DEPARTMENT OF HEALTH AND HUMAN SERVICES

Centers for Disease Control and Prevention

Draft Guideline for Infection Control in Health Care Personnel, 1997

AGENCY: Centers for Disease Control and Prevention (CDC), Department of Health and Human Services (DHHS).

ACTION: Notice.

SUMMARY: This notice is a request for review of and comment on the Draft Guideline for Infection Control in Health Care Personnel, 1997. The guideline consists of two parts: Part 1. ''Infection Control Issues for Health Care Personnel, an Overview'' and Part 2. ''Recommendations for Prevention of Infections in Health Care Personnel'', and was prepared by the Hospital Infection Control Practices Advisory Committee (HICPAC), the National Center for Infectious Diseases (NCID), the National Immunizations Program, and the National Institute of Occupational Safety and Health (NIOSH), CDC.

DATES: Written comments on the draft document must be received on or before October 17, 1997.

ADDRESSES: Comments on this document should be submitted in writing to the CDC, Attention: PHG Information Center, Mailstop E–68, 1600 Clifton Road, N.E., Atlanta, Georgia 30333. To order copies of the **Federal Register** containing the document, contact the U.S. Government Printing Office, Order and Information Desk, Washington, DC 20402–9329, telephone (202) 512–1800. In addition, the **Federal Register** containing this draft document may be viewed and photocopied at most libraries designated as U.S. Government Depository Libraries and at many other public and academic libraries that receive the **Federal Register** throughout the country. Addresses and telephone numbers of the U.S. Government Depository Libraries are available by fax by calling U.S. Fax Watch at (202) 512–1716 and selecting option 5 from the main menu. The **Federal Register** is also available online on the Superintendent of Documents home page at: http://www.access.gpo.gov/su__docs.

FOR FURTHER INFORMATION CONTACT: The CDC Fax Information Center, telephone (888) 232–3299 and order document number 370160 or, for voice information, call the PH Guideline Information Center, telephone (888) 232–3228, then press 2, 2, 3, 2, 2, 1, 5 to go directly to the guideline information.

SUPPLEMENTARY INFORMATION: This 2-part document updates and replaces the previously published CDC Guideline for Infection Control in Hospital Personnel (Infect Control 1983 [Special Supplement]; 4 [Suppl]: 326–349). Part 1, ''Infection Control Issues for Health Care Personnel, an Overview'' serves as the background for the consensus recommendations of the Hospital Infection Control Practices Advisory Committee (HICPAC) that are contained in Part 2, ''Recommendations for Prevention of Infections in Health Care Personnel''.

HICPAC was established in 1991 to provide advice and guidance to the Secretary and the Assistant Secretary for Health, DHHS; the Director, CDC, and the Director, NCID regarding the practice of hospital infection control and strategies for surveillance, prevention, and control of nosocomial infections in U.S. hospitals. The committee also advises CDC on periodic updating of guidelines and other policy statements regarding prevention of nosocomial infections.

The Guideline for Infection Control in Hospital Personnel, 1997 is the fourth in a series of CDC guidelines being revised by HICPAC and NCID, CDC.

Dated: September 2, 1997.

Joseph R. Carter,

Acting Associate Director for Management and Operations, Centers for Disease Control and Prevention (CDC).

Draft Guideline for Infection Control in Health Care Personnel, 1997

Executive Summary

This guideline updates and replaces the previous edition of the 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 exposures 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 also addresses issues related to latex hypersensitivity and provides recommendations to prevent sensitization and reactions among health care personnel.

Part I. Infection Control Issues for Health Care Personnel, an Overview

A. 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 6,000 hospitals. However, health care is increasingly being provided outside of hospitals in facilities such as nursing homes, freestanding surgical and outpatient centers, emergency care clinics, and in patients, homes or during pre-hospital emergency care. Hospital-based personnel and personnel who provide health care outside of hospitals may acquire infections from or transmit infections to patients or other personnel, household members, or other community contacts.

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, mortuary personnel, nurses, nursing assistants, physicians, technicians, students and trainees, contractual staff not employed by the health care facility, and persons not directly involved in patient care (e.g., clerical, dietary, housekeeping, maintenance, and volunteer personnel) but potentially exposed to infectious agents. In general, health care personnel, in or outside of 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.

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 (1). 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 bathing, performing 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 of time or 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 Centers for Disease Control and Prevention (CDC) published the Guideline for Infection Control in Hospital Personnel (2). 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 of hospitals; (b) management of exposures; (c) prevention of transmission of infections in microbiologic and biomedical laboratories; and, (d) because of the common use of latex barriers to prevent infections, prevention of latex hypersensitivity reactions. As in the 1982 Guideline, readers are frequently referred to the Guideline for Isolation Precautions in Hospitals (1) and other published guidelines and recommendations for precautions that health care personnel may use when caring for patients, or handling patient equipment or specimens (3, 4).

B. 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 individual 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.

C. 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. 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 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, e.g., history of chickenpox, rubella, measles, mumps, hepatitis, immunodeficiency, dermatologic conditions (including chronic draining or open wounds), and risk factors or treatment for tuberculosis. 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 counts, serologic tests for syphilis, urinalysis, chest x-rays) or 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, ongoing programs (e.g., tuberculosis 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, since 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 of preventive measures that will reduce those risks. Furthermore, educational materials need to be appropriate in content and vocabulary to the educational level, literacy, and language of the employee. 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. Preventing 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 (5). Also, programs in which the employer bears the cost of vaccination have had higher personnel vaccination rates than have programs without such support.

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) (6, 7). 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 (6–20); and (c) recommendations for immunization of immunocompromised persons (Table 2) (14, 21). The recommendations in this guideline have been adapted from the ACIP recommendations (7). In addition, individual states and professional organizations have regulations or recommendations on the vaccination of health care personnel (22).

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 (i.e., 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 (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 following 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. Decisions on work restrictions are based on the mode of transmission and the epidemiology of the disease (Table 3). 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. 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. Notification of emergency response personnel possibly exposed to selected infectious disease is mandatory (1990 Ryan White Act, Subtitle B, 42 U.S.C 300ff–80).

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 individualized information regarding (a) the risk and prevention of occupationally acquired infections; (b) the risk of illness or other adverse outcome following exposures; (c) management of exposures, including the risks and benefits of postexposure prophylaxis regimens; (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, data base 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) record-keeping requirements for occupational injuries and illnesses (23). In addition, the 1991 OSHA Occupational Exposure to Bloodborne Pathogens; Final Rule (24) 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.

More recently, OSHA developed enforcement policies that require the recording and reporting of positive tuberculin skin test results (25). It would be beneficial to health care organizations and personnel if the principles of record keeping and confidentiality mandated by OSHA were expanded to other work-related exposures and incidents, immunizations, tuberculosis screening, and investigation and management of nosocomial outbreaks.

D. 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. However, only those infectious diseases that occur frequently in the health care setting or are most important to personnel are discussed below.

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 is based upon 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.

CDC has periodically issued and updated recommendations for prevention of transmission of bloodborne pathogens in health care settings that provide detailed information and guidance (26–36). Also, in 1991, OSHA published a bloodborne pathogen standard, based on the concept of Universal Precautions, to prevent occupational exposure to bloodborne pathogens (24). In essence, the use of Standard Precautions (which incorporates Universal Precautions), including appropriate handwashing and barrier precautions to prevent contact with blood and body fluids and using techniques and devices that reduce percutaneous injury, will reduce the risk of transmission of bloodborne pathogens (1, 27, 37–42).

The risk posed to patients from 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 (43). Subsequently, Congress mandated that each state implement the CDC guidelines or equivalent as a condition for continued federal public health funding to that state. While 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 (44–48). Approximately 1,000 health care personnel were estimated to have become infected with HBV in 1994. This is a 90% decline since 1985, attributable to the use of vaccine and adherence to other preventive measures (e.g., Standard Precautions) (49). During the past decade, an estimated 100 to 200 health care personnel have died annually from HBV infection (49). The risk of acquiring HBV infection from occupational exposure is dependent on the nature and frequency of exposure to blood or body fluids containing blood (44, 48). The risk of infection is at least 30% after a percutaneous exposure to blood from a hepatitis B e antigen-positive source (49).

HBV is transmitted by percutaneous or mucosal exposure to blood and serum-derived body fluids from persons who either are have acute or chronic HBV infection. The incubation period is 45 to 180 days. Any person with blood positive 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 (7, 8, 36). 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 (24). Provision of vaccine during training for health care professions before such blood exposure occurs may increase the vaccination rates among personnel and prevent infection among trainees who are at increased risk of unintentional injuries while learning techniques.

Prevaccination serologic screening for susceptibility to HBV infection is not indicated for persons being vaccinated, unless the health care organization considers screening to be cost-effective. Postvaccination screening for antibody to HBsAg (anti-HBs) is advised for personnel at ongoing risk of blood exposure, to determine if 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 be evaluated to determine if they are HBsAg positive. Revaccinated persons should be tested for anti-HBs at the completion of the second vaccine series (7). If they do not respond, no further vaccination series should be given and they should be evaluated for the presence of HBsAg (e.g., possible chronic HBV infection).

Vaccine-induced antibodies decline gradually over time, and up to 60% of those who initially respond to vaccination will lose detectable anti-HBs over 12 years (50). Booster doses of vaccine are not 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 (51).

The need for postexposure prophylaxis and/or vaccination depends on the HBsAg status of the source of the exposure as well as the immunization status of the person exposed (Table 4) (36). Vaccine should be offered following any exposure in an unvaccinated person, and, if the source is known to be HBsAg positive, hepatitis B immune globulin (HBIG) should be given, preferably within 24 hours. The effectiveness of HBIG given >7 days after HBV exposure is unknown (6, 8, 36). If the exposed person is known not to have responded to a 3 dose vaccine series, a single dose of HBIG and a dose of hepatitis B vaccine needs to be given as soon as possible after the exposure. If the exposed person is known not to have responded to a 3 dose vaccine series or to revaccination, two doses of HBIG need to be given, one doses 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 (52,53). 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%–4% occurred among health care personnel who were occupationally exposed to blood (53).

A case-control study of patients with acute non-A, non-B hepatitis, conducted before the identification of HCV, showed a significant association between acquiring disease and health care employment, specifically, patient care or laboratory work (54). Seroprevalence studies among hospital-based health care personnel have shown anti-HCV seroprevalence rates of 1% to 2% (55–58). In a study that assessed risk factors for infection in health care personnel, a history of accidental needlesticks was independently associated with anti-HCV positivity (55).

Several case reports have documented transmission of HCV infection from anti-HCV-positive patients to health care personnel as a result of accidental needlesticks or cuts with sharp instruments (59, 60). In follow-up studies of health care personnel who sustained percutaneous exposures to blood from anti-HCV positive patients, the incidence of anti-HCV seroconversion averaged 1.8% (range, 0%–7%) (61–64). In a study in which HCV RNA polymerase chain reaction methods were used to measure HCV

infection, the incidence of HCV infection was 10% (64).

The incubation period for hepatitis C is 6–7 weeks, and nearly all persons with acute infection develop chronic HCV infection with persistent viremia and have the potential for transmission of HCV to others.

Serologic assays to detect antibody to HCV (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, or 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 (up to 50%) of false-positive results (30, 53). 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 (IG) for postexposure prophylaxis after occupational exposure to hepatitis C virus has been difficult to assess (65–67), postexposure prophylaxis with IG does not appear to be effective in preventing HCV infection. Current IG 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 IG manufactured from anti-HCV-screened plasma and administered one hour after exposure to HCV did not prevent infection or disease (68). Thus, available data do not support the use of IG for postexposure prophylaxis of hepatitis C and its use is not recommended. There is no information regarding the use of antiviral agents, such as alpha interferon, in the postexposure setting, and such prophylaxis is not recommended (33, 69).

Health care institutions should consider implementing recommended policies and procedures for follow-up for HCV infection after percutaneous or mucosal exposures to blood (69).

d. Human Immunodeficiency Virus. Nosocomial transmission of HIV infection from patients to health care personnel may occur following percutaneous or, infrequently, mucocutaneous, exposure to blood or body fluids containing blood. Based on 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% (70–74). 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 presence of visible blood on the device prior to injury; a procedure that involved a needle placed directly in the patient's vein or artery; or deep injury. Transmission of HIV infection also was associated with injuries in which the source patient was terminally ill with acquired immunodeficiency syndrome (AIDS); this may be attributable to the increased titer of HIV in blood that is known to accompany late stages of illness, or possibly 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 use of zidovudine postexposure may be protective for health care personnel (71).

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 (75). Most personnel who acquire infection following percutaneous exposure develop HIV antibody 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 (29). In 1996, provisional recommendations for postexposure chemoprophylaxis were published, reflecting current scientific knowledge on the efficacy of postexposure prophylaxis and the use of antiretroviral therapies (76). The U.S. Public Health Service will periodically review scientific information on antiretroviral therapies and will publish updated recommendations for their use as postexposure prophylaxis as necessary.

2. Conjunctivitis

Conjunctivitis can be caused by a variety of bacteria and viruses. However, 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 (EKC) in the community and health care settings. Nosocomial outbreaks have primarily occurred in eye clinics or offices, but have also been reported in newborn intensive care units and long term care facilities (77–81). Patients and health care personnel have acquired and transmitted EKC during these outbreaks. The incubation period ranges from 5 to 12 days and shedding of virus occurs from late in the incubation period up to 14 days after onset of disease (78). 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 (77, 78).

Infected personnel should not provide patient care for the duration of symptoms following onset of EKC (77, 78) 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 persons with AIDS (82–88). 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% versus 2%) (89–95). In areas where there are patient populations with 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 (87, 89–92, 94, 96–99). In addition, epidemiologic studies that included DNA testing of viral strains have demonstrated that personnel who acquired CMV infection while providing care to CMV-infected infants did not acquire their infection from the CMV-infected patients (83, 87, 90, 100–102).

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 (95, 103–106). Transmission via the hands of personnel or infected person(s) also has been suggested (87, 107). The incubation period for person-to-person transmission is not known. Although

CMV can survive on environmental surfaces and other objects for short periods of time (108), there is no evidence that the environment plays a role in the transmission of infection (87).

Because infection with CMV during pregnancy may have adverse effects on the fetus, protecting women of childbearing age from persons who are excreting the virus is of primary concern. However, the risk of occupational transmission to female health care personnel is no greater than the risk among the general public (89, 96, 109). While a majority of fetal infections follow primary maternal infection, fetal infection may follow maternal reinfection or reactivation. 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 (110); 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 (109, 111); (c) no currently available vaccines (112–115) or prophylactic therapy (116–120) can provide protection against primary infection; and (d) no studies clearly indicate that personnel may be protected by transfer to areas with less contact with patients likely to be reservoirs for CMV infection (83, 87, 89–91, 96, 99, 121). Counseling of female personnel of childbearing age on the risk of transmission of CMV in both nonoccupational and occupational environments may help allay their fears (122).

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. (1, 109, 123).

4. Diphtheria

Nosocomial transmission of diphtheria among patients and personnel has been reported (124–126). Diphtheria is currently a rare disease in the United States; during 1980–1994 only 41 diphtheria cases were reported (127), however, community outbreaks of diphtheria have occurred in the past (128), and clusters of infection may occur in communities where diphtheria was previously endemic (129). In addition diphtheria epidemics have been occurring since 1990 in the New Independent States of the former Soviet Union (130–132) and in Thailand (133). At least 20 imported cases of diphtheria have been reported in countries in Europe (132, 134) and two cases occurred in U.S. citizens visiting or working in the Russian Federation and Ukraine (135). Health care personnel are not at substantially higher risk than the general adult population for acquiring diphtheria; however, there is the 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 2–5 days. Patients with diphtheria are usually infectious for ≥2 weeks, but communicability can persist for several months (136). 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 two cultures taken ≥24 hours apart are negative (1).

Limited serosurveys conducted since 1977 in the United States indicate that 22%–62% of adults 18–39 years of age may lack protective diphtheria antibody levels (137–141). Prevention of diphtheria is best accomplished by maintaining high levels of diphtheria immunity among children and adults (17, 130, 131). Immunization with tetanus and diphtheria toxoid (Td) is recommended every 10 years for all adults who have completed the primary immunization series (7, 17) (Table 1). Health care personnel need to consider obtaining Td immunization from their health care providers (7).

To determine if 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 (142). Although the efficacy of antimicrobial prophylaxis in preventing secondary disease has not been proven, prophylaxis with either a single IM injection of benzathine penicillin (1.2 million units) or oral erythromycin (1 g/day) for 7 days has been recommended (17). Follow-up nasopharyngeal cultures for *C. diphtheriae* need to be obtained after antimicrobial therapy is completed. If the organism has not been eradicated, a 10-day course of erythromycin needs to be given (142). In addition, previously immunized, exposed personnel need to receive a dose of Td if they have not been vaccinated within the previous 5 years (17).

Exclusion from duty is indicated for personnel with *C. diphtheriae* infection or those identified as asymptomatic carriers until antimicrobial therapy is completed and nasopharyngeal cultures 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) (143–161). Nosocomial transmission of agents that cause gastrointestinal infections usually results from contact with infected individuals (143, 154, 156, 162); from consumption of contaminated food, water, or other beverages (143, 159, 162); or from exposure to contaminated objects or environmental surfaces (145, 146, 163). Airborne transmission of small round-structured viruses (Norwalk-like viruses) has been postulated but not proven (157, 158, 164–167). Inadequate handwashing by health care personnel (168) 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 Precautions (1) will minimize the risk of transmitting enteric pathogens (160, 169).

Laboratory personnel who handle infectious materials may also 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 (144, 155). Several typhoid vaccines are available for use in laboratory workers who regularly work with cultures or clinical materials containing *S. typhi* (170). The oral live-attenuated Ty21a vaccine, the IM Vi capsular polysaccharide (ViCPS) vaccine, or the subcutaneous inactivated vaccine may be given (170) (Table 1). 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(170).

Personnel who develop an acute gastrointestinal illness, defined as vomiting and/or diarrhea (i.e., ≥3 loose stools in a 24-hour period) 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) (158, 171, 172). 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., newborns, the elderly, 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 (155, 163, 173, 174).

After resolution of some acute bacterial gastrointestinal illnesses, some personnel may have persistent carriage of the infectious agent. However, once the person has clinically recovered and is having formed stools, the risk of transmission of enteric pathogens is minimal when there is adherence to Standard Precautions (1, 160). In addition, appropriate antimicrobial therapy may eradicate fecal carriage of *Shigella* (175) or *Campylobacter* (176). However, antimicrobial or antiparasitic therapy may not eliminate carriage of *Salmonella* (177) or *Cryptosporidium*. Moreover, antimicrobials may prolong excretion of *Salmonella* (178) and lead to emergence of resistant strains (179). However, transmission of *Salmonella* to patients from personnel who are asymptomatic carriers of *Salmonella* has not been well documented (160). In general, antimicrobial therapy is not recommended unless the person is at high risk for severe disease (180). When antibiotics are given, stool cultures should be obtained ≥48 hours after completion of antibiotic therapy.

Restriction from patient care or food-handling is indicated for personnel with diarrhea or acute gastrointestinal symptoms, regardless of the causative agent (1, 163). Some local and state agencies have regulations that require work exclusion for health care personnel and/or food handlers who have gastrointestinal infections caused by *Salmonella* or *Shigella*. These regulations may require that such personnel be restricted from duty until ≥2 consecutive stool cultures obtained ≥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 (181–190). 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 (181, 183, 185, 191); (b) not washing hands after handling an infected infant (183, 191, 192) and (c) sharing food, beverages, or cigarettes with patients, their families, or other staff (181, 183);.

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 has rarely been reported to be transmitted by transfusion of blood products (185, 193, 194). The incubation period for HAV is 15–50 days. Fecal excretion of HAV is greatest during the incubation period of disease before the onset of jaundice (195). 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 they are potentially infective (182, 195). Fecal shedding of HAV, formerly believed to continue only for up to 2 weeks after onset of dark urine (195), has been shown to occur for up to 6 months after diagnosis of infection in premature infants (181). Anicteric infection is typical in young children and infants (196).

Personnel can protect themselves and others from infection with HAV by following Standard Precautions (1). Foodborne transmission of hepatitis A is not discussed in this guideline, but has occurred in health care settings (197, 198).

Two inactivated hepatitis A vaccines, HAVRIX® and VAQTA®, are now available and provide long-term preexposure protection against clinical infection with >94% efficacy (196). Serologic surveys among health care personnel have not shown an elevated prevalence of HAV infection compared with control populations (47, 184, 199, 200); therefore, routine administration of vaccine in health care personnel is not recommended. Vaccine may be useful for personnel working 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 (7). Immune globulin (IG) given within 2 weeks following an HAV exposure is >85% effective in preventing hepatitis A virus infection (196) and may be advisable in some outbreak situations (7, 196).

Restriction from patient care or food-handling is indicated for personnel with HAV infection. They may return to regular duties 1 week following onset of illness (7).

7. Herpes Simplex

Nosocomial transmission of herpes simplex viruses (HSV) is rare. Nosocomial transmission has been reported in nurseries (201–203) and intensive care units (204, 205) 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 with either primary or recurrent lesions or from virus-containing secretions, such as saliva, vaginal secretions, or amniotic fluid (202, 204, 206). Exposed areas of skin are the most likely sites of nosocomial infection, particularly when minor cuts, abrasions, or other skin lesions are present (205). The incubation period of HSV is 2–14 days (207). The duration of viral shedding has not been well defined (208).

Personnel may develop an herpetic infection of the fingers (herpetic whitlow or paronychia) from exposure to contaminated oral secretions (205, 206). 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 (205). Less frequently, personnel may develop mucocutaneous infection on other body sites from contact with infectious body secretions (209).

Personnel with active infection of the hands (herpetic whitlow) can potentially transmit HSV infection to patients with whom they have contact (206). Transmission of HSV from personnel with orofacial HSV infection to patients has also been infrequently documented (201); however, the magnitude of the risk is unknown (203, 210). Although asymptomatic infected persons can shed the virus, they are less infectious than persons with active lesions (208, 211).

Personnel can protect themselves from acquiring HSV by adhering to Standard Precautions (1). 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, excluding them from the care of patients at high risk for serious disease (e.g., neonates, patients with severe malnutrition, patients with severe burns or eczema, and immunocompromised patients) should be considered. 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 if 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 (212–221). From 1985 through 1991, approximately 3,000 (4%) of all reported episodes of measles in the United States were probably acquired in a medical facility; of these, >700 (25%) occurred in health care personnel, many of whom were not vaccinated (7). Data have suggested that health care personnel have a 13-fold greater risk of measles compared with the general population (7). Of the 2,765 episodes of measles reported during 1992–95, 385 (13.9%) occurred in health care settings (213, 222).

Measles is transmitted both by large droplets during close contact between infected and susceptible persons and by the airborne route (221, 223). Measles is highly transmissible and frequently misdiagnosed during the prodromal stage. The incubation period for measles is 5–21 days. Immunocompetent persons with measles shed the virus from the nasopharynx, beginning with the prodrome until 3–4 days after rash onset; immunocompromised persons with measles may shed virus for extended periods of time (224).

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; (c) adherence to airborne precautions for suspected and proven cases of measles (1); and (d) vaccination of patients in medical settings, especially emergency rooms.

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. Furthermore, 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%-9% of health care personnel born before 1957 may not be immune (225, 226). Furthermore, during 1985–1989, 29% of all measles cases in U.S. health care personnel occurred in those born before 1957 (213). 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 prior measles disease; (b) documentation of receipt of one dose of live measles vaccine; or (c) serologic evidence of measles immunity (7). 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 upon testing should be considered susceptible). Persons born between 1957 and 1984 who received childhood measles immunization were given only one dose of vaccine in their infancy and may require a second dose of vaccine (6).

Serologic screening for measles immunity is not necessary prior to administering measles vaccine unless the medical facility considers it cost-effective or the person to be vaccinated requests it (227–229). When serologic screening before vaccination is done, tracking systems are needed to ensure that those identified as susceptibles are subsequently vaccinated in a timely manner (229). 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 develop measles; they need to be excluded from duty for 4 days after the rash appears. Likewise, personnel nonimmune to measles need to be excluded from duty for 5 days after the first exposure to 21 days following the last exposure to measles.

9. Meningococcal Disease

Community-acquired meningococcal disease typically is 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 (13). In contrast, epidemic meningococcal disease has, since the early 1990s, been caused increasingly by Serogroup C (13, 230, 231).

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 (1, 232–234) or through handling laboratory specimens (235). Lower respiratory infections caused by *N. meningitidis* may present a greater risk of transmission than either meningococcemia or meningitis (234, 236), especially if the patient has an active, productive cough (236). The risk of personnel acquiring meningococcal disease from casual contact (e.g., cleaning rooms or delivering food trays) appears to be negligible (236).

N. meningitidis infection is likely transmitted by large droplets; the incubation period is from 2–10 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 (1).

Postexposure prophylaxis is advised for persons who have had intensive, unprotected contact (i.e., without wearing a mask) with infected patients (e.g., intubating, resuscitating, or closely examining the oropharynx of patients) (13). Antimicrobial prophylaxis can eradicate carriage of *N. meningitidis* and prevent infections in personnel who have unprotected exposure to patients with meningococcal infections (237,238).

Because secondary cases of *N. meninigitidis* occur rapidly (within the first week) following exposure to persons with meningococcal disease (239), it is important to begin prophylactic therapy immediately after an intensive, unprotected exposure, often before results of antimicrobial testing are available. Prophylaxis administered >14 days after exposure is probably of limited or no value (13). Rifampin (600 mg orally every 12 hours for 2 days) is effective in eradicating nasopharyngeal carriage of *N.*

meningitidis (237). Ciprofloxacin (500 mg orally) and ceftriaxone (250 mg IM) in single-dose regimens are also effective in reducing nasopharyngeal carriage of *N. meningitidis* and are reasonable alternatives to the multidose rifampin regimen (13, 238). These antimicrobials may be useful in situations where infections are caused by rifampin-resistant meningococci or when rifampin is contraindicated. Rifampin and ciprofloxacin are not recommended for pregnant women (13, 240, 241).

The quadrivalent A,C,Y,W–135 polysaccharide vaccine has been used successfully to control community outbreaks caused by Serogroup C (13, 230, 231, 240), but its use is not recommended for postexposure prophylaxis in health care settings (13). However, preexposure vaccination may be considered for laboratory personnel who routinely handle soluble preparations of *N. meningitidis* (13, 235).

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. Healthy persons may have nasopharyngeal carriage of *N. meningitidis* (237, 242–244). Nosocomial transmission from carriers to personnel has not been reported.

10. Mumps

Mumps transmission has occurred in hospitals and long-term-care facilities housing adolescents and young adults (245, 246). 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 16–18 days. The virus may be present in saliva for 6–7 days before parotitis and may persist for up to 9 days after onset of disease. Exposed personnel may be infectious for 12–25 days after their exposure and many infected persons remain asymptomatic (247). Droplet precautions are recommended for patients with mumps; such precautions should be continued for 9 days after the onset of parotitis (1).

An effective vaccination program is the best approach to preventing nosocomial mumps transmission (10). Vaccination with mumps virus vaccine is recommended, unless otherwise contraindicated, for all those who are susceptible to mumps (10, 248); combined MMR vaccine is the vaccine of choice (249), 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) (10). Most persons born before 1957 are likely to have been infected naturally and may be considered to be immune, even if they may not have had clinically recognized mumps. Outbreaks among highly vaccinated populations have occurred and have been attributed to primary vaccine failure (250).

Work restrictions are necessary for personnel who develop 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.

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 develop 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 develop aplastic crisis. Immunodeficient patients (e.g., patients with leukemia or AIDS) may become chronically infected with B19 and develop chronic anemia (251, 252).

Transmission of B19 to health care personnel from infected patients appears to be rare. 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 controls (253, 254). In another investigation of health care personnel exposed to an undetected patient with chronic B19 infection, none of the susceptible employees became infected (255). Personnel have acquired infection while working in laboratories or during the care of patients with B19-associated sickle cell aplastic crises (256–261).

B19 may be transmitted via contact with infected persons, fomites, or large droplets (253, 262, 263). The incubation period is variable, depending on the clinical manifestation of disease, and ranges from 6–10 days (252). 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, up to 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 (264, 265). Because of the seriousness of 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 (1).

Isolation precautions are not indicated for most patients with erythema infectiousum because they are past their period of infectiousness at the time of clinical illness (259, 264). However, patients in aplastic crisis due to 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 known or suspected to be chronically infected with B19 should be placed on Droplet Precautions on admission and for the duration of hospitalization (1, 256). Work restrictions are not necessary for personnel exposed to B19.

12. Pertussis

Nosocomial transmission of *Bordetella pertussis* has involved both patients and personnel; unimmunized children are at greatest risk (266–270). 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 (267, 269, 271, 272). 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 housestaff (82%) and ward nurses (71%) and lowest in nurses with administrative responsibilities (35%) (267).

Pertussis is highly contagious: Secondary attack rates exceed 80% in susceptible household contacts (273–275). *B. pertussis* transmission occurs by contact with respiratory secretions or large aerosol droplets from the

respiratory tract of infected persons. The incubation period is usually 7–10 days. The period of communicability starts at the onset of the catarrhal stage and extends into the paroxysmal stage. Preventing 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 nonspecific 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 presenting with an acute cough lasting ≥7 days, particularly if accompanied by paroxysms of coughing, inspiratory whoop, or post-tussive vomiting (270, 271).

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 (1); (c) exclusion of infectious personnel from work; and (d) administration of postexposure prophylaxis to persons exposed to infectious patients (269). Patients with suspected or confirmed pertussis who are admitted to the hospital need to be placed on Droplet Precautions until they improve clinically and have received antimicrobial therapy for at least 5 days.

Vaccination of adolescents and adults with whole-cell *B. pertussis* vaccine is not recommended (17) because local and systemic reactions have been observed more frequently in these groups than in children. Acellular pertussis vaccine is immunogenic in adults and has a lower risk of adverse events than does whole-cell vaccine (270, 276). However, the acellular vaccine has not been licensed for use in persons ≥7 years old. Because immunity among vaccine recipients wanes 5–10 years after the last vaccine dose (usually given at 4–6 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 qid po) or trimethoprim-sulfamethoxazole (1 tablet bid) has been used for this purpose. The efficacy of such prophylaxis has not been well documented, but studies suggest that it may minimize transmission (17, 269, 277, 278). There are no data on the efficacy of newer macrolides (clarithomycin or azithromycin) for prophylaxis of 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 case of indigenously acquired wild-virus poliomyelitis occurred in the United States in 1979. Since then, all of the cases of endemic poliomyelitis reported in the United States (5–10 endemic cases/year) have been related to the administration of oral polio vaccine (OPV) (19). 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, and usually 7 to 21 days for paralytic polio (279). 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 following onset of symptoms.

Vaccine-associated poliomyelitis may occur in the recipient (7–21 days after vaccine administration) or susceptible contacts of the vaccine recipient (20–29 days after vaccine administration) (280). Adults have a slightly increased risk of vaccine-associated paralytic polio after receipt of OPV; therefore, inactivated poliovirus vaccine (IPV) should be used when adult immunization is warranted (6, 14, 19). Also, because immunocompromised persons may be at greater risk for developing polio 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 (6, 14, 19, 279).

Health care personnel who may have contact with patients excreting wild virus (e.g., imported poliomyelitis case) and laboratory personnel handling specimens containing poliovirus should receive a complete series of polio vaccine, or if previously vaccinated, they may require a booster dose of either IPV or OPV (6, 19). 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 if they have previously completed a polio vaccine series (19).

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 (281). Laboratory and animal care personnel who are exposed to infected animals, their tissues and excretions are at risk for the disease. Also, rabies transmission to laboratory personnel has been reported in vaccine production and research facilities following exposure to high-titered infectious aerosols (282, 283). Theoretically, rabies may be transmitted to health care personnel from exposures (bite and non-bite) to saliva from infected patients, but no cases have been documented following these types of exposures (284).

It is also possible that rabies can be transmitted when other potentially infectious material (such as brain tissue) comes in contact with nonintact skin or mucous membranes. Bites that penetrate the skin, especially bites to the face and hands, pose the greatest risk of transmission of rabies virus from animals to humans (20). The incubation period for rabies is usually 1 to 3 months but longer periods have been reported (285).

Exposures to rabies can be minimized by adhering to Standard Precautions when caring for persons with suspected or confirmed rabies (1) and by using proper biosafety precautions in laboratories (3). 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 (3, 20). 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 who breeds the animals.

Postexposure prophylaxis has been administered to health care personnel following exposures to patients with rabies (285–287) (Table 1) but decisions regarding postexposure prophylaxis should be made on a case-by-case basis

after discussion with public health authorities (20).

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 (288–295).

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 develop the rash 14–16 days after exposure. The disease is most contagious when the rash is erupting, but virus may be shed from 1 week before to 5–7 days after the onset of the rash (296). 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; therefore, when caring for such patients it is advisable to use Contact Precautions for the first year of life, unless nasopharyngeal and urine cultures are negative for rubella virus after 3 months of age (1).

Ensuring immunity among all health care personnel (male and female) is the most effective way to eliminate nosocomial transmission of rubella (6, 7, 12, 248, 297). 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; or (b) laboratory evidence of immunity (persons with indeterminate levels are considered susceptible). A history of past 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 recommended for those health care personnel that do not have laboratory evidence of immunity (7). In addition, birth before 1957 is not considered acceptible evidence of rubella immunity for women of childbearing age (7). Voluntary immunization programs are usually inadequate to ensure personnel protection (298, 299). 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 (227–229). 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 (229). Likewise, during rubella outbreaks, serologic screening is not necessary. The ACIP states that rubella vaccination is contraindicated among pregnant women, but administering rubella vaccine to women not known to be pregnant is justifiable without prevaccination screening (12); pregnant women who are already immune to rubella are not at increased risk for adverse advents (300). MMR trivalent vaccine is the vaccine of choice for rubella, especially when the recipient also is likely to be susceptible to measles and/or mumps (Table 2).

Work restrictions are necessary for personnel who develop 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 7th 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 because their skin may become hyperkeratotic, and pruritus may not be present, which also makes diagnosis difficult. In conventional scabies 10–15 mites are present, while in crusted scabies thousands of mites are harbored in the skin, increasing the potential for transmission (301, 302).

Nosocomial outbreaks of scabies have occurred in a variety of health care settings including intensive care units (303), rehabilitation centers (304), long-term care facilities (305–307), hospital wards (308, 309), and a health care laundry (310). 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 (303, 304, 306–308, 310–315).

Nosocomial transmission of scabies occurs primarily through skin-to-skin contact with an infested person (301, 316, 317). Personnel have acquired scabies while performing patient-care duties such as sponge-bathing, lifting, or applying body lotions (301, 302, 312, 318). Transmission by casual contact, such as by holding hands, or via innaminate objects, such as infested bedding, clothes, or other fomites, has been reported infrequently (310, 319, 320).

The use of Contact Precautions when taking care of infested patients prior to application of scabicides can decrease the risk of transmission to personnel (1, 302). 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 (301, 302, 321, 322).

Recommendations for treatment and control of scabies in health care institutions have been published previously (301, 302, 321–325). The recommended topical scabicides include permethrin cream (5%), crotamiton (10%), or lindane (1%) lotion; resistance to lindane has been reported (321, 324). Single-dose oral ivermectin has recently been shown to be an effective therapy for scabies (313, 325, 326), but has not received Federal Drug Administration approval for this purpose.

Most infested health care workers have typical scabies with low mite loads (311, 327); a single correct application of a scabicide is adequate and immediately decreases the risk of transmission (316–318, 328–331). If personnel remain symptomatic after initial treatment, a repeat application of scabicide may be needed in 7–10 days. Persistent symptoms likely represent newly hatched mites rather than new infestation. Patients with crusted scabies may require repeated treatments and should be observed for recurrence of the mite infestation (301, 302, 306, 321). Personnel who are exposed to scabies, but lack signs of infestation, do not require prophylactic treatment with scabicides.

Restrictions from patient care are indicated for personnel infested with scabies until after they receive initial treatment. They should be advised to report for further evaluation if symptoms do not subside.

b. Pediculosis. Pediculosis infestation is caused by three species of lice: *Pediculus humanus capitus* (human head louse), *Pediculus humanus corporis* (human body louse), or *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, while not common, has occurred (301).

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 (301, 322, 332). The drugs recommended for treatment include permethrin cream 1%, pyrethrins with piperonyl butoxide, malathion 0.5%, or lindane 1% (323–325, 332). 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 infested with pediculosis until after they receive initial treatment. If symptoms do not subside following initial treatment, they should be advised to report for further evaluation.

17. Staphylococcus aureus Infection and Carriage

Staphylococcal carriage and infection occur frequently in humans. In hospitals the most important sources of *S. aureus* are infected and colonized patients. Previously, methicillin-susceptible (but penicillin-resistant) *S. aureus* (MSSA) accounted for most staphylococcal infections. However, in recent years, methicillin-resistant *S. aureus* (MRSA) has accounted for approximately 80% of all *S. aureus* isolates reported to the National Nosocomial Surveillance System (333–335). The epidemiology of MRSA does not appear to differ from that of MSSA, except that outbreaks of MRSA tend to occur more frequently among elderly or immunocompromised patients or among patients with severe underlying conditions (333, 336).

Nosocomial transmission of *S. aureus* occurs primarily via the hands of personnel, which can become contaminated by contact with the colonized or infected body sites of patients (333, 337). Hospital personnel who are infected or colonized with *S. aureus* also can serve as reservoirs and disseminators of *S. aureus* (338–341) and infected dietary personnel have been implicated in staphylococcal food poisoning (342). The role of contaminated environmental surfaces in transmission of *S. aureus* remains controversial, although heavy contamination of fomites may facilitate transmission to patients via personnel hands (333).

The incubation period for *S. aureus* infections varies by type of disease: foodborne illness is 30 minutes to 6 hours; bullous impetigo is 1–10 days; toxic-shock syndrome is usually 2 days; and other types of infections it is variable (343).

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 (333). 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 (339). Nasal carriers with upper respiratory symptoms can disseminate the organism more effectively (339). Carriage of *S. aureus* in the nares has been shown to correspond to hand carriage (334) 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 cultures 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 is eradicated (333, 338, 344–346).

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 (345, 347–358). Resistant *S. aureus* strains have emerged following the use of the above oral or topical antimicrobial agents for eradication of *S. aureus* colonization (16, 202, 345, 349, 359–361). Thus, antimicrobial treatment to eradicate carriage may be best if limited to personnel carriers who are 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 (1).

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

Group *A Streptococcus* (GAS) has been transmitted from infected patients to health care personnel following contact with infected secretions (362–364), and the infected personnel have subsequently developed 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 infections (365–371) and foodborne transmission of GAS causing pharyngitis (372). In these outbreaks GAS carriage was documented in the pharynx (364, 367, 373), the skin (364, 365), the rectum (364, 370), and the female genital tract of the infected personnel (364, 369, 374).

The incubation period for GAS pharyngitis is 2–5 days, and is 7–10 days for impetigo. The incubation period is variable for other GAS infections (375).

Culture surveys to detect GAS carriage among personnel are not warranted unless personnel are epidemiologically linked to cases of nosocomial infection (373). In instances where thorough epidemiologic investigation has implicated personnel in nosocomial transmission, cultures may be obtained from skin lesions, the pharynx, rectum, and vagina; GAS isolates obtained from personnel and patients can be serotyped to determine strain relatedness (368). Treatment of personnel carriers needs to be individualized 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 over long periods of time (364–366, 369). 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 (364, 369, 370, 374). Nosocomial transmission of GAS to personnel can be prevented by adherence to Standard Precautions or other transmission-based precautions as needed (1).

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 (376, 377). In some areas in the USA, the incidence and prevalence of multidrug-resistant *Mycobacterium tuberculosis* (MDR–TB) also have increased, and nosocomial MDR–TB outbreaks have occurred (378–384). The increased risk of occupational acquisition of TB by health care personnel has been reported for decades and it dramatically decreased following the introduction of effective antituberculous drugs (385, 386). Skin-test conversion rates among health care personnel following routine skin testing have ranged from 0.11 % to 10%. Among health care personnel with known exposure to an infectious TB patient or involved in prolonged nosocomial outbreaks of TB, the skin-test conversion rates have ranged from 18% to 55% (378–380, 383, 384, 386–393).

The transmission of TB in health care facilities has been primarily caused by incomplete implementation of recommended TB infection control measures (388). In 1994, CDC published detailed recommendations for the prevention of transmission of TB in health care settings, Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health Care Facilities, 1994 (377). A summary of the recommendations pertaining to personnel health follow.

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 (377, 394). A risk assessment should include the following: (a) Review of the community TB profile; (b) review of the number of TB patients 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 antitubercular therapy regimens; (f) an observational review of TB infection control practices; and (g) evaluation of the function and maintenance of environmental controls (377).

Transmission of TB can be minimized by developing and implementing an effective TB-control program based on a hierarchy of controls, namely, (a) administrative controls, (b) engineering controls, and (c) personal respiratory protection (377, 379, 381, 386, 388, 394, 395).

b. TB screening program. A tuberculosis 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.

Baseline PPD testing of all personnel [including personnel with a history of Bacille Calmette-Guérin vaccination (BCG)] during their pre-employment physical examination or when applying for hospital privileges will identify personnel who have been previously infected. For the baseline testing a two-step procedure can be used to minimize the likelihood of confusing reactivity from an old infection (boosting) with reactivity from a recent infection (conversion). Criteria used for interpretation of a PPD test reaction may vary depending on the (a) purpose (diagnostic or epidemiologic) of the test; (b) prevalence of TB infection in the population being tested; (c) immune status of the host; and (d) previous receipt of TB immunization. Detailed recommendations have been published for performing and interpreting skin tests (377, 396, 397).

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

It is also important to obtain an initial chest x-ray on 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 x-ray examinations on asymptomatic PPD test-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 x-ray 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 convert their PPD test; those persons, if infected, are at increased risk of developing active TB (e.g., HIV-infected or otherwise severely immunocompromised persons).

d. Management of personnel after exposure to TB. It is important to perform PPD tests on personnel as soon as possible after TB exposures are recognized. Such immediate PPD testing establishes a baseline by which to monitor subsequent PPD tests. A PPD test, performed 12 weeks after the last exposure, will indicate if 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 evaluated for active TB. If active TB is not diagnosed, preventive therapy should be considered (377).

e. Preventive therapy. For workers with positive PPD tests who were likely exposed to drug-susceptible TB, preventive therapy with isoniazid is indicated, unless there are contraindications to such therapy (377, 397). Alternative preventive regimens have been proposed for persons who have positive PPD tests following exposure to drug-resistant TB (398).

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) 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 time period and that their sputum AFB smears continue to be negative.

Work restrictions are not necessary for personnel receiving preventive treatment for latent TB (positive PPD test 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 they develop symptoms suggestive of TB.

g. Considerations for Bacille Calmette-Guérin 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 (399, 400), the role of BCG vaccination in the prevention and control of TB in the country has been re-evaluated (401). 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 meta-analyses of 18 and 26 BCG studies, respectively, indicate that the efficacy of BCG vaccine in preventing serious TB in children is high (>80%) and suggested 50% efficacy in adults (399, 400); however, the protective efficacy of the vaccine in adolescents and adults, including health care personnel and HIV-infected children and adults, has not been determined (401).

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 not been successful in controlling nosocomial transmission (401). BCG vaccination often results in local adverse effects (such as muscular soreness, erythema, purulent drainage, 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 (402–404). 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 (405–408), BCG vaccination is not recommended for immunocompromised personnel (401).

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* (401). PPD-test reactivity caused by BCG vaccination wanes with time (409–411) and is unlikely to persist >10 years after vaccination in the absence of infection with *M. tuberculosis* (409, 410). After a person has been vaccinated with BCG, the presence or size of a PPD-test reaction cannot be used as a predictor of BCG vaccine efficacy in the vaccine recipient (412, 413), or as a determinant as to whether the reaction is caused by infection with *M. tuberculosis* or the prior BCG vaccination (414). However, a BCG-vaccinated person who has a PPD test reaction of ≥10 mm induration is considered infected with TB, especially if the vaccinee is a contact of a person with infectious TB, is from a country with high prevalence of TB, or is continually exposed to populations in which the prevalence of TB is high (401).

20. Vaccinia (Smallpox)

Because of the effective use of smallpox vaccine (vaccinia virus vaccine), the World Health Organization declared 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 (2–21 days after vaccination); thus, susceptible persons may acquire vaccinia from a recently vaccinated person (415–418). Covering the vaccination site and washing hands following contact with the vaccination site (including bandages) will prevent transmission. Recently, recombinant vaccinia viruses have been engineered. There is a theoretical 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 the recombinant vaccine recipients. Infections also have been reported among laboratory personnel who handle viral cultures or materials contaminated with vaccinia or recombinant viruses (16, 155).

Smallpox vaccination 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 (7, 16). Personnel who receive the vaccine may continue to have contact with patients if the vaccination site is covered and handwashing is maintained (16).

21. Varicella

Nosocomial transmission of varicella-zoster virus (VZV) is well recognized (419–430). 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. However, certain persons are at higher risk for severe disease and secondary complications; they include pregnant women, premature infants born to varicella-susceptible mothers; infants born at <28 weeks gestation or weighing 1000 grams, regardless of maternal immune status; and immunocompromised patients (11). During 1990–1994, while <5% of varicella cases occurred among adults 20 years old, they accounted for 55% of varicella-related deaths.

The incubation period for varicella is usually 14 to 16 days, but may be from 10 to 21 days after exposure. In persons who receive postexposure varicella-zoster immune globulin the incubation period may be up to 28 days after exposure. Transmission of infection may occur from 2 days before onset of rash and usually up to 5 days after rash onset, although, in immunocompromised persons transmission may occur during the period of eruption of lesions (431).

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 (11, 432). Personnel with localized zoster may not 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 (428).

VZV is transmitted by the contact with infected lesions and, in hospitals, airborne transmission from patients with varicella or zoster to susceptible persons who had no direct contact with the infected patient has occurred (432–436). Adherence to Airborne and Standard Precautions when caring for patients with known or suspected VZV infection can reduce the risk of transmission to personnel (1). Management of clusters of VZV infection in health care settings also generally includes (a) isolation of patients with varicella and of exposed susceptible patients (1); and (b) control of air flow (negative pressure) in isolation rooms (435–437).

a. Varicella screening and vaccination. Serologic tests have been used to assess the accuracy of reported histories of chickenpox (299, 429, 438–440). In adults, a history of varicella is highly predictive of serologic immunity (97% to 99% seropositive). The majority of 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 (7, 11).

A variety of methods have been used for detecting of varicella antibody, but a commercially available latex agglutination test will provide prompt, sensitive and specific serologic results at a reasonable cost. Routine testing for varicella immunity following 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 following exposures may be warranted. In addition, vaccinated persons who are exposed to varicella but lack antibody may be retested in 5–6 days to determine if they are antibody positive after the second test and, therefore, unlikely to develop varicella (11).

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 (11, 293, 441, 442). Effective varicella vaccination programs require two doses of vaccine to achieve high seroconversion rates in adults (441); 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 7–10 years after vaccination (11). Cases of varicella have occurred among vaccinees following exposure to wild-type virus (''breakthrough infection''). Data from vaccine trials indicate that 1% to 4% of vaccine recipients per year develop chickenpox, depending on the vaccine lot and interval following vaccination (7, 11). However, vaccinated persons have milder disease (e.g., afebrile; a mean of 50 skin lesions which are often not vesicular; and shorter duration of illness) compared with unvaccinated individuals (e.g., febrile with several hundred vesicular lesions) (443, 444), 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 following vaccination of 2141 children between 1981 and 1989 in 10 different trials (7) 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 (7).

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 developed a non-localized rash (median, 5 lesions) after the first injection, and 0.9% of adolescents and adults developed a non-localized 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 and possibly related to the occurrence of rash following vaccination (445, 446). Tertiary transmission of vaccine virus to a second healthy sibling of a vaccinated leukemic child has also occurred (99). 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 develop a varicella-like rash following 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 develop 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 infective 10 to 21 days after exposure and exclusion from duty is indicated from the 10th day after the first exposure through the 21st day after the last exposure, or if varicella occurs, until all lesions dry and crust (Table 3) (248).

If vaccinated health care personnel are exposed to varicella, they may be serotested immediately after exposure to assess the presence of antibody (442). 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, and/or rash) develop.

Vaccination should be considered for exposed unvaccinated health care personnel without documented immunity (430, 442). However, because the efficacy of postexposure vaccination is unknown, persons vaccinated following an exposure should be managed as previously recommended for unvaccinated persons.

The use of postexposure varicella zoster immune globulin (VZIG) is not recommended for routine use among immunocompetent health care personnel (11). 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 (11, 447). Postexposure use of acyclovir may be effective and less costly than the use of VZIG in some susceptible persons (447). However, additional data concerning the efficacy of acyclovir for postexposure prophylaxis are needed before such use can be recommended (7, 11, 430, 448).

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 (449).

a. Influenza. Nosocomial transmission of influenza has been reported in acute and long-term care facilities (450–455). Transmission has occurred from patients to health care personnel (452, 454), from health care personnel to patients (456), and among health care personnel (455, 457–462) .

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 (19, 279, 463). While the extent of transmission by virus-contaminated hands or fomites is not known, it is not the primary mode of transmission (463).

The incubation period of influenza is usually 1–5 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 up to 7 days after illness onset (464–466). Persons at greatest risk for influenza-related complications include (a) persons ≥65 years of age; (b) residents of nursing homes and other chronic-care facilities; (c) persons with chronic pulmonary or cardiovascular conditions; and (d) persons with diabetes mellitus (15). Adherence to Droplet Precautions may prevent nosocomial transmission (1).

Administration of influenza vaccine to health care personnel, including pregnant women (7), before the beginning of each influenza season can help to (a) reduce the risk of influenza infection to health care personnel; (b) prevent transmission of influenza from personnel to persons at high risk of complications; and (c) reduce personnel absenteeism during community outbreaks. Innovative methods may be needed to increase influenza immunization rates among health care personnel (467). 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 (468).

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 following personnel vaccination or, in unvaccinated personnel, for the duration of influenza activity in the community (15, 449, 469, 470).

b. Respiratory Syncytial Virus (RSV). Nosocomial transmission of 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 develop more severe disease. Because RSV infection can also occur simultaneously with other respiratory viruses, it may go unrecognized (471, 472). Nosocomial transmission has been reported most frequently among newborn and pediatric patients (473, 474), but outbreaks associated with substantial morbidity and mortality have been reported among adults in bone marrow transplant centers (475), intensive care units (476), and long-term care facilities (477, 478).

RSV is present in large numbers in the respiratory secretions of symptomatic persons infected with the virus and can be transmitted directly via large droplets during close contact with such persons, or indirectly via hands or fomites that are contaminated with RSV. Hands can become contaminated through handling of infected persons' respiratory secretions or contaminated fomites, and transmit RSV by touching the eyes or nose (449). The incubation period ranges from 2–8 days; 4–6 days is most common. In general, infected persons shed the virus for 3–8 days, but young infants may shed virus for as long as 3–4 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 (479, 480).

E. 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 infection with viruses such as parvovirus, varicella, and rubella has been associated with abortion, congenital anomaly, and mental retardation. Other diseases in which the infectious agent may be transmitted to the fetus include cytomegalovirus, 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 of 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 prior to 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 (480–482).

F. 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 (3, 16, 48, 144, 155, 235, 483, 484). Furthermore, newer technologies that require the use of large and/or concentrated specimens may further increase the risk of occupationally acquired infections among laboratory personnel (485).

In a review of laboratory-acquired infections from 1950–1974 >4000 laboratory associated infections were documented in the United States (483) 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 due to a wide variety of other pathogens (155, 483, 484). More recently, viral agents have accounted for a larger proportion of laboratory associated infections than have bacterial infections (484–489).

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 (3, 4, 485, 490–492).

In addition to biosafety precautions, preventive measures (e.g., immunizations and postexposure prophylaxis) also may 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 biosafety procedures to prevent transmission of infectious agents.

G. Emergency Response Personnel

Emergency medical technicians, firemen, 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 U.S.C. 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 meningococcemia. CDC published a list of diseases for which emergency response employees must be informed of an exposure (493).

H. Latex Hypersensitivity

Since the introduction of Universal Precautions, the use of latex gloves has become commonplace in health care settings (494, 495). The increased use of latex gloves has been accompanied by increasing reports of allergic reactions to natural rubber latex among health care personnel (496–501).

Natural rubber latex is a combination of heat and water-soluble proteins derived from the tree *Hevea braziliensis*. However, total protein concentrations and allergenicity are not always directly correlated (502), suggesting that total protein concentrations are not necessarily a measure of the allergenic properties of latex gloves. Latex gloves may be labeled ''hypoallergenic'', but this designation refers to nonlatex additives in gloves and does not reflect reduced allergenicity to latex (503). In one study, nearly 50% (11/24) of the lots of hypoallergenic gloves tested had measurable latex allergen (504). The FDA has proposed labeling of all the medical devices that contain natural rubber latex (505). Also, the total protein content of latex gloves may vary considerably from brand to brand and lot to lot (502, 504). Currently, the amount of latex allergen exposure required to produce sensitization or to elicit reactions in previously sensitized persons is unknown.

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. Powdered gloves have higher levels of these proteins than powder-free gloves. 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 individuals (506); 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 (497). 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.

Reactions to latex gloves may be localized or systemic and include dermatitis, conjunctivitis, rhinitis, urticaria, angioedema, asthma, and anaphylaxis (507–510). The majority of 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 (495, 500, 511–513). It may be 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 (496, 514); only a subset of health care personnel reporting glove-associated skin irritation will have immunoglobulin E (IgE) antibodies specific for latex (511, 515–517).

In contrast, systemic reactions to natural rubber latex, including urticaria, are mediated by anti-latex IgE antibodies (507, 518, 519) 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 (518, 520–522). Asthmatic responses to latex may occur early (<8 hours) or late (>8 hours) following exposure (523–525).

Local reactions (i.e., irritant or allergic contact dermatitis) account for the majority of reported reactions among health care personnel (496, 499). The risk of progression from localized to systemic reactions is unknown.

a. Prevalence and risk factors. In studies of health care personnel, the reported prevalence of IgE-mediated allergy to latex vary considerable ranging from 2.9%–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 (516, 517, 520, 526, 527). The prevalence detected in some studies also has been biased by enrollment or testing of only symptomatic personnel (497, 501). However, it is estimated that a minority of health care personnel, even if symptomatic, seek medical evaluation or treatment for latex-allergic conditions. 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 (499, 527). 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/clerical workers to 38% (5/13) among dental residents/assistants (499). 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 a four fold higher prevalence than did general ward nurses (5.6% vs 1.2%) (527). 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 (528).

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) (496, 514, 516, 517, 520, 526, 527), nonwhite race (79, 526), elevated total IgE levels (517), allergy to cosmetic powders or foods (529), years or status (full vs part-time) of employment, and frequency and/or duration of glove use (496, 514, 520, 527). Coexistent allergy to certain fruits (e.g., bananas [(530, 531)], avocados [(532, 533), pears, and chestnuts (534)) also has been described in latex-allergic health care personnel.

Skin irritation, eczematous dermatitis (514, 527) (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/identification. Diagnosis of personnel with latex allergy 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-medicated allergy (514, 517).

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 (SPT) 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 have been associated with adverse outcomes (535), suggesting that these nonstandardized reagents may not be safe for routine use. In Europe, where a standardized SPTallergen has been developed, SPT has been used successfully.

Currently, only one immunoassay has been FDA approved for detection of latex-specific IgE antibodies in blood. The FDA has recommended that this assay be used as a confirmatory test, rather than screening, for persons in whom latex allergy is suspected, based on clinical history and findings. Levels of detectable antibody appear to be associated with symptoms (497, 517), but, as with other allergens, the correlation between serum concentrations of latex-specific IgE antibodies and symptom severity is unpredictable (497, 514). Clinical screening, in which the worker is questioned about allergy to latex products and risk factors for latex allergy, may help to identify those in whom further diagnostic testing should be considered.

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 vinyl or cloth liners) 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 its 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 co-workers 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 to more effective and less costly than either laminar flow or high-efficiency particulate air-filtered glove-changing stations in reducing latex aeroallergens (528). For personnel with systemic manifestations to latex, workplace restriction or reassignment may be necessary.

I. The Americans With Disabilities Act

The Americans with Disabilities Act (ADA) provides guidelines for hiring and placing employees with disabilities as defined in the Act (536–539). 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 this would create undue hardship because of significant difficulty or expense.

The provisions of the ADA need to be incorporated into infection 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 of 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. Upon 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 need also 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

A. Introduction

In this document, the term health care personnel refers to all the 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, nursing assistants, laboratory personnel, mortuary 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 (e.g., volunteer, dietary, housekeeping, maintenance, and clerical personnel) but potentially exposed to infectious agents.

As in previous CDC guidelines, each recommendation is categorized on the basis of existing scientific data, theoretical rationale, applicability, and 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 based on 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 theoretical rationale, or definitive studies applicable to some, but not all, hospitals.

No Recommendation, Unresolved Issue. Practices for which insufficient evidence or consensus regarding efficacy exists.

B. Elements of a Personnel Health Service for Infection Control

1. Coordinated Planning and Administration

a. Coordinate policy-making and planning among the hospital administration, personnel health service, infection control personnel, clinical services and 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) other work-related infections and exposures; and (4) when appropriate, results of epidemiologic investigations. *Category IB*

c. Develop protocols to assure coordination between the personnel health program and the infection control program of the institution. *Category IB*

2. Placement evaluation

a. Before personnel begin duty or are given a new work assignment, obtain their health inventories. Include in the inventories the following: (1) immunization status or history of vaccine preventable diseases (e.g., chickenpox, measles, mumps, rubella, hepatitis B); (2) history of any conditions that may predispose personnel to acquiring or transmitting infectious diseases (e.g., immunosuppressive condition or therapy, tuberculosis, dermatologic conditions, chronic draining infections or open wounds, or chronic infections). *Category IB*

b. For infection control, perform directed physical and laboratory examinations on personnel, as may be determined from the results of the health inventory. Include examinations to detect conditions that might increase the likelihood of transmitting disease to patients, or unusual susceptibility to infection, and 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 (540). *Category IB*

e. Conduct routine screening for tuberculosis by using the intradermal (Mantoux), intermediate strength (5 TU) 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. Include the infection control aspects of personnel health and the proper use of the personnel health service in the initial job orientation and ongoing in-service education of personnel. *Category IB*

(1) Ensure that the following topics are included in the initial training on infection control: (a) handwashing; (b) modes of transmission of infection and importance of complying with standard and isolation precautions; (c) 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 of time (e.g., a cough that persists for >2 weeks, gastrointestinal illness, or febrile illness with fever of >103 °F lasting more than 2 days) and hospitalizations resulting from febrile or other contagious diseases; (d) tuberculosis control; (e) importance of complying with Standard Precautions and reporting exposure to blood and body fluids to prevent transmission of bloodborne pathogens; (g) importance of cooperating with infection control personnel during outbreak investigations; and (h) importance of personnel screening and immunization programs. *Category IB*

(2) Ensure that all personnel know that if they have medical conditions (e.g., immunosuppression) or receive medical treatment that render them more susceptible to or more likely to transmit infections, they can follow recommendations to greatly reduce their risk for transmitting or acquiring infections, e.g., request for work reassignment. *Category IB*

b. Make specific written policies and procedures for control of infections in health care personnel readily available. *Category IB*

c. Provide personnel, annually, and whenever the need arises, with in-service training and education on infection control that are 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. *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. *Category IB*

b. Establish a readily available mechanism for personnel to obtain advice about illnesses they may acquire from or transmit to patients. *Category IB*

c. Evaluate job-related and community-acquired illnesses or important exposures and postexposure prophylaxis, when indicated. *Category IB*

d. 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 therapeutic or prophylactic management for illnesses caused by or following exposures to transmissible infections. 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 data base, preferably computerized, that allows tracking of personnel immunizations, screening tests, and assessment of trends of infections and diseases in personnel. Copies of these records are to be available to the individual. *Category IB*

d. Periodically review and assess 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 (23, 24). *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 (1) and other guidelines (377). *Category IB*

D. Immunization of Health Care Personnel, General Recommendations

1. Ensure that persons administering immunizing agents are: (a) familiar with the general ACIP recommendations and recommendations on immunizing adults; (b) well informed about indications, storage, dosage, preparation, side effects, and contraindications for each of the vaccines, toxoids, and immune globulins used (6, 7, 22); and (c) kept updated on professional organization recommendations regarding vaccination of health care personnel (Tables 1 and 2). *Category IB*

2. 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*

3. Ensure that persons administering immunizing agents are familiar with state and local regulations regarding vaccinations for health care personnel. *Category IB*

4. Formulate a written comprehensive policy on immunizing health care personnel. *Category IB*

5. Develop a data base of employee specific information on history of vaccine preventable diseases and status of vaccine administration. *Category IB*

6. Develop a list of needed immunizations for each employee during screening and an individual plan to provide the necessary vaccines. *Category IB*

7. In the absence of a known occupational exposure, provide personnel with on-site service 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*

8. Provide vaccine to personnel who may have occupational exposure to uncommon diseases 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 follow-up 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) removal of personnel from contact with patients; and (c) clearance for work after an infectious disease 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

a. Ensure that health care personnel are familiar with precautions to prevent occupational transmission of bloodborne pathogens (1, 4, 26, 27, 35). *Category IA*

b. Follow state and federal guidelines and strategies for determining the need for work restrictions for health care personnel infected with bloodborne pathogens (43). *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 (7, 8, 36). *Category IA*

(2) Before vaccinating personnel, do not routinely perform serologic screening for hepatitis B vaccine unless the health care organization considers screening cost-effective or the potential vaccinee requests it (7). *Category IA*

(3) Conduct post vaccination 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) (7) (Tables 1 and 4). *Category IB*

(5) Test staff in chronic dialysis centers who do not respond to the hepatitis B vaccine for hepatitis B surface antigen (HBsAg) and antibody to hepatitis B surface antigen (anti-HBs) semi-annually (541). *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 positive (Table 6). *Category IA*

(7) Follow current recommendations for postexposure prophylaxis following percutaneous or mucous membrane exposure to blood and body fluids that is known or suspected to be at high risk for being HBsAg-positive (Table 4) (36). *Category IA*

b. Hepatitis C. (1) Do not administer immune globulin (IG) to personnel who have exposure to blood or body fluids positive for antibody to hepatitis C virus (33, 69). *Category IB*

(2) Consider implementing policies for postexposure follow-up for health care personnel who have had a percutaneous or mucosal exposure to blood containing antibody to hepatitis C virus at baseline and 6 months (69). *Category IB*

c. Human Immunodeficiency Virus (HIV). Follow current recommendations for postexposure prophylaxis following percutaneous or mucocutaneous exposure to suspected or known HIV-infected blood or body fluids containing blood (29, 76). *Category IB*

2. Conjunctivitis

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

3. Cytomegalovirus (CMV)

a. Do not restrict personnel from work who contract illnesses suspected or proven to be due to CMV (109). *Category IB*

b. Educate all patient-care personnel about careful handwashing and exercising care to prevent their body fluids from contacting other persons to reduce their risk of transmitting infections such as CMV to patients or other personnel (89, 123). *Category IA*

c. 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). *Category IA*

4. Diphtheria

a. Encourage vaccination with tetanus and diphtheria toxoid (Td) every 10 years for health care personnel (7, 17) (Table 1). *Category IB*

b. Obtain nasopharyngeal cultures from exposed personnel and monitor for signs and symptoms of diphtheria for 7 days (156). *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 personnel who have not been vaccinated within the previous 5 years (17, 156) (Table 1). *Category IB*

d. Repeat nasopharyngeal cultures of personnel found to have positive cultures at ≥2 weeks following completion of antimicrobial therapy. Repeat antimicrobial therapy if personnel remain culture positive (156). *Category IB*

e. Exclude exposed personnel and those identified as asymptomatic carriers from duty until antimicrobial therapy is completed *and* two nasopharyngeal cultures obtained ≥24 hours apart are negative (156) (Table 3). *Category IB*

5. Gastroenteritis

a. Vaccinate and provide booster doses of vaccine, following published guidelines, to microbiology laboratory personnel who work with *Salmonella typhi* on a regular basis (144, 155). *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) that may be accompanied by other symptoms (such as fever, abdominal cramps, or bloody stools), from contact with patients or food-handling (1, 163) (Table 3). *Category IB*

c. Consult local and state health authorities regarding regulations for the exclusion of patient-care personnel or food-handlers with enteric infections from contact with patients or food-handling, respectively. *Category IB*

d. Determine the etiology of gastrointestinal illness among personnel who care for patients at high risk of severe disease. *Category IB*

e. Allow personnel infected with enteric pathogens to return to work after their symptoms resolve if local regulations do not require exclusion from duty for designated pathogens for specified time periods or until negative cultures are available. *Category II*

f. Ensure that personnel, including those who are immunocompromised, 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 (160, 542). *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 (HAV)

a. Do not routinely administer inactivated hepatitis A vaccine to health care personnel. Susceptible personnel working in areas where hepatitis A is highly endemic should be vaccinated to prevent acquisition of community acquired infection (7, 196). *Category IB*

b. Do not routinely administer immune globulin (IG) as prophylaxis for personnel providing care or who are exposed to a patient with hepatitis A (196). *Category IB*

c. Administer IG (0.02 ml/kg) to personnel who have had oral exposure to fecal excretions from a person acutely infected with hepatitis A virus (196) (Table 1). *Category IA*

d. In documented outbreaks involving transmission of HAV from patient to patient or from patient to health care worker, use of IG in persons with close contact with infected persons may be indicated. Contact the local health department regarding control measures (Table 1). *Category IB*

e. Exclude personnel who have acute hepatitis A from work until 1 week after the onset of jaundice (Table 3). *Category IA*

7. Herpes Simplex Virus

a. Exclude personnel with primary or recurrent orofacial herpes simplex infections from the care of high-risk patients, including newborns, intensive care unit patients, patients with severe burns or eczema, or severely immunocompromised patients, until the lesions are crusted (201, 210) (Table 3). *Category IB*

b. Exclude personnel with herpes simplex infections of the fingers or hands (herpetic whitlow) from contact

with patients until their lesions are healed (206). *Category IB*

8. Measles

a. Ensure that all personnel have documented immunity to measles.

(1) Consider administering measles vaccine* to persons born in 1957 or later unless they have evidence of measles immunity. *Category IA*

(2) Administer measles vaccine* to personnel born before 1957 if they do not have evidence of measles immunity and are at risk of occupational exposure to measles (6, 213, 225, 226) (Table 1). *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 (6, 9, 227–229, 543). *Category IA*

(4) Administer postexposure measles vaccine* to measles-susceptible personnel who have contact with persons with measles, within 72 hours after the exposure. During the period 5 days after the first exposure until 21 days after the last exposure, exclude exposed, vaccinated personnel from duty (6) (Tables 1–3). *Category IA*

b. Exclude exposed unvaccinated personnel from duty from the 5th day after the first exposure until the 21st day after the last exposure to measles, regardless of whether they receive postexposure vaccine, if they do not have documented immunity to measles (9, 229) (Table 3). *Category IB*

c. Exclude personnel who develop measles from patient contact for 4 days after rash develops or for the duration of their acute illness, whichever is longer (9, 229) (Table 3). *Category IB*

9. Meningococcal Disease

a. Do not administer routinely meningococcal vaccine to health care personnel (13). *Category IB*

b. Consider vaccination of laboratory personnel who are routinely exposed to *Neisseria meninigitidis* in solutions that may be aerosolized (13) (Table 1). *Category IB*

c. Immediately offer antimicrobial prophylaxis to personnel who have had any of the following types of contact with a patient with meningococcal disease prior to administration of antibiotics: (a) Intensive, unprotected (i.e., without the use of proper precautions), close, face-to-face contact with a patient with meningococcal disease; (b) contact with the patient's oropharyngeal secretions; or (c) a needlestick from a patient with meningococcal disease (13) (Table 1). *Category IB*

d. Do not routinely give quadrivalent A,C,Y, W-135 meningococcal vaccines for postexposure prophylaxis (13) (Table 1). *Category II*

e. Administer meningococcal vaccine to personnel (and other persons likely to have contact with infected persons) to control Serogroup C outbreaks following consultation with public health authorities (13). *Category IB*

f. Consider preexposure vaccination of personnel who routinely handle soluble preparations in *N. meningitidis* (13). *Category II*

10. Mumps

a. Administer mumps vaccine* to all personnel without documented evidence of mumps immunity unless otherwise contraindicated (7, 250) (Table 1). *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 (10). *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 (7, 544) (Table 3). *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 (264, 265) (Table 6). *Category IB*

b. Do not routinely exclude pregnant personnel from caring for patients with parvovirus B19. *Category IB*

12. Pertussis

a. Do not administer whole-cell pertussis vaccine to personnel (Table 1). *Category IB*

b. *No Recommendation* for routine administration of an acellular pertussis vaccine to health care personnel. *Unresolved Issue*

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 cultures or other tests are negative for pertussis and the clinical course is suggestive of an alternate diagnosis (277, 278) (Table 1). *Category II*

d. Exclude personnel who develop symptoms (e.g., unexplained rhinitis or acute cough) following known exposure to pertussis from patient care until 5 days after the start of appropriate therapy (Table 3). *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; or (2) laboratory personnel who handle specimens that might contain wild polioviruses or who do cultures to amplify virus (19) (Table 1). *Category IA*

b. For personnel above, 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 (IPV) rather than oral polio vaccine (OPV) for completion of the series (19) (Table 1). *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 pre-exposure vaccination to personnel who work with rabies virus or infected animals in rabies diagnostic or research activities with rabies virus (3, 20) (Table 1). *Category IA*

b. After consultation with public health authorities, give a full course of anti-rabies treatment to personnel who either have been bitten by a human with rabies or have scratches, abrasions, open wounds, or mucous membranes contaminated with saliva or other potentially infective material from a human with rabies. In those previously vaccinated individuals, postexposure therapy is abbreviated to only include a single dose of vaccine on days 0 and 3 (285–287) (Table 1). *Category IB*

15. Rubella

a. Vaccinate all personnel without documented immunity to rubella with

* (Measles-mumps-rubella [MMR] trivalent vaccine 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.)

* (Measles-mumps-rubella [MMR] trivalent vaccine 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.)

rubella vaccine.* (7, 300) (Table 1) *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 (229). *Category IB*

d. Do not administer rubella vaccine* to susceptible personnel who are pregnant or might become pregnant within 3 months of vaccination (7) (Table 1). *Category IA*

e. Administer rubella vaccine* in the postpartum period to female personnel not known to be immune. *Category IA*

f. Exclude personnel who are exposed to rubella from duty from the 7th day after the first exposure through the 21st day after the last exposure (Table 3). *Category IB*

g. Exclude personnel who develop rubella from duty until 7 days after the beginning of the rash (Table 3). *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 (302). *Category IA*

b. Evaluate exposed personnel for louse infestation and provide appropriate therapy for confirmed pediculosis (325). *Category IA*

c. Do not routinely provide prophylactic scabicide treatment of personnel who have had contact with patients or other persons with scabies (301, 302, 308, 321, 329) (Table 1). *Category II*

d. Do not routinely provide prophylactic pediculicide treatment of personnel who have had contact with patients or other persons with pediculosis. *Category II*

e. Exclude personnel with either confirmed or suspected scabies or lice infestation from contact with patients until after they receive appropriate initial treatment or are found not to have scabies or pediculosis, respectively (302) (Table 3). Exclude personnel with confirmed scabies from the care of immunocompromised patients until after the second treatment, unless they wear gowns and gloves for patient contact. *Category IB*

17. Staphylococcal Disease or Carriage

a. Obtain appropriate cultures and exclude personnel from patient care or food handling if they have a draining lesion suspected to be due to *Staphylococcus aureus*, until the infections have been ruled out or personnel have received adequate therapy and their infections have resolved (Table 3). *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 the person is responsible for disseminating the organism in the health care setting (Table 3). *Category IB*

18. Group A Streptococcal Disease or Carriage

a. Obtain appropriate cultures, and exclude personnel from patient care or food handling if they have draining lesions that are suspected to be due to Streptococcus, until streptococcal infection has been ruled out or the worker has received adequate therapy for 24 hours (364–366, 369) (Table 3). *Category IB*

b. Do not routinely exclude personnel with suspected or confirmed carriage of group A *Streptococcus* from patient care or foodhandling unless it is shown epidemiologically that the person is responsible for disseminating the organism in the health care setting (Table 3). *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 (377). *Category IB*

b. TB Screening Program. (1) Include all health care personnel who have potential for exposure to *M. tuberculosis* in a purified protein derivative (PPD) skin-test program (377). *Category IA*

(2) Maintain confidentiality regarding the medical condition of personnel. *Category IA*

(3) Administer PPD tests by using the intracutaneous (Mantoux) method of administration of 5 tuberculin units (0.1 ml) of purified protein derivative (377, 397). *Category IB*

(4) Do not use the Tine or other tests to administer PPD (397). *Category IB*

(5) 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 (377). *Category IB*

(6) Ensure that the administration, reading, and interpretation of PPD tests are performed by specified trained personnel. *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*. Include those with a history of BCG vaccination (377). *Category IB*

(2) Perform two-step, baseline PPD tests on newly employed health care personnel who are negative on 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 their facility (377). *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 (377). *Category IB*

d. Follow-up (Repeat) PPD. (1) Perform periodic follow-up PPD tests on all health care personnel (with negative baseline PPD test result) who have the potential for exposure to *M. tuberculosis* (377). *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 (377). *Category IB*

(3) Exempt from follow-up-PPD tests: personnel with documented history of positive baseline PPD test result or adequate treatment for tuberculosis (377). *Category IB*

(4) Interpret follow-up-PPD test results as outlined in the Guidelines for Preventing the Transmission of Mycobacterium tuberculosis in Health-Care Facilities, 1994 (377). *Category IB*

(5) 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 (377). *Category IB*

(b) Perform chest x-ray examinations on personnel with a positive PPD-test result as part of the evaluation for active TB (377). *Category IB*

(c) Do not repeat chest x-rays unless symptoms suggestive of TB develop, if

* (Measles-mumps-rubella [MMR] trivalent vaccine 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.)

the initial chest x-ray examination is negative (377). *Category IB*

(d) 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 (377). *Category IB*

(e) Do not require routine chest x-rays for asymptomatic, PPD-negative workers (377). *Category IB*

e. Preventive therapy. 1) Offer preventive therapy to the following personnel, regardless of age, who convert 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; or (e) injecting-drug users (377, 397). *Category IB*

(2) Offer preventive therapy to personnel with positive PPD reactions who do not have the above risk factors, if they are <35 years of age (397). *Category IA*

(3) Provide preventive therapy to personnel through the occupational health service or refer them to the health department or other 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-skin test results. When the result of this PPD test is negative, administer a second test 12 weeks after the exposure (377). *Category IB*

(2) Do not perform PPD tests or chest x-rays on personnel with prior positive PPD-test results unless they have symptoms suggestive of active TB (377). *Category IB*

(3) Consider retesting immunocompromised health care personnel who are potentially exposed to M. tuberculosis at least every 6 months (377). *Category II*

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 there have been three consecutive sputum smears collected on different days negative for acid-fast bacilli (AFB). After personnel return to work, obtain periodic documentation from their health care provider that effective drug therapy has been maintained for the recommended time period and that sputum smears remain AFB negative (377) (Table 3). *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 smears are AFB negative (377). *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 and/or larynx. *Category IB*

(5) Do not restrict personnel from their usual work activities if they are receiving preventive therapy because of positive PPD tests (377). *Category IB*

(6) Do not exclude personnel from the workplace who have positive PPD-test results and cannot take or do not accept or complete a full course of preventive therapy. Instruct them to seek prompt evaluation if symptoms suggestive of TB develop (377). *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 (377). *Category II*

(2) Upon 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 (377). *Category II*

i. BCG 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 (401). 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 (401). *Category II*

(2) Counsel health care personnel who are being considered to receive 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 multi-drug resistant TB infections; (d) the risks of drug toxicity with multi-drug prophylactic regimens; and (e) the fact that BCG vaccination interferes with the diagnosis of newly acquired TB infection (401). *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 smallpox vaccination is current to within 10 years for 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 humans (7, 16) (Table 1). *Category IB*

b. Consider administering vaccinia vaccine to personnel who provide clinical care to recipients of recombinant vaccinia virus vaccines (7, 16) (Table 1). *Category II*

c. Do not administer vaccinia vaccine to personnel with immunosuppression or eczema, or who are pregnant (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 they follow handwashing practices (16). *Category IB*

21. Varicella

a. Administer varicella vaccine to susceptible personnel, especially those that will have contact with persons at high risk for serious complications (7, 11) (Table 1). *Category IA*

b. Before vaccinating personnel with varicella vaccine, do not perform serologic screening for varicella of persons with negative or uncertain history of varicella, unless the institution considers it cost-effective (7). *Category IB*

c. Do not routinely perform post vaccination testing of personnel for antibodies to varicella (133). *Category IB*

d. *No Recommendation* for administering postexposure varicella vaccination for the protection of exposed, susceptible personnel (7). *Unresolved Issue*

e. Develop guidelines for managing health care personnel who receive varicella vaccine, e.g., consider precautions for personnel who develop a rash following their 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. *Category IB*

f. Develop written guidelines for postexposure management of vaccinated or susceptible personnel who are exposed to wild-type varicella (7). *Category IB*

g. Exclude personnel from work who have onset of varicella or zoster at least until all lesions have dried and crusted (1) (Table 3). *Category IB*

h. Exclude personnel from duty, following exposure to varicella or zoster, who are not known to be immune to varicella (by history or serology), beginning on the 10th day after the first exposure until the 21st day after the last exposure (7) (Table 3). *Category IB*

i. Perform serologic screening for immunity to varicella on exposed personnel who have not had varicella or are unvaccinated against varicella (7, 16). *Category IB*

j. Consider performing serologic screening for immunity to varicella on exposed, vaccinated personnel whose antibody status is not known. If the test is negative, retest 5–6 days following exposure for anamnestic response. *Category IB*

k. 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 (7) (Table 3). *Category IB*

l. Do not routinely give varicella-zoster immune globulin (VZIG) to exposed 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 twenty-eighth day after the last exposure (7, 16) (Tables 1 and 3). *Category IB*

22. Viral Respiratory Infections

a. Administer influenza vaccine annually to all personnel, including pregnant women, before the influenza season, unless otherwise contraindicated (7, 15) (Table 1). *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, and vaccination of personnel who did not receive vaccine prior to influenza infections in the community in conjunction with antiviral postexposure prophylaxis for 2 weeks following vaccination (1, 449) (Table 1). *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 (1) (Table 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 non-occupational or occupational environments (122). Provide such women with information on Standard and Transmission-Based Precautions appropriate for each infection (1, 123) (Table 6). *Category IB*

b. Do not routinely exclude women, on the basis only 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) (480–482) (Table 6). 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 U.S.C. 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 cultures 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 by 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; and (3) measuring the impact of preventive measures. Educational materials and activities should be provided to inform personnel about the manifestations and potential risk of latex allergy. *Category IB*

b. Purchasers should consider barrier effectiveness and worker acceptance (e.g., comfort, fit) when selecting gloves for use in the health care organization. When nonlatex gloves are selected, they should have comparable barrier effectiveness to latex gloves (494). *Category IB*

c. Provide workers with a list of nonlatex glove alternatives or, if possible, low-allergen latex gloves that are available within the organization. *Category IB*

d. Question all personnel for symptoms suggestive of latex allergy (e.g., localized dermatitis, workplace-related asthma) during preemployment and periodic evaluations (520). Use serologic tests only for confirmation in those who, based on clinical history, are suspected to be latex allergic. *Category IB*

e. Avoid the use of *all* latex products in personnel with a history of systemic reactions to latex. *Category IB*

f. Use nonlatex gloves or powder-free latex gloves, or double-glove with cloth or vinyl gloves beneath latex gloves for personnel with localized reactions to latex (e.g., irritant or allergic contact dermatitis). *Category IB*

g. Consider targeted substitution of nonlatex gloves and/or powder-free latex gloves in areas of the facility or among groups where glove use is high (e.g., operative suite, nursing) or in areas where large numbers of personnel have developed latex allergy (499, 527, 528). *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 to reduce latex aeroallergens. *Unresolved issueReferences*

1. Garner JS, Hospital Infection Control Practices Advisory Committee. Guideline for isolation precautions in hospitals. Infect Control Hosp Epidemiol 1996; 17:53–80.

2. Williams WW. CDC Guideline for infection control in hospital personnel. Infect Control 1983; 4:326–349.

3. CDC/National Institutes for Health. Biosafety in microbiological and biomedical laboratories. 3rd ed. U.S. Government Printing Office, 1993.

4. 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 1991; 11(No.14):1–214.

5. Heseltine PNR, Ripper M, Wohlford P. Nosocomial rubella—consequences of an outbreak and efficacy of a mandatory immunization program. Infect Control 1997; 6:371–374.

6. Centers for Disease Control and Prevention. Update on adult immunization: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1991; 40(RR–12):1–94.

7. Centers for Disease Control and Prevention, Advisory Committee on Immunization Practices and the Hospital Infection Control Practices Advisory Commitee. Immunization of Health Care Workers. MMWR 1997; In press.

8. Centers for Disease Control. Protection against viral hepatitis: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1990; 39(RR–2):1–27.

9. Centers for Disease Control. Measles prevention: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1989; 38(S–9):1–18.

10. Centers for Disease Control. Mumps Prevention: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1989; 38:388–392–397–400.

11. Centers for Disease Control and Prevention. Prevention of varicella: recommendations of the Advisory Committee on Immunizations Practices (ACIP). MMWR 1996; 45(RR–1):1–36.

12. Centers for Disease Control. Rubella prevention: recommedations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1990; 39(RR–15):1–18.

13. Centers for Disease Control and Prevention. Control and prevention of meningococcal disease: evaluation and management of suspected outbreaks; 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 1997; 46(RR–5):1–21.

14. 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 1996; 45(RR–12):1–35.

15. Centers for Disease Control and Prevention. Prevention and control of influenza: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997; 46(RR–9):1–25.

16. Centers for Disease Control. Vaccinia (smallpox) vaccine: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1991; 40(RR–14):1–10.

17. Centers for Disease Control. Diphtheria, tetanus, pertussis: recommendations for vaccine use and other preventive measures: recommendations of the Immunization Practices Advisory Committee (ACIP). MMWR 1991; 40(RR–10):1–28.

18. Centers for Disease Control and Prevention. Prevention of pneumococcal disease: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1997; 46(RR–8):1–24.

19. 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 1997; 46(RR–3):1–25.

20. Centers for Disease Control. Rabies prevention—United States, 1991: recommendations of the Immunizations Practices Advisory Committee (ACIP). MMWR 1991; 40(RR–3):1–19.

21. Centers for Disease Control and Prevention. Recommedations of the Advisory Committee on Immunization Practices (ACIP): use of vaccines and immune globulins in persons with altered immunocompetence. MMWR 1993; 42(RR–4):1–18.

22. 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.

23. Anonymous. Record keeping guidelines for occupational injuries and illnesses: the Occupational Safety and Health Act of 1970 and 29 CFR 1904.OMB no. 120–0029. 1986; Washington, DC:

24. Department of Labor.Occupational Safety and Health Administration. Occupational exposure to bloodborne pathogens; final rule. Fed Reg 1991; 56:64004–64182.

25. U.S.Department of Labor., Occupational Safety and Health Administration. Enforcement procedures and scheduling for occupational exposure to tuberculosis. OSHA Instruction 1996; CPL 2.106:

26. Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. MMWR 1987; 36(S–2):1S–18S.

27. Centers for Disease Control. Update: universal precautions for prevention of tranmission of human immunodeficiency virus, hepatitis B virus, and other bloodborne pathogens in health-care settings. MMWR 1988; 37:377–382, 387–388.

28. 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 1989; 38(S–6):1–36.

29. Centers for Disease Control. Public Health Service statement on management of occupational exposure to human immunodeficiency virus, including considerations regarding zidovudine postexposure use. MMWR 1990; 39(RR–1):1–14.

30. Centers for Disease Control. Public Health Service inter-agency guidelines for screening donors of blood, plasma, organs, tissues, and semen for evidence of hepatitis B and hepatitis C. MMWR 1991; 40(RR–4):13–14.

31. Centers for Disease Control and Prevention. Recommended infection control practices for dentistry. MMWR 1993; 42(RR–8):1–12.

32. Centers for Disease Control and Prevention. Human immunodeficiency virus transmission in household settings-United States. MMWR 1994; 43:347–353, 356.

33. Centers for Disease Control and Prevention. Risk of acquiring hepatitis C for health-care workers and recommendations for prophylaxis and follow-up after occupational exposure. Hepatitis Surveillance Report 1996; 56:3–7.

34. CDC/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, D.C. U.S. Government Printing Office, 1993:116–121.

35. 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 1988; 37 (S–4):19–22.

36. 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 1991; 40(RR–13):1–25.

37. 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 1997; 46:21–25.

38. 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 1997; 46:25–29.

39. Centers for Disease Control. Recommendations for prevention of HIV transmission in health-care settings. MMWR 1987; 36 (No. 2S):3S–18S.

40. Centers for Disease Control and Prevention. Patient exposure to HIV during nuclear medicine procedures. MMWR 1992; 41:575–578.

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: Lippencott-Raven, 1997:655–664.

42. Rhodes RS, Bell DM, eds. Prevention of transmission of bloodborne pathogens. Surg Clin North Am 1995; 75:1047–1217.

43. Centers for Disease Control. Recommendations for preventing transmission of human immunodeficiency virus and hepatitis B virus to patients during exposure-prone invasive procedures. MMWR 1991; 40(RR–8):1–9.

44. Thomas DL, Factor SH, Kelen GD, et al. Viral hepatitis in health care personnel at the Johns Hopkins Hospital: Seroprevalence and risk factors for hepatitis B virus and hepatitis C virus infection. Arch Intern Med 1993; 153:1705–1712.

45. Dienstag JL, Ryan DM. Occupational exposure to hepatitis B virus in hospital personnel: infection or immunization? Am J Epidemiol 1982; 115:26–39.

46. Shapiro CN, Tokars JI, Chamberland ME, et al. Use of the hepatitis B vaccine and infection with hepatitis B and C among orthopaedic surgeons. J Bone Joint Surg 1996; 78:1791–1800.

47. Gibas A, Blewett DR, Schoenfield DA, et al. Prevalence and incidence of viral hepatitis in health care workers in the prehepatitis B vaccination era. Am J Epidemiol 1992; 136:603–610.

48. Hadler SC, Doto IL, Maynard JE, et al. Occupational risk of hepatitis B infection in hospital workers. Infect Control 1985; 6:24–31.

49. Shapiro CN. Occupational risk of infection with hepatitis B and hepatitis C virus. Surg Clin North Am 1995; 75:1047–1056.

50. Hadler SC, Margolis HS. Hepatitis B immunization: Vaccine types, efficacy, and indications for immunization. In: Remington JS, Swartz MN, editors. Current Topics in Infectious Diseases. Boston: Blackwell Scientific Publications, 1992:282

51. 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; 175:674–677.

52. Alter MJ, Coleman PJ, Alexander WJ, et al. Importance of heterosexual activity in the transmission of hepatitis B and non-A, non-B hepatitis. JAMA 1989; 262:1201–1205.

53. Alter MJ. The detection, transmission, and outcome of hepatitis C virus infection. Infect Agents Dis 1993; 2:155–166.

54. Alter MJ, Gerety RJ, Smallwood L, et al. Sporadic non-A, non-B hepatitis: frequency and epidemiology in an urban United States population. J Infect Dis 1982; 145:886–893.

55. Polish LB, Tong MJ, Co RL, et al. Risk factors for hepatitis C virus infection among health care personnel in a community hospital. Am J Infect Control 1993; 21:196–200.

56. Cooper BW, Krusell A, Tilton RC, et al. Seroprevalence of antibodies to hepatitis C virus in high-risk hospital personnel. Infect Control Hosp Epidemiol 1992; 13:82–85.

57. Campello C, Majori S, Poli A, et al. Prevalence of HCV antibodies in health-care workers from northern Italy. Infection 1992; 20:224–226.

58. Nishimura Y, Yamaguchi K, Williams NP, et al. Antibodies to hepatitis C virus in Japanese blood donors and in hospital personnel. Transfusion 1990; 30:667–668.

59. Herbert AM, walker DM, Kavies KJ, et al. Occupationally acquired hepatitis C virus infection. Lancet 1992; 339:305

60. Tsude K, Fujiyama S, Sato S, et al. Two cases of accidental transmission of hepatitis C to medical staff. Hepato-Gastroenterol 1992; 39:73–75.

61. Zuckerman J, Clewley G, Griffiths P, et al. Prevalence of hepatitis C antibodies in clinical health-care workers. Lancet 1994; 343:1618–1620.

62. Petrosilla N, Puro V, Ipolito G, and the Italian Study Group on Blood-borne Occupational Risk in Dialysis. Prevalence of hepatitis C antibodies in health-care workers. Lancet 1994; 344:339–340.

63. Lanphear BP, Linneman CC, Cannon CG, et al. Hepatitis C virus infection in health care workers: risk of exposure and infection. Infect Control Hosp Epidemiol 1994; 15:745–750.

64. Mitsui T, Iwano K, Masuko K, et al. Hepatitis C virus infection in medical personnel after needlestick accident. Hepatology 1992; 16:1109–1114.

65. Knodell RG, Conrad ME, Ginsberg AL, Bell CJ. Efficacy of prophylactic gamma-globulin in preventing non-A, non-B post-transfusion hepatitis. Lancet 1976; 1:557–561.

66. Seeff LB, Zimmerman HJ, Wright EC, 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; 72:111–121.

67. Sanchez-Quijano A, Pineda JA, Lissen E, et al. Prevention of post-transfusion non-A, non-B hepatitis by non-specific immunoglobulin in heart surgery patients. Lancet 1988; 1:1245–1249.

68. Krawczynski K, Alter MJ, Govindarajan S, et al. Studies on protective efficacy of hepatitis C immunoglobulins (HCIG) in experimental hepatitis C virus infection [abstract]. Hepatology 1993; 18:110A

69. Centers for Disease Control. Recommendations for follow-up of health-care workers after occupational exposure to hepatitis C virus. MMWR 1997; 46:603–606.

70. Tokars JI, Marcus R, Culver DH, et al. Surveillance of HIV infection and zidovudine use among health care workers after occupational exposure to HIV-infected blood. Ann Intern Med 1993; 118:913–919.

71. Centers for Disease Control and Prevention. Case-control study of HIV serovonversion in health-care workers after percutaneous exposure to HIV-infected blood-France, United Kingdom, and United States, January 1988–August 1994. MMWR 1995; 44:929–933.

72. 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, 1995:2632–2656.

73. Puro V, Ippolito G, Guzzanti E, et al. Zidovudine prophylaxis after accidental exposure to HIV: the Italian experience. AIDS 1992; 6:963–969.

74. Chamberland ME, Ciesielski CA, Howard RJ, Fry DE, Bell DM. Occupational risk of infection with human immunodeficiency virus. Surg Clin North Am 1995; 75:1057–1070.

75. 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: Lippencott-Raven, 1997:645–654.

76. Centers for Disease Control and Prevention. Update: provisional Public Health Service recommendations for chemoprophylaxis after occupational exposure to HIV. MMWR 1996; 45:468–472.

77. Centers for Disease Control. Epidemic keratoconjunctivitis in an opthalmology clinic—California. MMWR 1990; 39:598–601.

78. Ford E, Nelson KE, Warren D. Epidemiology of epidemic keratoconjunctivitis. Epidemiol Rev 1987; 9:244–261.

79. Birenbaum E, Linder N, Varsano N, et al. Adenovirus type 8 conjunctivitis outbreak in a neonatal intensive care unit. Arch Dis Child 1993; 68:610–611.

80. Warren D, Nelson KE, Farrar JA, et al. A large outbreak of epidemic keratoconjunctivitis: problems in controlling spread. J Infect Dis 1989; 160:938–943.

81. Jernigan JA, Lowry BS, Hayden FG, et al. Adenovirus type 8 epidemic keratoconjunctivitis in an eye clinic: risk factors and control. J Infect Dis 1993; 167:1307–1313.

82. Adler SP. Molecular epidemiology of cytomegalovirus: a study of factors affecting transmission among children at three day-care centers. Pediatr Infect Dis J 1991; 10:584–590.

83. Adler SP, Bagget J, Wilson M. Molecular epidemiology of cytomegalovirus in a nursery: Lack of evidence for nosocomial transmission. J Pediatr 1986; 108:117–123.

84. Meyers JD, Fluornoy N, Thomas ED. Nonbacterial pneumonia after allogeneic marrow transplantation: a review of ten years' experience. Rev Infect Dis 1982; 3:1119–1132.

85. Winston DJ, Gale RP, Meyer DV, et al. Infectious complications of human bone marrow transplantation. Med 1979; 58:1–31.

86. Brady MT, Demmler GJ, Reis S. Factors associated with cytomegalovirus excretion in hospitalized children. Am J Infect Control 1988; 16:41–45.

87. Demmler GJ, Yow MD, Spector SA, et al. Nosocomial cytomegalovirus infections within two hospitals caring for infants and children. J Infect Dis 1987; 156:9–16.

88. Rubin RH, Wolfson JS, Cosimi AB, et al. Infection in the renal transplant recipient. Am J Med 1981; 70:405–411.

89. Ahlfors K, Ivarsson SA, Johnson T, et al. Risk of cytomegalovirus infection in nurses and congenital infection in their offspring. Acta Paediatr Scand 1981; 70:819–823.

90. Dworsky ME, Welch K, Cassady G, et al. Occupational risk for primary

cytomegalovirus infection among pediatric health-care workers. N Engl J Med 1983; 309:950–953.
91. Yeager AS. Longitudinal, serological study of cytomegalovirus infections in nurses and in personnel without patient contact. J Clin Microbiol 1975; 2:448–452.
92. Gerberding KL, 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.
93. Blackman JA, Murph JR, Bale JF. Risk of cytomegalovirus infection among educators and health care personnel serving disabled children. Pediatr Infect Dis J 1987; 6:725–729.
94. Tolkoff-Rubin NE, Rubin RH, Keller EE, et al. Cytomegalovirus infection in dialysis patients and personnel. Ann Intern Med 1978; 89:625–628.
95. Adler SP. Hospital transmission of cytomegalovirus. Infect Agents Dis 1992; 1:43–49.
96. Balfour CL, Balfour HH. Cytomegalovirus is not an occupational risk for nurses in renal transplant and neonatal units. JAMA 1986; 256:1909–1914.
97. Brady MT, Demmler GJ, Anderson DC. Cytomegalovirus infection in pediatric house officers: susceptibility to, and new rate of primary infection. Infect Control 1987; 8:329–332.
98. Lipscomb JA, Linneman CC, Hurst PF, Myers MG, Stringer W, Moore P, et al. Prevalence of cytomegalovirus antibody in nursing personnel. Infect Control 1984; 5:513–518.
99. Friedman HM, Lewis MR, Nemerosky DM. Acquisition of cytomegalovirus infection among female employees at a pediatric hospital. Pediatr Infect Dis J 1984; 3:233–235.
100. Hokeberg I, Grillner L, Reisenfeld T, et al. No evidence of hospital-acquired cytomegalovirus on environmental surfaces. Pediatr Infect Dis J 1988; 7:812–814.
101. Yow MD, Lakeman AD, Stagno S. Use of restriction enzymes to investigate the source of a primary cytomegalovirus infection in a pediatric nurse. Pediatrics 1982; 70:713–716.
102. Wilfert CM, Huang EA, Stagno S. Restriction endonuclease analysis of cytomegalovirus deoxyribonucleic acid as an epidemiologic tool. Pediatrics 1982; 70:717–721.
103. Spector SA. Transmission of cytomegalovirus among infants in hospital documented by restriction-endonuclease-digestion analyses. Lancet 1983; 2:378–381.
104. Pass RF, Hutto C, Lyon MD, et al. Increased rate of cytomegalovirus infection among daycare center workers. Pediatr Infect Dis J 1990; 9:465–470.
105. 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; 314:1414–1418.
106. Adler SP. Cytomegalovirus and child day care. Evidence for an increased infection rate among day-care workers. N Engl J Med 1989; 321:1290–1296.
107. Hutto C, Little EA, Ricks R. Isolation of cytomegalovirus from toys and hands in a day care center. J Infect Dis 1986; 154:527–530.
108. Faix RG. Survival of cytomegalovirus on environmental surfaces. J Pediatr 1985; 106:649–652.
109. Onorato IM, Morens DM, Martone WJ, Stansfield SK. Epidemiology of cytomegaloviral infections: recommendations for prevention and control. Rev Infect Dis 1995; 7:479–497.
110. 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, 1997:187–191.
111. Stagno S, Pass RF, Dworsky ME, et al. Maternal cytomegalovirus infection and perinatal transmission. Clin Obstet Gynecol 1982; 25:563–576.
112. Plotkin SA, Starr SE, Friedman HM, et al. Vaccines for the prevention of human cytomegalovirus infection. Rev Infect Dis 1990; 12 (Suppl 7):S827-S838.
113. 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.
114. Plotkin SA, Starr SE, Friedman HM, et al. Effect of Towne live vaccine on cytomegalovirus disease in patients receiving renal transplants: A controlled trial. Ann Intern Med 1990; 114:525–531.
115. Fleisher GR, Starr SE, Friedman HM, et al. Vaccination of pediatric nurses with live attenuated cytomegalovirus. Am J Dis Child 1982; 136:294–296.
116. Snydman DR, Werner BG, Heinz-Lacey B, et al. Use of cytomegalovirus immune globulin to prevent cytomegalovirus disease in renal-transplant recipients. N Engl J Med 1987; 317:1049–1054.
117. Bowden RA, Fisher LD, Rogers K, et al. Cytomegalovirus (CMV)-specific intravenous immunoglobulin for the prevention of primary CMV infection and disease after marrow transplant. J Infect Dis 1991; 164:483–487.
118. Meyers JD, Reed EC, Shepp DH, et al. Acyclovir for prevention of cytomegalovirus infection and disease after allogeneic marrow transplantation. N Engl J Med 1988; 318:70–75.
119. Goodrich JM, Kmori M, Gleaves CA, et al. Early treatment with ganciclovir to prevent cytomegalovirus disease after allogeneic bone marrow transplantation. N Engl J Med 1991; 325:1601–1607.
120. Bailey TC, Trulock EP, Ettinger NA, et al. Failure of prophylactic ganciclovir to prevent cytomegalovirus disease in recipients of lung transplants. J Infect Dis 1992; 265:548–552.
121. Balcarek KB, Bagley R, Cloud GA. Nosocomial cytomegalovirus infections within two hospitals caring for infants and children. J Infect Dis 1987; 145:9–16.
122. Finney JW, Miller KM, Adler SP. Changing protective and risky behaviors to prevent child-to-parent transmission of cytomegalovirus. J Appl Behav Anal 1993; 26:471–472.
123. Hatherly LI. Is primary cytomegalovirus infection an occupational hazard for obstetric nurses? A serological study. Infect Control 1986; 7:452–455.
124. Anderson GS, Penfold JB. An outbreak of diphtheria in a hospital for the mentally subnormal. J Clin Path 1973; 26:606–615.
125. Gray RD, James SM. Occult diphtheria infection in a hospital for the mentally subnormal. Lancet 1973; 1(812):1105–1106.
126. Palmer SR, Balfour AH, Jephcott AE. Immunisation of adults during an outbreak of diphtheria. Brit Med J Clin Research Ed 1983; 286:624–626.
127. Centers for Disease Control and Prevention. Respiratory diphtheria in the United States, 1980–1995. Am J Pub Health 1997; In press.
128. 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.
129. Centers for Disease Control and Prevention. Toxigenic Corynebacterium diphtheriae—northern plains Indian community, August–October 1996. MMWR 1997; 46:506–510.
130. Centers for Disease Control and Prevention. Update: Diphtheria epidemic—new independant states of the former Soviet Union, January 1995–March 1996. MMWR 1997; 45:693–697.
131. Centers for Disease Control and Prevention. Diphtheria epidemic—new independent states of the former Soviet Union, 1990–1994. MMWR 1995; 44:177–181.
132. 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; 347:1739–1744.
133. Centers for Disease Control and Prevention. Diphtheria outbreak—Saraburi Province, Thailand, 1994. MMWR 1996; 45:271–273.
134. Lumio J, Jahkola M, Vuento R, Haikala O, Eskila J. Diphtheria after visit to Russia. Lancet 1993; 342:53–54.
135. Centers for Disease Control and Prevention. Diphtheria acquired by U.S. citizens in the Russian Federation and Ukraine—1994. MMWR 1995; 44:243–244.
136. 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, 1997:191–195.
137. Sargent RK, Rossing TH, Dowton SB, Breyer MD, Levine L, Weinstein L. Diphtheria immunity in Massachusetts—a study of three urban patient populations. Am J Med Sci 1984; 287:37–39.
138. Weiss BP, Strassburg MA, Feeley JC. Tetanus and diphtheria immunity in an elderly population in Los Angeles County. Am J Public Health 1983; 73:802–804.
139. Crossley K, Irvine P, Warren JB, Lee BK, Mead K. Tetanus and diphtheria immunity in urban Minnesota adults. JAMA 1979; 242:2298–3000.
140. Ruben FL, Nagel J, Fireman P. Antitoxin responses in the elderly to tetanus-diphtheria (Td) immunization. Am J Epidemiol 1978; 108:145–149.

141. Koblin BA, Townsend T. Immunity to diphtheria and tetanus in inner-city women of childbearing age. Am J Public Health 1989; 79:1297–1298.

142. 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.

143. Steere AC, Craven PJ, Hall WJ, III, et al. Person-to-person spread of Salmonella typhimurium after a hospital common-source outbreak. Lancet 1975; 1:319–322.

144. Blaser MJ, Hickman FW, Farmer JJ, III, et al. Salmonella typhi: the laboratory as a reservoir of infection. J Infect Dis 1980; 142:934–938.

145. Standaert SM, Hutcheson RH, Schaffner W. Nosocomial transmission of Salmonella gastroenteritis to laundry workers in a nursing home. Infect Control Hosp Epidemiol 1994; 15:22–26.

146. Toivanen P, Toivanen A, Olkkonen L, Aantaa S. Hospital outbreak of Yersinia enterocolitica infection. Lancet 1973; April 14:801–803.

147. Ratnam S, Mercer E, Picco B, et al. A nosocomial outbreak of diarrheal disease due to Yersinia enterocolitica serotype 0:5, biotype 1. J Infect Dis 1982; 145:242–247.

148. Anglim AM, Farr BM. Nosocomial gastrointestinal tract infections. In: Mayhall CG, editor. Hospital epidemiology and infection control. Baltimore: Williams & Wilkens, 1996:196–219.

149. Mitchell DK, Pickering LK. Nosocomial gastrointestinal tract infections in pediatric patients. In: Mayhall CG, editor. Hospital epidemiology and infection control. Baltimore: Williams & Wilkens, 1996:506–523.

150. McGowan JE. Nosocomial infections in diagnostic laboratories. In: Mayhall CG, editor. Hospital epidemiology and infection control. Baltimore: Wlliams & Wilkens, 1996:883–892.

151. Kurtz JB, Lee TW, Pickering D. Astrovirus associated gastroenteritis in a children's ward. J Clin Pathol 1977; 30:948–952.

152. Dryjanski J, Gold JW, Ritchie MT, Kurtz RC, Lim SL, Armstrong D. Cryptosporidiosis: case report in a health team worker. Am J Med 1986; 80:751–752.

153. Lewis DC, Lightfoot WD, Cubitt WD, et al. Outbreaks of astrovirus type 1 and rotovirus gastroenteritis in a geriatric in-patient population. J Hosp Infect 1989; 14:9–14.

154. Koch KL, Phillips DJ, Aber RC, Current WL. Cryptosporidiosis in hospital personnel: evidence for person-to-person transmission. Ann Intern Med 1985; 102:593–596.

155. Pike RM. Laboratory-associated infections: summary and analysis of 3921 cases. Health Lab Sci 1976; 13:105–114.

156. 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; 17:587–592.

157. Gellert GA, Waterman SH, Ewert D, et al. An outbreak of acute gastroenteritis caused by a small round structured virus in a geriatric convalescent facility. Infect Control Hosp Epidemiol 1990; 11:459–464.

158. Chadwick PR, McCann R. Transmission of a small round structured virus by vomiting during a hospital outbreak of gastroenteritis. J Hosp Infect 1994; 26:251–259.

159. Linneman CC, Jr, Cannon CG, Staneck JL, McNeely BL. Prolonged hospital epidemic of salmonellosis: use of trimethoprim-sulfamethoxazole for control. Infect Control 1985; 6:221–225.

160. Tauxe RV, Hassan LF, Findeisen KO, Sharrar RG, Blake PA. Salmonellosis in nurses: Lack of transmission to patients. J Infect Dis 1988; 157:370–373.

161. Guerrant RL. Cryptosporidiosis: an emerging, highly infectious threat. Emerging Infect Dis 1997; 3:51–57.

162. Schroeder SA, Aserkoff B, Brachman PS. Epidemic salmonellosis in hospitals and institutions. N Engl J Med 1968; 279:674–678.

163. Centers for Disease Control. Viral agents of gastroenteritis. MMWR 1990; 39;(RR–5):1–24.

164. Caul EO. Small round structured viruses: airborne transmission and hospital control. Lancet 1994; 343:1240–1242.

165. Noah ND. Airborne transmission of a small round structured virus. Lancet 1994; 343:608–609.

166. Sawyer LA, Murphy JJ, Kaplan JE, 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; 127:1261–1271.

167. Sharp TW, Hyams KC, Watts D, et al. Epidemiology of norwalk virus during an outbreak of acute gastroenteritis aboard a US aircraft carrier. J Med Virol 1995; 45:61–67.

168. Dobbeling BN, Stanley GL, Sheetz CT, Pfaller MA, Houston AK, Annis L, et al. Comparitive efficacy of alternative hand-washing agents in reducing nosocomial infections in intensive care units. N Engl J Med 1992; 327:88–93.

169. Black DE, Dykes AC, Anderson KE, et al. Handwashing to prevent diarrhea in day care centers. Am J Epidemiol 1982; 113:445–451.

170. Centers for Disease Control and Prevention. Typhoid immunization: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR 1994; 43(RR14):1–7.

171. Ho MS, Glass RI, Monroe SS, et al. Viral gastroenteritis aboard a cruise ship. Lancet 1989; October 21:961–965.

172. Kilgore PE, Belay ED, Hamlin DM, 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; 173:787–793.

173. Grohmann GJ, Jr., Glass RI, Pereira H, et al. Enteric viruses and diarrhea in HIV-infected patients. N Engl J Med 1993; 329:14–20.

174. Centers for Disease Control. Recommendations for collection of laboratory specimens associated with outbreaks of gastroenteritis. MMWR 1990; 39(RR–14):1–13.

175. Salam MA, Bennish ML. Antimicrobial therapy for shigellosis. Rev Infect Dis 1991; 13(Suppl 4):S332–S341.

176. Allos BM, Blaser MJ. Campylobacter jejuni and the expanding spectrum of related infections. Clin Infect Dis 1995; 20:1092–1099.

177. Buchwald DS, Blaser MJ. A review of human salmonellosis: II. Duration of excretion following infection with nontyphi Salmonella. Rev Infect Dis 1984; 6:345–356.

178. Aserkoff B, Bennett JV. Effect of antibiotic therapy in acute salmonellosis in the fecal excretion of Salmonellae. N Engl J Med 1969; 281:636–640.

179. Pavia AT, Shipman LD, Wells JG, et al. Epidemiologic evidence that prior antimicrobial exposure decreases resistance to infection by antimicrobial-sensitive Salmonella. J Infect Dis 1990; 161:255–260.

180. 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, 1995:2013–2033.

181. Rosenblum LS, Villarino ME, Nainan OV, 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; 164:476–482.

182. Carl M, Kantor RJ, Webster HM, et al. Excretion of hepatitis A virus in the stools of hospitalized patients. J Med Virol 1982; 9:125–129.

183. Drusin LM, Sohmer M, Groshen SL, et al. Nosocomial hepatitis A infection in a pediatric intensive care unit. Arch Dis Child 1987; 62:690–695.

184. Baptiste R, Koziol DE, Henderson DK. Nosocomial transmission of hepatitis A in an adult population. Infect Control 1987; 8:364–370.

185. Azimi PH, Roberto RR, Guralnik J, et al. Transfusion-acquired hepatitis A in a premature infant with secondary nosocomial spread in an intensive care nursery. Am J Dis Child 1986; 140:23–27.

186. Goodman RA, Carder CC, Allen JR, et al. Nosocomial hepatitis A transmission by an adult patient with diarrhea. Am J Med 1982; 73:220–226.

187. Skidmore SJ, Gully PR, Middleton JD, et al. An outbreak of hepatitis A on a hospital ward. J Med Virol 1985; 17:175–177.

188. Klein BS, Michaels JA, Rytel MW, et al. Nosocomial hepatitis A: a multinursery outbreak in Wisconsin. JAMA 1984; 252:2716–2721.

189. Krober MS, Bass JW, Brown JD, et al. Hospital outbreak of hepatitis A: risk factors for spread. Pediatr Infect Dis 1984; 3:296–299.

190. Reed CM, Gustafson TL, Siegel J, et al. Nosocomial transmission of hepatitis A from a hospital-acquired case. Pediatr Infect Dis 1984; 3:300–303.

191. Doebbeling BN, Li N, Wenzel RP. An outbreak of hepatitis A among health care workers: risk factors for transmission. Am J Public Health 1993; 83:1679–1684.

192. Watson JC, Fleming DC, Borella AJ, et al. Vertical transmission of hepatitis A resulting in an outbreak in a neonatal intensive care unit. J Infect Dis 1993; 167:567–571.

193. Noble RC, Kane MA, Reeves SA, et al. Posttransfusion hepatitis A in a neonatal

intensive care unit. JAMA 1984; 252:2711–2715.
194. 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 1992; 11:122–123.
195. 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; 141:151–156.
196. 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 1996; 45(RR–15):1–30.
197. Meyers JD, Romm FJ, Tihen WS. Food-borne hepatitis A in a general hospital. JAMA 1975; 231:1049–1053.
198. Eisenstein AB, Aach RD, Jacobsen W, et al. An epidemic of infectious hepatitis in a general hospital: probable transmission by contaminated orange juice. JAMA 1963; 185:171–174.
199. 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; 7:143–148.
200. Kashiwagi S, Hayashi J, Ikematsu H, et al. Prevalence of immunologic markers of hepatitis A and B infection in hospital personnel in Miyazaki Prefecture, Japan. Am J Epidemiol 1985; 122:960–969.
201. 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 1984; 3:153–156.
202. Linneman CC, Buchman TG, Light IJ, et al. Transmission of herpes-simplex virus type 1 in a nursery for the newborn: identification of isolates by DNA ''fingerprinting''. Lancet 1978; 1:964–966.
203. Kleiman MB, Schreimer RL, Eitzen H, et al. Oral herpesvirus infection in nursery personnel: infection control policy. Pediatrics 1982; 70:609–612.
204. Buchman TG, Roizman B, Adams G, et al. Restriction endonuclease fingerprinting of herpes simplex virus DNA: A novel epidemiological tool applied to a nosocomial outbreak. J Infect Dis 1978; 138:488–498.
205. Greaves WL, Kaiser AB, Alford RH, et al. The problem with herpetic whitlow among hospital personnel. Infect Control 1980; 1:381–385.
206. Adams G, Stober BH, Keenyslide RA, Kooton TM, Buchman TG, Roizman B, et al. Nosocomial herpetic infections in a pediatric intensive care unit. Am J Epidemiol 1981; 113:126–132.
207. 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, 1997:266–276.
208. Pereira FA. Herpes simplex: evolving concepts. J Am Acad Dermatol 1996; 35:503–520.
209. 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; 117:584–586.
210. Turner R, Shehab Z, Osborne K, et al. Shedding and survival of herpes simplex virus from ''fever blisters''. Pediatrics 1982; 70:547–549.
211. Spruance SL, Overall JC, Jr., Kern ER, et al. The natural history of recurrent herpes simplex labialis: implications for antiviral therapy. N Engl J Med 1977; 297:69.
212. Davis RM, Orenstein WA, Frank JA, et al. Transmission of measles in medical settings, 1980 through 1984. JAMA 1986; 255:1295–1298.
213. Atkinson WL, Markowitz LE, Adams NC, et al. Transmission of measles in medical settings—United States, 1985–1989. Am J Med 1991; 91(suppl 3B):320S–324S.
214. Raad II, Sheretz RJ, Rains CS, et al. The importance of nosocomial transmission of measles in the propogation of a community outbreak. Infect Control Hosp Epidemiol 1989; 10:161–166.
215. Istre GR, McKee PA, West GR, et al. Measles spread in hospital settings: an important focus of disease transmission? Pediatrics 1987; 79:356–358.
216. Dales LG, Kizer KW. Measles transmission in medical facilities. West J Med 1985; 142:415–416.
217. 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; 77:1222–1224.
218. Rivera ME, Mason WH, Ross LA, et al. Nosocomial measles infection in a pediatric hospital during a community-wide epidemic. J Pediatr 1991; 119:183–186.
219. Rank EL, Brettman L, Katz-Pollack H, et al. Chronology of a hospital-wide measles outbreak: lessons learned and shared from an extraordinary week in late March 1989. Am J Infect Control 1992; 209:315–318.
220. Watkins NM, Smith RP, St. Germain DL, et al. Measles (rubeola) infection in a hospital setting. Am J Infect Control 1987; 15:201–206.
221. Remington PL, Hall WN, Davis IH, et al. Airborne transmission of measles in a physician's office. JAMA 1985; 253:1574–1577.
222. Atkinson WL. Measles and health care workers. Infect Control Hosp Epidemiol 1994; 15:5–7.
223. Bloch AB, Orenstein WA, Ewing WM. Measles outbreak in a pediatric practice: airborne transmission in an office setting. Pediatrics 1985; 75:676–683.
224. American Academy of Pediatrics. Summaries of Infectious Diseases: Measles. In: Peter G, editor. 1997 Redbook: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics, 1997:334–357.
225. Braunstein H, Thomas S, Ito R. Immunity to measles in a large population of varying age. Am J Dis Child 1990; 144:296–298.
226. Smith E, Welch W, Berhow M, et al. Measles susceptibility of hospital employees as determined by ELISA. Clin Res 1990; 38:183A.
227. Subbarao EK, Amin S, Kumar ML. Prevaccination serologic screening for measles in health care workers. J Infect Dis 1991; 163:876–878.
228. Sellick J, Longbine D, Schiffeling R, et al. Screening hospital employees for measles is more cost-effective than blind immunization. Ann Intern Med 1992; 116:982–984.
229. Grabowsky M, Markowitz LE. Serologic screening, mass immunization, and implications for immunization programs. J Infect Dis 1991; 164:1237–1238.
230. Jackson LA, Schuchat A, Reeves NW, et al. Serogroup C meningococcal outbreaks in the United States. An emerging threat. JAMA 1995; 273:383–389.
231. Houck P, Patnode M, Atwood R, et al. Epidemiologic characteristics of an outbreak of serogroup C meningococcal disease and the public health response. Publ Health Rep 1995; 110:343–349.
232. Centers for Disease Control. Laboratory-acquired menigococcemia—California and Massachusetts. MMWR 1991; 40:46–47–55.
233. Centers for Disease Control. Nosocomial meningococcemia—Wisconsin. MMWR 1978; 27:358–363.
234. Rose HD, Lenz IE, Sheth NK. Meningococcal pneumonia: A source of nosocomial infection. Arch Intern Med 1981; 141:575–577.
235. Centers for Disease Control. Laboratory-acquired meningococcemia—California and Massachusetts. MMWR 1991; 40:46–47–55.
236. Cohen MS, Steere AC, Baltimore R, et al. Possible nosocomial transmission of group Y Neisseria meningitidis among oncology patients. Ann Intern Med 1979; 91:7–12.
237. Broome CV. The carrier state: Neisseria meningitidis. J Antimicrob Chemother 1986; 18:25–34.
238. Gaunt PN, Lambert BE. Single dose ciprofloxacin for the eradication of pharyngeal carriage of Neisseria meningiditis. J Antimicrob Chemother 1988; 21:489–496.
239. Munford RS, Taunay A, de Morais JS, et al. Spread of meningococcal infection within households. Lancet 1974; 1:1275–1278.
240. American Academy of Pediatrics. Meningococcal disease prevention and control strategies for practice-based physicians. Pediatrics 1996; 97:404–411.
241. Riedo FX, Plikaytis BD, Broome CV. Epidemiology and prevention of meningococcal disease. Pediatr Infect Dis J 1995; 14:643–657.
242. Griffis JM. Epidemic meningococcal disease: synthesis of a hypothetical immuno-epidemiologic model. Rev Infect Dis 1982; 4:159–172.
243. Caugant DA, Hoiby EA, Magnus P, et al. Asymptomatic carriage of Neisseria meningitidis in a randomly sampled population. J Clin Microbiol 1994; 32:323–330.
244. Caugant DA, Hoiby EA, Rosenqvist LO, et al. Transmission of Neisseria meningitidis from a population of asymptomatic carriers. Epidemiol Infect 1992; 109:241–253.
245. Wharton M, Cochi SL, Hutcheson RH, et al. Mumps transmission in hospitals. Arch Intern Med 1990; 150:47–49.

246. Fischer PR, Brunetti C, Welch V, et al. Nosocomial mumps: report of an outbreak and its control. Am J Infect Control 1996; 24:13–18.
247. American Academy of Pediatrics. Summaries of Infectious Diseases: Mumps. In: Peter G, editor. 1997 Redbook: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics, 1997:366–369.
248. Williams WW, Preblud SR, Reichelderfer PS, Hadler SC. Vaccines of importance in the hospital setting. Infect Dis Clin N Amer 1989; 3:701–722.
249. Koplan JP, Preblud SR. A benefit-cost analysis of mumps vaccine. Am J Dis Child 1982; 136:362–364.
250. Hersh BS, Fine PEM, Kent WK, et al. Mumps outbreak in a highly vaccinated population. J Pediatr 1991; 119:187–193.
251. Anderson LJ, Török TJ. The clinical spectrum of human parvovirus B19 infections. Curr Clin Topics Infect Dis 1991; 11:267–280.
252. Török TJ. Parvovirus B19 and human disease. Advances Intern Med 1992; 37:431–455.
253. Dowell SF, Török TJ, Thorp JA, et al. Parvovirus B19 infection in hospital workers: Community or hospital acquisition. J Infect Dis 1995; 172:1076–1079.
254. Ray SM, Erdman DD, Berschling JD, et al. Nosocomial exposure to parvovirus B19: low risk of transmission to healthcare workers. Infect Control Hosp Epidemiol 1997; 18:109–114.
255. Koziol DE, Kurtzman G, Ayub J, et al. Nosocomial human parvovirus B19 infection: lack of transmission from a chronically infected patient to hospital staff. Infect Control Hosp Epidemiol 1992; 13:343–348.
256. Bell LM, Noides SJ, Stoffman P, et al. Human parvovirus B19 infection among hospital staff members after contact with infected patients. N Engl J Med 1989; 321:485–491.
257. Evans JP, Rossiter MA, Kumaran TO, et al. Human parvovirus aplasia: case due to cross infection in a ward. Br Med J 1984; 288:681
258. Cohen BJ, Courouce AM, Schwartz TF. Laboratory infection with parvovirus B19 (letter). J Clin Pathol 1988; 41:1027–1028.
259. Anderson LJ, Gillespie SM, Török TJ, et al. Risk of infection following exposures to human parvovirus B19. Behring Inst Mitt 1990; 85:60–63.
260. Pillay D, Patou G, Hurt S, et al. Parvovirus B19 outbreak in a children's ward. Lancet 1992; 339:107–109.
261. Harrison J, Jones DE. Human parvovirus B19 in health care workers. Occup Med 1995; 45:93–96.
262. Anderson MJ, Lewis E, Kidd IM, et al. An outbreak of erythema infectiousum associated with human parvovirus infection. J Hyg 1984; 93:85–93.
263. Chorba T, Coccia P, Holman RC, et al. The role of parvovirus B19 in aplastic crisis and erythema infectiosum. J Infect Dis 1986; 154:383–393.
264. Török TJ. Human parvovirus B19. In: Remington JS, Klein JO, editors. Infectious diseases of the fetus and newborn infant. 4th ed. Philadelphia: WB Saunders, 1995:668–702.
265. Törökk TJ. Human parvovirus B19 infections in pregnancy. Pediatr Infect Dis J 1990; 9:772–776.
266. Kurt TL, Yeager AS, Guennette S, Dunlop S. Spread of pertussis by hospital staff. JAMA 1972; 221:264–267.
267. Linneman CC, Ramundo M, Perlstein PH, et al. Use of pertussis vaccine in an epidemic involving hospital staff. Lancet 1975; 2:540–543.
268. Valenti WM, Pincus PH, Messner MK. Nosocomial pertussis: possible spread by a hospital visitor. Am J Dis Child 1980; 134:520–521.
269. Christie C, Glover AM, Willke MJ, et al. Containment of pertussis in the regional pediatric hospital during the greater Cincinnati epidemic of 1993. Infect Control Hosp Epidemiol 1995; 16:556–563.
270. Shefer A, Dales L, Nelson M, et al. Use and safety of acellular pertussis vaccine among adult hospital staff during an outbreak of pertussis. J Infect Dis 1995; 171:1053–1056.
271. Deville JG, Cherry JD, Christenson PD, et al. Frequency of unrecognized Bordatella pertussis in adults. Clin Infect Dis 1995; 21:639–642.
272. Nennig ME, Shinefield HR, Edwards KM, et al. Prevalence and incidence of adult pertussis in an urban population. JAMA 1996; 275:1672–1674.
273. Mortimer EA, Jr. Pertussis vaccine. In: Plotkin SA, Mortimer EA, editors. Vaccines. Philadelphia: WB Saunders, 1994:91–137.
274. Mortimer EA, Jr. Pertussis and its prevention: a family afffair. J Infect Dis 1990; 161:473–479.
275. Deen JL, Mink CA, Cherry JD, et al. Household contact study of Bordatella pertussis infections. Clin Infect Dis 1995; 21:1211–1219.
276. Edwards KM, Decker MD, Graham BS, et al. Adult immunization with acellular pertussis vaccine. JAMA 1993; 269:53–56.
277. Weber DJ, Rutala WA. Management of healthcare workers exposed to pertussis. Infect Control Hosp Epidemiol 1994; 15:411–415.
278. Halsey NA, Welling MA, Lehman RM. Nosocomial pertussis: a failure of erythromycin treatment and prophylaxis. Am J Dis Child 1980; 134:521–522.
279. American Academy of Pediatrics. Summaries of infectious diseases: poliovirus infections. In: Peter G, editor. 1997 Redbook: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics, 1997:424–433.
280. Centers for Disease Control and Prevention. Paralytic poliomyelitis—United States, 1980–1994. MMWR 1997; 46:79–83.
281. Fishbein DB, Robinson LE. Current concepts: rabies. N Engl J Med 1997; 329:1632–1638.
282. Winkler WG, Fashinell TR, Leffingwell L, et al. Airborne rabies transmission in a laboratory worker. JAMA 1973; 226:1219–1221.
283. Centers for Disease Control. Rabies in a laboratory worker—New York. MMWR 1977; 26:183–184.
284. Helmick CG, Tauxe RV, Vernon AA. Is there a risk to contacts of patients with rabies? Rev Infect Dis 1987; 9:511–518.
285. Centers for Disease Control and Prevention. Human rabies—New Hampshire, 1996. MMWR 1997; 46:267–270.
286. Centers for Disease Control and Prevention. Human rabies—Connecticut, 1995. MMWR 1996; 45:207–209.
287. Centers for Disease Control and Prevention. Human rabies—Washington, 1995. MMWR 1995; 44:625–627.
288. Greaves WL, Orenstein WA, Stetler HC, et al. Prevention of rubella transmission in medical facilities. JAMA 1982; 248:861–864.
289. Centers for Disease Control. Rubella in hospitals—California. MMWR 1983; 32:37–39.
290. Poland GA, Nichol KL. Medical students as sources of rubella and measles outbreaks. Arch Intern Med 1990; 150:44–46.
291. Storch GA, Gruber C, Benz B, et al. A rubella outbreak among dental students: description of the outbreak and analysis of control measures. Infect Control 1985; 6:150–156.
292. Strassburg MA, Stephenson TG, Habel LA, et al. Rubella in hospital employees. Infect Control 1984; 5:123–126.
293. Fliegel PE, Weinstein WM. Rubella outbreak in a prenatal clinic: management and prevention. Am J Infect Control 1982; 10:29–33.
294. Strassburg MA, Imagawa DT, Fannin SL, et al. Rubella outbreak among hospital personnel. Obstet Gynecol 1981; 57:283–288.
295. Gladstone JL, Millian SJ. Rubella exposure in an obstetric clinic. Obstet Gynecol 1981; 57:182–186.
296. American Academy of Pediatrics. Summaries of Infectious Diseases: Rubella. In: Peter G, editor. 1997 Redbook: Report of the Committe on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics, 1997:456–462.
297. Polk FB, White JA, DeGirolami PC, et al. An outbreak of rubella among hospital personnel. N Engl J Med 1980; 303:541–545.
298. Sachs JJ, Olson B, Soter J, et al. Employee rubella screening programs in Arizona hospitals. JAMA 1983; 249:2675–2678.
299. Heseltine PNR, Ripper M, Wohlford P. Nosocomial rubella—consequences of an outbreak and efficacy of a mandatory immunization program. Infect Control 1985; 6:371–374.
300. Preblud SR. Some current issues related to the rubella vaccine. JAMA 1985; 254:253–256.
301. Lettau LA. Nosocomial transmission and infection control aspects of parasitic and ectoparasitic diseases part III. Ectoparasites/summary and conclusions. Infect Control Hosp Epidemiol 1991; 12:179–185.
302. 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, 1985:139–156.
303. Jucowics P, Ramon ME, Don PC, et al. Norwegian scabies in an infant with acquired immunodeficiency syndrome. Arch Derm 1989; 125:1670–1671.
304. Hench C, Paulson SS, Stevens DA, et al. Scabies outbreak on a spinal cord injury unit. Rehab Nursing 1994; 19:21–23.
305. Arlian LG, Estes SA, Vyszenski-Moher DL. Prevalemce of Sarcoptes scabei in the

homes and nursing homes of scabietic patients. J Am Acad Dermatol 1988; 19:806–811.

306. Jimenez-Lucho VE, Fallon F, Caputo C, et al. Role of prolonged surveillance in the eradication of nosocomial scabies in an extended care Veterans Affairs medical center. Am J Infect Control 1995; 23:44–49.

307. Degelau J. Scabies in long-term care facilities. Infect Control Hosp Epidemiol 1992; 13:421–425.

308. 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; 31:668–684.

309. Bannatyne RM, Patterson TA, Wells BA, et al. Hospital outbreak traced to a case of Norwegian scabies. Can J Infect Control 1992; 7:111–113.

310. Thomas MC, Giedinghagen DH, Hoff GL. Brief report: An outbreak of scabies among employees in a hospital-associated commercial laundry. Infect Control 1987; 8:427–429.

311. Clark J, Friesen DL, Williams WA. Management of an outbreak of Norwegian scabies. Am J Infect Control 1992; 20:217–222.

312. Centers for Disease Control. Scabies in health-care facilities-Iowa. MMWR 1988; 37:178–179.

313. Corbett EL, Crossley I, Holton J, et al. Crusted (''Norwegian'') scabies in a specialist HIV unit: successful use of ivermectin and failure to prevent nosocomial transmission. Genitourinary Medicine 1996; 72:115–117.

314. Orkin M. Scabies in AIDS. Sem Dermatol 1993; 12:9–14.

315. Portu JJ, Santamaria JM, Zubero Z, et al. Atypical scabies in HIV-positive patients. J Am Acad Dermatol 1996; 34:915–917.

316. Gooch JJ, Strasius SR, Beamer B, et al. Nosocomial outbreak of scabies. Arch Derm 1978; 114:897–898.

317. Belle EA, D'Souza TJ, Zarzour JY, et al. Hospital epidemic of scabies: diagnosis and control. Can J Pub Health 1979; 70:133–135.

318. Taplin D, Rivera A, Walker JG, et al. A comparitive trial of three treatment schedules for the eradication of scabies. J Am Acad Dermatol 1983; 9:550–554.

319. Lempert KD, Baltz PS, Welton WA, et al. Pseudouremic pruritis: A scabies epidemic in a dialysis unit. Am J Kidney Dis 1985; 5:117–119.

320. Haydon JR, Caplan RM. Epidemic scabies. Arch Derm 1971; 103:168–173.

321. Estes SA, Estes J. Therapy of scabies: nursing homes, hospitals, and the homeless. Sem Dermatol 1993; 12:26–33.

322. Sargent SJ. Ectoparasites. In: Mayhall CG, editor. Hospital Epidemiology and Infection Control. Baltimore: Williams & Wilkens, 1996:465–472.

323. Centers for Disease Control and Prevention. 1993 sexually transmitted diseases treatment guidelines. MMWR 1993; 42(No. RR–14):93–97.

324. 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–S109.

325. Anonymous. Drugs for parasitic infections. Med Lett Drugs Ther 1995; 37:102–109.

326. Mienking TL, Taplan D, Hermida JL, et al. The treatment of scabies with ivermectin. N Engl J Med 1995; 333:26–30.

327. Hopper AH, Salisbury J, Jegadeva AN, et al. Epidemic Norwegian scabies in a geriatric unit. Age Aging 1990; 19:125–127.

328. Taplin D, Arrue C, Walker JG, et al. Eradication of scabies with a single treatment schedule. J Am Acad Dermatol 1983; 9:546–550.

329. Yonosky D, Ladia L, Gackenheimer L, et al. Scabies in nursing homes: An eradication program with permethrin 5% cream. J Am Acad Dermatol 1990; 23:1133–1136.

330. Schultz MW, Gomez M, Hansen RC, et al. Comparative study of 5% permethrin cream and 1% lindane lotion for the treatment of scabies. Arch Derm 1990; 126:167–170.

331. Marty P, Gari-Toussaint M, LeFichoux Y. Efficacy of ivermectin in treatment of an epidemic of sarcoptic scabies. Ann Trop Med Parasitol 1994; 88:453.

332. Juranek DD. Pediculosis capitis in school children. In: Orkin M, Maiback HI, editors. Cutaneous Infestations and Insect Bites. New York, NY: Dekker, 1985:199–211.

333. Boyce JM. Methicillin-resistant Staphylococcus aureus in hospitals and long-term care facilities: microbiology, epidemiology, and preventive measures. Infect Control Hosp Epidemiol 1992; 13:725–737.

334. Wenzel RP. Healthcare workers and the incidence of nosocomial infection: can treatment of one influence the other? A brief review. J Chemother 1994; 6(Supp 4):33–37.

335. Panlilio AL, Culver DH, Gaynes RP, et al. Methicillin-resistant Staphylococcus aureus in U.S. hospitals, 1975–1991. Infect Control Hosp Epidemiol 1992; 13:582–586.

336. Boyce JM. Methicillin-resistant Staphylococcus aureus: Detection, epidemiology and control measures. Infect Dis Clin N Amer 1989; 3:901–913.

337. Boyce JM. Should we vigorously try to contain and control methicillin-resistant Staphylococcus aureus? Infect Control Hosp Epidemiol 1991; 12:46–54.

338. Boyce JM, Opal SM, Byone-Potter G, et al. 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.

339. Sherertz RJ, Reagan DR, Hampton KD, et al. A cloud adult: the Staphylococcus aureus-virus interaction revisited. Ann Intern Med 1996; 124:539–547.

340. 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; 7:487–490.

341. Kreiswirth BN, Kravitz GR, Schlievert PM, Novick RP. Nosocomial transmission of a strain of Staphylococcus aureus causing toxic shock syndrome. Ann Intern Med 1986; 195:704–707.

342. Villarino ME, Vugia DJ, Bean NH, Jarvis WR, Hughes JM. Foodborne disease prevention in health care facilties. In: Bennett JV, Brachman PS, editors. Hospital Infections. 3rd ed. Boston: Little, Brown and Company, 1992:345–358.

343. American Academy of Pediatrics. Summaries of Infectious Diseases: Staphylococcal Infections. In: Peter G, editor. 1997 Redbook: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics, 1997:476–482.

344. Boyce JM, Landry M, Deetz TR, et al. Epidemiologic studies of an outbreak of nosocomial methicillin-resistant Staphylococcus aureus infections. Infect Control 1981; 2:110–116.

345. Walsh TJ, Standiford HD, Reboli AC, 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 efect of host factors on outcome. Antimicrob Agents Chemother 1993; 37:1334–1342.

346. Mulligan ME, Murray-Leisure KA, Ribner BS, 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; 94:313–328.

347. Reboli AC, John JF, Platt CG, et al. 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; 11:291–296.

348. Reagan DR, Doebbeling BN, Pfaller MA, et al. Elimination of coincident Staphylococcus aureus nasal and hand carriage with intranasal application of mupirocin calcium ointment. Ann Intern Med 1991; 114:101–106.

349. Chambers HF. Treatment of infection and colonization caused by methicillin-resistant Staphylococcus aureus. Infect Control Hosp Epidemiol 1991; 12:29–35.

350. Kauffman CA, Terpenning MS, He X, 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; 94:371–378.

351. Wenzel RP, Nettleman MD, Jones RN, et al. Methicillin-resistant Staphylococcus aureus: implications for the 1990s and effective control measures. Am J Med 1991; 91(Suppl 3B):221–227.

352. 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; 17:466–474.

353. Doebbeling BN, Reagan DR, Pfaller MA, et al. Long-term efficacy of intranasal mupirocin ointment. A prospective cohort study of Staphylococcus aureus carriage. Arch Intern Med 1994; 154:1505–1508.

354. Smith SM, Eng RH, Tecson-Tumang F. Ciprofloxacin therapy for methicillin-resistant Staphylococcus aureus infections and colonization. Antimicrob Agents Chemother 1989; 33:181–184.

355. Darouiche R, Wright C, Hamill R, et al. Eradication of colonization by methicillin-resistant and topical mupirocin. Antimicrob Agents Chemother 1991; 35:1612–1615.

356. Arathoon EG, Hamilton JR, Platt CG, et al. 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; 34:1655–1659.

357. Bartzokas CA, Paton JH, Gibson MF, et al. Control and eradication of methicllin-resistant Staphylococcus aureus on a surgical unit. N Engl J Med 1984; 311:1422–1425.

358. Ward TT, Winn RE, Hartstein AL, et al. Observations relating to an inter-hospital outbreak of methicillin-resistant Staphylococcus aureus: role of antimicrobial therapy in infection control. Infect Control 1981; 2:453–459.

359. Strasbaugh LJ, Jacobson C, Sewell DL, et al. Antimicrobial therapy for methicillin-resistant Staphylococcus aureus colonization for residents and staff of a veterans affairs nursing home care unit. Infect Control Hosp Epidemiol 1992; 13:151–159.

360. Miller MA, Dascal A, Portnoy J, et al. Development of mupirocin resistance among methicillin-resistant Staphylococcus aureus after widespread use of nasal mupirocin ointment. Infect Control Hosp Epidemiol 1996; 17:811–813.

361. 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 1997; 17:813–816.

362. Valenzuela TD, Hooton TM, Kaplan EL, et al. Transmission of toxic strep syndrome from an infected child to a firefighter during CPR. Ann Emerg Med 1991; 20:90–92.

363. Rammelkamp CH, Mortimer EA, Wolinsky E. Transmission of streptococcal and staphylococcal infection. Ann Intern Med 1964; 60:753–758.

364. Weber DJ, Rutala WA, Denny FW, Jr. Management of healthcare workers with pharyngitis or suspected streptococcal infections. Infect Control Hosp Epidemiol 1996; 17:753–761.

365. 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; 323:968–972.

366. Viglionese A, Nottebart VF, Bodman HA, et al. Recurrent group A streptococcal carriage in a health care worker associated with widely separated nosocomial outbreaks. Am J Med 1991; 91:329S–333S.

367. Paul SM, Genese C, Spitalny K. Postoperative group A B-hemolytic steptococcus outbreak with the pathogen traced to a member of a healthcare worker's household. Infect Control Hosp Epidemiol 1990; 11:643–646.

368. Ridgway EJ, Allen KD. Clustering of group A streptococcal infections on a burns unit: important lessons in outbreak management. J Hosp Infect 1993; 25:173–182.

369. Berkelman RL, Martin D, Graham DR, et al. Streptococcal wound infections caused by a vaginal carrier. JAMA 1982; 247:2680–2682.

370. Schaffner W, Lefkowitz LB, Jr., Goodman JS, et al. Hospital outbreak of infections with group A streptococci traced to an asymptomatic anal carrier. N Engl J Med 1969; 280:1224–1225.

371. Richman DD, Breton SJ, Goldmann DA. Scarlet fever and group A streptococcal surgical wound infection traced to an anal carrier. J Pediatr 1977; 90:387–390.

372. Decker MD, Lavely GB, Hutcheson RHJ, Schaffner W. Food-borne streptococcal pharyngitis in a hospital pediatrics clinis. JAMA 1986; 253:679–681.

373. Stromberg A, Schwan A, Cars O. Throat carrier rates of beta-hemolytic streptococci among healthy adults and children. Scand J Infect Dis 1988; 20:411–417.

374. Stamm WE, Feeley JC, Facklam RR. Wound infection due to group A streptococcus traced to a vaginal carrier. J Infect Dis 1978; 138:287–292.

375. American Academy of Pediatrics. Summaries of Infectious Diseases: Group A Streptococcal Infections. In: Peter G, editor. 1997 Redbook: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: American Academy of Pediatrics, 1997:483–494.

376. Barnes PF, Bloch AB, Davidson PT, et al. Tuberculosis in patients with human immunodeficiency syndrome. N Engl J Med 1991; 324:1644–1650.

377. Centers for Disease Control and Prevention. Guidelines for preventing the transmission of Mycobacterium tuberculosis in health-care facilities, 1994. MMWR 1994; 43(RR–13):1–132.

378. 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; 326:1514–1521.

379. Stroud LA, Tokars JI, Grieco MH, et al. Evaluation of infection control measures in preventing the nosocomial transmission of multidrug-resistant Mycobacterium tuberculosis in a New York City hosptial. Infect Control Hosp Epidemiol 1995; 16:141–147.

380. Beck-Sague 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; 268:1280–1286.

381. Wenger PN, Otten J, Breeden A, et al. Control of nosocomial transmission of multidrug-resistant Mycobacterium tuberculosis among healthcare workers and HIV-infected patients. Lancet 1995; 345:235–240.

382. 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; 267:2632–2635.

383. Pearson ML, Jereb JA, Frieden TR, et al. Nosocomial transmission of mutidrug-resistant Mycobacterium tuberculosis: a risk to patients and health care workers. Ann Intern Med 1992; 117:191–196.

384. 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.

385. Sepkowitz KA. Tuberculosis and the health care worker: a historical perspective. Ann Intern Med 1994; 120:71–79.

386. Menzies D, Fanning A, Yuam L, et al. Tuberculosis among health care workers. N Engl J Med 1995; 332(2):92–98.

387. Zaza S, Blumberg HM, Beck-Sague C, et al. Nosocomial transmission of Mycobacterium tuberculosis: Role of heatlh care workers in outbreak propagation. J Infect Dis 1995; 172:1542–1549.

388. Jarvis WR. Nosocomial transmission of multi-drug resistant Mycobacterium tuberculosis. Am J Infect Control 1995; 23:146–151.

389. Ikeda RM, Birkhead GS, DiFerinando G, Bornstein DL, Dooley SW, Kubica GP, et al. Nosocomial tuberculosis: an outbreak of a strain resistant to seven drugs. Infect Control Hosp Epidemiol 1995; 16:152–159.

390. Ussery XT, Bierman JA, Valway S., et al. Transmission of multidrug-resistant Mycobacterium tuberculosis among persons exposed in a medical examiner's office, New York. Infect Control Hosp Epidemiol 1995; 16:160–165.

391. Hutton MD, Stead WW, Cauthen GM, Bloch AB, Ewing WM. Nosocomial transmission of tuberculosis associated with a draining abscess. J Infect Dis 1990; 161:286–295.

392. 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; 119:594–595.

393. Rattner SL, Fleischer JA, Davidson BL. Tuberculin positivity and patient contact in healthcare workers in the United States. Infect Control Hosp Epidemiol 1996; 17:369–371.

394. Pugliese G, Tapper ML. Tuberculosis control in health care. Infect Control Hosp Epidemiol 1996; 17:819–827.

395. Maloney SA, Pearson ML, Gordon MT, et al. Efficacy of control measures in preventing nosocomial transmission of multidrug-resistant tuberculosis to patients and health care workers. Ann Intern Med 1995; 122:90–95.

396. American Thoracic Society, Centers for Disease Control. Diagnostic standards and classification of tuberculosis. Am Rev Respir Dis 1990; 142:725–735.

397. Centers for Disease Control. Screening for tuberculosis and tuberculosis infection in high-risk populations and the use of preventive therapy for tuberculous infection in the United States. MMWR 1990; 39(RR–8):1–12.

398. Centers for Disease Control and Prevention. Management of persons exposed to multidrug-resistant tuberculosis. MMWR 1992; 41(RR–11):59–71.

399. Colditz G, Brewer T, Berkey C, et al. Efficacy of BCG vaccine in the prevention of tuberculosis. JAMA 1994; 271:698–702.

400. Rodrigues L, Diwan D, Wheeler J. Protective effect of BCG against tuberculosis meningitis and miliary tuberculosis: A meta-analysis. Int J Epidemiol 1993; 22:1154–1158.

401. 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 Avisory Committee on Immunization Practices. MMWR 1996; 45(RR–4):1–18.

402. Lotte A, Wasz-Hockert O, Poisson N, et al. Second IUATLD study on complications induced by intradermal BCG vaccination. Bull Int Union Tuberc 1988; 63:47–59.

403. Caglayan S, Yegin O, Kayran K, et al. Is medical therapy effective for regional lymphadenitis following BCG vaccination? Am J Dis Child 1987; 141:1213–1214.

404. Brewer T, Colditz G. Bacille Calmette-Guerin vaccination for prevention of tuberculosis in health care workers. Clin Infect Dis 1995; 20:136–142.

405. Centers for Disease Control and Prevention. Disseminated Mycobacterium bovis infection from BCG vaccination of a patient with acquired immunodeficiency syndrome. MMWR 1985; 16:227–228.

406. Ninane J, Grymonprez A, Burtonboy G, et al. Disseminated BCG in HIV infection. Arch Dis Child 1988; 63:1268–1269.

407. Smith E, Thybo S, Bennedsen J. Infection with Mycobacterium bovis in an patient with AIDS: a late complication of BCG vaccination. Scand J Infect Dis 1992; 24:109–110.

408. von Reyn CF, Clements CJ, Mann JM. Human immunodeficiency virus infection and routine childhood immunisation. Lancet 1987; 2:669–672.

409. Comstock GW, Edwards LB, Nabangxang H. Tuberculin sensitivity eight to fifteen years after BCG vaccination. Am Rev Respir Dis 1971; 103:572–275.

410. Guld J, Waaler H, Sundaresan TK, et al. 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; 39:829–836.

411. Orefici G, Scopeti F, Grandolfo ME, et al. Study of a BCG vaccine: influence of dose and time. Boll Ist Sieroter Milan 1982; 39:829–836.

412. Fine PEM, Ponnighaus JM, Maine NP. The relationship between delayed type hypersensitivity and protective immunity induced by mycobacterial vaccines in man. Lepr Rev 1986; 57 (suppl):275–283.

413. Fine PEM, Sterne JAC, Ponnighaus JM, Rees RJW. Delayed-type hypersensitivity, mycobacterial vaccines and protective immunity. Lancet 1994; 344:1245–1249.

414. American Thoracic Society/Centers for Disease Control. The tuberculin test. Am Rev Respir Dis 1981; 124:356–363.

415. Lane JM, Ruben FL, Neff JM, Millar JD. Complications of smallpox vaccination, 1968: results of ten statewide surveys. J Infect Dis 1970; 122:303–309.

416. Centers for Disease Control and Prevention. Contact spread of vaccinia from a recently vaccinated Marine—Louisiana. MMWR 1984; 33:37–38.

417. Centers for Disease Control. Contact spread of vaccinia from a National Guard vaccinee—Wisconsin. MMWR 1985; 34:182–183.

418. Centers for Disease Control and Prevention. Vaccinia outbreak—Newfoundland. MMWR 1981; 30:453–455.

419. Meyers JD, MacQuarrie MB, Merigan TC, et al. Varicella. Part 1: Outbreak in oncology patients at a children's hospital. West J Med 1979; 130:196–199.

420. 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; 93:414–419.

421. Baltimore RS. Infections in the pediatric intensive care unit. Yale J Biol Med 1984; 57:185–197.

422. Gustafson TL, Shebab A, Brunell PA. Outbreak of varicella in a newborn intensive care nursery. Am J Dis Child 1984; 138:548–550.

423. 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.

424. Weitekamp MR, Schan P, Aber RC. An algorithm for the control of varicella-zoster virus. Am J Infect Control 1985; 13:193–198.

425. Alter SJ, Hammond JA, McVey CJ, et al. Susceptibility to varicella-zoster virus among adults at high risk for exposure. Infect Control 1986; 7:448–451.

426. Krasinski K, Holzman RS, LaCouture R, et al. Hospital experience with varicella-zoster virus. Infect Control 1986; 7:312–316.

427. Haiduven-Griffiths D, Fecko H. Varicella in hospital personnel: a challenge for the infection control practitioner. Am J Infect Control 1987; 15:207–211.

428. Weber DJ, Rutala AW, Parham C. Impact and costs of varicella prevention in a university hospital. Am J Public Health 1988; 78:19–23.

429. 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.

430. Weber DJ, Rutala WA, Hamilton H. Prevention and control of varicella-zoster infections in healthcare facilities. Infect Control Hosp Epidemiol 1996; 17:694–705.

431. American Academy of Pediatrics. Summaries of Infectious Diseases: Varicella. In: Peter G, editor. 1997 Redbook: Report of the Committee on Infectious Diseases. 24th ed. Elk Grove Village, IL: Americn Academy of Pediatrics, 1997:-573.

432. Josephson A, Gombert M. Airborne transmission of nosocomial varicella from localized zoster. J Infect Dis 1988; 158:238–241.

433. Asano Y, Iwayama S, Miyata T, Yazaki T, Ozaki T, Tsukuki 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; 23:157–161.

434. Sawyer MH, Chamberlin CJ, Wu YN, et al. Detection of varicella-zoster virus DNA in air samples from hospital room. J Infect Dis 1994; 169:91–94.

435. LeClair JM, Zaia JA, Levin MJ, et al. Airborne transmission of chickenpox in a hospital. N Engl J Med 1980; 302:450–453.

436. Gustafson TL, Lavely GB, Brawner ER, Jr., et al. An outbreak of airborne nosocomial varicella. Pediatrics 1982; 70:550–556.

437. Anderson JD, Bonner M, Scheifele DW, et al. Lack of nosocomial spread of varicella in a pediatric hospital with negative pressure ventilated patient rooms. Infect Control 1985; 6:120–121.

438. 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; 15:347–351.

439. Kelley PW, Petruccelli 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; 266:2724–2729.

440. Struewing JP, Hyams KC, Tueller JE, et al. The risk of measles, mumps, and varicella among young adults: a serosurvey of US Navy and Marine Corps recruits. Am J Public Health 1993; 83:1717–1720.

441. Gershon AA, Steinberg SP, LaRussa P, et al. Immunization of healthy adults with live attenuated varicella vaccine. J Infect Dis 1988; 1:132–137.

442. Gardner P, Eickhoff T, Poland GA, et al. Adult immunizations [update]: recommendations of the American College of Physicians. Ann Intern Med 1996; 124:35–40.

443. 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.

444. Bernstein HH, Rothstein EP, Pennridge Pediatric Associates, Watson BM, Reisenger KS, Blatter MM, et al. Clinical survey of natural varicella compared with breakthrough varicella after immunization with live attenuated Oka/Merck varicella vaccine. Pediatrics 1993; 92:833–837.

445. Weibel RE, Neff BJ, Kuter BJ, Guess HA, Rothenberger CA, Fitzgerald AJ, et al. Live attenuated varicella vaccine: efficacy trial in healthy chilren. N Engl J Med 1984; 310:1409–1415.

446. Tsolia M, Gershon AA, Steinberg SP, et al. 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; 116:185–189.

447. Centers for Disease Control and Prevention. Varicella-related deaths among adults—United States, 1997. MMWR 1997; 46:409–412.

448. Wallace MR, Bowler WA, Murray NB, Brodine SK, Oldfield ELI. Treatment of adult varicella with oral acyclovir: a randomized, placebo-controlled trial. Ann Intern Med 1992; 117:358–363.

449. Centers for Disease Control, Hospital Infection Control Practices Advisory Committee. Guideline for prevention of nosocomial pneumonia. Infect Control Hosp Epidemiol 1994; 15:587–627.

450. Balkovic ES, Goodman RA, Rose FB, et al. Nosocomial influenza A(H1N1) infection. Am J Med Tech 1980; 46:318–320.

451. Blumenfeld HL, Kilbourne ED, Louria DB, et al. Studies on influenza pandemic of 1957–1958. I. An epidemiologic, clinical and serologic investigation of an intrahospital epidemic, with a note on vaccine efficacy. J Clin Invest 1959; 38:199–212.

452. Kapila R, Lintz DI, Tecson FT, et al. A nosocomial outbreak of influenza A. Chest 1977; 71:576–579.

453. Kimball AM, Foy HM, Cooney MK, et al. Isolation of respiratory and influenza viruses from the sputum of patients

hospitalized with pneumonia. J Infect Dis 1983; 147:181–184.
454. Van Voris LP, Belshe RB, Shaffer JL. Nosocomial influenza B virus infection in the elderly. Ann Intern Med 1982; 96:153–158.
455. Pachucki CT, Walsh Pappas SA, Fuller GF, et al. Influenza A among hospital personnel and patients: implications for recognition, prevention, and control. Arch Intern Med 1990; 149:77–80.
456. Centers for Disease Control. Suspected nosocomial influenza cases in an intensive care unit. MMWR 1988; 37:3–4, –9.
457. Hammond GW, Cheang M. Absenteeism among hospital staff during an influenza epidemic: implications for immunoprophylaxis. Can Med Assoc J 1984; 131:449–452.
458. Horman JT, Stetler HC, Israel E, et al. An outbreak of influenza A in a nursing home. Am J Public Health 1986; 76:501–504.
459. Patriarca PA, Weber JA, Parker RA, et al. Risk factors for outbreaks of influenza in nursing homes: a case-control study. Am J Epidemiol 1986; 124:114–119.
460. Centers for Disease Control and Prevention. Outbreak of influenza A in a nursing home—New York, December, 1991–January, 1992. MMWR 1992; 41:129–131.
461. Gross PA, Rodstein M, LaMontagne JR, et al. Epidemiology of acute respiratory illness during an influenza outbreak in a nursing home. Arch Intern Med 1988; 148:559–561.
462. Cartter ML, Renzullo PO, Helgerson SD, et al. Influenza outbreaks in nursing homes: how effective is influenza vaccine in the institutionalized elderly? Infect Control Hosp Epidemiol 1990; 11:473–478.
463. Bean B, Moore BM, Sterner B, et al. Survival of influenza viruses on environmental surfaces. J Infect Dis 1982; 146:47–51.
464. Kilbourne ED. Influenza. New York, NY: Plenum Publishing, 1987.
465. Hall CB, Douglas RG. Nosocomial influenza infection as a cause of intercurrent fevers in infants. Pediatrics 1975; 55:673–677.
466. Noble GR. Epidemiological and clinical aspects of influenza. In: Beare AS, editor. Applied Influenza Research. Boca Raton, FL: CDC Press, 1982:11–49.
467. Adal KA, Flowers RH, Anglim AM, et al. Prevention of nosocomial influenza. Infect Control Hosp Epidemiol 1996; 17:641–648.
468. Nichol KL, Margolis KL, Lind A, et al. Side effects associated with influenza vaccination in healthy working adults: a randomized, placeo-controlled trial. Arch Intern Med 1996; 156:1546–1550.
469. Arden NH, Patrirca PA, Fasana MB, et al. The roles of vaccination and amantadine prophylaxis in controlling an outbreak of influenza A (H3N2) in a nursing home. Arch Intern Med 1988; 148:865–868.
470. Pachucki CT, Pappas SA, Fuller GF, Krause SL, Lentino JR, Schaaff DM. Influenza A among hospital personnel and patients; implications for recognition, prevention, and control. Arch Intern Med 1989; 149:77–80.
471. Falsey AR, Cunningham CK, Barker WH, et al. Respiratory syncytial virus and influenza A infections in the hospitalized elderly. J Infect Dis 1995; 172:389–394.
472. Valenti WM, Clarke TA, Hall CB, et al. Concurrent outbreaks of rhinovirus and respiratory syncytial virus in an intensive care nursery: epidemiology and associated risk factors. J Pediatr 1982; 100:722–726.
473. Hall CB. Respiratory syncytial virus: its transmission in the hospital environment. Yale J Biol Med 1982; 55:219–223.
474. Snydman DR, Greer C, Meissner HC, et al. Prevention of nosocomial transmission of respiratory syncytial virus in a newborn nursery. Infect Control Hosp Epidemiol 1988; 9:105–108.
475. Harrington RD, Hooton TM, Hackman RC, et al. An outbreak of respiratory syncytial virus in a bone marrow transplant center. J Infect Dis 1992; 165:987–993.
476. Guidry GG, Black-Payne CA, Payne DK, et al. Respiratory syncytial virus infection among intubated adults in a university medical intensive care unit. Chest 1991; 100:1377–1384.
477. Falsey AR. Noninfluenza respiratory virus infection in long-term care facilities. Infect Control Hosp Epidemiol 1991; 12:602–608.
478. Sorvillo FJ, Huie SF, Strassburg MA, et al. An outbreak of respiratory syncytial virus pneumonia in a nursing home for the elderly. J Infect 1984; 9:252–256.
479. Valenti WM, Hruska JF, Menegus MA, et al. Nosocomial viral infections: III. guidelines for prevention and control of exanthematous viruses, gastroenteritis viruses, picornaviurses, and uncommonly seen viruses. Infect Control 1980; 2:38–49.
480. 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, 1996:22–1–22–8.
481. Valenti WM. Infection control and the pregnant health care worker. Nurs Clin North Am 1993; 28:673–686.
482. Shortridge-McCauley LA. Reproductive hazards: an overview of exposures to health care workers. AAOHN Journal 1995; 43:614–621.
483. Pike RM. Past and present hazards of working with infectious agents. Arch Pathol Lab Med 1978; 102:333–336.
484. Pike RM. Laboratory-associated infections: incidence, fatalities, causes, and prevention. Ann Rev Microbiol 1979; 33:41–66.
485. Favero MS. Biological hazards in the laboratory. Lab Med 1987; 18:665–670.
486. Jacobson JT, Orlob RB, Clayton JL. Infections acquired in clinical laboratories in Utah. J Clin Micro 1985; 21:486–489.
487. Grist NR, Emslie JA. Infections in British clinical laboratories, 1986–1987. J Clin Pathol 1989; 42:677–681.
488. Vesley D, Hartmann HM. Laboratory-acquired infections and injuries in clinical laboratories: a 1986 survey. Am J Pub Health 1988; 78:1213–1215.
489. Grist NR, Emslie JA. Association of Clinical Pathologists' survey of infection in British clinical laboratories, 1970–1989. J Clin Pathol 1994; 47:391–394.
490. Gilchrist MJR. Laboratory safety management. In: Isenberg HI, editor. Clinical microbiology procedures handbook. Washington, D.C. American Society for Microbiology, 1992:
491. Gilchrist MJR, Hindler J, Fleming D. Laboratory safety management update—aerosol borne microorganisms. In: Isenberg HI, editor. Clinical microbiology procedures handbook. Washington, D.C. American society for Microbiology, 1994:
492. 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, D.C. American Society for Microbiology, 1995:
493. Centers for Disease Control and Prevention. Implementation of provisions of the Ryan White Comprehensive AIDS Resources Emergency Act regarding emergency response employees. Fed Reg 1994; 59(54):13418–13428.
494. Centers for Disease Control. Update: human immunodeficiency virus infection in health care workers exposed to the blood of infectious patients. MMWR 1987; 36:285–289.
495. Hamann CP. Natural rubber latex protein sensitivity in review. Contact Dermatitis 1993; 4:4–21.
496. Zaza S, Reeder JM, Charles LE, et al. Latex sensitivity among perioperative nurses. AORN J 1994; 60:806–812.
497. Bubak ME, Reed CE, Fransway AF, et al. Allergic reactions to latex among health-care workers. Mayo Clin Proc 1992; 67:1075–1079.
498. Berky ZT, Luciano WJ, James WD. Latex glove allergy: a survey of the US Army Dental Corps. JAMA 1992; 268:2695–2697.
499. Yassin MS, Lierl MB, Fischer TJ, et al. Latex allergy in hospital employees. Ann Allergy 1994; 72:245–249.
500. Fisher AA. Allergic contact reactions in health personnel. J Allergy Clin Immunol 1992; 90:729–738.
501. Hunt LW, Fransway AF, Reed CE, et al. An epidemic of occupational allergy to latex involving health care workers. J Occup Environ Med 1995; 37:1204–1209.
502. Alenius H, Makinen-Kiljunes S, Turjanmaa K, et al. Allergen and protein content of latex gloves. Ann Allergy 1994; 73:315–320.
503. Field EA. Hypoallergenic gloves. Int Dental J 1995; 45:339–346.
504. Yunginger JW, Jones RT, Fransway AF, et al. Extractable latex allergens and proteins in disposable medical gloves and other rubber products. J Allergy Clin Immunol 1994; 93:836–842.
505. Food and Drug Administration. Latex-containing devices; user labeling. Fed Regist 1996; 61:32617–32621.
506. Jaeger D, Kleinhans D, Czuppon AB, et al. Latex-specific proteins causing immediate-type cutaneous, nasal, bronchial, and systemic reactions. J Allergy Clin Immunol 1992; 89:759–768.
507. Sussman GL, Tario S, Dolovich J. The spectrum of IgE-mediated responses to latex. JAMA 1991; 265:2844–2847.
508. Cormio L, Turjanmaa K, Talja M, et al. Toxicity and immediate allergenicity of latex gloves. Clin Exp Allergy 1993; 23:618–623.
509. Hamann CP, Kick SA. Update: immediate and delayed hypersensitivity to natural rubber latex. Cutis 1993; 52:307–311.
510. Ownby DR. Manifestation of latex allergy. Immun Allergy Clin North Am 1995; 15:31–43.
511. Estlander T, Jolanski R, Kanerva L. Dermatitis and urticaria from rubber and

plastic gloves. Contact Dermatitis 1985; 14:20–25.

512. Conde-Salazar L, del-Rio E, Guimaraens D, et al. Type IV allergy to rubber additives: a 10-year study of 686 cases. J Am Acad Dermatol 1993; 29:176–180.

513. Heese A, Hintzenstern J, Peters KP, et al. Allergic and irritant reactions to rubber gloves in medical health services. J Am Acad Dermatol 1991; 25:831–839.

514. Lagier F, Vervloet D, Lhermet I, et al. Prevalence of latex allergy in operating room nurses. J Allergy Clin Immunol 1992; 90(3 pt 1):319–322.

515. Gerber AC, Jorg W, Zbinden S, et al. Severe intraoperative anaphylaxis to surgical gloves: latex allergy, an unfamiliar condition. Anesthesiology 1989; 71:800–802.

516. Arellano R, Bradley J, Sussman G. Prevalance of latex sensitization among hospital physicians occupationally exposed to latex gloves. Anesthesiology 1992; 77:905–908.

517. Kaczmarek RJ, Silverman BJ, Gross TP, et al. Prevalence of latex-specific IgE antibodies in hospital personnel. Ann Allergy Asthma Immunol 1996; 76:51–56.

518. Marcos C, Lazaro M, Fraj J, et al. Occupational asthma due to latex surgical gloves. Ann Allergy 1991; 67:319–323.

519. Frosch PJ, Wahl R, Bahmer FA, et al. Contact urticaria to rubber gloves is IgE-mediated. Contact Dermatitis 1986; 14:241–245.

520. Vandenplas O, Delwiche J, Evrard G, et al. Prevalence of occupational asthma due to latex among hospital personnel. Am J Respir Crit Care Med 1995; 151:54–60.

521. Tarlo SM, Wong L, Roos J, et al. Occupational asthma caused by latex in a surgical glove manufacturing plant. J Allergy Clin Immunol 1990; 85:626–631.

522. Seaton A, Cherrie B, Turnbull J. Rubber glove asthma. Br Med J 1988; 296:531–532.

523. O'Byrne PM, Dolovich J, Hargreave FE. Late asthmatic responses. Am Rev Respir Dis 1987; 136:130–131.

524. De Zotti R, Larese F, Fiorito A. Asthma and contact urticaria from latex gloves in a hospital nurse. Br J Ind Med 1992; 49:596–598.

525. Brugnami G, Marabini A, Siracusa A, et al. Work-related late asthmatic response induced by latex allergy. J Allergy Clin Immunol 1995; 96:457–464.

526. Grzybowski M, Ownby DR, Peyser PA, et al. The prevalence of anti-latex IgE antibodies among registered nurses. J Allergy Clin Immunol 1996; 98:535–544.

527. Turjanmaa K. Incidence of immediate allergy to latex gloves in hospital personnel. Contact Dermatitis 1987; 17:270–275.

528. Swanson MC, Bubak ME, Hunt LW, et al. Quantification of occupational latex aerollergens in a medical center. J Allergy Clin Immunol 1994; 94:455–451.

529. Shield SW, Blaiss MS. Prevalence of latex sensitivity in children evaluated for inhalant allergy. Allergy Proc 1992; 13:129–131.

530. M'Raihi L, Cahrpin D, Pons A, et al. Cross-reactivity between latex and banana. J Allergy Clin Immunol 1991; 87:129–130.

531. Kurup VJ, Kelly T, Elms N, et al. Cross-reactivity of food allergens in latex allergy. Allergy Proc 1994; 15:211–216.

532. Blanco C, Carrillo T, Castillo R, et al. Avocado hypersensitivity. Allergy 1994; 49:454–459.

533. Ahlroth M, Alenius H, Turjanmaa K, et al. Cross-reacting allergens in natural rubber latex and avocado. J Allergy Clin Immunol 1995; 96:167–173.

534. Fernandez de Corres L, Moneo I, Munoz D, et al. Sensitization from chestnuts and bananas in patients with urticaria and anaphylaxis from contact with latex. Ann Allergy 1993; 70:35–39.

535. Kelly KJ, Kurup V, Zacharisen M, et al. Skin and serologic testing in the diagnosis of latex allergy. J Allergy Clin Immunol 1993; 91:1140–1145.

536. Equal Employment Opportunity Commission. Equal employment opportunity for individuals with disabilities. 29 CFR 1630. Fed Reg 1991; 56:35726–35753.

537. Bureau of National Affairs. Title VII Jurisdiction: Equal Opportunity Commission Compliance Manual. 1986; 147–149. Washington, D.C.

538. Department of Justice. Title II Technical Assistance Manual: The Americans with Disabilities Act. 1993; 1–12. Washington, D.C.

539. Department of Justice. Title III Technical Assistance Manual: The Americans with Disabilities Act. 1993; 2–14. Washington, D.C.

540. Khuri-Bulos NA, Khalaf MA, Shehabi A, et al. Foodhandler-associated *Salmonella* outbreak in a university hospital despite routine surveillance cultures of kithchen employees. Infect Control Hosp Epidemiol 1994; 15:311–314.

541. Moyer LA, Alter MJ, Favero MS. Hemodialysis-associated hepatitis B: revised recommendations for serologic screening. Semin Dialysis 1990; 3:201–204.

542. Pether JVS, Scott RHD. *Salmonella* carriers: are they dangerous? A study to identify finger contamination with Salmonellae by convalescent carriers. J Infect 1982; 5:81–88.

543. Stover BH, Kuebler CA, Cost KM, et al. Measles-mumps-rubella immunization of susceptible hospital employees during a community measles outbreak: cost-effectiveness and protective efficacy. Infect Control Hosp Epidemiol 1994; 15:20–23.

544. Polder JA, Tablan OC, Williams WW. Personnel health services. In: Bennett JV, Brachman PS, eds. editors. Hospital Infections. 3rd ed. Boston: Little, Brown and Company, 1992:31–61.

BILLING CODE 4163–18–P

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

A. 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 weeks apart; third dose 5 months after second; 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 months 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 years of age.	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 subcutaneously (SC); second dose at least 1 month later.	Health care personnel born in or after 1957 without documentation of a) receipt of two doses of live vaccine on or after their first 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 following 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) with a vaccine of unknown type should be revaccinated with two doses of live measles vaccine.

* Persons immunocompromised because of immune deficiency diseases, HIV infection (who should primarily not receive BCG, OPV, and yellow fever vaccines), leukemia, lymphoma or generalized malignancy or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

A. Immunizing Agents Strongly Recommended for Health Care Personnel (con't)

Generic name	Primary/booster dose schedule	Indications	Major precautions and contraindications	Special considerations
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 following 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 first 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 following receipt of neomycin.	Women pregnant when vaccinated or who become pregnant within 3 months of vaccination should be counseled on the theoretical 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 weeks apart if ≥13 years of age.	Health care personnel without reliable history of varicella or laboratory evidence of varicella immunity.	Pregnancy, immunocompromised* state, history of anaphylactic reaction following receipt of neomycin or gelatin. Salicylate use should be avoided for 6 weeks after vaccination.	Because 71%-93% of persons without a history of varicella are immune, serologic testing prior to vaccination may be cost-effective.

* Persons immunocompromised because of immune deficiency diseases, HIV infection (who should primarily not receive BCG, OPV, and yellow fever vaccines), leukemia, lymphoma or generalized malignancy or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

B. 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) multidrug-resistant tuberculosis is prevalent, b) a strong likelihood of infection exists, and c) full implementation of TB infection control precautions has been inadequate in controlling the spread of infection. NOTE: BCG should be used after consultation with local and/or state health department	Immunocompromised* state and pregnancy	In the United States, tuberculosis-control efforts are directed towards early identification and treatment of cases of active tuberculosis, and preventive therapy with isoniazid for PPD converters.
Hepatitis A vaccine	Two doses of vaccine IM either (HAVRIX®) 6-12 months apart or (VAQTA®) 6 months apart.	Not routinely indicted for health care personnel in the USA. 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 the risk of infection is high	May be useful in certain outbreak situations (see text).
Pneumococcal polysaccharide vaccine (23 valent)	One dose IM or SC; revaccination recommended for those at highest risk ≥5 years after the first dose	Adults who are at increased risk of pneumococcal disease and its complications because of underlying health conditions; older adults, especially those who are ≥65 of age and healthy.	The safety of the vaccine in pregnant women has not been evaluated; it should not be given during pregnancy unless the risk of infection is high.	Previous recipients of any type of pneumococcal polysaccharide vaccine who are at highest risk of fatal infection or antibody loss may be revaccinated ≥5 years after the first dose.

* Persons immunocompromised because of immune deficiency diseases, HIV infection (who should primarily not receive BCG, OPV, and yellow fever vaccines), leukemia, lymphoma or generalized malignancy or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

B. Other Immunizing Agents Available for Health Care Personnel in Special Circumstances (con't)

Generic name	Primary/booster dose schedule	Indications	Major precautions and contraindications	Special considerations
Polio vaccine	IPV, two doses SC given 4-8 weeks apart, followed by a third dose at 6-12 months after the second 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. The safety of the vaccine in pregnant women has not been evaluated; it should not be given during pregnancy.	Use only IPV for immunosuppressed persons or personnel who care for immunosuppressed patients. If immediate protection against polio 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, and 28. or HDCV, intradermally (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 (Reference 20).
Tetanus and diphtheria (toxoids [Td])	Two doses intramuscularly (IM) 4 weeks apart; third dose 6-12 months after second dose; booster every 10 years.	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 following previous dose of Td vaccine should not be given further routine or emergency doses of Td for 10 years.	

* Persons immunocompromised because of immune deficiency diseases, HIV infection (who should primarily not receive BCG, OPV, and yellow fever vaccines), leukemia, lymphoma or generalized malignancy or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

B. Other Immunizing Agents Available for Health Care Personnel in Special Circumstances (con't)

Generic name	Primary/booster dose schedule	Indications	Major precautions and contraindications	Special considerations
Typhoid vaccines: IM, SQ, and oral	One 0.5-ml dose IM; booster doses of 0.5 ml every 2 years. (Vi CPS) OR Two 0.5 ml doses SC, 4 or more weeks apart; boosters of 0.5 ml SC or 0.1 ml intradermally every 3 years if exposure continues. OR Four oral doses on alternate days. (Ty 21a) Vaccine manufacturer's recommendation: revaccination with the entire four-dose series every 5 years.	Personnel in laboratories who frequently work with *Salmonella typhi*.	History of severe local or systemic reaction to a previous dose of typhoid vaccine. Ty 21a 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 years.	Personnel who directly handle cultures of, or animals contaminated with recombinant vaccinia viruses, or orthopox viruses (monkeypox, cowpox, vaccinia, etc.) that infect humans.	Pregnancy, presence or history of eczema, or immunodeficiency in the 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.

* Persons immunocompromised because of immune deficiency diseases, HIV infection (who should primarily not receive BCG, OPV, and yellow fever vaccines), leukemia, lymphoma or generalized malignancy or immunosuppressed as a result of therapy with corticosteroids, alkylating drugs, antimetabolites, or radiation.

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

C. Diseases for Which Postexposure Prophylaxis may be Indicated for Health Care Personnel

Disease	Prophylaxis	Indications	Major precautions and contraindications	Special considerations
Diphtheria	Benzathine penicillin, 1.2m units IM, single dose or erythromycin (1g/d) PO x 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 years.
Hepatitis A	One IM dose of IG 0.02 ml/kg given within 2 weeks 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 weeks after MMR vaccine, or within 3 weeks 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 a different body site). If the hepatitis B series has not been started, a second dose of HBIG should be given 1 month after the first dose.	HBV-susceptible health care personnel with percutaneous or mucous-membrane exposure to blood known to be HBsAg positive. (See Table 5)		
Meningitis	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., resuscitating, intubating, or closely examining the oropharynx of patients)	Rifampin and ciprofloxacin are not recommended during pregnancy.	
Pertussis	Erythromycin, 500 mg qid PO, OR Trimethoprim-sulfamethoxazole 1 tablet bid PO For 14 days following exposure	Personnel with direct contact with respiratory secretions or large aerosol droplets from the respiratory tract of infected persons.		

Table 1. Immunobiologics and Schedules for Health Care Personnel (adapted from ACIP recommendations, reference 7)

C. Diseases for Which Postexposure may be Indicated for Health Care Personnel (con't)

Disease	Prophylaxis	Indications	Major precautions and contraindications	Special considerations
Rabies	For those never vaccinated HRIG 20 IU/kg, one half infiltrated around would AND HDCV or RVA vaccine, 1.0 ml, IM (deltoid area), one each on days 0, 3, 7, 14, and 28.	Personnel who have been bitten by a human or animal with rabies or 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 RCV vaccine, 1.0 ml, IM, on days 0 and 3. No HRIG is necessary.
Varicella-zoster	VZIG for persons ≤50 kg: 125 u/10kg IM; for persons >50kg: 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 or complications, such as pregnant women or immunocompromised.		Serologic testing may help in assessing whether to administer VZIG. If varicella is prevented by the use of VZIG, vaccine should be offered later.

[†] 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
(adapted from APIC recommendations, reference 7)

Vaccine	Pregnancy	HIV Infection	Severe immuno-suppression*	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, Inactivated**	UI	UI	UI	UI	UI	UI	UI
Polio, Oral**	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

* 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 the 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 polio viruses. 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.

R=Recommended; **C**= Contraindicated; **UI**=Use if indicated

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 (adapted from APIC recommendations, reference 7)

Disease/problem	Work restriction	Duration	Category
Conjunctivitis	Restrict from patient contact	Until discharge ceases	II
Cytomegalovirus infections	No restriction		II
Diarrheal diseases Acute stage (diarrhea with other symptoms)	Restrict from patient contact or food-handling	Until symptoms resolve	IB
Convalescent stage *Salmonella* spp.	Restrict from care of high-risk patients	Until symptoms resolve. Consult with local and state health authorities regarding the 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, newborns, and immunocompromised patients	Until symptoms resolve	II
Hepatitis A	Restrict from patient contact 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;* 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. The panel should review and recommend the procedures the worker can perform, taking into account the specific procedure as well as the skill and technique of the worker.†	Until HBeAg is negative	II

* Unless epidemiologically liked to transmission of infection

† See Section D.1.a.

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 (adapted from ACIP recommendations, reference 7) (con't)

Disease/problem	Work restriction	Duration	Category
Hepatitis C	No recommendation		Unresolved issue
Herpes simplex			
Genital	No restriction		II
Hands (herpetic whitlow)	Restrict from patient contact.	Until lesions heal	IA
Orofacial	Restrict from care of high-risk patients.	Until lesions heal	II
Human immunodeficiency virus	Do not perform exposure-prone invasive procedures until counsel from an expert review panel has been sought. The panel should review and recommend the procedures the worker can perform, taking into account the specific procedure as well as the skill and technique of the worker. Standard Precautions should always be observed.†		II
Measles			
Active	Exclude from duty	Until 4 days after the rash appears	IA
Postexposure (Susceptible personnel)	Exclude from duty	From the 5th day after the first exposure through the 21st day after the last exposure and/or 4 days after the rash appears	IB
Mumps			
Active	Exclude from duty	Until 9 days after onset of parotitis	IB
Postexposure (Susceptible personnel)	Exclude from duty	From the 12th day after the first exposure through the 26th day after the last exposure or until 9 days after onset of parotitis	II

† See section D.1.a.

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 (adapted from ACIP recommendations, reference 7) (con't)

Disease/problem	Work restriction	Duration	Category
Pertussis			
Active	Exclude from duty	From the beginning of the catarrhal stage through the third week after onset of paroxysms or until 5 days after start of effective antimicrobial therapy	IB
Postexposure			
(Asymptomatic personnel)	No restriction, prophylaxis recommended		II
(Symptomatic personnel)	Exclude from duty	Until 5 days after start of effective antimicrobial therapy	IB
Rubella			
Active	Exclude from duty	Until 5 days after the rash appears	IA
Postexposure (Susceptible personnel)	Exclude from duty	From the 7th day after the first exposure through the 21st day after the last exposure	IB
Scabies or pediculosis infestation	Restrict from patient contact	Until treated	IB
Staphylococcus aureus			
Active, draining skin lesions	Restrict from contact with patients and patient materials or food-handling	Until lesions have resolved	IB
Carrier state	No restriction, unless personnel are shown epidemiologically to be disseminating the organism.		IB
Streptococcal infection, Group A	Restrict from patient care or food handling	Until 24 hours after adequate treatment started	IB
Tuberculosis	Exclude from duty	Until proven non-infectious	IA

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 (adapted from ACIP recommendations, reference 7) (con't)

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 the 10th day after the first exposure through the 21st day (28th day if VZIG was given) after the last exposure	IA
Zoster			
Localized, in normal 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 the 10th day after the first exposure through the 21st day (28th day if VZIG was given) after the 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† during community outbreak of RSV and influenza	Until acute symptoms resolve	IB

* 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 4. Recommendation for Postexposure Prophylaxis for Percutaneous or Permucosal Exposure to Hepatitis B Virus, United States

Exposed Person	Source HBsAg* Positive	Source HBsAg Negative	Source Not Tested or Unknown
Unvaccinated	HBIG** x 1 and initiate HB vaccine†	Initiate HB vaccine	Initiate HB vaccine
Previously vaccinated			
Known responder	No treatment	No treatment	No treatment
Known nonresponder	HBIG x 2 or HBIG x 1 and initiate revaccination	No treatment	If known high-risk source treat as if source were HBsAg positive
Response unknown	Test exposed for anti-HBs‡ 1. If adequate¶, no treatment 2. If inadequate¶, HBIG x 1 and vaccine booster	No treatment	Test exposed for anti-HBs 1. If adequate, no treatment 2. If inadequate, vaccine booster

* HBsAg = Hepatitis B surface antigen
** HBIG = Hepatitis B immune globulin; dose 0.06 mg/kg IM
† HB vaccine = Hepatitis B vaccine
‡ anti-HBs = antibody to hepatitis B surface antigen
¶ adequate anti-HBs is ≥ 10 mIU/ml

Table 5. Selected Reported Etiologic Agents Causing Community or Nosocomially Acquired Gastrointestinal Infections in Developed Countries

Agent	Community-acquired Agents in Patients	Nosocomially-acquired Agents in Patients	Nosocomially-acquired Agents in Health Care Personnel
Bacterial			
Bacillus cereus	++	0	0
Campylobacter species	++++	+	0
Clostridium difficile	+	++++	+
Clostridium perfringins	+	+	0
Diarrheogenic *Escherichia coli*	++++	++	+
Salmonella species	+++	++	+
Shigella species	++	+	+
Staphyloccus aureus, toxigenic	+++	+++	0
Yersinia enterocolitica	+	+	+
Viral			
Adenovirus	++	+	+
Astrovirus	*	*	?
Calicivirus (Norwalk and norwalk-like viruses or SRSVs)	*	*	*
Coxsackievirus	++	+	+
Rotavirus	++++	++++	++
Fungal			
Candida species	+	+	0
Cryptococcus neoformans	++	+	0
Parasitic			
Cryptosporidium	++	+	+
Cyclospora	++	0	0
Entamoeba histolytica	++	+	0
Giardia lamblia	++	+	0
Isospora belli	+	0	0
Strongyloides	+	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

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. CMV	Hearing loss; congenital syndrome*	15% after primary maternal infection; symptomatic 5%	Antibody protects against clinical disease; routine screening not recommended	Standard Precautions
2. Hepatitis B	Hepatitis; hepatocellular cancer as adult	HBeAg pos. 90% HBeAg neg. 25%	Anti-HBsAg Anti-HBeAg	Vaccine (safe during pregnancy); Standard Precautions
3. Hepatitis C	Hepatitis	0%-15%	Anti-HCV; HCV RNA in reference labs	ISG no longer contains anti-HCV and is 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. HIV	AIDS by 2-3 years of age	8%-30%	Antibody by ELISA, Western blot; PCR	Avoid high-risk behaviors; consider postexposure prophylaxis following high risk needlestick; zidovudine intrapartum and post-natal for HIV- positive 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, IgG antibody prepregnancy; antibody protective	Droplet Precautions

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

† Live-virus vaccines are given routinely prior to pregnancy.

Table 6. Pregnant Health Care Personnel: Pertinent Facts to Guide Management of Occupational Exposures to Infectious Agents (con/t)

Agent	Potential Effect on Fetus	Rate of Perinatal Transmission	Maternal Screening	Prevention
9. Rubella	Congenital syndrome*	45%-50% overall; 90% in first 12 weeks	Antibody	Vaccine†; Droplet Precautions for acute infection; Contact Precautions for congenital rubella
10. Tuberculosis	Hepatomegaly, pulmonary, CNS	Rare	Skin test	INH ± Ethambutol; 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

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

† Live-virus vaccines are given routinely prior to pregnancy.

Adapted from Siegel JD. Risk and exposure for the pregnant health-care worker. In: Olmstead RN, ed. APIC infection control and applied epidemiology: principles and practices/Association for Professionals in Infection Control and Epidemiology, Inc. St. Louis: Mosby-Year Book, Inc.;1996.22-2-22-3 (Table 22-1).

[FR Doc. 97–23677 Filed 9–5–97; 8:45 am]

BILLING CODE 4163–18–C