992;13:343-8.
269. Evans JP, Rossiter MA, Kumaran TO, Marsh GW, Mortimer PP. Human parvovirus aplasia: case due to cross infection in a ward. *BMJ* 1984;288:681.
270. Cohen BJ, Courouce AM, Schwartz TF, Okochi K, Kurtzman GJ. Laboratory infection with parvovirus B19 [letter]. *J Clin Pathol* 1988;41:1027-8.
271. Anderson LJ, Gillespie SM, Török TJ, Hurwitz ES, Tsou J, Gary GW. Risk of infection following exposures to human parvovirus B19. *Behring Inst Mitt* 1990;85:60-3.
272. Anderson MJ, Lewis E, Kidd IM, Hall SM, Cohen BJ. An outbreak of erythema infectiosum associated with human parvovirus infection. *J Hyg* 1984;93:85-93.
273. Chorba T, Coccia P, Holman RC, Tattersall P, Anderson LJ, Sudman J, et al. The role of parvovirus B19 in aplastic crisis and erythema infectiosum. *J Infect Dis* 1986;154:383-93.
274. Török TJ. Human parvovirus B19. In: Remington JS, Klein JO, editors. *Infectious diseases of the fetus and newborn infant*. 4th ed. Philadelphia: WB Saunders; 1995. p. 668-702.
275. Török TJ. Human parvovirus B19 infections in pregnancy. *Pediatr Infect Dis J* 1990;9:772-6.
276. Kurt TL, Yeager AS, Guennette S, Dunlop S. Spread of pertussis by hospital staff. *JAMA* 1972;221:264-7.
277. Linneman CC Jr, Ramundo N, Perlstein PH, Minton SD, Englander GS. Use of pertussis vaccine in an epidemic involving hospital staff. *Lancet* 1975;2:540-3.
278. Valenti WM, Pincus PH, Messner MK. Nosocomial pertussis: possible spread by a hospital visitor. *Am J Dis Child* 1980;134:520-1.
279. Christie C, Glover AM, Willke MJ, Marx ML, Reising SF, Hutchinson NM. Containment of pertussis in the regional pediatric hospital during the greater Cincinnati epidemic of 1993. *Infect Control Hosp Epidemiol* 1995;16:556-63.
280. Shefer A, Dales L, Nelson M, Werner B, Baron R, Jackson R. Use and safety of acellular pertussis vaccine among adult hospital staff during an outbreak of pertussis. *J Infect Dis* 1995;171:1053-6.
281. Deville JG, Cherry JD, Christenson PD, Pineda E, Leach CT, Kuhls TL, et al. Frequency of unrecognized *Bordetella pertussis* in adults. *Clin Infect Dis* 1995;21:639-42.
282. Nennig ME, Shinefield HR, Edwards KM, Black SB, Fireman BH. Prevalence and incidence of adult pertussis in an urban population. *JAMA* 1996;275:1672-4.
283. Mortimer EA Jr. Pertussis vaccine. In: Plotkin SA, Mortimer EA Jr, editors. *Vaccines*. Philadelphia: WB Saunders; 1994. p. 91-137.
284. Mortimer EA Jr. Pertussis and its prevention: a family affair. *J Infect Dis* 1990;161:473-9.
285. Deen JL, Mink CA, Cherry JD, Christenson PD, Pineda EF, Lewis K, et al. Household contact study of *Bordetella pertussis* infections. *Clin Infect Dis* 1995;21:1211-9.
286. Edwards KM, Decker MD, Graham BS, Mozzatesta J, Scott J, Hackell J. Adult immunization with acellular pertussis vaccine. *JAMA* 1993;269:53-6.
287. Weber DJ, Rutala WA. Management of healthcare workers exposed to pertussis. *Infect Control Hosp Epidemiol* 1994;15:411-5.
288. Halsey NA, Welling MA, Lehman RM. Nosocomial pertussis: a failure of erythromycin treatment and prophylaxis. *Am J Dis Child* 1980;134:521-2.
289. Centers for Disease Control and Prevention. Paralytic poliomyelitis—United States, 1980-1994. *MMWR Morb Mortal Wkly Rep* 1997;46:79-83.
290. American Academy of Pediatrics. Summaries of infectious diseases: poliovirus infections. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 424-33.
291. Fishbein DB, Robinson LE. Current concepts: rabies. *N Engl J Med* 1997;329:1632-8.
292. Winkler WG, Fashinell TR, Leffingwell L, Howard P, Conomy JP. Airborne rabies transmission in a laboratory worker. *JAMA* 1973;226:1219-21.
293. Centers for Disease Control. Rabies in a laboratory worker—New York. *MMWR Morb Mortal Wkly Rep* 1977;26:183-4.
294. Helmick CG, Tauxe RV, Vernon AA. Is there a risk to contacts of patients with rabies? *Rev Infect Dis* 1987;9:511-8.
295. Centers for Disease Control and Prevention. Human rabies—New Hampshire, 1996. *MMWR Morb Mortal Wkly Rep* 1997;46:267-70.
296. Centers for Disease Control and Prevention. Human rabies—Connecticut, 1995. *MMWR Morb Mortal Wkly Rep* 1996;45:207-9.
297. Centers for Disease Control and Prevention. Human rabies—Washington, 1995. *MMWR Morb Mortal Wkly Rep* 1995;44:625-7.
298. Greaves WL, Orenstein WA, Stetler HC, Preblud SR, Hinman AR, Bart KJ. Prevention of rubella transmission in medical facilities. *JAMA* 1982;248:861-4.
299. Centers for Disease Control. Rubella in hospitals—California. *MMWR Morb Mortal Wkly Rep* 1983;32:37-9.
300. Poland GA, Nichol KL. Medical students as sources of rubella and measles outbreaks. *Arch Intern Med* 1990;150:44-6.
301. Storch GA, Gruber C, Benz B, Beaudoin J, Hayes J. A rubella outbreak among dental students: description of the outbreak and analysis of control measures. *Infect Control* 1985;6:150-6.
302. Strassburg MA, Stephenson TG, Habel LA, Fannin SL. Rubella in hospital employees. *Infect Control* 1984;5:123-6.
303. Fliegel PE, Weinstein WM. Rubella outbreak in a prenatal clinic: management and prevention. *Am J Infect Control* 1982;10:29-33.

304. Strassburg MA, Imagawa DT, Fannin SL, Turner JA, Chow AW, Murray RA, et al. Rubella outbreak among hospital employees. *Obstet Gynecol* 1981;57:283-8.
305. Gladstone JL, Millian SJ. Rubella exposure in an obstetric clinic. *Obstet Gynecol* 1981;57:182-6.
306. American Academy of Pediatrics. Summaries of infectious diseases: rubella. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 456-62.
307. Polk FB, White JA, DeGirolami PC, Modlin JF. An outbreak of rubella among hospital personnel. *N Engl J Med* 1980;303:541-5.
308. Sachs JJ, Olson B, Soter J, Clark C. Employee rubella screening programs in Arizona hospitals. *JAMA* 1983;249:2675-8.
309. Preblud SR. Some current issues relating to the rubella vaccine. *JAMA* 1985;254:253-6.
310. Lettau LA. Nosocomial transmission and infection control aspects of parasitic and ectoparasitic diseases part III. Ectoparasites/summary and conclusions. *Infect Control Hosp Epidemiol* 1991;12:179-85.
311. Juranek DD, Currier RW, Millikan LE. Scabies control in institutions. In: Orkin M, Maiback HI, editors. Cutaneous infestations and insect bites. New York, NY: Dekker; 1985. p. 139-56.
312. Jucowics P, Ramon ME, Don PC, Stone RK, Bamji M. Norwegian scabies in an infant with acquired immunodeficiency syndrome. *Arch Dermatol* 1989;125:1670-1.
313. Hench C, Paulson SS, Stevens DA, Thompson JD. Scabies outbreak on a spinal cord injury unit. *Rehabil Nurs* 1994;19:21-3.
314. Jimenez-Lucho VE, Fallon F, Caputo C, Ramsey K. Role of prolonged surveillance in the eradication of nosocomial scabies in an extended care Veterans Affairs medical center. *AJIC Am J Infect Control* 1995;23:44-9.
315. Degelau J. Scabies in long-term care facilities. *Infect Control Hosp Epidemiol* 1992;13:421-5.
316. Lerche NW, Currier RW, Juranek DD, Baer W, Dubay NJ. Atypical crusted "Norwegian" scabies: report of nosocomial transmission in a community hospital and an approach to control. *Cutis* 1983;31:668-84.
317. Lempert KD, Baltz PS, Welton WA, Whittier FC. Pseudouremic pruritus: a scabies epidemic in a dialysis unit. *Am J Kidney Dis* 1985;5:117-9.
318. Thomas MC, Giedinghagen DH, Hoff GL. Brief report: an outbreak of scabies among employees in a hospital-associated commercial laundry. *Infect Control* 1987;8:427-9.
319. Centers for Disease Control. Scabies in health-care facilities—Iowa. *MMWR Morb Mortal Wkly Rep* 1988;37:178-9.
320. Orkin M. Scabies in AIDS. *Semin Dermatol* 1993;12:9-14.
321. Lam S, Brennessel D. Norwegian scabies and HIV infection—case report and literature review. *Infect Dis Clin Practice* 1993;3:169-73.
322. Gooch JJ, Strasius SR, Beamer B, Reiter MD, Correll GW. Nosocomial outbreak of scabies. *Arch Dermatol* 1978;114:897-8.
323. Corbett EL, Crossley I, Holton J, Levell N, Miller RF, DeCock KM. Crusted ("Norwegian") scabies in a specialist HIV unit: successful use of ivermectin and failure to prevent nosocomial transmission. *Genitourin Med* 1996;72:115-7.
324. Taplin D, Rivera A, Walker JG, Roth WI, Reno D, Meinking T. A comparative trial of three treatment schedules for the eradication of scabies. *J Am Acad Dermatol* 1983;9:550-4.
325. Arlian LG, Estes SA, Vyszynski-Moher DL. Prevalence of *Sarcoptes scabiei* in the homes and nursing homes of scabietic patients. *J Am Acad Dermatol* 1988;19:806-11.
326. Estes SA, Estes J. Therapy of scabies: nursing homes, hospitals, and the homeless. *Semin Dermatol* 1993;12:26-33.
327. Sargent SJ. Ectoparasites. In: Mayhall CG, editor. Hospital epidemiology and infection control. Baltimore: Williams & Wilkins; 1996. p. 465-72.
328. Centers for Disease Control and Prevention. 1998 Guidelines for treatment of sexually transmitted diseases *MMWR Morb Mortal Wkly Rep* 1998; 47(RR-1):105-8.
329. Brown S, Becher J, Brady W. Treatment of ectoparasitic infections: review of the English-language literature, 1982-1992. *Clin Infect Dis* 1995;20(suppl 1):S104-9.
330. Anonymous. Drugs for parasitic infections. *Med Lett Drugs Ther* 1995;37:102, 105.
331. American Academy of Pediatrics. Summaries of infectious diseases: scabies. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 27th ed. Elk Grove (IL): American Academy of Pediatrics; 1997. p. 468-70.
332. Mienking TL, Taplan D, Hermida JL, Pardo R, Kerdel FA. The treatment of scabies with ivermectin. *N Engl J Med* 1995;333:26-30.
333. Hopper AH, Salisbury J, Jegadeva AN, Scott B, Bennett GCS. Epidemic Norwegian scabies in a geriatric unit. *Age Ageing* 1990;19:125-7.
334. Taplin D, Arrue C, Walker JG, Roth WI, Rivera A. Eradication of scabies with a single treatment schedule. *J Am Acad Dermatol* 1983;9:546-50.
335. Juranek DD. *Pediculosis capitis* in school children: epidemiologic trends, risk factors, and recommendations for control. In: Orkin M, Maiback HI, editors. Cutaneous infestations and insect bites. New York, NY: Dekker; 1985. p. 199-211.
336. Wenzel RP. Healthcare workers and the incidence of nosocomial infection: can treatment of one influence the other? A brief review. *J Chemother* 1994;6(suppl 4):33-7, 39-40.
337. Panlilio AL, Culver DH, Gaynes RP, Banerjee S, Henderson TS, Tolson JS, et al. Methicillin-resistant *Staphylococcus aureus* in U.S. hospitals, 1975-1991. *Infect Control Hosp Epidemiol* 1992;13:582-6.
338. Boyce JM. Methicillin-resistant *Staphylococcus aureus*: Detection, epidemiology and control measures. *Infect Dis Clin North Am* 1989;3:901-13.
339. Boyce JM. Methicillin-resistant *Staphylococcus aureus* in hospitals and long-term care facilities: microbiology, epidemiology, and preventive measures. *Infect Control Hosp Epidemiol* 1992;13:725-37.
340. Boyce JM. Should we vigorously try to contain and control methicillin-resistant *Staphylococcus aureus*? *Infect Control Hosp Epidemiol* 1991;12:46-54.
341. Boyce JM, Opal SM, Byone-Potter G, Medeiros AA. Spread of methicillin-resistant *Staphylococcus aureus* in a hospital after exposure to a health care worker with chronic sinusitis. *Clin Infect Dis* 1993;17:496-504.
342. Sherertz RJ, Reagan DR, Hampton KD, Robertson KL, Streed SA, Hoen HM, et al. A cloud adult: the *Staphylococcus aureus*-virus interaction revisited. *Ann Intern Med* 1996;124:539-47.
343. Belani A, Sherertz RJ, Sullivan ML, Russel BA, Reumen PD. Outbreak of staphylococcal infection in two hospital nurseries traced to a single nasal carrier. *Infect Control* 1986;7:487-90.

344. Kreiswirth BN, Kravitz GR, Schlievert PM, Novick RP. Nosocomial transmission of a strain of *Staphylococcus aureus* causing toxic shock syndrome. *Ann Intern Med* 1986;105:704-7.
345. Villarino ME, Vugia DJ, Bean NH, Jarvis WR, Hughes JM. Foodborne disease prevention in health care facilities. In: Bennett JV, Brachman PS, editors. *Hospital infections*. 3rd ed. Boston: Little, Brown and Company; 1992. p. 345-58.
346. Layton MC, Perez M, Heald P, Patterson JE. An outbreak of mupirocin-resistant *Staphylococcus aureus* on a dermatology ward associated with an environmental reservoir. *Infect Control Hosp Epidemiol* 1993;14:369-75.
347. American Academy of Pediatrics. Summaries of infectious diseases: staphylococcal infections. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 476-82.
348. Boyce JM, Landry M, Deetz TR, DuPont HL. Epidemiologic studies of an outbreak of nosocomial methicillin-resistant *Staphylococcus aureus* infections. *Infect Control* 1981;2:110-6.
349. Walsh TJ, Standiford HD, Reboli AC, John JF, Mulligan ME, Ribner BS, et al. Randomized double-blinded trial of rifampin with either novobiocin or trimethoprim sulfamethoxazole against methicillin-resistant *Staphylococcus aureus* colonization: prevention of antimicrobial resistance and effect of host factors on outcome. *Antimicrob Agents Chemother* 1993;37:1334-42.
350. Mulligan ME, Murray-Leisure KA, Ribner BS, Standiford HC, John JF, Korvick JA, et al. Methicillin-resistant *Staphylococcus aureus*: a consensus review of the microbiology, pathogenesis, and epidemiology with implications for prevention and management. *Am J Med* 1993;94:313-28.
351. Reboli AC, John JF, Platt CG, Cantley JR. Methicillin-resistant *Staphylococcus aureus* outbreak at a Veterans' Affairs medical center: importance of carriage of the organism by hospital personnel. *Infect Control Hosp Epidemiol* 1990;11:291-6.
352. Reagan DR, Doebbeling BN, Pfaller MA, Sheetz CT, Houston AK, Hollis RJ, et al. Elimination of coincident *Staphylococcus aureus* nasal and hand carriage with intranasal application of mupirocin calcium ointment. *Ann Intern Med* 1991;114:101-6.
353. Chambers HF. Treatment of infection and colonization caused by methicillin-resistant *Staphylococcus aureus*. *Infect Control Hosp Epidemiol* 1991;12:29-35.
354. Kauffman CA, Terpenning MS, He X, Zaring LT, Ramsey MA, Jorgensen KA, et al. Attempts to eradicate methicillin-resistant *Staphylococcus aureus* from a long-term-care facility with the use of mupirocin ointment. *Am J Med* 1993;94:371-8.
355. Wenzel RP, Nettleman MD, Jones RN, Pealler MA. Methicillin-resistant *Staphylococcus aureus*: implications for the 1990s and effective control measures. *Am J Med* 1991;91(suppl 3B):221S-7S.
356. Doebbeling BN, Breneman DL, Neu HC, Aly R, Yangco BG, Holley HP Jr, et al. Elimination of *Staphylococcus aureus* nasal carriage in health care workers: analysis of six clinical trials with calcium mupirocin ointment. *Clin Infect Dis* 1993;17:466-74.
357. Doebbeling BN, Reagan DR, Pfaller MA, Houston AK, Hollis RJ, Wenzel RP. Long-term efficacy of intranasal mupirocin ointment. A prospective cohort study of *Staphylococcus aureus* carriage. *Arch Intern Med* 1994;154:1505-8.
358. Smith SM, Eng RH, Tecson-Tumang F. Ciprofloxacin therapy for methicillin-resistant *Staphylococcus aureus* infections or colonization. *Antimicrob Agents Chemother* 1989;33:181-4.
359. Darouiche R, Wright C, Hamill R, Koza M, Lewis D, Markowski J. Eradication of colonization by methicillin-resistant *Staphylococcus aureus* by using oral minocycline-rifampin and topical mupirocin. *Antimicrob Agents Chemother* 1991;35:1612-5.
360. Arathoon EG, Hamilton JR, Hench CE, Stevens DA. Efficacy of short courses of oral novobiocin-rifampin in eradicating carrier state of methicillin-resistant *Staphylococcus aureus* and in vitro killing studies of clinical isolates. *Antimicrob Agents Chemother* 1990;34:1655-9.
361. Bartzokas CA, Paton JH, Gibson MF, Graham F, McLoughlin GA, Croton RS. Control and eradication of methicillin-resistant *Staphylococcus aureus* on a surgical unit. *N Engl J Med* 1984;311:1422-5.
362. Ward TT, Winn RE, Hartstein AL, Sewell DL. Observations relating to an inter-hospital outbreak of methicillin-resistant *Staphylococcus aureus*: role of antimicrobial therapy in infection control. *Infect Control* 1981;2:453-9.
363. Boyce JM, Jackson MM, Pugliese G, Batt MD, Fleming D, Garner JS, et al. Methicillin-resistant *Staphylococcus aureus* (MRSA): a briefing for acute care hospitals and nursing facilities. *Infect Control Hosp Epidemiol* 1994;15:105-15.
364. Strasbaugh LJ, Jacobson C, Sewell DL, Potter S, Ward TT. Antimicrobial therapy for methicillin-resistant *Staphylococcus aureus* colonization in residents and staff of a Veterans Affairs nursing home care unit. *Infect Control Hosp Epidemiol* 1992;13:151-9.
365. Miller MA, Dascal A, Portnoy J, Mendelson J. Development of mupirocin resistance among methicillin-resistant *Staphylococcus aureus* after widespread use of nasal mupirocin ointment. *Infect Control Hosp Epidemiol* 1996;17:811-3.
366. Santos KRN, Fonseca LS, Filho PPG. Emergence of high-level mupirocin resistance in methicillin-resistant *Staphylococcus aureus* isolated from Brazilian university hospitals. *Infect Control Hosp Epidemiol* 1996;17:813-6.
367. Valenzuela TD, Hooton TM, Kaplan EL, Schlievert PM. Transmission of toxic strep syndrome from an infected child to a firefighter during CPR. *Ann Emerg Med* 1991;20:90-2.
368. Rammelkamp CH, Mortimer EA, Wolinsky E. Transmission of streptococcal and staphylococcal infection. *Ann Intern Med* 1964;60:753-8.
369. Weber DJ, Rutala WA, Denny FW Jr. Management of health-care workers with pharyngitis or suspected streptococcal infections. *Infect Control Hosp Epidemiol* 1996;17:753-61.
370. Mastro TD, Farley TA, Elliott JA, Facklam RR, Perks JR, Hadler JL, et al. An outbreak of surgical-wound infections due to group A *Streptococcus* carried on the scalp. *N Engl J Med* 1990;323:968-72.
371. Viglionese A, Nottebart VF, Bodman HA, Platt R. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. *Am J Med* 1991;91(suppl 3B):329S-33S.
372. Paul SM, Genese C, Spitalny K. Postoperative group A β -hemolytic *Streptococcus* outbreak with the pathogen traced to a member of a healthcare worker's household. *Infect Control Hosp Epidemiol* 1990;11:643-6.
373. Ridgway EJ, Allen KD. Clustering of group A streptococcal infections on a burns unit: important lessons in outbreak management. *J Hosp Infect* 1993;25:173-82.
374. Berkelman RL, Martin D, Graham DR, Mowry J, Freisem R, Weber JA, et al. Streptococcal wound infections caused by a vaginal carrier. *JAMA* 1982;247:2680-2.

375. Schaffner W, Lefkowitz LB Jr, Goodman JS, Koenig MG. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. *N Engl J Med* 1969;280:1224-5.
376. Richman DD, Breton SJ, Goldmann DA. Scarlet fever and group A streptococcal surgical wound infection traced to an anal carrier. *J Pediatr* 1977;90:387-90.
377. Decker MD, Lavelly GB, Hutcheson RHJ, Schaffner W. Food-borne streptococcal pharyngitis in a hospital pediatrics clinic. *JAMA* 1986;253:679-81.
378. Stromberg A, Schwan A, Cars O. Throat carrier rates of beta-hemolytic streptococci among healthy adults and children. *Scand J Infect Dis* 1988;20:411-7.
379. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A *Streptococcus* traced to a vaginal carrier. *J Infect Dis* 1978;138:287-92.
380. American Academy of Pediatrics. Summaries of infectious diseases: group A streptococcal infections. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 483-94.
381. Barnes PF, Bloch AB, Davidson PT, Snider DE. Tuberculosis in patients with human immunodeficiency syndrome. *N Engl J Med* 1991;324:1644-50.
382. Centers for Disease Control and Prevention. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities, 1994. *MMWR Morb Mortal Wkly Rep* 1994;43(RR-13):1-132.
383. Edlin BR, Tokars JI, Grieco MH, Crawford JT, Williams J, Sordillo EM, et al. An outbreak of multidrug-resistant tuberculosis among hospitalized patients with the acquired immunodeficiency syndrome. *N Engl J Med* 1992;326:1514-21.
384. Stroud LA, Tokars JI, Grieco MH, Crawford JT, Culver DH, Edlin BR, et al. Evaluation of infection control measures in preventing the nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis* in a New York City hospital. *Infect Control Hosp Epidemiol* 1995;16:141-7.
385. Beck-Sagué CM, Dooley SW, Hutton MD, Otten J, Breeden A, Crawford JT, et al. Hospital outbreak of multidrug-resistant *Mycobacterium tuberculosis* infections: factors in transmission to staff and HIV-infected patients. *JAMA* 1992;268:1280-6.
386. Wenger PN, Otten J, Breeden A, Orfas E, Beck-Sagué CM, Jarvis WR. Control of nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis* among healthcare workers and HIV-infected patients. *Lancet* 1995;345:235-40.
387. Dooley SW, Villarino ME, Lawrence M, Salinas L, Amil S, Rullan JV, et al. Nosocomial transmission of tuberculosis in a hospital unit for HIV-infected patients. *JAMA* 1992;267:2632-5.
388. Pearson ML, Jereb JA, Frieden TR, Crawford JT, Davis BJ, Dooley SW, et al. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*: a risk to patients and health care workers. *Ann Intern Med* 1992;117:191-6.
389. Cleveland JL, Kent J, Gooch BF, Valway SE, Marianos DW, Butler WR, et al. Multidrug-resistant *Mycobacterium tuberculosis* in an HIV dental clinic. *Infect Control Hosp Epidemiol* 1995;16:7-11.
390. Ridzon R, Kenyon T, Luskin-Hawk R, Schultz C, Valway S, Onorato IM. Nosocomial transmission of human immunodeficiency virus and subsequent transmission of multidrug-resistant tuberculosis in a healthcare worker. *Infect Control Hosp Epidemiol* 1997;18:422-3.
391. Jereb JA, Klevens M, Privett TD, Smith PJ, Crawford JT, Sharp VL, et al. Tuberculosis in health care workers at a hospital with an outbreak of multidrug-resistant *Mycobacterium tuberculosis*. *Arch Intern Med* 1995;155:854-9.
392. Sepkowitz KA. Tuberculosis and the health care worker: a historical perspective. *Ann Intern Med* 1994;120:71-9.
393. Menzies D, Fanning A, Yuan L, Fitzgerald M. Tuberculosis among health care workers. *N Engl J Med* 1995;332:92-8.
394. McKenna MT, Hutton MD, Cauthen G, Onorato IM. The association between occupation and tuberculosis: a population based survey. *Am J Respir Crit Care Med* 1996;154:587-9.
395. Zaza S, Blumberg HM, Beck-Sagué C, Haas WH, Woodley CL, Pineda M, et al. Nosocomial transmission of *Mycobacterium tuberculosis*: role of health care workers in outbreak propagation. *J Infect Dis* 1995;172:1542-9.
396. Jarvis WR. Nosocomial transmission of multidrug-resistant *Mycobacterium tuberculosis*. *AJIC Am J Infect Control* 1995;23:146-51.
397. Ikeda RM, Birkhead GS, DiFerdinando GT Jr, Bornstein DL, Dooley SW, Kubica GP, et al. Nosocomial tuberculosis: an outbreak of a strain resistant to seven drugs. *Infect Control Hosp Epidemiol* 1995;16:152-9.
398. Ussery XT, Bierman JA, Valway S, Seitz TA, DiFerdinando GT Jr, Ostroff SM. Transmission of multidrug-resistant *Mycobacterium tuberculosis* among persons exposed in a medical examiner's office, New York. *Infect Control Hosp Epidemiol* 1995;16:160-5.
399. Hutton MD, Stead WW, Cauthen GM, Bloch AB, Ewing WM. Nosocomial transmission of tuberculosis associated with a draining abscess. *J Infect Dis* 1990;161:286-95.
400. Kramer F, Sasse SA, Simms JC, Leedom JM. Primary cutaneous tuberculosis after a needlestick injury from a patient with AIDS and undiagnosed tuberculosis. *Ann Intern Med* 1993;119:594-5.
401. Rattner SL, Fleischer JA, Davidson BL. Tuberculin positivity and patient contact in healthcare workers in the urban United States. *Infect Control Hosp Epidemiol* 1996;17:369-71.
402. Chaisson R, Benson C. Tuberculosis. New York: McGraw-Hill; 1995.
403. Selwyn PA, Hartel D, Lewis VA, Schoenbaum EE, Vermund SH, Klein RS. A prospective study of the risk of tuberculosis among intravenous drug users with human immunodeficiency virus infection. *N Engl J Med* 1989;320:545-50.
404. Pugliese G, Tapper ML. Tuberculosis control in health care. *Infect Control Hosp Epidemiol* 1996;17:819-27.
405. Maloney SA, Pearson ML, Gordon MT, DelCastillo R, Boyle JF, Jarvis WR. Efficacy of control measures in preventing nosocomial transmission of multidrug-resistant tuberculosis to patients and health care workers. *Ann Intern Med* 1995;122:90-5.
406. American Thoracic Society, Centers for Disease Control. Diagnostic standards and classification of tuberculosis. *Am Rev Respir Dis* 1990;142:725-35.
407. Centers for Disease Control and Prevention. Screening for tuberculosis and tuberculosis infection in high-risk populations: recommendations of the Advisory Council for the Elimination of Tuberculosis. *MMWR Morb Mortal Wkly Rep* 1995;44(RR-11):19-34.
408. Centers for Disease Control and Prevention. Anergy skin testing and preventive therapy for HIV-infected persons: revised recommendations. *MMWR Morb Mortal Wkly Rep* 1997;46(RR-15):1-10.

409. Centers for Disease Control and Prevention. Management of persons exposed to multidrug-resistant tuberculosis. *MMWR Morb Mortal Wkly Rep* 1992;41(RR-11):59-71.
410. Rodrigues L, Diwan D, Wheeler J. Protective effect of BCG against tuberculosis meningitis and miliary tuberculosis: a meta-analysis. *Int J Epidemiol* 1993;22:1154-8.
411. Colditz GA, Brewer TF, Berkey CS, Wilson ME, Burdick E, Fineberg HV, et al. Efficacy of BCG vaccine in the prevention of tuberculosis: meta-analysis of the published literature. *JAMA* 1994;271:698-702.
412. Centers for Disease Control and Prevention. The role of BCG vaccine in the prevention and control of tuberculosis in the United States: a joint statement by the Advisory Council for the Elimination of Tuberculosis and the Advisory Committee on Immunization Practices. *MMWR Morb Mortal Wkly Rep* 1996;45(RR-4):1-18.
413. Lotte A, Wasz-Hockert O, Poisson N, Engbaek H, Landmann H, Quast U. Second IUATLD study on complications induced by intradermal BCG vaccination. *Bull Int Union Tuberc Lung Dis* 1988;63:47-59.
414. Caglayan S, Yegin O, Kayran K, Timocin N, Kasirga E, Gun M. Is medical therapy effective for regional lymphadenitis following BCG vaccination? *Am J Dis Child* 1987;141:1213-4.
415. Brewer T, Colditz G. Bacille Calmette-Guérin vaccination for prevention of tuberculosis in health care workers. *Clin Infect Dis* 1995;20:136-42.
416. Centers for Disease Control and Prevention. Disseminated *Mycobacterium bovis* infection from BCG vaccination of a patient with acquired immunodeficiency syndrome. *MMWR Morb Mortal Wkly Rep* 1985;34:227-8.
417. Ninane J, Grymonprez A, Burtonboy G, Francois A, Cornu G. Disseminated BCG in HIV infection. *Arch Dis Child* 1988;63:1268-9.
418. Smith E, Thybo S, Bennedsen J. Infection with *Mycobacterium bovis* in a patient with AIDS: a late complication of BCG vaccination. *Scand J Infect Dis* 1992;24:109-10.
419. von Reyn CF, Clements CJ, Mann JM. Human immunodeficiency virus infection and routine childhood immunisation. *Lancet* 1987;2:669-72.
420. Comstock GW, Edwards LB, Nabangxang H. Tuberculin sensitivity eight to fifteen years after BCG vaccination. *Am Rev Respir Dis* 1971;103:572-5.
421. Guld J, Waaler H, Sundaresan TK, Kaufmann PC, Dam HG. The duration of BCG-induced tuberculin sensitivity in children, and its irrelevance for revaccination: results of two 5-year prospective studies. *Bull World Health Organ* 1968;39:829-36.
422. Orefici G, Scopetti F, Grandolfo ME, Annes I, Kissopoulos A. Study of a BCG vaccine: influence of dose and time. *Boll Ist Sieroter Milan* 1982;61:24-8.
423. Fine PEM, Pönnighaus JM, Maine NP. The relationship between delayed type hypersensitivity and protective immunity induced by mycobacterial vaccines in man. *Lepr Rev* 1986;57(suppl 2):275-83.
424. Fine PEM, Sterne JAC, Pönnighaus JM, Rees RJW. Delayed-type hypersensitivity, mycobacterial vaccines and protective immunity. *Lancet* 1994;344:1245-9.
425. American Thoracic Society, Centers for Disease Control. The tuberculin test. *Am Rev Respir Dis* 1981;124:356-63.
426. Lane JM, Ruben FL, Neff JM, Millar JD. Complications of smallpox vaccination, 1968: results of ten statewide surveys. *J Infect Dis* 1970;122:303-9.
427. Centers for Disease Control. Contact spread of vaccinia from a recently vaccinated Marine—Louisiana. *MMWR Morb Mortal Wkly Rep* 1984;33:37-8.
428. Centers for Disease Control. Contact spread of vaccinia from a National Guard vaccinee—Wisconsin. *MMWR Morb Mortal Wkly Rep* 1985;34:182-3.
429. Centers for Disease Control. Vaccinia outbreak—Newfoundland. *MMWR Morb Mortal Wkly Rep* 1981;30:453-5.
430. Meyers JD, MacQuarrie MB, Merigan TC, Jennison MH. Nosocomial varicella. Part 1: outbreak in oncology patients at a children's hospital. *West J Med* 1979;130:196-9.
431. Morens DM, Bregman DJ, West CM, Green MH, Mazur MH, Dolin R, et al. An outbreak of varicella-zoster virus infection among cancer patients. *Ann Intern Med* 1980;93:414-9.
432. Baltimore RS. Nosocomial infections in the pediatric intensive care unit. *Yale J Biol Med* 1984;57:185-97.
433. Gustafson TL, Shebab A, Brunell PA. Outbreak of varicella in a newborn intensive care nursery. *Am J Dis Child* 1984;138:548-50.
434. Hyams PJ, Stuewe MCS, Heitzer V. Herpes zoster causing varicella (chicken pox) in hospital employees: cost of a casual attitude. *Infect Control* 1984;12:2-5.
435. Weitekamp MR, Schan P, Aber RC. An algorithm for the control of varicella-zoster virus infection. *Am J Infect Control* 1985;13:193-8.
436. Alter SJ, Hammond JA, McVey CJ, Myers MG. Susceptibility to varicella-zoster virus among adults at high risk for exposure. *Infect Control* 1986;7:448-51.
437. Krasinski K, Holzman RS, LaCouture R, Florman A. Hospital experience with varicella-zoster virus. *Infect Control* 1986;7:312-6.
438. Haiduven-Griffiths D, Fecko H. Varicella in hospital personnel: a challenge for the infection control practitioner. *Am J Infect Control* 1987;15:207-11.
439. Weber DJ, Rutala WA, Parham C. Impact and costs of varicella prevention in a university hospital. *Am J Public Health* 1988;78:19-23.
440. McKinney WP, Horowitz MM, Battiola RJ. Susceptibility of hospital-based health care personnel to varicella-zoster virus infections. *Am J Infect Control* 1989;17:26-30.
441. Weber DJ, Rutala WA, Hamilton H. Prevention and control of varicella-zoster infections in healthcare facilities. *Infect Control Hosp Epidemiol* 1996;17:694-705.
442. American Academy of Pediatrics. Summaries of infectious diseases: varicella-zoster infections. In: Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997. p. 573-85.
443. Asano Y, Iwayama S, Miyata T, Yazaki T, Ozaki T, Tsuzuki K, et al. Spread of varicella in hospitalized children having no direct contact with an indicator zoster case and its prevention by a live vaccine. *Biken J* 1980;23:157-61.
444. Sawyer MH, Chamberlin CJ, Wu YN, Aintablian N, Wallace MR. Detection of varicella-zoster virus DNA in air samples from hospital rooms. *J Infect Dis* 1994;169:91-4.
445. LeClair JM, Zaia JA, Levin MJ, Congdon RG, Goldmann DA. Airborne transmission of chickenpox in a hospital. *N Engl J Med* 1980;302:450-3.
446. Gustafson TL, Lavelly GB, Brawner ER Jr, Hutcheson RH, Wright PF, Schaffner W. An outbreak of airborne nosocomial varicella. *Pediatrics* 1982;70:550-6.
447. Josephson A, Gombert M. Airborne transmission of nosocomial varicella from localized zoster. *J Infect Dis* 1988;158:238-41.

448. Ferson MJ, Bell SM, Robertson PW. Determination and importance of varicella immune status of nursing staff in a children's hospital. *J Hosp Infect* 1990;15:347-51.
449. Kelley PW, Petruccioli BP, Stehr-Green P, Erickson RL, Mason CJ. The susceptibility of young adult Americans to vaccine-preventable infections: a national survey of US Army recruits. *JAMA* 1991;266:2724-9.
450. Struewing JP, Hyams KC, Tueller JE, Gray GC. The risk of measles, mumps, and varicella among young adults: a serosurvey of US Navy and Marine Corps recruits. *Am J Public Health* 1993;83:1717-20.
451. Gershon AA, Steinberg SP, LaRussa P, Ferrara A, Hammerschlag M, Gelb L. Immunization of healthy adults with live attenuated varicella vaccine. *J Infect Dis* 1988;158:132-7.
452. Gardner P, Eickhoff T, Poland GA, Gross P, Griffin M, LaForce F, et al. Adult immunizations: recommendations of the American College of Physicians. *Ann Intern Med* 1996;124:35-40.
453. White CJ, Kuter BJ, Ngai A, Hildebrand CS, Isganitis KL, Patterson CM, et al. Modified cases of chicken pox after varicella vaccination: correlation of protection with antibody response. *Pediatr Infect Dis J* 1992;11:19-23.
454. Bernstein HH, Rothstein EP, Watson BM, Reisinger KS, Blatter MM, Wellman CO, et al. Clinical survey of natural varicella compared with breakthrough varicella after immunization with live attenuated Oka/Merck varicella vaccine. *Pediatrics* 1993;92:833-7.
455. Weibel RE, Neff BJ, Kuter BJ, Guess HA, Rothenberger CA, Fitzgerald AJ, et al. Live attenuated varicella vaccine: efficacy trial in healthy children. *N Engl J Med* 1984;310:1409-15.
456. Tsohia M, Gershon AA, Steinberg SP, National Institute of Allergy and Infectious Diseases Varicella Vaccine Collaborative Study Group. Live attenuated varicella vaccine: evidence that the vaccine virus is attenuated and the importance of skin lesions is transmission of varicella-zoster virus. *J Pediatr* 1990;116:184-9.
457. Centers for Disease Control and Prevention. Varicella-related deaths among adults—United States, 1997. *MMWR Morb Mortal Wkly Rep* 1997;46:409-12.
458. Wallace MR, Bowler WA, Murray NB, Brodine SK, Oldfield ECI. Treatment of adult varicella with oral acyclovir: a randomized, placebo-controlled trial. *Ann Intern Med* 1992;117:358-63.
459. Centers for Disease Control and Prevention, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of nosocomial pneumonia. *Infect Control Hosp Epidemiol* 1994;15:587-627.
460. Balkovic ES, Goodman RA, Rose FB, Borel CO. Nosocomial influenza A (H1N1) infection. *Am J Med Tech* 1980;46:318-20.
461. Evans ME, Hall KL, Berry SE. Influenza control in acute care hospitals. *AJIC Am J Infect Control* 1997;25:357-62.
462. Kapila R, Lintz DI, Tecson FT, Ziskin L, Louria DB. A nosocomial outbreak of influenza A. *Chest* 1977;71:576-9.
463. Kimball AM, Foy HM, Cooney MK, Allan ID, Matlock M, Plourde JJ. Isolation of respiratory syncytial and influenza viruses from the sputum of patients hospitalized with pneumonia. *J Infect Dis* 1983;147:181-4.
464. Van Voris LP, Belshe RB, Shaffer JL. Nosocomial influenza B virus infection in the elderly. *Ann Intern Med* 1982;96:153-8.
465. Pachucki CT, Walsh Pappas SA, Fuller GF, Krause SL, Lentino JR, Schaoff DM. Influenza A among hospital personnel and patients: implications for recognition, prevention, and control. *Arch Intern Med* 1989;149:77-80.
466. Centers for Disease Control. Suspected nosocomial influenza cases in an intensive care unit. *MMWR Morb Mortal Wkly Rep* 1988;37:3-4, 9.
467. Hammond GW, Cheang M. Absenteeism among hospital staff during an influenza epidemic: implications for immunoprophylaxis. *Can Med Assoc J* 1984;131:449-52.
468. Horman JT, Stetler HC, Israel E, Sorley O, Schiper MT, Joseph JM. An outbreak of influenza A in a nursing home. *Am J Public Health* 1986;76:501-4.
469. Patriarca PA, Weber JA, Parker RA, Orenstein WA, Hall WN, Kendal AP, et al. Risk factors for outbreaks of influenza in nursing homes: a case-control study. *Am J Epidemiol* 1986;124:114-9.
470. Centers for Disease Control and Prevention. Outbreak of influenza A in a nursing home—New York, December 1991-January 1992. *MMWR Morb Mortal Wkly Rep* 1992;41:129-31.
471. Gross PA, Rodstein M, LaMontagne JR, Kaslow RA, Saah AJ, Wallenstein S, et al. Epidemiology of acute respiratory illness during an influenza outbreak in a nursing home. *Arch Intern Med* 1988;148:559-61.
472. Cartter ML, Renzullo PO, Helgersson SD, Martin SM, Jekel JF. Influenza outbreaks in nursing homes: how effective is influenza vaccine in the institutionalized elderly? *Infect Control Hosp Epidemiol* 1990;11:473-8.
473. Bean B, Moore BM, Sterner B, Peterson LR, Gerding DN, Balfour HH Jr. Survival of influenza viruses on environmental surfaces. *J Infect Dis* 1982;146:47-51.
474. Kilbourne ED, editor. *Influenza*. New York: Plenum Medical Book; 1987.
475. Hall CB, Douglas RG. Nosocomial influenza infection as a cause of intercurrent fevers in infants. *Pediatrics* 1975;55:673-7.
476. Noble GR. Epidemiological and clinical aspects of influenza. In: Beare AS, editor. *Applied influenza research*. Boca Raton (FL): CRC Press; 1982. p. 11-49.
477. Adal KA, Flowers RH, Anglim AM, Hayden FG, Titus MG, Coyner BJ, et al. Prevention of nosocomial influenza. *Infect Control Hosp Epidemiol* 1996;17:641-8.
478. Nichol KL, Margolis KL, Lind A, Murdoch M, McFadden R, Hauge M, et al. Side effects associated with influenza vaccination in healthy working adults: a randomized, placebo-controlled trial. *Arch Intern Med* 1996;156:1546-50.
479. Arden NH, Patriarca PA, Fasano MB, Lui KJ, Harmon MW, Kendal AP, et al. The roles of vaccination and amantadine prophylaxis in controlling an outbreak of influenza A (H3N2) in a nursing home. *Arch Intern Med* 1988;148:865-8.
480. Falsey AR, Cunningham CK, Barker WH, Kouides RW, Yeun JB, Menegus M, et al. Respiratory syncytial virus and influenza A infections in the hospitalized elderly. *J Infect Dis* 1995;172:389-94.
481. Valenti WM, Clarke TA, Hall CB, Menegus MA, Shapiro DL. Concurrent outbreaks of rhinovirus and respiratory syncytial virus in an intensive care nursery: epidemiology and associated risk factors. *J Pediatr* 1982;100:722-6.
482. Hall CB. Respiratory syncytial virus: its transmission in the hospital environment. *Yale J Biol Med* 1982;55:219-23.
483. Snyderman DR, Greer C, Meissner HC, McIntosh K. Prevention of nosocomial transmission of respiratory syncytial virus in a newborn nursery. *Infect Control Hosp Epidemiol* 1988;9:105-8.
484. Harrington RD, Hooton TM, Hackman RC, Storch GA, Osborne B, Gleaves CA, et al. An outbreak of respiratory syncytial virus in a bone marrow transplant center. *J Infect Dis* 1992;165:987-93.

485. Guidry GG, Black-Payne CA, Payne DK, Jamison RM, George RB, Bocchini JA Jr. Respiratory syncytial virus infection among intubated adults in a university medical intensive care unit. *Chest* 1991;100:1377-84.
486. Falsey AR. Noninfluenza respiratory virus infection in long-term care facilities. *Infect Control Hosp Epidemiol* 1991;12:602-8.
487. Sorvillo FJ, Huie SF, Strassburg MA, Butsumyo A, Shandera WX, Fannin SL. An outbreak of respiratory syncytial virus pneumonia in a nursing home for the elderly. *J Infect* 1984;9:252-6.
488. Valenti WM, Hruska JF, Menegus MA, Freeburn MJ. Nosocomial viral infections: III. Guidelines for prevention and control of exanthematous viruses, gastroenteritis viruses, picornaviruses, and uncommonly seen viruses. *Infect Control* 1980;2:38-49.
489. Siegel JD. Risks and exposures for the pregnant health-care worker. In: Olmstead RN, editor. *APIC infection control and applied epidemiology: principles and practice*. St Louis: Mosby; 1996. p. 22-1, 22-8.
490. Valenti WM. Infection control and the pregnant health care worker. *Nurs Clin North Am* 1993;28:673-86.
491. Shortridge-McCauley LA. Reproductive hazards: an overview of exposures to health care workers. *AAOHN J* 1995;43:614-21.
492. Pike RM. Past and present hazards of working with infectious agents. *Arch Pathol Lab Med* 1978;102:333-6.
493. Pike RM. Laboratory-associated infections: incidence, fatalities, causes, and prevention. *Annu Rev Microbiol* 1979;33:41-66.
494. Favero MS. Biological hazards in the laboratory. *Lab Med* 1987;18:665-70.
495. Jacobson JT, Orlob RB, Clayton JL. Infections acquired in clinical laboratories in Utah. *J Clin Microbiol* 1985;21:486-9.
496. Grist NR, Emslie JAN. Infections in British clinical laboratories, 1986-87. *J Clin Pathol* 1989;42:677-81.
497. Vesley D, Hartmann HM. Laboratory-acquired infections and injuries in clinical laboratories: a 1986 survey. *Am J Public Health* 1988;78:1213-5.
498. Grist NR, Emslie JAN. Association of Clinical Pathologists' survey of infection in British clinical laboratories, 1970-1989. *J Clin Pathol* 1994;47:391-4.
499. Gilchrist MJR, Hindler J, Fleming DO. Laboratory safety management. In: Isenberg HI, editor. *Clinical microbiology procedures handbook*. Washington, DC: American Society for Microbiology; 1992. p. xxix-xxxvii.
500. Gilchrist MJR. Biosafety precautions for airborne pathogens. In: Fleming DO, Richardson JH, Tulis JJ, Vesley D, editors. *Laboratory safety principles and practices*. 2nd ed. Washington, DC: American Society for Microbiology; 1995. p. 67-76.
501. Centers for Disease Control and Prevention. Implementation of provisions of the Ryan White Comprehensive AIDS Resources Emergency Act regarding emergency response employees. *Federal Register* 1994;59(54):13418-28.
502. Hamann CP. Natural rubber latex protein sensitivity in review. *Contact Dermatitis* 1993;4:4-21.
503. Zaza S, Reeder JM, Charles LE, Jarvis WR. Latex sensitivity among perioperative nurses. *AORN J* 1994;60:806-12.
504. Bubak ME, Reed CE, Fransway AF, Yunginger JW, Jones RT, Carlson CA, et al. Allergic reactions to latex among health-care workers. *Mayo Clin Proc* 1992;67:1075-9.
505. Berky ZT, Luciano WJ, James WD. Latex glove allergy: a survey of the US Army Dental Corps. *JAMA* 1992;268:2695-7.
506. Yassin MS, Lierl MB, Fischer TJ, O'Brian K, Cross J, Steinmetz C. Latex allergy in hospital employees. *Ann Allergy* 1994;72:245-9.
507. Fisher AA. Allergic contact reactions in health personnel. *J Allergy Clin Immunol* 1992;90:729-38.
508. Hunt LW, Fransway AF, Reed CE, Miller LK, Jones RT, Swanson MC, et al. An epidemic of occupational allergy to latex involving health care workers. *J Occup Environ Med* 1995;37:1204-9.
509. Sussman GL, Tario S, Dolovich J. The spectrum of IgE-mediated responses to latex. *JAMA* 1991;265:2844-7.
510. Cormio L, Turjanmaa K, Talja M, Anderson LC, Ruutu M. Toxicity and immediate allergenicity of latex gloves. *Clin Exp Allergy* 1993;23:618-23.
511. Hamann CP, Kick SA. Update: immediate and delayed hypersensitivity to natural rubber latex. *Cutis* 1993;52:307-11.
512. Ownby DR. Manifestation of latex allergy. *Immun Allergy Clin North Am* 1995;15:31-43.
513. Estlander T, Jolanski R, Kanerva L. Dermatitis and urticaria from rubber and plastic gloves. *Contact Dermatitis* 1985;14:20-5.
514. Conde-Salazar L, del-Rio E, Guimaraens D, Gonzalez DA. Type IV allergy to rubber additives: a 10-year study of 686 cases. *J Am Acad Dermatol* 1993;29:176-80.
515. Heese A, Hintzenstern J, Peters KP, Koch HU, Hornstein OP. Allergic and irritant reactions to rubber gloves in medical health services. *J Am Acad Dermatol* 1991;25:831-9.
516. Lagier F, Vervloet D, Lhermet I, Poyen D, Charpin D. Prevalence of latex allergy in operating room nurses. *J Allergy Clin Immunol* 1992;90:319-22.
517. Gerber AC, Jorg W, Zbinden S, Seger RA, Dangel PH. Severe intraoperative anaphylaxis to surgical gloves: latex allergy, an unfamiliar condition. *Anesthesiology* 1989;71:800-2.
518. Arellano R, Bradley J, Sussman G. Prevalence of latex sensitization among hospital physicians occupationally exposed to latex gloves. *Anesthesiology* 1992;77:905-8.
519. Kaczmarek RG, Silverman BG, Gross TP, Hamilton RG, Kessler E, Arrowsmith-Lowe JT, et al. Prevalence of latex-specific IgE antibodies in hospital personnel. *Ann Allergy Asthma Immunol* 1996;76:51-6.
520. Marcos C, Lazaro M, Fraj J, Quirce S, de la Hoz B, Fernandes-Rivas M, et al. Occupational asthma due to latex surgical gloves. *Ann Allergy* 1991;67:319-23.
521. Frosch PJ, Wahl R, Bahmer FA, Maasch HJ. Contact urticaria to rubber gloves is IgE-mediated. *Contact Dermatitis* 1986;14:241-5.
522. Vandenplas O, Delwiche JP, Evrard G, Aimont P, VanDerBrempt X, Jamart J, et al. Prevalence of occupational asthma due to latex among hospital personnel. *Am J Respir Crit Care Med* 1995;151:54-60.
523. Tarlo SM, Wong L, Roos J, Booth N. Occupational asthma caused by latex in a surgical glove manufacturing plant. *J Allergy Clin Immunol* 1990;85:626-31.
524. Seaton A, Cherrie B, Turnbull J. Rubber glove asthma. *BMJ* 1988;296:531-2.
525. O'Byrne PM, Dolovich J, Hargreave FE. Late asthmatic responses. *Am Rev Respir Dis* 1987;136:130-1.
526. De Zotti R, Larese F, Fiorito A. Asthma and contact urticaria from latex gloves in a hospital nurse. *Br J Ind Med* 1992;49:596-8.

527. Brugnami G, Marabini A, Siracusa A, Abbritti G. Work-related late asthmatic response induced by latex allergy. *J Allergy Clin Immunol* 1995;96:457-64.
528. Alenius H, Mäkinen-Kiljunen S, Turjanmaa K, Palosuo T, Reunala T. Allergen and protein content of latex gloves. *Ann Allergy* 1994;73:315-20.
529. Yunginger JW, Jones RT, Fransway AF, Kelso JM, Warner MA, Hunt LW. Extractable latex allergens and proteins in disposable medical gloves and other rubber products. *J Allergy Clin Immunol* 1994;93:836-42.
530. Food and Drug Administration. Latex-containing devices; user labeling. *Federal Register* 1996;61:32617-21.
531. Jaeger D, Kleinhans D, Czuppon AB, Baur X. Latex-specific proteins causing immediate-type cutaneous, nasal, bronchial, and systemic reactions. *J Allergy Clin Immunol* 1992;89:759-68.
532. Grzybowski M, Ownby DR, Peyser PA, Johnson CC, Schork MA. The prevalence of anti-latex IgE antibodies among registered nurses. *J Allergy Clin Immunol* 1996;98:535-44.
533. Turjanmaa K. Incidence of immediate allergy to latex gloves in hospital personnel. *Contact Dermatitis* 1987;17:270-5.
534. Swanson MC, Bubak ME, Hunt LW, Yunginger JW, Warner MA, Reed LE. Quantification of occupational latex aeroallergens in a medical center. *J Allergy Clin Immunol* 1994;94:445-51.
535. Shield SW, Blaiss MS. Prevalence of latex sensitivity in children evaluated for inhalant allergy. *Allergy Proc* 1992;13:129-31.
536. M'Raihi L, Cahrpin D, Pons A, Bongrand P, Vervloet D. Cross-reactivity between latex and banana. *J Allergy Clin Immunol* 1991;87:129-30.
537. Kurup VJ, Kelly T, Elms N, Kelly K, Fink J. Cross-reactivity of food allergens in latex allergy. *Allergy Proc* 1994;15:211-6.
538. Blanco C, Carrillo T, Castillo R, Quirarte J, Cuevas M. Avocado hypersensitivity. *Allergy* 1994;49:454-9.
539. Ahlroth M, Alenius H, Turjanmaa K, Mäkinen-Kiljunen S, Reunala T, Palosuo T. Cross-reacting allergens in natural rubber latex and avocado. *J Allergy Clin Immunol* 1995;96:167-73.
540. Fernandez de Corres L, Moneo I, Munoz D, Bernaloa G, Fernandez E, Audicana M, et al. Sensitization from chestnuts and bananas in patients with urticaria and anaphylaxis from contact with latex. *Ann Allergy* 1993;70:35-9.
541. Kelly KJ, Kurup V, Zacharisen M, Resnick A, Fink JN. Skin and serologic testing in the diagnosis of latex allergy. *J Allergy Clin Immunol* 1993;91:1140-5.
542. Equal Employment Opportunity Commission. Equal employment opportunity for individuals with disabilities. 29 CFR 1630. *Federal Register* 1991;56:35726-53.
543. Bureau of National Affairs. Title VII jurisdiction: Equal Opportunity Commission compliance manual. Washington, DC: Bureau of National Affairs; 1986. p. 147-9.
544. Department of Justice. Title II technical assistance manual: the Americans with Disabilities Act. Washington, DC: Department of Justice; 1993. p. 1-12.
545. Department of Justice. Title III technical assistance manual: the Americans with Disabilities Act. Washington, DC: Department of Justice; 1993. p. 2-14.
546. Korniewicz DM, Kirwin M, Larson E. Do your gloves fit the task? *Am J Nurs* 1991;91:38-40.
547. Korniewicz DM. Effectiveness of glove barriers used in clinical settings. *Medsurg Nurs* 1992;1:29-32.
548. Korniewicz DM, Laughon BE, Butz A, Larson E. Integrity of vinyl and latex procedure gloves. *Nurs Res* 1989;38:144-6.
549. Korniewicz DM. Barrier protection of latex. *Immune Allergy Clin North Am* 1995;15:123-7.

Appendix A. Recommended readings for infection control in health care personnel

- Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. *Infect Control Hosp Epidemiol* 1996;17:53-80.
- Centers for Disease Control and Prevention, National Institutes for Health. Biosafety in microbiological and biomedical laboratories. 3rd ed. Atlanta: US Department of Health and Human Services, Public Health Service; 1993.
- Centers for Disease Control and Prevention. Immunization of health-care workers: recommendations of the Advisory Committee on Immunization Practices (ACIP) and the Hospital Infection Control Practices Advisory Committee (HIC-PAC). *MMWR Morb Mortal Wkly Rep* 1997;46(RR-18):1-42.
- Centers for Disease Control. Update on adult immunization: recommendations of the Immunization Practices Advisory Committee (ACIP). *MMWR Morb Mortal Wkly Rep* 1991;40(RR-12):1-94.
- US Department of Labor, Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens; final rule. CFR Part 1910.1030. *Federal Register* 1991;56:64004-182.
- Centers for Disease Control. Update: universal precautions for prevention of transmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. *MMWR Morb Mortal Wkly Rep* 1988;37:377-82, 387-8.
- Centers for Disease Control. Protection against viral hepatitis: recommendations of the Advisory Committee on Immunization Practices (ACIP). *MMWR Morb Mortal Wkly Rep* 1990;39(RR-2):1-27.
- Centers for Disease Control and Prevention. Public Health Service (PHS) guidelines for the management of health care worker exposures to HIV and recommendations for postexposure prophylaxis. *MMWR Morb Mortal Wkly Rep*. In press 1998.
- Centers for Disease Control and Prevention. Guidelines for preventing the transmission of *Mycobacterium tuberculosis* in health-care facilities, 1994. *MMWR Morb Mortal Wkly Rep* 1994;43(RR-13):1-132.
- Centers for Disease Control, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of nosocomial pneumonia. *Infect Control Hosp Epidemiol* 1994;15:587-627.
- US Department of Labor, Occupational Health and Safety Administration. Record keeping guidelines for occupational injuries and illnesses: the Occupational Safety and Health Act of 1970 and 29 CFR 1904. OMB no. 120-0029. Washington, DC: US Department of Labor; 1986.
- US Department of Labor, Occupational Health and Safety Administration. Criteria for recording on OSHA form 200. OSHA instruction 1993; standard 1904. Washington, DC: US Department of Labor; 1993.
- American Public Health Association. Benenson AS, editor. Control of communicable diseases manual. 16th ed. Washington, DC: American Public Health Association. 1995.
- American Academy of Pediatrics. Peter G, editor. 1997 red book: report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village (IL): American Academy of Pediatrics; 1997.