

response and subsequent signs and symptoms. Such reproduction injures the host by causing cellular damage from microorganism-producing toxins or intracellular multiplication or by competing with the host's metabolism.

The host's immune response may compound the tissue damage; such damage may be localized (e.g., as in infected pressure ulcers) or systemic. However, in some instances, microorganisms may be present in the tissues of the host and yet not cause symptomatic disease. This process is called *colonization of organisms*. The person with colonization may be a carrier and transmit the organisms to others but does not have detectable symptoms of infection.

The development of an infection begins with transmission of an infectious organism (agent, pathogen, pathogenic agent) and depends on a complex interaction of the pathogen, an environment conducive to transmission of the organism, and the susceptibility of the human host. Even after successful transmission of a pathogen, the host may experience more than one possible outcome.

The pathogen may merely contaminate the body surface and be destroyed by first-line defenses such as intact skin or mucous membranes that prevent further invasion, or a subclinical infection may occur in which no apparent symptoms are evident other than an identifiable immune response of the host. A rise in the titer of antibody directed against the infecting agent is often the only detectable response. Antibiotic treatment is not necessary, although infection control procedures remain in force to prevent spreading the bacteria to others.

A third possible outcome is the development of a clinically apparent infection in which the host-parasite interaction causes obvious injury and is accompanied by one or more clinical symptoms. This outcome is called *infectious disease* and ranges in severity from mild to fatal depending on the organism and the response and underlying health of the host.⁷²

The period between the pathogen entering the host and the appearance of clinical symptoms is called the *incubation period*. This period may last from a few days to several months, depending on the causative organism and type of disease. Disease symptoms herald the end of the incubation period. A *latent infection* occurs after a microorganism has replicated but remains dormant or inactive in the host, sometimes for years (e.g., tuberculosis, herpes zoster). The host may harbor a pathogen in sufficient quantities to be shed at any time after latency and toward the end of the incubation period. This time period when an organism can be shed is called the *period of communicability*.

From this concept of communicability, communicable diseases can be defined as any disease whereby the causative agent may pass or be carried from one person to another directly or indirectly. It usually precedes symptoms and continues through part or all of clinical disease, sometimes extending to convalescence; but it is important to note that an asymptomatic host can still transmit a pathogen. The communicable period, like the incubation period and mode of transmission, varies with different pathogens and different diseases.¹⁴⁴

Types of Organisms

A great variety of microorganisms are responsible for infectious diseases, including viruses, mycoplasmas, bacteria, rickettsiae, chlamydiae, protozoa, fungi (yeasts and molds), helminths (e.g., tapeworms), mycobacteria, and prions. All microorganisms can be distinguished by certain intrinsic properties such as shape, size, structure, chemical composition, antigenic makeup, growth requirements, ability to produce toxins, and ability to remain alive (viability) under adverse conditions such as drying, sunlight, or heat.

These properties provide the basis for identification and classification of the organisms. Knowledge of the properties permits diagnosis of a specific pathogen in specimens of body fluids, secretions, or exudates. All these properties are important to consider when looking for ways to interfere with the mechanisms of transmission.

Viruses are subcellular organisms made up only of a ribonucleic acid (RNA) or a deoxyribonucleic acid (DNA) nucleus covered with proteins. They are the smallest known organisms, visible only through an electron microscope. Viruses are completely dependent on host cells and cannot replicate unless they invade a host cell and stimulate it to participate in the formation of additional virus particles.

The estimated 400 viruses that infect human beings are classified according to their size, shape (spherical, rod shaped, or cubic), or means of transmission (respiratory, fecal, oral, or sexual). Viruses are not susceptible to antibiotics. However, antiviral medications can mitigate (moderate) the course of the viral illness. For example, acyclovir, an antiviral medication used for herpesvirus, interferes with DNA synthesis, causing decreased viral replication and decreasing the time of lesional healing.

Mycoplasmas are unusual, self-replicating bacteria that have no cell wall components and very small genomes. For this reason, antibiotics that are active against bacterial cell walls have no effect on mycoplasmas. At present, mycoplasmas remain sensitive to some antibiotics. They require a strict dependence on the host for nutrition and sustenance and are able to pass through many bacteria-retaining filters or barriers because they are very small.¹⁰

Bacteria are single-celled microorganisms with well-defined cell walls that can grow independently on artificial media without the need for other cells. Bacteria can be classified according to shape. Spherical bacterial cells are called *cocci*, rod-shaped bacteria are called *bacilli*, and spiral-shaped bacteria are called *spirilla* or *spirochetes*.

Bacteria can also be classified according to their response to staining (gram positive, gram negative, or acid fast), motility (motile or nonmotile), tendency toward capsulation (encapsulated or nonencapsulated), and capacity to form spores (sporulating or nonsporulating) (Fig. 8-1).

Bacteria can also be classified according to whether oxygen is needed to replicate and develop (aerobic) or whether they can sustain life in an oxygen-poor (anaerobic) environment. Anaerobic bacteria are organisms that require reduced oxygen tension for growth.

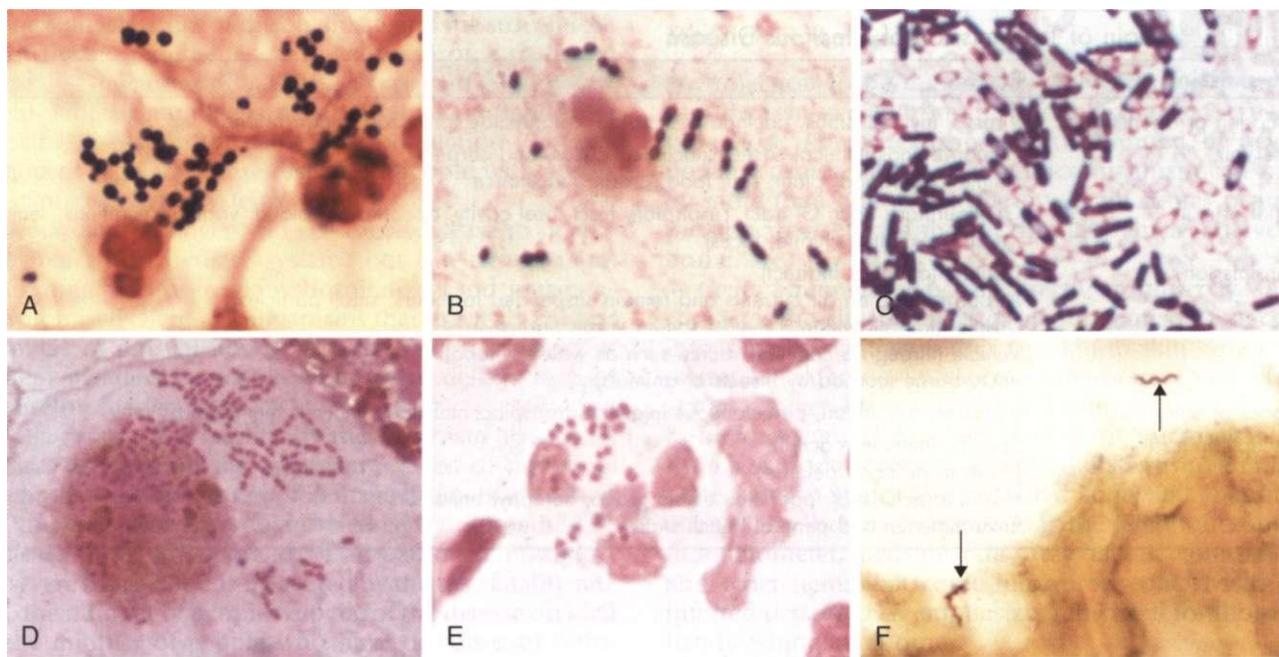


Figure 8-1

A variety of bacterial morphology. **A**, Gram stain of sputum from person with pneumonia. There are gram-positive cocci in clusters (*Staphylococcus aureus*) with degenerating neutrophils. **B**, Gram stain of sputum from an individual with pneumonia. Gram-positive, elongated cocci in pairs and short chains (*Streptococcus pneumoniae*) and a neutrophil are seen. **C**, Gram stain of *Clostridium sordellii* grown in culture. A mixture of gram-positive and gram-negative rods, many of which have subterminal spores (clear areas), are present. *Clostridia* species often stain as both gram positive and negative, although they are true gram-positive bacteria. **D**, Gram stain of a bronchoalveolar lavage specimen showing gram-negative intracellular rods typical of *Enterobacteriaceae* such as *Klebsiella pneumoniae* and *E. coli*. **E**, Gram stain of urethral discharge from someone with gonorrhea. Many gram-negative diplococci (*Neisseria gonorrhoeae*) are present within a neutrophil. **F**, Silver stain of brain tissue from a person with Lyme disease meningoencephalitis. Two helical spirochetes (*Borrelia burgdorferi*) are indicated by arrows. The panels are at different magnifications. (A to C, Reprinted from Kumar V, Abbas AK, Fausto N: *Robbins and Cotran: pathologic basis of disease*, ed 7, Philadelphia, 2005, Saunders, courtesy Dr. Kenneth Van Horn. D, Courtesy Karen Krisher, Clinical Microbiology Institute, Wilsonville, OR.)

Normal human flora are primarily anaerobic, and disease can be produced when these normal organisms are displaced from their usual tissue sites (e.g., mouth, skin, large bowel, female genital tract) into other tissues or closed body spaces.¹²⁸ Other common anaerobic organisms include the spore-forming bacilli such as *Clostridium botulinum* or *C. tetani* that thrive in a strictly anaerobic environment.

Rickettsiae are primarily animal pathogens that generally produce disease in human beings through the bite of an insect vector such as a tick, flea, louse, or mite. They are small, gram negative, obligate intracellular organisms that often cause life-threatening infections. Like viruses, these microorganisms require a host for replication. Three categories of the family Rickettsiaceae are *Rickettsia*, *Coxiella*, and *Bartonella*.¹⁹

Chlamydiae are smaller than rickettsiae and bacteria but larger than viruses. They, too, depend on host cells for replication, but unlike viruses they always contain both DNA and RNA and are susceptible to antibiotics.

Protozoa have a single cell unit or a group of nondifferentiated cells loosely held together and not forming tissues. They have cell membranes rather than cell walls, and their nuclei are surrounded by nuclear membranes. Larger parasites include roundworms and flatworms. *Fungi* are unicellular to filamentous organisms possessing hyphae (filamentous outgrowths) surrounded by cell

walls and containing nuclei (eukaryocyte). Fungi show relatively little cellular specialization and occur as yeasts (single-cell, oval-shaped organisms) or molds (organisms with branching filaments). Depending on the environment, some fungi may occur in both forms. Fungal diseases in human beings are called *mycoses*.

Prions are proteinaceous, infectious particles consisting of proteins but without nucleic acids. These particles are transmitted from animals to human beings and are characterized by a long latent interval in the host. When reactivated, they cause a rapidly progressive deteriorating state in the host (e.g., Creutzfeldt-Jakob disease, bovine spongiform encephalopathy or "mad cow disease").⁴³

The Chain of Transmission

Infection begins with transmission of a pathogen to the host. Successful transmission depends on a pathogenic agent, a reservoir, a portal of exit from the reservoir, a mode (mechanism) of transmission, a portal of entry into the host, and a susceptible host. This sequence of events is called the *chain of transmission* (Table 8-2).

Nosocomial infections are infections acquired during hospitalization. In the United States, about 5% of people who enter the hospital without infection will acquire a nosocomial infection. Transmission can be through any

Table 8-2 Chain of Transmission of Infectious Disease

Transmission Chain	Factors
Pathogen or agent	Viruses, mycoplasmas, bacteria, rickettsiae, chlamydiae, protozoa, fungi, prions
Reservoir	Human beings (clinical cases, subclinical cases, and carriers) Animal, arthropod, plant, soil, food, organic substance
Portal of exit	Genitourinary tract, GI tract, respiratory tract, oral cavity, open lesion, blood, vaginal secretions, semen, tears, excretions (urine, feces)
Transmission	Contact (direct or indirect) Airborne (float on air currents and remain suspended for hours; small particles) Droplet (fall out within 3 feet of source; large particles) Vehicle (through a common source such as water or food) Vectorborne (carried by insects or animals)
Modes of entry	Ingestion, inhalation, percutaneous injection, transplacental entry, mucous membranes
Susceptible host	Specific immune reactions Nonspecific body defenses Host characteristics: age, sex, ethnic group, heredity, behaviors Environmental and general health status

of the possible routes discussed in this section. Nosocomial infections result in prolongation of hospital stays, increase in cost of care, significant morbidity, and a mortality rate of about 5%.¹⁷

The most common infections are urinary tract infections (usually associated with Foley catheters or urologic procedures) and bloodstream infections. Bloodstream infections can occur as a result of indwelling intravenous (IV) catheters or surgical wound infections, abscesses, pneumonia (especially in intubated individuals or those with altered levels of consciousness), and GI and genitourinary infections.

In general, increases in nosocomial infections can be related to more frequent use of invasive devices for monitoring or therapy, more colonization and infection by multidrug-resistant organisms (both viral and bacterial), and greater debilitation and severity of illness of hospitalized clients who acquire these infections.

The increased use of invasive and surgical procedures, immunosuppressants, antibiotics, and the lack of hand-washing predispose people to such infections and superinfections. At the same time, the growing number of personnel who come into contact with the client makes the risk of exposure greater.

Prevention is of critical importance in controlling nosocomial infections. The concept of standard precautions emphasizes that all clients must be treated as though each one has a potential bloodborne, transmissible disease; thus all body secretions are handled with care to prevent disease. Handwashing has been cited as the easiest and most effective means of preventing nosocomial infection and must be done routinely even when gloves are used.¹² See further discussion under Control of Transmission in this chapter.

Pathogens

Humans coexist with many microorganisms in complex, mutually beneficial relationships. Even so, many organisms are parasitic, maintaining themselves at the expense

of their host. Some parasites arouse a pathologic response in the host and are called *pathogens* or *pathogenic agents*. A *pathogen* is defined as any microorganism that has the capacity to cause disease.¹²⁶ As such, pathogens are ineffective parasites because they stimulate a disease response, which may harm the host and eventually kill the pathogen.

The ability of a pathogen to stimulate an immune response in the host (antigenicity) varies greatly among organisms, depending on the site of invasion, the number of pathogenic organisms, and the dissemination of organisms in the body. The immune status of a person plays the largest role in determining the risk for infection and the ability of the host to combat organisms that have gained entry.

The mode of action of a pathogen refers to how the organism produces a pathologic process. Great variation exists among the various pathogens. Some intracellular pathogens, like viruses, invade cells and interfere with cellular metabolism, growth, and replication, whereas others invade and cause hyperplasia and cell death. Yet other organisms, such as the influenza virus, have the potential to alter their antigenic characteristics. This virus is capable of extensive gene rearrangements, resulting in significant changes in surface antigen structure. This ability allows new strains to evade host antibody responses directed at earlier strains.

Some viruses (e.g., all members of the herpesvirus group) cause a persistent latent infection that can be reactivated in certain circumstances. HIV causes immunosuppression by destroying helper T lymphocytes. Some pathogens, such as the tetanus bacillus, produce a toxin that interferes with intercellular responses. Some bacteria, such as diphtheria and tetanus, secrete water-soluble antigenic exotoxins that are quickly disseminated in the blood, causing potentially severe systemic and neurologic manifestations. Larger parasites such as roundworms cause anemia and interfere with the function of the GI system.

The characteristics of the organism and the susceptibility of the host influence the likelihood of a pathogen producing infectious disease and the type of disease produced. Not all pathogens have an equal probability of inducing disease in the same host population. *Principal* pathogens regularly cause disease in people with apparently intact defense systems.

Opportunist pathogens do not cause disease in people with intact host defense systems but can clearly cause devastating disease in many hospitalized and immunocompromised clients.¹²⁶ Organisms that may be harmless members of normal flora in healthy people may act as virulent invaders in people with severe defects in host defense mechanisms.⁹¹

Pathogenicity, the ability of the organism to induce disease, depends on the organism's speed of reproduction in the host, the extent of damage it causes to tissues, and the strength of any toxin released by the pathogen. *Virulence* refers to the potency of the pathogen in producing severe disease and is measured by the case fatality rate (i.e., the number of people who die of the disease divided by the number of people who have the disease). Virulence provides a quantitative measure of pathogenicity. The amount and destructive potential of released toxin are closely related to virulence.

Reservoir

A reservoir is an environment in which an organism can live and multiply, such as an animal, plant, soil, food, or other organic substance or combination of substances. The reservoir provides the essentials for survival of the organism at specific stages in its life cycle. Some parasites have more than one reservoir, such as the yellow fever virus, which can maintain life in human beings and other animals. Some parasites require more than one reservoir at different growth stages and still others, such as most sexually transmitted organisms, require only a human reservoir.

Human and animal reservoirs can be symptomatic or asymptomatic carriers of the pathogen. A carrier maintains an environment that promotes growth, multiplication, and shedding of the parasite without exhibiting signs of disease. Hepatitis is a common example of this carrier state in human beings.

Portal of Exit

The portal of exit is the place from which the parasite leaves the reservoir. Generally, this is the site of growth of the organism and corresponds to the system of entry into the next host. For example, the portal of exit for GI parasites is usually the feces, and the portal of entry into a new host is the mouth. Exceptions to the case include hookworm eggs, which are shed in the feces but enter through the skin of a person walking barefoot in soil containing hatched eggs.

Common portals of exit include secretions and fluids (e.g., respiratory secretions, blood, vaginal secretions, semen, tears), excretions such as urine and feces, open lesions, and exudates such as pus from an open wound or ulcer. Some organisms, such as HIV, have more than one portal of exit. Knowledge of the portal of exit is essential for preventing transmission of a pathogen.

Mode of Transmission

For infection to be transmitted, the invading organism must be transported from the infected source to a susceptible host. Microorganisms are transmitted by several possible routes, and the same microorganism can travel by more than one route. The five main routes of transmission are contact, airborne, droplet, vehicle, and vector borne.

Contact transmission occurs directly or indirectly. Direct contact is the direct transfer of microorganisms that come into physical contact either by skin-to-skin contact or mucous membrane-to-mucous membrane contact (e.g., sexual contact, biting, touching, kissing).

Indirect contact involves transfer of microorganisms from a source to a host by passive transfer from an inanimate, intermediate object, called a *fomite*. Inanimate objects can include items such as the telephone, sphygmomanometer, bedside rails, tray tables, countertops, and other items that come into direct contact with the infected person, thus emphasizing the need for thorough handwashing at all times.

An example of indirect transmission is transfer of human immunodeficiency virus from a contaminated source to a host through a needlestick. Another example of indirect contact includes oral-fecal transmission by the ingestion of enteric pathogens from a food prepared by a person who does not wash his or her hands.⁷²

Airborne transmission occurs when disease-causing organisms are so small (less than 5 microns) that they are capable of floating on air currents within a room and remain suspended in the air for several hours. They are often propelled from the respiratory tract through coughing or sneezing. A host then inhales the particles directly into the respiratory tract (e.g., tuberculosis, chickenpox, rubella, measles).⁵⁴

Droplet transmission is different from airborne transmission because droplets are larger particles (greater than 5 microns) than airborne particles and they do not remain suspended in air but fall out within 3 feet of the source. They are produced when a person coughs or sneezes and then travel only a short distance. A common example of droplet-spread infection is influenza. Those people who are in closest proximity to the infected source have the highest risk for infection.⁷²

Vehicle transmission occurs when infectious organisms (e.g., salmonellosis) are transmitted through a common source (e.g., contaminated food, water, and IV fluid) to many potential susceptible hosts. *Vectorborne* transmission of infectious organisms involves insects and/or animals that act as intermediaries between two or more hosts. Lyme disease and Rocky Mountain spotted fever are examples of vectorborne diseases.

Portal of Entry

A pathogen may enter a new host by ingestion, inhalation, or bites or through contact with mucous membranes, percutaneously or transplacentally. Infectious diseases vary as to the number of organisms and the duration of exposure required to start the infectious process in a new host.

Host Susceptibility

Each person has his or her own susceptibility to infectious disease, and this susceptibility can vary throughout time. A susceptible host has personal characteristics and behaviors that increase the probability of an infectious disease developing.

Biologic and personal characteristics such as age, sex, ethnicity, and heredity influence this probability. General health and nutritional status, hormonal balance, and the presence of concurrent disease also play a role. Likewise, living conditions and personal behaviors such as drug use, diet, hygiene, and sexual practices influence the risk of exposure to pathogens and resistance once exposed.

Older adults in hospitals and long-term care facilities are already susceptible hosts, especially if poorly nourished. Immunosuppressive agents and corticosteroids decrease the body's ability to resist infection. Inadequate or absent handwashing or other breaches of aseptic technique result in spread of microorganisms from health care workers (HCWs) to clients and between individuals receiving health care.

Surfaces of equipment can become contaminated and then transmit microorganisms that cause infection. Incorrect isolation procedures such as leaving doors open to rooms in which airborne precautions are in effect or not using masks increase the risk of transmitting organisms that cause nosocomial infections.

The presence of underlying medical disorders (e.g., malignancy, diabetes, renal failure, acquired immune deficiency syndrome [AIDS], and cirrhosis) decreases T-cell- and B-cell-mediated immune function. Breaches of body integrity such as nasogastric and chest tubes, intubation, urinary catheters, and IV devices impair the body's defense mechanisms, decreasing the ability of the integumentary, GI, genitourinary, and respiratory systems to resist invasion by microorganisms.¹⁷⁴

Lines of Defense. Susceptibility is also influenced by the presence of anatomic and physiologic defenses, sometimes called *lines of defense*. The *first-line defenses* are external, such as intact skin and mucous membranes; oil and perspiration on skin; cilia in respiratory passages; gag and coughing reflexes; peristalsis in the GI tract; and the flushing action of tears, saliva, and mucus.

These first-line defenses act to inhibit invasion of pathogens and remove them before they have an opportunity to multiply. The chemical composition of body secretions such as tears and sweat, together with the pH of saliva, vaginal secretions, urine, and digestive juices, further prevents or inhibits growth of organisms. Compromise in any of these natural defenses increases host susceptibility to pathogen invasion.

Another important first-line defense is the normal flora of microorganisms that inhabit the skin and mucous membranes in the oral cavity, GI tract, and vagina. These organisms occur naturally and usually coexist with their host in a mutually beneficial relationship. Through a mechanism called *microbial antagonism* they control the replication of potential pathogens.

The importance of this mechanism is evident when it is disturbed, as happens when extensive antibiotic therapy destroys normal flora in the oral or vaginal cavity, result-

ing in *Candida albicans*, an overgrowth of yeast. Some normal flora can become pathogenic under specific conditions such as immunosuppression or displacement of the pathogen to another area of the body. Displacement of normal flora is a common cause of nosocomial infections. This can occur when *Escherichia coli*, ordinarily normal flora in the GI tract, invade the urinary tract. Invasive procedures increase the risk of displacing these organisms.

The *second-line defense*, the inflammatory process, and the *third-line defense*, the immune response, share several physiologic components. These include the lymphatic system, leukocytes, and a multitude of chemicals, proteins, and enzymes that facilitate the internal defenses.

Once a microorganism penetrates the first line of defense, the inflammatory response is initiated. Inflammation is a local reaction to cell injury of any type whether from physical, chemical, or thermal damage, or microbial invasion. As a response to microbial injury, inflammation is aimed at preventing further invasion by walling off, destroying, or neutralizing the invading organism.

The early inflammatory response is protective, but it can continue for sustained periods in some infections, leading to granuloma formation. The production of new leukocytes may be stimulated for weeks or months and is reflected in an elevated white blood cell count. However, sustained inflammation can become chronic and result in destruction of healthy tissues. Extensive necrosis from persistent inflammation can increase tissue susceptibility to the infectious agent or provide an ideal setting for invasion by other pathogens.

The first- and second-line defenses are nonspecific; that is, they operate against all infectious agents in the same way. In contrast, the immune system responds in a specific manner to individual pathogens as long as the organism has antigenic characteristics. Generally, antigens are proteins, large polysaccharides, or large lipoprotein complexes that stimulate an immune response.

Not all microorganisms are antigenic, but some are bound by complement or other host-produced substances to form an antigen that elicits an immune response. An immune response is triggered after foreign materials have been cleared from an area of inflammation. For specific details regarding cell-mediated versus humoral immune responses, see Chapter 7.

Control of Transmission

Much can be done to prevent transmission of infectious diseases, including the use of barriers and isolation; comprehensive immunizations, including the required immunization of travelers to or emigrants from endemic areas; drug prophylaxis; improved nutrition, living conditions, and sanitation; and correction of environmental factors.

Breaking the transmission chain at any of these links can help control transmission of infectious diseases. The link most amenable to control varies with the characteristics of the organism, its reservoirs, the type of pathologic response it produces, and the available technology for control. The general goal is to break the chain at the most cost-effective point or points—that is, the point at which the greatest number of people can be protected.

with available technology and the least amount of resources.

Isolation and barriers can be used to prevent the transmission of microorganisms from infected or colonized people to other unaffected people. In hospital or institutional settings, the purpose of isolating individuals or residents is to prevent the transmission of colonized or infectious microorganisms among clients, visitors, and HCWs. In 1996 the Centers for Disease Control and Prevention (CDC) and the Hospital Infection Control Practices Advisory Committee issued a revision of the isolation guidelines.

The new guidelines outline a two-tiered approach with standard precautions that apply to all clients and transmission-based precautions that apply to anyone with documented or suspected infection or colonization with specific microorganisms.

These *transmission-based precautions* are defined according to the major modes of transmission of infectious agents (contact, airborne, and droplet) in the health care setting (Table 8-3). Standard precautions have replaced universal precautions. Barrier precautions stipulate that gloves should be worn to touch any of the following: blood; all body fluids; secretions and excretions except sweat, regardless of whether these are visibly bloody; nonintact skin; and mucous membranes. Infectious waste is defined as free-flowing blood or body fluids present in sufficient quantity to drip, splash, or flake from dressings or containers (Box 8-3).

Hands must be washed immediately after gloves are removed and between clients. For procedures that are likely to generate splashes or sprays of body fluid, a mask with eye protection or a face shield and gown should be worn. Gowns should be of a material that prevents penetration by microorganisms and subsequent contamination of the skin or clothing.^{54,179} Needles should not be recapped, bent, or broken, but should be disposed of in a puncture-resistant container.⁴⁵

Handwashing is an integral part of reducing transmission of harmful microbes in a health care setting. Streptococci and coagulase-negative staphylococci are the most common bacteria residing on the skin. HCWs can

transiently obtain other pathogens after patient care or from the areas immediately surrounding a client, which can then be transmitted to other clients. Transient organisms are more likely to be transmitted to others than are resident flora of the skin.

Handwashing significantly reduces transient bacteria on HCW hands and the risk of transmitting infection. Different studies have been done looking at actual barriers to proper handwashing, while others concentrated on perceived barriers.

In one large study, nonadherence to recommended handwashing was highest in areas where adherence was crucial (such as intensive care units), whereas adherence was highest in areas where there was a lower need for stringent hand hygiene. Perceived reasons for nonadherence included skin irritation caused by the detergents, high client/HCW ratio (less time), unavailable or inconvenient location of supplies, forgetfulness, glove use, and incorrect information regarding proper hand hygiene.¹²¹

The CDC has reevaluated hand hygiene and made recommendations allowing HCWs to achieve more realistic goals of proper hand hygiene.¹² Soap (antimicrobial or non-antimicrobial) and water should be used when hands are visibly soiled with blood or other body fluid, before and after eating, and after using the restroom (Box 8-4).

If hands are not visibly soiled, decontamination may be accomplished by using an alcohol-based rub for the following instances: before and after routine contact with a client, before putting on gloves and after removing gloves for a nonsurgical procedure or client contact, and after contact with a body fluid or skin that is not intact and before moving to a clean part of the same person. During outbreaks of *C. difficile-associated disease* (CDAD),

Box 8-4

INDICATIONS FOR HANDWASHING AND HAND ANTISEPSIS

Handwashing with soap (microbial or not antimicrobial) and water:

- When hands are visibly soiled with blood or body fluids
- Before eating
- After using the restroom
- After proven or suspected exposure to *Bacillus anthracis*

Decontaminate hands with alcohol-based rub:

- After exposure to body fluids or excretions but hands not visibly soiled
- After having direct contact with a client
- Before and after putting on gloves for client care
- Before and after putting on gloves for a nonsurgical procedure
- After contact with intact client skin
- After attending to a contaminated body site and before moving to a clean body site on the same client
- After contact with objects in client area

Modified from Boyce JM, Pittet D: Guideline for hand hygiene in health-care settings. Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force, *MMWR* 51(RR-16):1-44, 2002.

Box 8-3

INFECTIOUS AND SAFE WASTE

Infectious Waste

- Blood and components
- All disposable sharps (used or unused)
- Urine, stool, or emesis if visibly contaminated with blood
- Vaginal secretions
- Semen
- CSF
- Synovial fluid
- Pericardial fluid (mediastinal tubes)
- Amniotic fluid

Safe Waste

- Cotton balls, adhesive bandages
- Latex gloves, masks, or other personal protective devices
- Nasal secretions
- Sputum
- Feces
- Urine
- Vomit
- Tears
- Sweat

Table 8-3 Type of Transmission-Based Precautions and Prevention Guidelines

Type of Precaution	Type of Microorganism	Measures Taken
Standard precautions	Bloodborne pathogens; applies to all clients	See Appendix A
Airborne precautions	Microorganism transmitted by small-particle residue; can suspend in the air and be dispersed by air currents (e.g., coughing, sneezing, talking) Examples: Measles (rubeola) <i>M. tuberculosis</i> (MDRT) VZV (chickenpox, disseminated shingles)	Private room with monitored negative airflow ROOM DOOR CLOSED and client in room Respiratory protection when entering room (specialized, approved filter masks in suspected or known tuberculosis) Restrict entry of certain susceptible people (immunosuppressed, pregnant women) when rubella or varicella is suspected or known Limited transport of client from room (only when essential); place surgical mask on restricted individual transported
Droplet precautions	Microorganisms transmitted by large-particle droplets about 3 feet from the source; generated by sneezing, coughing, talking, or performing procedures Examples: Invasive <i>Haemophilus influenza</i> B, including meningitis, pneumonia, epiglottitis, sepsis Invasive <i>Neisseria meningitidis</i> Diphtheria, pertussis <i>Mycoplasma pneumoniae</i> Pneumonia plaque Streptococcal pharyngitis, pneumonia, or scarlatina in infants and young children Adenovirus Influenza, RSV Mumps, rubella Parvovirus B19	Private room or house with others with same infection Door may remain open Wear mask when working within 3 feet of affected person Limit client transport to only when necessary; place surgical mask on person when transported
Contact precautions	Microorganisms that can be transmitted by direct contact with the client (hand or skin-to-skin contact) or indirect contact (touching environmental surfaces or client care items) Examples: GI, respiratory; skin, or wound infections Multiple-drug-resistant bacteria (MRSA, VRE) Enteric infections (low infectious dose or prolonged environmental survival) (<i>C. difficile</i>) Diapered or incontinent clients (enterohemorrhagic <i>E. coli</i> , <i>Shigella</i> ; hepatitis A; rotavirus) In infants and young children RSV Parainfluenza virus Enteroviral infections Highly contagious skin infections or those that may occur on dry skin (diphtheria; HSV; impetigo; noncontained abscesses, cellulitis, or pressure ulcers; pediculosis; scabies; herpes zoster [disseminated]) Viral hemorrhagic conjunctivitis Viral hemorrhagic infections (e.g., Ebola)	Private room Glove when entering room Change gloves after having contact with infective material that may contain high concentrations of microorganism (e.g., fecal material, wound drainage) Remove gloves before leaving the client environment and wash hands immediately with antimicrobial soap or waterless antiseptic After removing gloves and washing hands, DO NOT TOUCH potentially contaminated surfaces or materials Wear a clean gown when entering the room if you anticipate substantial contact with the contaminated materials or surfaces (particularly if the person is incontinent, or has diarrhea, an ileostomy, colostomy, or noncontained draining wound) Remove gown before leaving; do not touch contaminated areas Limit transport of person from room Dedicate use of noncritical client care items to only this person (e.g., stethoscope) Disinfect equipment with approved disinfectant if to be used with other people

MDRT, Multiple-drug-resistant tuberculosis; RSV, respiratory syncytial virus; MRSA, methicillin-resistant *Staphylococcus aureus*; VRE, vancomycin-resistant enterococci.

Modified from Centers for Disease Control and Prevention: Guidelines for infection control in health care personnel, *Am J Infect Control* 26(3):289-343, 1998.

Box 8-5**PROPER HAND HYGIENE TECHNIQUE****Alcohol-Based Rubs**

Using alcohol-based hand rubs may reduce contamination better than soap and water. Apply product in the palm of the hand and rub hands together, remembering to cover all areas of the hands, including the back of the hands and webs of the fingers, until dry.

Washing Hands with Soap and Water

Wet hands before beginning, then add soap. Rub hands together vigorously for at least 15 to 20 seconds, covering all areas of the hands and fingers. Rinse soap from hands and dry completely with a disposable towel. Turn the water off using the towel.

Special Considerations

Jewelry may sequester gram-negative organisms, but more studies are needed to determine if this translates to increased transmission of the organisms.

Artificial nails or extenders may harbor high concentrations of CoNS and gram-negative rods. Although more studies are needed to determine if artificial nails increase the likelihood of transmitting organisms, the CDC recommends that artificial nails or extenders not be worn when in contact with clients at high risk for infection.

Wear gloves if potentially coming into contact with blood, body fluids, mucous membranes, or nonintact skin.

For more information on hand hygiene and infection control, visit the web sites for the CDC (<http://www.cdc.gov>) and the Association for Professionals in Infection Control and Epidemiology (<http://www.apic.org>).

Modified from Boyce JM, Pittet D: Guideline for hand hygiene in healthcare settings. Recommendations of the Healthcare Infection Control Practices Advisory Committee and the HICPAC/SHEA/APIC/IDSA Hand Hygiene Task Force, *MMWR* 51(RR-16):1-44, 2002.

washing hands with an antimicrobial soap and water after removing gloves is advised (Box 8-5).¹³

No recommendations are available for the use of non-alcohol-based rubs since data are lacking. Antimicrobial towelettes are an alternative but not a substitute for hand-washing with soap and water or alcohol-based rubs.

Other pertinent recommendations have been made by the CDC in regard to wearing jewelry or gloves and having long or artificial nails (acrylics). Although rings have been shown to harbor organisms, more studies are needed to determine if this leads to an increase in transmission of these microbes; the CDC has not made specific recommendations in this area.

The Occupational Safety and Health Administration has mandated that gloves be worn by HCWs when caring for clients when they may be exposed to blood or body fluids that can be contaminated with blood. Gloves have been shown to reduce contamination of hands as well as reduce the transmission of pathogens by HCWs. However, gloves do not completely protect against hand contamination; therefore decontamination of the hands after use is essential.

Available studies suggest that the areas under the fingernails contain significant amounts of bacteria, espe-

dally coagulase-negative staphylococci and gram-negative rods. Artificial nails harbor even larger concentrations than natural nails. It is unknown if long or artificial nails contribute to transmission of these organisms, although a few reports have linked outbreaks of *Pseudomonas* to artificial nails worn by nurses. More studies are needed to determine the significance of artificial nails, but the CDC recommends that artificial nails should not be worn if the HCW is in contact with clients at high risk for infection.

Immunization, by decreasing host susceptibility, can now control many diseases, including diphtheria, tetanus, pertussis, measles, mumps, rubella, some forms of meningitis, poliomyelitis, hepatitis A and B, pneumococcal pneumonia, influenza (certain strains), and rabies. Each year the CDC issues updated information on the vaccine and antiviral agents available for controlling influenza during the current influenza season.

Vaccines, which contain live but attenuated (weakened) or killed microbes, induce active immunity against bacterial and viral diseases by stimulating antibody formation.

These molecules lock onto specific proteins made by a virus or bacterium, which are often those proteins lodged in the microbe's outer coat. Once antibodies attach to an invading microbe, other immune defenses are evoked to destroy it. Immune globulins contain previously formed antibodies from hyperimmunized donors or pooled plasma and provide temporary passive immunity. Passive immunization is generally used when active immunization is life threatening or when complete protection requires both active and passive immunization (e.g., immunoglobulins used for hepatitis B or for tetanus) (see Table 7-1).

Side effects to immunization can occur, but the incidence of significant adverse effects of immunization among human beings remains very small.^{1,3,4} The potential increase in susceptibility to influenza and death from respiratory illness in high-risk people (e.g., those with rheumatoid arthritis, the aging adult, and chronically ill or immunosuppressed individuals) suggests that the influenza and pneumococcal vaccines should include these groups in standard immunization programs.³

Prophylactic antibiotic therapy may prevent certain diseases and is usually reserved for people at high risk of exposure to dangerous infections (e.g., *Pneumocystis carinii* pneumonia in clients with HIV/AIDS, postexposure of HCWs to percutaneous contamination from individuals with HIV, preoperatively before joint replacement surgery).⁵⁶

Antibiotic-resistant bacteria are on the rise in part because antibiotics have been misused and overused. This is only a small part of the total picture regarding this issue. Other components of this problem include the extensive use of antibiotics in animals later consumed or whose products are consumed (e.g., milk products). Crowded conditions (e.g., day care centers, hospitals, military barracks, and prisons) and prior antibiotic therapy are the principal factors predisposing to colonization and disease.

Some bacteria, such as enterococci, which cause wound and blood infections, have developed mutant strains that

do not respond to any antibiotic therapy. Enterococcal infections are primarily limited to hospitals. At present, certain strains of pneumococcus are resistant to only one antibiotic, so other antibiotics remain effective against these pneumococci. If those strains become resistant to other (currently effective) antibiotics, there may be no treatment available. Before 1980, only a few cases of pneumococcus were resistant to penicillin, but within a year strains resistant to penicillin were reported everywhere and are now common throughout the world.

Because resistance to many commonly used antibiotics has developed, including resistance to vancomycin, a search is underway for novel and more effective antimicrobial agents. Cellular enzymes such as synthetases that are universal and essential for cellular viability are being studied as one of the targets for new antibiotics.¹³⁶ Experiments are also underway to find molecules that are effective in binding small, untranslated RNA, causing plasmid elimination and reducing resistance.¹⁷¹ Gatifloxacin, a newer fluoroquinolone, continues to retain susceptibility to most gram-negative organisms, but increasing resistance to *Streptococcus* spp., including *S. pneumoniae*, is worrisome.^{46,111}

In April 2000 the Food and Drug Administration (FDA) approved linezolid for the treatment of individuals with infections caused by gram-positive bacteria. Linezolid comes from the first completely new class of antibiotics to reach hospitals in 35 years, the oxazolidinones. Linezolid is indicated for adults and children in the treatment of numerous gram-positive organisms, including MRSA and *Enterococcus* spp.¹¹⁹

Although studies are lacking, linezolid may have efficacy in treating *M. tuberculosis*, including strains resistant to the usual antituberculous agents.⁴⁴ Current research is focused on the epidemiology of resistant bacteria, including investigation about the receptors and mechanisms of cellular action and resistance. The study and potential use of inhibitors of resistance mechanisms is an area that might prove useful as a new direction in drug development.^{101,185}

*Improved nutrition, living conditions, and sanitation through the use of disinfection, sterilization, and antiinfective drugs can inactivate pathogens such as *S. aureus*. This is important because drug-resistant strains of *S. aureus* have developed. One strain of staphylococcus, MRSA, is resistant to most antibiotics.*

Over the past 10 years, MRSA and other resistant strains of *S. aureus* have become the most common causes of hospital and community-acquired infections. MRSA usually develops when multiple antibiotics are used in the treatment of infection and in older adults who are debilitated, having surgery or multiple invasive procedures, or being treated in critical care units.

Transmission to a new portal of entry can be prevented by environmental disinfection, use of barrier precautions (gloves, masks, condoms), proper handling of food, and protection from vectors. Decreasing host susceptibility can be achieved through personal hygiene and avoidance of high-risk behaviors (unsafe sex practices, injection drug use, recapping needles) and effective handwashing.

Maintaining the first-line defense is an important consideration for the client whose health status has already

been compromised by disease or diagnostic and treatment procedures. Some ways to accomplish this include preoperative and postprocedure assistance to encourage deep breathing; coughing, ambulation, and skin care; maintaining adequate hydration, fluid, and electrolyte balance; and providing proper nutrition to strengthen resistance.

Correction of environmental factors, particularly water treatment; food and milk safety programs; and control of animals, vectors, rodents, sewage, and solid wastes, can best eradicate nonhuman environments (reservoirs) and thus control pathogens.

Other prevention methods in this category include proper handling and disposal of secretions, excretions, and exudates; isolation of infected clients (doors must remain closed, especially in negative-pressure rooms); and quarantine of contacts.

The CDC has recommended specific transmission precautions based on knowledge of the transmission chain for individual infections. The precautions were designed to prevent transmission of pathogens among hospitalized people, HCWs, and visitors (see Table 8-3). Specific recommendations have been made for individual diseases.

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-1

Control of Transmission

The CDC has set up guidelines for the care of all clients regarding precautions against the transmission of infectious disease (see Appendix A). These should be used with all clients regardless of their disease status. All blood and body fluids are potentially infectious and should be handled as such (see Box 8-3).

All clients receiving therapy (and thus in contact with HCWs) may be asymptomatic hosts during the period of communicability. The careful use of precautionary measures severely limits the transmission of any disease. In addition, each hospital has transmission-based precautions organized according to categories of transmission routes to prevent the spread of infectious disease to others. Every HCW must be familiar with these procedures and follow them carefully. Transmission prevention guidelines and accompanying basic isolation procedures are provided in Table 8-3.

HCWs should be concerned about improving their resistance and decreasing their susceptibility to infectious diseases. Maintaining an adequate immunization status is one approach. Every HCW should be adequately immunized against hepatitis B, measles, mumps, rubella, polio, tetanus, diphtheria, and varicella. The most recent CDC recommendations for immunization of HCWs are given in Table 8-4.

Second to immunization, handwashing is the most effective disease-preventing measure anyone can practice. Despite compelling evidence that proper handwashing can reduce the transmission of pathogens to patients and clients and the spread of antimicrobial

Table 8-4 CDC Recommendations for Immunization of HCWs

Vaccine	Schedule
HBV	Give recombinant vaccine (IM) in a three-dose series; obtain anti-HBV serologic testing 1 to 2 months after last dose
Influenza	Annual vaccination; live, attenuated vaccine available for healthy, nonpregnant HCWs aged 49 years and younger (nasal spray); inactivated vaccine given IM for any HCW, especially if in contact with immunocompromised clients
MMR	For anyone born in 1957 or later without serologic evidence of immunity or prior vaccination; contraindicated in pregnancy
VZV	HCWs who have no serologic proof of immunity, prior vaccination, or history of chickenpox; contraindicated in pregnancy
DTaP	Recommended for all adults with booster every 10 years; tetanus prophylaxis advised for HCWs in wound management; advised after needlestick injury. New ACIP recommendations for DTaP in adults and HCWs are pending release.
Meningococcal	Recommended for HCWs in direct contact with respiratory secretions from infected persons without proper use of precautions; microbiologists who are routinely exposed to <i>N. meningitidis</i> ; postexposure prophylaxis advised.
BCG	Not used in the United States as prophylaxis for tuberculosis; foreign-born HCWs who have received this vaccine outside the United States must be aware that it does not provide lifelong immunity.

IM, Intramuscular; DTaP, tetanus and diphtheria; ACIP, Advisory Committee on Immunization Practices; BCG, Bacille Calmette-Guérin.

For more complete information visit the Immunization Action Coalition (IAC) web site (<http://www.immunize.org/acip>) and check with the CDC for updates (www.cdc.gov).

Modified from Centers for Disease Control and Prevention: Immunization of health-care workers: recommendations of the advisory committee on immunization practices (ACIP) and the hospital infection control practices advisory committee (HICPAC), *MMWR* 46(RR-18):1-42, 1997.

resistance, the adherence of HCWs to recommended hand hygiene practices remains unacceptably low.¹⁶⁷ A single hand can carry 200 million organisms, including bacteria, viruses, and fungi. It takes a full 5 minutes of handwashing to cleanse 99% of the bacteria from fingernails, thumbs, palm creases, and backs of the hands. The average wash-and-rinse in the hospital setting is less than 10 seconds, and the dominant hand is often underwashed.¹⁸⁰

However, many HCWs suffer severe hand irritation, with cracking and bleeding as a consequence of frequent handwashing and glove use. Integumentary breakdown has major implications for nosocomial infection control and promotes the spread of blood-borne viruses. In 2002, the CDC provided new guidelines (see Boxes 8-3 and 8-4) concerning handwashing and appropriate situations for using soap and water or alcohol-based rubs.¹² An adjunct to handwashing is an alcohol gel fortified with a skin-protecting emollient (e.g., glycerin) or an alcohol-based towelette.

An alcohol-based hand rub requires less time, is microbiologically more effective than washing with soap and water, and is less irritating to the skin. Alcohol-based hand rubs can replace handwashing as the standard for hand hygiene in health care settings in all situations in which the hands are not visibly soiled.¹⁶⁵

Alcohol hand rinses may increase compliance with hand disinfection and are as effective as soaps; but used without the added skin protection, alcohol has a significant drying effect, leading to the same skin problems associated with frequent washing.⁹⁸ The use of lotions after hand sanitation may also decrease cracking of skin and lead to a reduction in bacterial shedding from hands.^{12,98}

Nosocomial Infections

Therapists can help prevent transmission of nosocomial infections from themselves to others (Table 8-5), from client to client, and from client to self by following standard precautions (see Appendix A) and guidelines as follows:

- Follow strict infection-control procedures. Make sure to identify each client's individual transmission precautions and procedures. When in doubt, ask the nursing staff regarding the status of the person in question.
- Strictly follow necessary isolation techniques. Doors must remain closed, especially in negative pressure rooms.
- Observe *all* clients for signs of infection (see Box 8-1), especially those people at high risk. Notify nursing or medical staff of these observations.
- Always follow proper handwashing technique or use an alcohol-based hand antiseptic, and encourage other staff members to do so as well. Take time to wash or disinfect hands before and after every client.
- Stay away from susceptible, high-risk clients when you have an obvious infection. Make arrangements for another therapist to treat that client until the contagious period has passed. If in doubt, consult a physician.
- Take special precautions with vulnerable clients—those with Foley catheters, mechanical ventilators, or IV lines and those recuperating from surgery. Specific tips for preventing infection in these situations are listed in Box 8-6.
- Avoid the use of acrylic nails, which harbor pathogens.⁹⁷

Continued.

Table 8-5 Summary of Important Recommendations and Work Restrictions for Personnel with Infectious Diseases

Disease/Condition	Work Restriction	Duration
Conjunctivitis	Restrict from client contact with the client's environment	Until discharge ceases
CMV infections	No restriction	
Diarrheal Diseases		
Acute stage (diarrhea with other symptoms)	Restrict from client contact, contact with the client's environment, and food handling	Until symptoms resolve
Convalescent stage, <i>Salmonella</i>	Restrict from care of high-risk clients	Until symptoms resolve; consult with local and state health authorities regarding need for negative stool cultures
Diphtheria	Exclude from duty	Until antimicrobial therapy completed and top cultures obtained ≥24 hours apart are negative
Enteroviral infections	Restrict from care of infants, neonates, and immunocompromised persons and their environment	Until symptoms resolve
Hepatitis A	Restrict from client contact, contact with client's environment, and food handling; use proper handwashing	Until 7 days of onset of jaundice
Hepatitis B		
Personnel with acute or chronic HBsAg who do not perform exposure-prone procedures	No restriction*; refer to state regulations; standard precautions should always be observed	
Personnel with acute or chronic HBeAg who perform exposure-prone procedures	Do not perform exposure-prone invasive procedures until counsel from an expert review panel has been sought; panel should review and recommend procedures the HCW can perform, taking into account specific procedures and skill and technique of worker; refer to state regulations	Until HBeAg (a marker of a high titer of virus) is negative
Hepatitis C	No recommendation	
Herpes Simplex	No restriction	
Genital	Restrict from client contact and contact with client's environment	Until lesions heal
Hands (herpetic whitlow)	Evaluate for need to restrict from care of high-risk clients	
Orofacial	Do not perform exposure-prone invasive procedures until counsel from an expert review panel has been sought; panel should review and recommend procedures the HCW can perform, taking into account specific procedures and skill and technique of the worker; standard precautions should always be observed; refer to state regulations	
HIV		
Active	Exclude from duty	Until 7 days after rash appears
Postexposure (susceptible personnel)	Exclude from duty	From fifth day after first exposure through twenty-first day after last exposure and/or 4 days after rash appears
Measles		
Active	Exclude from duty	Until 7 days after rash appears
Postexposure (susceptible personnel)	Exclude from duty	From twelfth day after first exposure through twenty-sixth day after last exposure or until 9 days after onset of parotitis
Meningococcal infections	Exclude from duty	Until 24 hours after start of effective therapy
Mumps		
Active	Exclude from duty	Until 9 days after onset of parotitis
Postexposure (susceptible personnel)	Exclude from duty	From twelfth day after first exposure through twenty-sixth day after last exposure or until 9 days after onset of parotitis
Pediculosis	Restrict from duty	Until treated and observed to be free of adult and immature lice

Table 8-5 Summary of Important Recommendations and Work Restrictions for Personnel with Infectious Diseases—cont'd

Disease/Condition	Work Restriction	Duration
Pertussis Active	Exclude from duty	From beginning of catarrhal stage through third week after onset of paroxysms or until 5 days after start of effective antimicrobial therapy
Postexposure (asymptomatic personnel)	No restriction, prophylaxis recommended	
Postexposure (symptomatic personnel)	Exclude from duty	Until 5 days after start of effective antimicrobial therapy
Rubella Active	Exclude from duty	Until 5 days after rash appears
Postexposure (susceptible personnel)	Exclude from duty	From seventh day after first exposure through twenty-first day after last exposure
Scabies	Restrict from client contact	Until cleared by medical evaluation
<i>S. aureus</i> Infection Active, draining skin lesions	Restrict from contact with client, client's environment, and food handling	Until lesions have resolved
Carrier state	No restriction unless HCW is epidemiologically linked to transmission of the organism	
Streptococcal infection, group A	Restrict from client care, contact with client's environment, and food handling	Until 24 hours after adequate treatment started
Tuberculosis Active disease	Exclude from duty	Until proved noninfectious
PPD converter	No restriction if not an active case and if cleared medically	
Varicella Active	Exclude from duty	Until all lesions dry and crust
Postexposure (susceptible personnel)	Exclude from duty	From tenth day after first exposure through twenty-first day (twenty-eighth day if VZIG given) after last exposure
Viral respiratory infections, acute febrile	Consider excluding from the care of high-risk clients [†] or contact with their environment during community outbreak of RSV influenza	Until acute symptoms resolve
Zoster Localized in healthy person	Cover lesions; restrict from care if high-risk clients [†]	Until lesions dry and crust
Generalized or localized immunosuppressed person	Restrict from client contact	Until lesions dry and crust
Postexposure (susceptible personnel)	Restrict from client contact	From tenth day after first exposure through twenty-first day (twenty-eighth day if VZIG given) after last exposure or, if varicella occurs, until all lesions dry and crust

PPD, Purified protein derivative; VZIG, varicella zoster immunoglobulin; RSV, respiratory syncytial virus.

Modified from Centers for Disease Control and Prevention: Guidelines for infection control in health care personnel, 1998, *Am J Infect Control* 26(3):289-343, 1998. No changes have been made since publication in 1998.

*Unless epidemiologically linked to transmission of infection.

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

[‡]High-risk patients defined by the Advisory Committee of Immunization Practices for complications of influenza.

Box 8-6**TIPS FOR PREVENTING INFECTION****Chest Tube**

- Prevent chest tube from kinking by carefully coiling the tubing on top of the bed and securing it to the bed linen, leaving room for the person to turn.

Tracheostomy

- Contact with secretions occurs with a tracheostomy; follow standard precautions. When direct contact is made and potential splash secondary to expelled secretions occurs, gown, mask, protective face wear, and gloves are needed.

Urinary Catheter

- Follow standard precautions for handwashing techniques.
- Do not allow the drainage bag spigot to come in contact with a contaminated surface.
- When the drainage tubing becomes disconnected, do not touch the ends of the tubing or catheter. Contact the nursing staff for reconnecting.
- Before turning, moving, or transferring a catheterized person, locate the proximal end of the tubing and either clamp it to the person's gown or hold it to allow necessary slack during movement. This will help prevent the catheter from accidentally and traumatically being pulled out.
- Whenever possible, avoid raising the drainage bag above the level of the person's bladder.
- If it becomes necessary to raise the bag during transfers, clamp the tubing but avoid prolonged clamping or kinking of the tubing (except during bladder conditioning).
- Avoid allowing large loops of tubing to dangle from the bedside, wheelchair, or walker.
- Drain all urine from tubing into the bag before the person exercises or ambulates.

IV Devices

- If you have exudative lesions or weeping dermatitis, refrain from all direct contact with IVs or invasive equipment until the condition resolves.
- Notify the nursing staff of any suspicious observations such as if the IV device is not dripping at a steady rate (either none at all or flowing very fast), if the IV bag is empty, or if blood is flowing from insertion of the IV catheter tip into the person's body out into the IV line.

Nasogastric and Feeding Tubes

- Care must be taken to avoid excessive movement or pulling and tugging of these tubes.
- Wash your hands before and after touching the entry point of the tube into the body.

Hydrotherapy

- Hydrotherapy for wound care (pulsatile lavage with suction, whirlpool) should be performed in a private treatment room with all walls and doors closed.
- Proper personal protective equipment must be worn when treating the client and/or cleaning hydrotherapy equipment.
- Whirlpool equipment should be cleaned before and after treatment.

Hydrotherapy and Therapeutic Pool Protocol

In the past, routine cultures of hydrotherapy and pool equipment were performed to identify and supposedly eliminate colonization of infectious bacteria. In this way the spread of infection was prevented from equipment to client and from client to client, especially in the acute care setting.

Now, under the outcomes-based management philosophy, infection control is cost driven so that the outcome is managed, as long as the outcome is what was predicted and intended or improving. For example, in the case of preventing the spread of infection through hydrotherapy or therapeutic pool equipment, good disinfection and cleaning procedures are practiced and monitored closely. This plan is both cost effective and accompanied by a high degree of safety.

Routine environmental cultures are not cost effective and therefore are not performed. Many organisms are present normally, and if present and no one is infected, these pathogens are not considered a functional problem. Under outcomes-based management, when an infectious problem develops, the cause is traced back to the source and eliminated at that point.

When using hydrotherapy (e.g., pulsatile lavage with suction, whirlpool) for wound care, clients should be treated in a private treatment room with all walls and doors closed. Proper personal protective equipment, such as masks, gloves, and eyewear, must be worn by the therapist when treating the individual and cleaning hydrotherapy equipment. Whirlpool equipment should be cleaned before and after treatment.^{**}

Home Health Care

Preventing spread of an infectious disease to the family, the home health therapist, and perhaps the community is a primary concern when preparing the client for return home. The therapist should work closely with the home health nurse and seek guidance if unsure how to handle a specific situation. A list of helpful hints for home health care includes the following^{††}:

- Handwashing is the best protection against transmission of infectious diseases, and it is essential after providing direct care and before touching anything when gloves are removed.
- Staff should leave extraneous clothing and equipment outside the client's area and take in only items that are needed.
- Equipment needed on a regular basis, such as the blood pressure cuff and stethoscope, should be in the room at the beginning of home health care. Stethoscopes are often contaminated with staphylococci and are therefore a potential vector of infection. Such contamination poses a risk to people with open wounds such as burns. This contamination is greatly reduced by frequent cleaning with alcohol or nonionic detergent; cleaning with anti-septic soap is only 75% effective in reducing the bacterial count.^{††}

- When it is no longer needed, equipment should be bagged or covered and taken to the appropriate area for decontamination and reprocessing. Disposable equipment should be contained, labeled, and discarded.
- The therapist should be adequately supplied with gloves, masks, gowns, and disposable plastic aprons. Some plastic bags of different sizes should be carried for the therapist's own use and to demonstrate to the client's family how to handle soiled linens and trash.
- Paper towels are useful when working in the client's area. Use them as a clean surface during care and to wipe your hands.
- Before going into the client's area, plan what to do and gather the items needed.
- It is important to remember that isolation or precautions can have a negative effect on the family. Help the family feel comfortable with the techniques needed for isolation. Encourage them to visit with the client and not just be with him or her during care.
- Should the client have a feces-borne infectious disease such as hepatitis A or salmonellosis, it is important to show the family how to bag and launder soiled linens. It is equally important to demonstrate how to bag and dispose of soiled paper products such as linen savers, which cannot be flushed down the toilet. Remind the family to wash their hands afterward, and the therapist should do so as well.
- If the client has hepatitis A or salmonellosis, the family and the client should be reminded not to handle raw food served to others, such as lettuce or tomatoes, until the physician determines the client is past the infectious stage.
- In treating clients with bloodborne illnesses, if the client accidentally sustains a cut, any spilled blood on inanimate objects or surfaces should be cleaned off with household bleach and water. Razors and toothbrushes should not be shared.
- The therapist should practice self-protection at all times. Use good handwashing technique and, when in doubt, ask for assistance from other, more knowledgeable health care staff.

Diagnosis of Infectious Diseases

Five basic laboratory techniques can be used in the diagnosis of infectious diseases: (1) direct visualization of the organism, (2) detection of microbial antigen, (3) a search for clues produced by the host immune response to specific microorganisms, (4) detection of specific microbial nucleotide sequences, and (5) isolation of the organism in culture. Each technique has its use and each has associated advantages and disadvantages.

In many infectious diseases, pathogenic organisms can be directly visualized by microscopic examination of

readily available tissue fluids, such as sputum, urine, and pus as well as pleural, peritoneal, and cerebrospinal fluid (CSF). Detection of specific antigens establishes the presence of some diseases such as meningitis, hepatitis B, and some respiratory and genitourinary tract infections.

Histopathologic examination of biopsied or excised tissue often reveals patterns of the host inflammatory response that can provide clues to narrow down the diagnostic possibilities. Some viral infections, such as skin or respiratory infections caused by herpesviruses or pneumonia due to CMV, produce characteristic changes in host cells visible on cytologic examination.

Recent techniques to amplify microbial DNA or RNA sequences for detection have been used to diagnose some infections and are expected to be developed enough to diagnose numerous other infectious diseases. Finally, isolation of a single microbe from an infected site is generally considered evidence that the infection is caused by this organism.

SPECIFIC INFECTIOUS DISEASES

Most infections are confined to specific organ systems. In this book, many of the important infectious disease entities are discussed in the specific chapter dealing with the affected anatomic area. Only the most commonly encountered infectious problems not covered elsewhere are included in this chapter.

Bacterial Infections

Clostridium difficile

Overview. *Clostridium difficile* (*C. difficile*, "C diff") is becoming an important public health issue as a cause of nosocomial and community-based diarrhea. It is an anaerobic, spore-forming bacillus recognized as occurring exclusively in the presence of exposure to antibiotics; it is the only anaerobe that poses a nosocomial risk.⁹

CDAD is increasingly recognized among residents of long-term care facilities but has especially been reported with increasing frequency in acute care or short-stay hospitals because of the high rates of antibiotic use in hospitals.¹⁰⁰

Incidence. CDAD rates and severity appear to be increasing rapidly in the United States and Canada and show no sign of decline. U.S. hospital discharges for which *C. difficile* was listed as a diagnosis doubled from 82,000 in 1996 to 178,000 in 2003, with the highest rate reported in adults aged 65 years or older. Incidence is an estimated 228 cases per 100,000 persons.¹⁵⁰

Etiology, Transmission, and Risk Factors. Transmission of *C. difficile* occurs primarily in health care facilities via the fecal-oral route following contamination of the hands of HCWs and patients with oral ingestion of the causative organism. Contamination of the patient care environment also plays an important role.¹⁶² Nonhuman reservoirs such as water, raw vegetables, and animals can also cause infection.⁵ Tube feeding is also a risk factor.³²

The reason for rising rates of CDAD is unknown. Several possible explanations have been offered, includ-

ing new and evolving patterns of antimicrobial drug use (e.g., increased use of fluoroquinolones), promotion of alcohol-based hand sanitizers (these may not be as effective as soap in removing *C. difficile*), and the emergence of more virulent strains of *C. difficile* better able to cause transmission and disease.¹⁰⁰

Age (65 years and older) is a definite risk factor, especially when linked with antibiotic use and residence in acute or long-term health care facilities, where exposure is increased through physical proximity of residents and with their health care providers, or admittance to a room that housed someone with CDAD during the last 10 to 14 days.^{27,52}

Older adults may also have decreased host defenses to protect them from CDAD such as decreased stomach acid as part of the aging process (achlorhydria) or decreased immune responsiveness. There is also an increased use of medications such as H₂ receptor blockers or proton-pump inhibitors, which are becoming increasingly recognized in association with CDAD.⁴²

Pathogenesis. Change in the protective flora of the enteric system induced by antibiotics may produce acute diarrhea by overgrowth and toxin production by *C. difficile*. Gastric acid constitutes a major defense mechanism against ingested pathogens. Loss of stomach acid has been associated with colonization of the normally sterile upper GI tract.

In the healthy person, the *C. difficile* organism is inactive in the spore form. It is assumed that antibiotic-induced change in the competing intestinal flora promotes a conversion from a spore state to the vegetative forms, which then replicate and produce toxins, causing cellular damage of the intestinal mucosa and increased gut permeability.⁹

A more virulent strain of *C. difficile* is associated with more frequent and more severe disease with higher rates of toxic megacolon, shock, and even death. The variant strains of *C. difficile* are resistant to fluoroquinolones, producing up to 23 times more toxins than other strains.⁹

Clinical Manifestations. CDAD is easily recognized by persistent diarrhea following antibiotic consumption. The patient may not have a fever, but an elevated white blood cell count is common. Manifestations of CDAD can range from uncomplicated diarrhea to sepsis and even death from toxic megacolon. Antibiotic-associated colitis is discussed further in Chapter 16.

MEDICAL MANAGEMENT

DIAGNOSIS AND TREATMENT. Diagnosis is typically confirmed by identifying toxins in the stool of the infected individual. Colonoscopy identifying pseudomembranous lesions present late in the disease may help identify difficult-to-diagnose cases.

Standard treatment consists of prompt discontinuation of the antibiotic agent with administration of oral metronidazole (Flagyl), an antibiotic effective against anaerobic bacteria. In some cases, oral vancomycin is the antimicrobial treatment of choice. Individuals with the fulminant form of colitis may not respond to this treatment. Management of diarrhea is essential to prevent electrolyte imbalance and subsequent sequelae.

PREVENTION. Prevention of this nosocomial infection is imperative to reduce patient morbidity and mortality and reduce health care costs associated with infection control, medication, and excess hospital days.

C. difficile is a bacteria commonly present in our everyday environment in natural water sources, soil, animals, and raw vegetables.⁵ CDAD is, by and large, a nosocomial disease and therefore most prevention and control efforts take place in the health care setting. Proven strategies include hand hygiene, barrier precautions, environmental disinfection, and antimicrobial stewardship.¹⁵⁰

Contact precautions are recommended to prevent the transmission of *C. difficile* in the health care setting consisting of using private rooms or rooms shared by CDAD patients, using gloves and gowns for all contact, and using disposable equipment or cleaning equipment between use with each patient.⁵⁵ The primary means of reducing risk is through the careful use of antimicrobial drugs.⁵⁸

Preventing oral ingestion of the *C. difficile* organism is important whenever suction devices are used in the oral cavity (mouth). A strong correlation has been noted between ventilator-associated pneumonia rates and CDAD rates in the critical care population, emphasizing again the importance of cleanliness of anything introduced into a patient's mouth and stomach.¹⁵¹

Staphylococcal Infections

Overview and Incidence. Staphylococci bacteria are among the most common bacterial pathogens normally residing on the skin. Although there are more than 30 species of staphylococci, only a few are clinically relevant.

Staphylococci can be characterized as coagulase (a surface enzyme that converts fibrinogen to fibrin) positive or negative. *Staphylococcus aureus* is, almost without exception, the only significant staphylococcal species that is coagulase positive.

Several coagulase-negative bacteria may be pathogenic, such as *S. epidermidis*, but all are often collectively referred to as coagulase-negative staph (CoNS). *S. aureus* is very virulent, whereas the forms of CoNS are less virulent but still cause significant human infections. These organisms are nonmotile and anaerobic. They are hardy and able to survive on inanimate objects for an extended period.

Staphylococci bacteria are the leading cause of nosocomial and community-acquired infections, accounting for about 13% of all hospital infections each year. This figure translates into approximately 2 million hospital infections annually, resulting in 60,000 to 80,000 deaths each year. Staphylococcal species are the most common cause of infections, affecting all ages and involving the blood, skin, lung, soft tissue, joints, and bones. They are a leading cause of infective endocarditis. *S. epidermidis* is the most common cause of prosthetic device infections.

Risk Factors. *S. aureus* spreads by direct contact with colonized surfaces or people. The most common location of human colonization of *S. aureus* are the nares (nasal passages), although the skin, axilla, perineum, vagina, and oropharynx can also be colonized. Approximately

Table 8-6 Staphylococcal Infections

Type	Predisposing Factors
Bacteremia	Infected surgical wounds Abscesses Infected IV or intra-arterial catheter sites; catheter tips Infected vascular grafts or prostheses Infected pressure ulcers Osteomyelitis Injection drug use Source unknown (primary bacteremia) Cellulitis Burns Immunosuppression Debilitating diseases (e.g., diabetes, renal failure) Infective endocarditis Cancer (leukemia) or neutropenia after chemotherapy or radiation
Pneumonia	Immunodeficiency (especially older adults and children younger than 2 years) Chronic lung disease and cystic fibrosis Malignancy Antibiotics that kill normal respiratory flora but spare <i>S. aureus</i> Viral respiratory infections, especially influenza Bloodborne bacteria spread to the lungs from primary sites of infections (e.g., heart valves, abscesses, pulmonary emboli) Recent bronchial or endotracheal suctioning or intubation
Enterocolitis	Broad-spectrum antibiotics as prophylaxis for bowel surgery or treatment of hepatic coma Elderly; newborns (associated with staphylococcal skin lesions)
Osteomyelitis	Hematogenous organisms (blood borne) Skin trauma Infection spreading from adjacent joint or other infected tissues <i>S. aureus</i> bacteraemia Orthopedic surgery or trauma Cardiothoracic surgery Usually occurs in growing bones, especially femur and tibia of children younger than 12 years Male sex
Food poisoning	Contaminated food
Skin infections	Decreased resistance Burns or pressure ulcers Decreased blood flow Skin contamination from nasal discharge Foreign bodies Underlying skin diseases such as eczema and acne Common in persons with poor hygiene living in crowded quarters

25% to 50% of healthy adults are intermittently or perpetually colonized with *S. aureus*.

Colonization occurs more frequently in individuals with diabetes who are insulin dependent, individuals who are HIV positive, clients receiving hemodialysis, IV drug users, and persons with chronic skin lesions. Infections occur more frequently in individuals who are colonized than those who are not (typically with their own colonized strain).

Individuals more likely to develop a staph infection include surgical and burn patients (from damaged skin); individuals with diabetes who require insulin (from needlesticks, perhaps decreased leukocyte function); anyone who is neutropenic (PMNs dangerously low); and anyone with prosthetics, chronic skin disease, rheu-

matoid arthritis, catheters, or on corticosteroid therapy (unable to control local infections sufficiently).

Predisposing factors are multiple and varied depending on disease location (Table 8-6). Transient contamination of HCWs' hands can also transmit the bacteria to clients and is an important cause of spreading bacteria in health care settings.¹⁵⁹

Pathogenesis. *S. aureus* cannot invade through intact skin or mucous membranes; infection usually begins with traumatic inoculation of the organism. Once inside the body, the organism is a virulent pathogen, secreting membrane-damaging enzymes and toxins that harm host tissues. Staphylococci stimulate a significant host immune response, forming a suppurative or pustular local response. If the bacteria are then able to evade local host

**Figure 8-2**

Staphylococcus skin abscess. (Reprinted from Braverman IM: *Skin signs of systemic disease*, ed 3, Philadelphia, 1998, W.B. Saunders.)

defenses, they can spread via the bloodstream to almost any location in the body. The bones, joints, kidney, lung, and heart valves are the most common sites of *S. aureus* infections.

Clinical Manifestations. When *S. aureus* is inoculated into a previously sterile site, infection usually produces suppuration and abscess formation. The abscesses range in size from microscopic to lesions several centimeters in diameter filled with pus and bacteria (Fig. 8-2).

Infective syndromes include osteomyelitis, infections of burns or surgical wounds,¹⁴³ respiratory tract infections (particularly in newborns and intubated or ICU patients), bacterial arthritis, septicemia, bacterial endocarditis, and toxic shock syndrome. Consumption of toxins produced by staphylococcal species in contaminated food is a common cause of food poisoning. Fever, chills, and symptoms associated with the affected area may accompany staphylococcal infection of any body part.

Acute *S. osteomyelitis*, usually in the bones of the legs, most commonly affects boys between ages 3 and 10 years, most of whom have a history of infection or trauma. Osteomyelitis presenting in the vertebrae affects adults older than age 50 years, often following staphylococcal infections of the skin or urinary tract, after prostatic surgery, or after pinning of a fracture. Clinical manifestations include abrupt onset of fever, shaking chills, pain and swelling over the infected area, restlessness, and headache (see the section on Osteomyelitis in Chapter 25).

Staphylococcus-associated skin infections include cellulitis (see the section on Cellulitis in Chapter 10), boil-like lesions (furuncles and carbuncles), and small macules that may develop into pus-filled vesicles. Associated symptoms may include mild or spiking fever and malaise. Scalded skin syndrome is typically seen in infants with exfoliation of skin secondary to staphylococcal-produced toxins.

CoNS is most often associated with prosthetic device infections: prosthetic cardiac valves and joints, vascular grafts, intravascular devices, and CNS shunts.¹⁴⁴ CoNS does not typically produce fulminant symptoms. Indi-

viduals may present with mild fever, pain, and slightly elevated leukocytosis with a subtle course of illness.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Gram Stain and culture of the organism from the infected site, blood, or other fluid is usually diagnostic; antibiotic sensitivity testing is important. Polymerase chain reaction (PCR)-based assays are becoming more available for rapid diagnosis of *S. aureus*, especially in areas where resistance is high. Since CoNS is ubiquitously found on the skin and is a common contaminant, the isolation of CoNS does not always indicate infection. In fact, blood cultures positive for CoNS are truly positive in only 10% to 25% of isolates.⁹⁶

Isolation of *S. aureus* in blood cultures usually confirms bacteremia, and several positive blood cultures can be diagnostic of endocarditis in the presence of a new murmur or echocardiogram demonstrating valvular vegetations. Treatment may include drainage of any abscesses, removal of prosthetic devices, administration of antibiotics, and supportive therapy for the specific type and site.

MRSA is resistant to available antimicrobials except vancomycin, making vancomycin the preferred drug for staphylococcal infections, especially nosocomial ones. Reports of reduced susceptibility to vancomycin among strains of *S. aureus* (VISA strains) are on the rise.^{23,49}

New classes of antistaphylococcal drugs have been developed to combat the multidrug-resistant *S. aureus*. Combinations of drugs have also been used to create multifaceted attacks to decrease resistance development.

Debate continues on the efficacy of preventing *S. aureus* using topical antimicrobial ointments to eradicate colonization in high-risk individuals. Studies are underway to develop an effective vaccine to prevent *S. aureus*. Although it may be several years before vaccines against *S. aureus* are available for human beings, immunization may represent an important step toward solving the problem of antimicrobial resistance.¹¹⁰

Prognosis is good with treatment, although antibiotic-resistant strains are increasingly associated with morbidity and mortality. Infective endocarditis with *S. aureus* remains a serious life-threatening illness, with 20% to 40% mortality. Visceral abscesses, osteomyelitis, and staphylococcal sepsis are illnesses that are potentially lethal.

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-2

Staphylococcal Infections

PREFERRED PRACTICE PATTERNS

7A: Prevention/Risk Reduction for Integumentary Disorder

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7E: Impaired Integumentary Associated with Skin Involvement Extending into Fascia, Muscle, or Bone, and Scar Formation (osteomyelitis, lymphangitis)

Some organisms such as *S. aureus* (and streptococci) are considered resident organisms because they are not easily removed by scrubbing and often can be cultured from the HCWs skin. Many HCWs carry *S. aureus* without sequelae and are able to shed organisms into nonintact skin areas of susceptible hosts, causing infections.

For the most part, good handwashing with soap or an alcohol-based rub is adequate in the therapy or home setting, but therapists need to consistently educate family members and caregivers about infection control through handwashing and environmental management.

Antimicrobial soaps that contain chemicals to kill transient and some resident organisms may be recommended, although debate continues over questions of long-term resistance. The choice of using an antimicrobial soap or plain soap is usually based on the need to reduce and maintain minimal counts of resident organisms and to mechanically remove transient organisms such as *Pseudomonas*, *Escherichia coli*, *Salmonella*, or *Shigella*. When working with people who are infected with drug-resistant, gram-positive cocci such as MRSA¹⁰¹ or VRE, antimicrobial soap may be recommended since some studies have shown that these organisms persist on hands until an antimicrobial product is used.⁶⁰

Some questions have been raised regarding isolation procedures for methicillin- or vancomycin-resistant clients who have been discharged from an isolation setting as an inpatient but are now returning to therapy as an outpatient. Is a separate area required? Are special precautions necessary?

Anyone with an active, resistant infection should not be discharged from an inpatient setting. However, if such a case is encountered, the therapist must remember that these organisms are spread by contact. Therefore the same germicidal cleaning measures used in a hospital or institutional setting are required.

All equipment that comes in direct contact with a draining area needs to be cleaned with an approved germicidal before and after use. Isolation (e.g., private room or separate area of the gym or clinic) is not required. American Physical Therapy Association infection control guidelines for hydrotherapy and physical therapy aquatic programs recommend that clients with MRSA may attend therapy programs provided the area of colonization can be contained. If it is in a wound, the drainage must be contained within the dressing without evidence of breakthrough.

Streptococcal Infections

Croup A Streptococci. *Streptococcus pyogenes*, the prototype of group A streptococci (GAS), is one of the most common bacterial pathogens of human beings of any age. It causes many diseases of diverse organ systems, ranging from skin infections to acute self-limited pharyngitis to postinfectious syndromes of rheumatic fever and poststreptococcal glomerulonephritis (Box 8-7).

Box 8-7

STREPTOCOCCAL INFECTIONS

Streptococcus pyogenes (group A streptococci)

Suppurative

- Streptococcal pharyngitis
- Scarlet fever (scarlatina)
- Impetigo (streptococcal pyoderma)
- Streptococcal gangrene (necrotizing fasciitis)
- Streptococcal cellulitis
- Streptococcal myositis
- Puerperal sepsis (following vaginal delivery or abortion)
- Toxic shock syndrome
- Pneumonia (rare)

Nonsuppurative

- Rheumatic fever
- Acute poststreptococcal glomerulonephritis

Streptococcus agalactiae (group B streptococci)

- Neonatal streptococcal infections
- Adult group B streptococcal infection

Streptococcus pneumoniae

- Pneumococcal pneumonia
- Otitis media
- Meningitis
- Endocarditis

The diseases caused by *S. pyogenes* may be considered in two categories: suppurative (formation of pus) and nonsuppurative. Suppurative diseases occur at sites where the bacteria invade and cause tissue necrosis and pus formation, inciting an acute inflammatory response. By contrast, nonsuppurative diseases are immune related and triggered by previous streptococcal infection.

GAS is typically transmitted via contact with respiratory droplets, although other, less-common mechanisms have been identified, such as foodborne. In health care settings, personnel may spread GAS after contact with clients who have infected secretions or may become infected themselves. The infected personnel subsequently acquire a variety of GAS-related illnesses (e.g., toxic shock-like syndrome, cellulitis, and pharyngitis).

HCWs who are GAS carriers have infrequently been linked to sporadic outbreaks of surgical site, postpartum, or burn wound infection and to foodborne transmission of GAS causing pharyngitis. Adherence to standard precautions or other transmission-based precautions can prevent nosocomial transmission of GAS to personnel. Restriction from client care activities and food handling is indicated for personnel with GAS (see Table 8-5).

Signs, symptoms, and complications of GAS depend upon the location of the infection.

Streptococcal Pharyngitis. Streptococcal pharyngitis, commonly known as *strep throat*, occurs most commonly in children and accounts for 15% to 36% of all sore throats in children.⁸⁷ It is also the only pharyngitis requiring antibiotic treatment. The infection occurs most commonly from October to April in children aged 5 to 10 years, but a recent increase has occurred among adults aged 30 to 50 years. This organism often colonizes in

throats of people with no symptoms; up to 20% of schoolchildren may be carriers (pets may also be carriers).

The incubation stage is 1 to 5 days. Clinical manifestations vary but may include a fever, sore throat with pain on swallowing (may be severe), beefy red pharynx, edematous tonsils with exudate, swollen lymph nodes along the jaw line, generalized malaise and weakness, anorexia, and occasional abdominal discomfort (particularly in children). Up to 40% of affected children may have symptoms too mild for diagnosis.

Complications have significantly been reduced with the advent of antibiotics but may include otitis media, sinusitis, peritonsillar or retropharyngeal abscess, bacteremia, endocarditis, meningitis, pneumonia, and osteomyelitis. Poststreptococcal sequelae include acute rheumatic fever or acute glomerulonephritis. Diagnosis is usually by rapid diagnostic kits, but if negative a throat culture (the gold standard) should be performed. Treatment is with antibiotics to avoid poststreptococcal syndromes.

Scarlet Fever. Scarlet fever usually follows untreated streptococcal pharyngitis but may also occur after wound infections. It is caused by a streptococcal strain that releases a pyrogenic exotoxin and is most common in children aged 2 to 10 years old.

The streptococcus is acquired by inhalation or direct contact with oral secretions and presents with a sore throat, fever, strawberry tongue (white-coated tongue with prominent red papillae), and a fine erythematous rash that blanches on pressure and has been described as feeling like sandpaper. The rash first appears on the upper chest and then spreads to the extremities, sparing the soles and palms. After 6 to 9 days there may be desquamation of the skin of the soles and palms. The cheeks may be flushed, with pallor around the mouth. Rarely complications may include high fever, arthritis, and jaundice.

Impetigo. Impetigo is principally caused by GAS, although other streptococcal or staphylococcal species may be involved. It occurs most commonly in children aged 2 to 5 years, especially in hot, humid weather. Predisposing factors include close contact in schools, overcrowded living quarters, poor skin hygiene, and minor skin trauma.

Colonization with GAS most often precedes the skin lesions, so good hygiene is essential. Small macules appear and rapidly develop into vesicles that become pustular and encrusted. Neither fever nor pain is typically a component of impetigo and if present suggests another diagnosis.

Scratching spreads infection and may develop into lymphadenitis or cellulitis. Lesions often affect the face although any area of the skin can be involved (see Chapter 10). Antibiotic treatment should cover both staphylococcal and streptococcal species. Untreated skin infections do not lead to rheumatic fever, although they can trigger poststreptococcal glomerulonephritis.

Erysipelas. Streptococcal species and other organisms (such as *S. aureus*) may cause a type of cellulitis that may cause an acute infection of the skin accompanied by fever and chills. The skin may be very red, shiny, and swollen

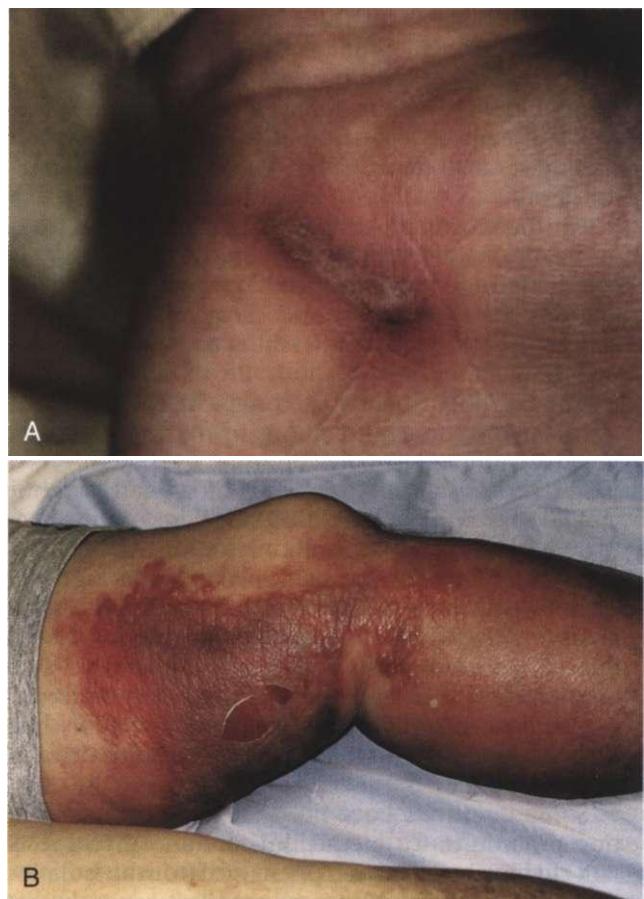


Figure 8-3

Cellulitis and lymphangitis. **A.** Infection in a wound is usually due to streptococcal bacteria, but often in combination with *Staphylococcus*. Infection can be local without streaking (cellulitis), local infection with streaking toward the heart (lymphangitis or blood poisoning), or pus forming (boil or abscess). This boy stuck a needle into a burn blister on his palm the previous day. Note the redness and the streaking. **B.** Erysipelas, a type of cellulitis, is more of a clinical diagnosis describing an infectious skin condition characterized by sharp, elevated, demarcated borders; redness; swelling; vesicles; bullae; fever; pain; and lymphadenopathy. Erysipelas affects the face and legs most often. It is almost always caused by GAS but can be caused by *Staphylococcus*. (A, Courtesy Bruce Argyle, Utah Mountain Biking, <http://www.utahmountainbiking.com/firstaid/infect.htm>. B, Reprinted from Black JM, Hawks JH: *Medical-surgical nursing: clinical management for positive outcomes*, ed 7, Philadelphia, 2006, W.B. Saunders.)

in appearance (Fig. 8-3). This type of cellulitis develops over a few hours; bullae may form in the affected areas after 2 to 3 days. The margins between normal and infected skin are well demarcated.

The most common areas affected include the face and legs, although erysipelas can occur anywhere. The disease is more common in women and may be especially severe in anyone with a debilitating condition.⁶⁶ Antibiotics that cover GAS are the treatment of choice but may need to be broadened to include staphylococcal species.

Streptococcal Cellulitis. Streptococcal cellulitis, an acute spreading inflammation of the skin and subcutaneous tissues, usually results from infection of burns, wounds, or other breaks in the skin, although in some

cases no entry site is noted. Recurrent episodes of cellulitis may occur in extremities in which lymphatic drainage has been impaired (e.g., postaxillary node dissection, site of saphenous vein harvest).

Lymphangitis may accompany cellulitis or may occur after clinically minor or unapparent skin infection. Lymphangitis is readily recognized by the presence of red, tender, linear streaks directed toward enlarged, tender regional lymph nodes. It is accompanied by systemic symptoms such as chills, fever, malaise, and headache (see the sections on Cellulitis in Chapter 10 and Lymphangitis in Chapter 13).

Streptococcal Necrotizing Fasciitis. Necrotizing fasciitis (NF) is a serious infection that progresses rapidly along fascial planes, usually in the legs, causing severe tissue damage as it spreads. The classification of soft-tissue necrotizing infections has changed over the years, and NF was previously known as streptococcal gangrene. There are different types of NF caused by various organisms but they often have overlapping features.

One classification separates NF into type I and type II. Type I is a polymicrobial infection that typically occurs following surgery when protective mucosa of the GI or genitourinary tract is damaged or when seeding occurs from an occult abscess. Gas formation may also form. *S. pyogenes* accounts for more than 60% of type II NF and often originates from a distal break in the skin or transient bacteremia. NF is a rare but serious complication of chickenpox and should be suspected in children with progressive pain, fever, and lethargy.³⁰

NF may be difficult to diagnose initially. Pain and fever are present but the overlying skin often is without abnormalities. The infection spreads rapidly, causing edema and tenderness. Changes later occur in the skin as thrombosis of blood vessels occurs. The skin turns a dark red color with accompanying induration. Bullae form and fill with dark fluid. Later the skin becomes friable and turns a maroon or black color consistent with ischemia.

The bacteria produce several pyogenic endotoxins, causing severe breakdown of tissue in multiple organs. At least 50% of affected individuals experience toxic shock syndrome with hypotension, nausea, vomiting, and delirium.³¹ There is often renal and hepatic compromise as well as pulmonary infiltrates, leading to respiratory distress. Mortality rate is high (30% to 70%).

Immediate surgery with aggressive debridement of all necrotic tissue along with appropriate intensive IV antibiotics are essential to save muscles and limbs.¹⁴¹ Gram stain and culture of the site is essential to identify the organism(s) and antimicrobial susceptibility. Multiple procedures and serial debridement may be necessary with secondary closure since the demarcation line of infection is difficult to identify.

Gram stain is a laboratory staining technique used to distinguish between two groups of bacteria by identifying differences in the structure of their cell walls. The Gram stain was named after Danish bacteriologist Christian Gram, who developed this technique. Gram stain has become an important laboratory tool, distinguishing between so-called gram-positive bacteria, which remain colored after the staining procedure, and gram-negative bacteria, which do not retain dye.

Streptococcal Myositis. Streptococcal myositis is a rare but potentially life-threatening entity characterized by severe pain and inflammation in the affected muscle with few abnormalities of overlying skin. Typically blunt, non-penetrating trauma or hematologic seeding of bacteria to the muscle leads to the infection. Two forms of streptococcal myositis have been reported. The first is a slower, less-virulent process, while the second is more fulminant with systemic symptoms, high fever, bacteremia, and a high mortality rate.¹⁶³

This condition can also be caused by other bacteria (usually *S. aureus*), mycobacteria, fungi, viruses, and protozoan forms. Clinical features of myositis and NF often overlap. Therapy includes aggressive surgical debridement and IV antibiotics (see the section on Myositis in Chapter 25 and Streptococcal Gangrene in this chapter).

Puerperal Sepsis. Puerperal sepsis follows abortion or normal delivery when streptococci colonizing the woman or transmitted from medical personnel invade the endometrium and surrounding structures, lymphatics, and bloodstream. The resulting endometritis and septicemia may be complicated by pelvic cellulitis, septic pelvic thrombophlebitis, peritonitis, or pelvic abscess. Before the antibiotic era and the benefits of handwashing between clients were known, this disease was more common and associated with a high mortality rate.

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-3

Streptococcal Infections

Health care personnel can transmit and acquire streptococcal infections. Guidelines for preventing transmission must be followed at all times (see Boxes 8-3 and 8-4 and Table 8-4).

PREFERRED PRACTICE PATTERNS

7A: Prevention /Risk Reduction for Integumentary Disorder

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone

Group B Streptococci. Group B streptococcal infection (*S. agalactiae*) is the leading cause of neonatal pneumonia, meningitis, and sepsis. The organism is also an infrequent cause of pyogenic infections in adults. Several thousand neonatal infections with group B streptococci occur in the United States each year, and about 3% to 4% of the infants with the infection die.¹⁴⁰ Group B streptococci are part of the normal vaginal flora and are found in 30% of women. Most newborns born to colonized women acquire the organism as they pass through the birth canal, but only 1% of these infants develop group B streptococcal infections.

Neonates who develop early infections (birth to 1 week) may present with hypotension, pneumonia (and respiratory distress), bacteremia, or meningitis. Late disease (1 week to 3 months) is acquired either at birth

or from contact with the infected mother or other personnel. These babies demonstrate fever, bacteremia, meningitis, and pneumonia.¹⁴⁰

Rapid administration of IV antibiotics is essential. The CDC recommends that pregnant women be screened for carriage of group B strep and appropriate antibiotics be given to prevent transmission to the baby.^{123,139} A vaccine is under development that would reduce the number of women and babies exposed to antibiotics.

Group B streptococci can cause peripartum infections in women, such as endometritis or chorioamnionitis. Resulting bacteremia can lead to endocarditis or meningitis. Appropriate antibiotics are required for treatment.

Streptococcus pneumoniae

Etiologic and Risk Factors. Pneumonia and other infections, such as sepsis, otitis media, and meningitis, can be caused by *S. pneumoniae* (pneumococcal pneumonia or pneumococcus) (see Box 8-6). This organism colonizes the oropharynx and can be found in 5% to 10% of healthy adults and 20% to 40% of children. Once colonized, the host can develop illness related to *S. pneumoniae* by spreading to the sinuses or eustachian tubes or inhaling the bacteria into the lungs. Hematogenous spread occurs, creating disease in other organs.

Transmission from person to person is by direct contact or inhalation of droplets of respiratory secretions. *S. pneumoniae* causes disease particularly in the very young and the old. It is the most common cause of community-acquired pneumonia and the most common cause of death by a vaccine-preventable bacterial disease.¹²⁴ Pneumococcal pneumonia often follows influenza or viral respiratory infections and is often seen in clients with chronic diseases or immunosuppression and in alcohol abusers. Other risk factors are included in Box 8-8.

S. pneumoniae is the most common cause of meningitis in adults, infants, and toddlers. Head trauma, CSF leaks, otitis media, and sinusitis may precede pneumococcal

meningitis, creating an extension of disease or opportunity for direct infection.²⁰ Pneumonia may lead to bacteremia with subsequent seeding of the meninges.

Clinical Manifestations. Clinical manifestations of pneumonia include acute onset of fever, chills, pleuritis with pleuritic chest pain, and dyspnea with productive cough or purulent sputum that may be blood tinged. Because pneumococcal disease occurs most commonly in the very young and the very old, the presenting features will vary.

Older adults may have only a slight cough or delirium but lack a fever. Complications from pneumococcal pneumonia may include empyema (about 2% of cases), bacteremia, sepsis, or meningitis. Infection of the meninges stimulates a robust inflammation, leading to increased intracranial pressure and brain edema with headache and nausea/vomiting, mental status changes, stiff neck, and fever.

The disease progresses rapidly over 24 to 48 hours and mortality rate is high without treatment. Rare complications of *S. pneumoniae* include pericarditis, endocarditis, peritonitis, and septic arthritis. Septic arthritis can occur in a natural or prosthetic joint or in damaged joints from rheumatoid arthritis; underlying chronic joint disease may delay diagnosis.

Pneumococcal (septic) arthritis secondary to this pathogen is relatively uncommon and occurs principally in older adults and individuals with underlying diseases (e.g., rheumatoid arthritis).¹⁶⁷ The clinical symptoms are similar to other forms of hematogenous pyogenic joint infections. Adjacent osteomyelitis involving the vertebral bones may be detected on radiologic examination.

Diagnosis, Treatment, and Prevention. Diagnosis of pneumococcal disease is by laboratory examination of sputum (pneumonia), CSF (meningitis), or blood (bacteremia) with Gram stain and culture of the organism. Treatment is with antibiotics that are effective against local pneumococcal strains and take into consideration resistance patterns in the community.

Currently, immunization for pneumococcal disease is available and is recommended in specific circumstances as defined by the CDC (see Box 8-7).¹²⁴ Adults aged 65 years and older should receive one dose of the vaccine, as should special groups such as Native Alaskans and certain Native American populations. Individuals aged 2 to 64 years with defined conditions such as immunocompromise, HIV, asplenia, chronic liver or renal dysfunction, pulmonary disorders (COPD), and diabetes mellitus should be vaccinated.

In the year 2000 a conjugate vaccine became available for young children and infants. Since its use, there has been a significant decrease in invasive pneumococcal disease in children.^{74,177} The pneumococcal polysaccharide vaccine available for adults appears to have reduced efficacy in the older adult in preventing pneumonia, although it has protection against bacteremia.⁷⁴

Overall the rate of antibiotic-resistant invasive pneumococcal infections has decreased in young children. As a bonus, the use of vaccines against pneumococcal bacteria in children has also reduced the rate of pneumococcal disease in adults because fewer bacteria are passed from children to adults.^{74,84}

Box 8-8

RISK FACTORS FOR PNEUMOCOCCAL DISEASE

- Age
 - Children younger than 2 years
 - Adults aged 65 years or older
- Recent episode of influenza or viral respiratory infection
- Chronic illness
 - Diabetes mellitus
 - Heart disease
 - Pulmonary disease
 - Renal disease
 - Liver disease
- Immunosuppression
 - HIV
 - Multiple myeloma
 - Leukemia
 - Lymphoma
 - Hodgkin's disease
 - Transplant recipients
 - Chronic use of corticosteroids
- Neurologic impairment (e.g., CSF leak)
- History of alcoholism
- Asplenia (absent, removed, or nonfunctioning spleen)

Gas Gangrene (Clostridial Myonecrosis)

Definition and Overview. *Gangrene* is the death of body tissue, usually associated with loss of vascular (nutritive, arterial circulation) supply and followed by bacterial invasion and putrefaction. The three major types of gangrene are dry, moist, and gas gangrene.

Dry and moist gangrene results from loss of blood circulation due to various causes; gas gangrene occurs in wounds infected by anaerobic bacteria, leading to gas production and tissue breakdown. This is a rare but severe and painful condition that usually follows trauma or surgery in which muscles and subcutaneous tissues become filled with gas and exudate. The disease spreads rapidly to adjacent tissues and can be fatal within hours of onset.

Pathogenesis. Fortunately, the anaerobic conditions necessary to foster clostridial growth are uncommon in human tissues and are produced only in the presence of extensive devitalized tissue, such as occurs with severe trauma, wartime injuries, and septic abortions.

Contributing factors include hypoxia from injury to blood vessels near the wound site, pressure dressings, tourniquets, local injection of vasoconstrictors, foreign bodies, damaged tissues from earlier injury, and concurrent microbial infections.

Gas gangrene is most often found in deep wounds, especially those in which tissue necrosis further reduces oxygen supply. Such necrosis releases both carbon dioxide and hydrogen subcutaneously, producing interstitial gas bubbles. Gas gangrene (*Clostridial myonecrosis*) is rare when wounds are promptly and thoroughly cleaned and debrided of traumatized tissue.

Clinical Manifestations. The incubation period for gas gangrene is less than 3 days after injury. Sudden, severe pain occurring at the site of the wound, which is tender and edematous, is an early sign and symptom of gas gangrene. The skin darkens because of hemorrhage and cutaneous necrosis. The lesion develops a thick discharge with a foul odor and may contain gas bubbles. Crepitus may be felt on palpation of the skin from the gas bubbles in muscles and subcutaneous tissue.

True gas gangrene produces myositis and anaerobic cellulitis, affecting only soft tissue. The skin over the wound may rupture, revealing dark-red or black necrotic muscle tissue accompanied by a foul-smelling watery or frothy discharge. Associated symptoms may include sweating, low-grade fever, and disproportionate tachycardia followed by hemolytic anemia, hypotension, and renal failure.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Prevention is the key to avoiding gas gangrene before treatment is required. To prevent gangrene in an open wound, the wound should be kept as clean as possible. Special wound care is particularly important in people with diabetes mellitus, malnutrition, and immunodeficiency.

Early, immediate intervention is necessary with surgical debridement and excision of necrotic tissue. Diagnosis can be established by frozen section of muscle. Gram stain and culture also verify the diagnosis but not as

rapidly. Radiographs may show evidence of gas formation, although gas may not be seen in early stages. Antibiotics are administered, but if significant gangrene develops amputation may be necessary. Hyperbaric oxygen therapy is controversial.¹⁶⁴

With prompt treatment, 80% of people with gas gangrene of the extremities survive; prognosis is poorer for gas gangrene in other sites such as the abdominal wall, uterus, or bowel.

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-4

Gas Gangrene

PREFERRED PRACTICE PATTERNS

7A: Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

Careful observation may result in early diagnosis. With any postoperative or posttraumatic injury, look for signs of ischemia such as cool skin; pallor or cyanosis; sudden, severe pain; sudden edema; and loss of pulses in the involved limb. Record carefully and immediately report these findings to the medical staff. Throughout this illness adequate fluid replacement is essential; assess pulmonary and cardiac functions often.

Special care to prevent skin breakdown is important, and meticulous wound care following surgery is imperative. To prevent gas gangrene, routinely take precautions to render all wound sites unsuitable for growth of *Clostridia* by attempting to keep granulation tissue viable; adequate debridement is imperative to reduce anaerobic growth conditions. Notify the physician immediately of any devitalized tissue. Position the client to facilitate drainage.

Psychologic support is critical, as these clients can remain alert until death, knowing that death is imminent and unavoidable. The therapist must be prepared for the foul odor from the wound and prepare the client emotionally for the large wound after surgical excision. Wound care requires sterile procedures to prevent spread of bacteria; dispose of drainage material and dressings in double plastic bags for incineration. No special cleaning measures are required after the client is discharged.

Pseudomonas

Overview. *Pseudomonas aeruginosa* is a major opportunistic pathogen and one of the most common hospital- (particularly ICU) and nursing home-acquired (nosocomial) pathogens. *Pseudomonas* is uncommon in community-acquired infections and healthy individuals. The organism infrequently colonizes human beings, but it can cause disease, particularly in the hospital environment,

where it is associated with pneumonia, wound infections,¹⁴³ urinary tract disease, and sepsis in debilitated people.

Burns, urinary catheterization, cystic fibrosis, chronic lung diseases, neutropenia associated with chemotherapy, and diabetes all predispose to infections with *P. aeruginosa*. It thrives on moist environmental surfaces, making swimming pools, whirlpool tubs, respiratory therapy equipment, flowers, endoscopes, and cleaning solutions prime targets for growth.

This organism produces several virulence factors and is inherently antibiotic resistant. Spread of the organism in a health care setting is by contact, typically from a reservoir as described above. HCWs have been known to pass the organism on their hands or under fingernails.¹² Colonization usually precedes infection, although colonization is not indicative of future infection. It is unclear which factors lead from colonization to invasive disease.¹²²

Pathogenesis. *P. aeruginosa* produces of an array of proteins, which allow it to attach to, invade, and destroy host tissues while avoiding host inflammatory and immune defenses. Injury to epithelial cells uncovers surface molecules that serve as binding sites for *P. aeruginosa*.

Many strains of this pathogen produce a proteoglycan that surrounds the bacteria, protecting them from mucociliary action, complement, and phagocytes. The organism releases extracellular enzymes, which facilitate tissue invasion and are partially responsible for the necrotizing lesions associated with *Pseudomonas* infections. This pathogen can also invade blood vessel walls and produce systemic pathologic effects through endotoxin and several systemically active exotoxins.

Clinical Manifestations. Signs and symptoms of *Pseudomonas* infection vary with the site of infection and the state of host defenses.¹²² If the host has the capacity to respond to the invading bacteria with neutrophils, an acute inflammatory response results.

The *Pseudomonas* organism often invades small arteries and veins, producing vascular thrombosis and hemorrhagic necrosis, particularly in the lungs and skin. Blood vessel invasion predisposes to bacteremia, dissemination, and sepsis. This bacterium causes infections of the respiratory tract (pneumonia), bloodstream, CNS, skin (see Fig. 8-4) and soft tissues, bone and joints, and other parts of the body.

Respiratory Tract Infections. Pneumonia caused by *P. aeruginosa* is one of the most common causes of nosocomial pneumonia.¹²⁸ The infection may be primary (contained to the lungs) or cause a bacteremia with metastasis of infection. Primary pneumonia is most often seen in clients with a predisposing history of chronic lung disease, congestive heart failure, or AIDS who are in a hospital setting. These individuals are often intubated and in the intensive care unit.

Signs and symptoms are typical of pneumonias seen with other organisms, such as dyspnea, fever, productive cough, low oxygenation, elevated white cell count, and delirium. Cavitary lesions may be seen on chest radiograph (particularly if the client has AIDS), and pleural effusions are common.

Pneumonia secondary to *P. aeruginosa* can be severe and life threatening. Bacteremic pneumonia begins as a respiratory tract infection but spreads to the bloodstream and metastasizes to other viscera, causing hemorrhaging and necrosis. This bacteremia is often associated with clients who are neutropenic and unable to control the infection. Symptoms include those of pneumonia with sepsis, which is rapid and severe. The chest radiograph may demonstrate necrotizing pneumonia with cavitary lesions.

Chronic infections with *Pseudomonas* are noted in children or young adults with cystic fibrosis. Over time there is chronic progression of symptoms with acute exacerbations of disease. Clients typically experience mucous plugging and airway inflammation, which predispose to *P. aeruginosa* infection. The bacteria then contribute to further mucous plugging and cause a supportive reaction leading to bronchiectasis and atelectasis. Episodes of pneumonia are seen more frequently as more lung is damaged and becomes fibrotic.

Bacteremia. Bacteremia may occur without prior pneumonia and is an important cause of serious, life-threatening bloodstream infections in clients with neutropenia. *P. aeruginosa* bacteremia is typically acquired in the hospital and may be primary (no identifiable source) or secondary to a focal infected site (e.g., skin, lungs, intravascular source, indwelling catheters, GI or urinary tracts).

As with other *Pseudomonas* infections, bacteremia is rapidly progressive without treatment, with high morbidity and mortality rates. Clients experience fever, tachypnea, tachycardia, hypotension, and delirium, which can lead to renal failure, acute respiratory distress syndrome, and death. Rarely characteristic lesions of *Pseudomonas* bacteremia, *ecthyma gangrenosum*, develop on the skin. These vesicles are initially hemorrhagic with progressive necrosis and ulceration. They occur as single lesions or in small groups.

Central Nervous System Infections. *Pseudomonas* infections of the CNS result from extension from a contiguous structure such as the ear, mastoid, or paranasal sinus; direct inoculation into the subarachnoid space or brain by means of head trauma, surgery, or invasive diagnostic procedures (e.g., lumbar punctures, spinal anesthesia, intraventricular shunts); and bacteremic spread from a distant site of infection such as the urinary tract, lung, or endocardium.

The clinical manifestations of *Pseudomonas* meningitis are like those of other forms of bacterial meningitis (see Chapter 29) and include fever, headache, stiff neck, nausea, and confusion. The onset of disease may be acute and occur suddenly or may be more gradual and insidious. Rapidly progressive and toxic disease is seen more frequently in clients who are bacteremic, while those without associated bacteremia may have few systemic signs and symptoms (e.g., those whose meningitis is related to neurosurgery or extension from a contiguous site of chronic infection).

Skin and Soft Tissue Infections. *Pseudomonas* disease of the skin and mucous membranes can result from primary or metastatic foci of infections. Common predisposing factors for primary skin and soft tissue infections are a



Figure 8-4

Pseudomonas. Blue-green color in a burn wound indicates infection by *Pseudomonas aeruginosa*. (Reprinted from Gould BE: *Pathophysiology for the health professions*, ed 3, Philadelphia, 2006, W.B. Saunders, courtesy Judy Knighton, Ross Tilley Burn Center, Sunnybrook and Women's College Health Center, Toronto, Ontario, Canada.)

breakdown in the integument, especially resulting from surgery, burns, trauma, and pressure ulcers; whirlpool use; and chemotherapy-induced neutropenia.

The wound is hemorrhagic and necrotic and rarely may have a characteristic fruity odor (sweet, grapelike odor) with a blue-green exudate that forms a crust on wounds (Fig. 8-4). *Pseudomonas* bacteria may produce distinctive skin lesions known as *ecthyma gangrenosum*, as described above, associated with *Pseudomonas* bacteremia.

Pseudomonas burn wound sepsis is a dreaded complication of extensive third-degree burns and is characterized by multifocal black or dark-brown discoloration of the burn eschar; degeneration of the underlying granulation tissue with rapid eschar separation and hemorrhage into subcutaneous tissue; edema, hemorrhage, and necrosis of adjacent healthy tissue; and erythematous nodular lesions on unburned skin.

Systemic manifestations may include fever, hypothermia, disorientation, hypotension, oliguria, ileus, or leukopenia. The diagnosis is based on clinical signs and symptoms; biopsy of the burn site, which demonstrates evidence of invading bacteria; and culture positive for *Pseudomonas*.

Bone and Joint Infections. *Pseudomonas* infections of the bones and joints result from hematogenous spread from other sites or extension from contiguous sites of infection. Contiguous infections are usually related to penetrating trauma, surgery, or overlying soft tissue

infections. Injection drug users may contaminate drugs or water with *Pseudomonas*, which often seeds the sternoclavicular or other joints.¹³¹

P. aeruginosa is the most common cause of osteochondritis of the foot following a puncture wound.⁸ Infection involves the cartilage of the small joints and the bones of the foot. Typically, the person experiences early improvement in pain and swelling following a puncture wound only to have the symptoms recur or worsen several days later. The average duration of symptoms before diagnosis is several weeks; fever and other systemic signs are usually absent. An area of superficial cellulitis is evident on the plantar surface of the foot, or there may merely be tenderness to deep palpation.

Bloodborne *Pseudomonas*, from injection drug use or pelvic surgery, appears to have a predilection for fibrocartilaginous joints such as the symphysis pubis.¹³⁰ Vertebral osteomyelitis caused by *P. aeruginosa* is occasionally associated with complicated urinary tract infections and genitourinary surgery or instrumentation.

This disease occurs most often in older adults and involves the lumbosacral spine. Physical signs include local tenderness and decreased range of motion in the spine; fever and other systemic symptoms are relatively uncommon. Mild neurologic deficits may be present (see further discussion in Chapter 27).

Other *Pseudomonas* Infections. *Pseudomonas* is noted to cause disease of the external ear, which may be benign ("swimmer's ear") or malignant (invasion of bone, soft tissue, and cartilage). *P. aeruginosa* infection of the cornea causes bacterial keratitis or corneal ulcers.

Corneal ulcers can progress rapidly with complications such as corneal perforation, anterior chamber involvement, and endophthalmitis. Native heart valves or prosthetic valves can become infected with *P. aeruginosa*, causing endocarditis. Infection of native valves is seen in injection drug users, and multiple valves may be involved.

Right-sided endocarditis may be complicated by septic pulmonary emboli causing pulmonary disease, while left-sided endocarditis may cause heart failure, brain abscess, or mycotic aneurysms. *P. aeruginosa* is the most common cause of nosocomial urinary tract infections often arising from urinary catheters, instrumentation, or surgery. The prostate or kidney stones may harbor the bacteria, resulting in recurrent infections.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Diagnosis requires isolation of the *Pseudomonas* organism in blood, spinal fluid, urine, exudate, or sputum culture. Antibiotic therapy is initiated immediately with two antipseudomonal drugs; local *Pseudomonas* infections or septicemia secondary to wound infection requires debridement or drainage of the infected wound. For older children with cystic fibrosis, intermittent, inhaled tobramycin has been used with some success at reducing hospitalizations and improving lung function, although long-term studies are needed.¹³²

P. aeruginosa infections are among the most aggressive human bacterial infections, often progressing rapidly to sepsis, especially in people with poor immunologic

resistance (e.g., premature infants; aging adults; and those with debilitating disease, burns, or wounds).

In local *Pseudomonas* infections, treatment is usually successful and complications are rare. Immediate medical intervention is necessary; septicemic *Pseudomonas* infections are associated with a high mortality rate. Medical management is directed according to the site of infection and may include antibiotics, surgery, pulmonary therapy, respiratory assistance if necessary, and other supportive measures dictated by the presence of septic shock and other complications.

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-5

Pseudomonas Infections

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

4E: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated with Localized Inflammation (arthropathy associated with infection)

5C: Impaired Motor Function and Sensory Integrity Associated with Nonprogressive Disorders of the Central Nervous System: Congenital Origin or Acquired in Infancy or Childhood (meningitis)

5D: Impaired Motor Function and Sensory Integrity Associated with Nonprogressive Disorders of the Central Nervous System: Acquired in Adolescence or Adulthood

6D: Impaired Aerobic Capacity/Endurance Associated with Cardiovascular Pump Dysfunction or Failure (endocarditis)

Reservoirs for *P. aeruginosa* are most often medical equipment or moist areas in the health care setting, such as sinks. However, the source of some outbreaks has been traced to HCWs' hands or nails.^{51,177} *P. aeruginosa* can be removed from the skin by following proper hand hygiene guidelines, which prevents further spread of the organism.^{12,133} Proper cleaning of any equipment in contact with mucous membranes or a moist environment is absolutely critical.¹⁰⁶

Anyone who is immunocompromised should be protected from exposure to this infection. Wound care requires strict sterile technique.

VIRAL INFECTIONS

Bloodborne Viral Pathogens

The bloodborne viruses that most endanger HCWs are the bloodborne pathogens hepatitis B virus (HBV), hepatitis C virus (HCV), and HIV. In 1991 the U.S. Congress passed the Bloodborne Pathogens Standard, prepared by

the Occupational Safety and Health Administration (OSHA) and written to help eliminate or minimize occupational exposure to HBV, HCV, HIV, and other bloodborne pathogens.¹⁶⁹

The guidelines are based on the use of standard precautions, including appropriate handwashing and barrier precautions, to reduce contact with body fluids potentially contaminated by these viruses. The use of safety devices and techniques to reduce the handling of sharp instruments can help in the reduction of significant contact with body fluids, particularly blood or blood-containing fluids.¹⁷⁹

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-6

Bloodborne Viral Pathogens

PREFERRED PRACTICE PATTERNS

See individual disease discussion.

Hepatitis B

See discussion on HBV in Chapter 17. Nosocomial transmission of HBV is a serious risk for HCWs. The risk of acquiring HBV infection from occupational exposure is dependent on the degree of exposure to blood and the presence of HBV e antigen (HBeAg) from the source. An approximate 22% to 31% chance exists of acquiring HBV after percutaneous (needlestick) contact with a hepatitis surface antigen (HBsAg) and HBeAg seropositive source.¹⁶⁹ Yet the rate of development of hepatitis from a needlestick in a HBeAg-negative, HBsAg-positive source was only 1% to 6%.

HBV can be transmitted to HCWs via percutaneous injuries or by direct or indirect contact with blood from an infected client. Blood contains the highest amount of infected particles and is the most efficient means of transmission. HBV in blood is able to survive up to 1 week on environmental surfaces. The incubation period is 45 to 180 days (average 60 to 90 days).

Preexposure HBV vaccination of HCWs who are at risk (e.g., those who work in an area likely to have contact with blood and body fluids) is strongly recommended and can prevent acquisition of HBV (see Table 8-4).²⁴ Once a HCW has been exposed, the HBsAg status of the person and vaccination and vaccine-response status of the exposed HCW must be determined. Postexposure prophylaxis (PEP) should then be given if the HCW is unvaccinated or nonresponsive to previous vaccine and the source is HBsAg positive.

Studies done in perinatal situations have shown that the combination of HBV immunoglobulin (HGIB) and HBV vaccine is 85% to 95% effective in preventing HBV as compared to either agent alone. Data show that HGIB and the HBV vaccine are effective as single PEP for preventing clinical disease in occupational exposures (70% to 75%).

Although data are not available comparing the combination PEP in occupational exposures, the current recommendations have been extrapolated from other studies and the combination presumed to

be as effective as either one alone.¹⁶⁹ HGIB and the vaccine are both tolerated well with only rare reports of anaphylaxis.

The OSHA bloodborne pathogen standard mandates that HBV vaccine and HGIB be made available, at the employer's expense, to all HCWs with potential occupational exposure. In addition, strict adherence to handwashing and standard precautions (see Appendix A) is critical in prevention of the transmission of hepatitis-contaminated body fluids. Transmission is also prevented by use of barriers during sexual activity and by not sharing personal or other items that may have blood on them.

For the HCW who is positive for HIV, HBV, or HCV, the CDC guidelines are based on the assumption that the risk of transmission to others is greatest during invasive procedures that include (for the therapist) sharp debridement or digital palpation of a needle tip (or other sharp instrument) in a poorly visualized or highly confined anatomic site.

Any therapist performing such procedures should determine his or her own HIV, HBeAg, and HCV antigen status. Those who are infected should not perform the procedures unless they have obtained guidance from an expert panel about when and how they may safely do so. Although the CDC does not mandate restricted practice, the requirement that clients of an infected HCW be notified of the provider's infection status before undergoing these procedures is a restriction on practice.⁵⁷

Some experts consider these practice guidelines and the idea of mandatory testing of all clinicians performing invasive procedures cost prohibitive and unnecessary given the fact that HCWs are far more likely than their clients to contract bloodborne pathogens from exposure to infected blood. Viewed in this context, extraordinary measures to protect clients undergoing these interventions may not be warranted, and the current CDC recommendations are considered overly conservative.^{18,57}

Hepatitis C

See the section on HCV in Chapter 17.

HCV is the most common etiologic agent in cases of non-A, non-B hepatitis in the United States. Sero-prevalence studies among HCWs have shown a significant association between acquisition of disease and health care employment, specifically client care or laboratory work. Accidental percutaneous injuries (needlesticks or cuts with sharp instruments) are the highest risk vehicle for transmission to HCWs from people with acute or chronic HCV.

The incubation period for HCV is 6 to 7 weeks, and nearly all individuals with acute infection will have chronic (more than 3 to 6 months' duration) HCV infection with persistent viremia and the potential for transmission to others over an extended period.

Serologic assays to detect HCV antibodies (not protective antibodies) are available and are used to determine source and HCW status after exposure. More than 75% of all people with HCV develop chronic hepatitis, and cirrhosis may develop in up to 25% of

those with chronic HCV. A risk of developing hepatocellular carcinoma (approximately 3% to 5% per year) exists, and a small percentage of people with HCV may progress to fulminant liver failure.¹¹⁸

Currently no vaccine against HCV is available, and no PEP can be recommended. PEP with immune globulin or antiviral agents does not appear to be effective in preventing HCV infection.¹⁶⁰ Follow-up HCV testing should be performed to verify if seroconversion occurs.

Strict adherence to handwashing and standard precautions (see Appendix A) is critical in prevention of transmission of hepatitis-contaminated body fluids. Transmission is also prevented by use of barriers during sexual activity and by not sharing personal or other items that may have blood on them.

Human Immunodeficiency Virus

See the section on HIV in Chapter 7.

Nosocomial transmission of HIV from clients to HCWs may occur after percutaneous or, infrequently, mucocutaneous exposure to blood and body fluids containing blood. According to prospective studies, the risk of acquiring HIV infection following percutaneous exposure has been estimated to be about 0.3% and 0.09% after mucous membrane exposure for HCWs.¹¹⁸ Nonintact skin exposure risk is not precisely known but estimated to be less than mucous membrane exposure.

HIV seroconversion among HCWs after percutaneous exposure to blood containing HIV is associated with the following factors: (1) presence of visible blood on the device prior to injury, (2) a procedure that involved needle placement into a person's vein or artery, or (3) deep injury with the contaminated device.

Transmission of HIV infection was also associated with injuries in which the source was terminally ill with AIDS, thus exposing the HCW to high titers of active virus. Assessing the viral load of the source of the exposure may be a surrogate for transmission risk, but studies are lacking and even very low viral titers do not negate the risk following exposure.¹¹⁸

In 2005 the CDC updated previously published provisional recommendations for postexposure chemoprophylaxis and use of antiretroviral therapies.¹¹⁸ HCWs who sustain needlestick or other high-risk exposures should be counseled and offered HIV baseline and follow-up blood testing as soon as possible and should then be treated with antiretroviral therapy per CDC protocol.

HCWs at high risk for acquiring HIV postexposure should be given at least three drugs; drug selection should also consider the source's viral resistance. Data are lacking concerning the number of drugs offered HCWs who are at a low risk of acquiring HIV. Two-drug PEP therapy may be sufficient in these cases.

Postexposure, the involved area of contact should be immediately washed thoroughly (not scrubbed) with antiseptic soap and then rinsed with water. Therapy with antiretrovirals should be initiated immediately and should be continued for 4 weeks.¹¹⁸

Continued.

Consultation with an infectious disease or HIV specialist should be quickly obtained for selection of an antiviral regimen. Since HCWs are not able to tolerate many of the antiviral medications because of side effects, careful monitoring should be provided. Prevention of transmission includes the use of meticulous handwashing, standard precautions, and the same precautions described above for the bloodborne hepatitis viruses.

Herpesviruses

Overview and Definition

The term *herpes* is derived from the Greek word *herpein*, which means "to creep." The word refers to the tendency for this type of viral infection to become chronic, latent, and recurrent. The known human herpesviruses (HHVs) are divided by genomic and biologic behavior into eight types (Box 8-9).

All herpesviruses are morphologically similar, but the biologic and epidemiologic features of each are distinct. Subclinical primary infection with the herpesviruses is more common than clinically symptomatic illness, and each type then persists in a latent state for the rest of the life of the host.

With the herpes simplex virus (HSV) and varicella-zoster virus (VZV), the virus remains latent in sensory ganglia and, upon reactivation, lesions appear in the distal sensory nerve distribution. Virus reactivation in immunocompromised hosts may lead to widespread lesions in affected organs such as the viscera or the CNS. Severe or fatal illness may occur in infants and the immunocompromised. Association with malignancies includes EBV with Burkett's lymphoma and nasopharyngeal carcinoma and HHV-8 with Kaposi's sarcoma and body cavity lymphoma.^{33,138}

Herpes Simplex Viruses Types 1 and 2

See Table 8-7.

Incidence, Etiologic Factors, and Risk Factors. Approximately 70% of Americans older than 12 years harbor HSV-1, which is usually responsible for cold sores; 20% older than 12 years have HSV-2, the principal cause of genital herpes.¹³⁵ Since 1966, the incidence of genital herpes has continued to rise.

Box 8-9

TYPES OF HERPESVIRUSES

Herpes simplex virus (HSV)	Type 1
Herpes simplex virus (genital herpes)	Type 2
Varicella-zoster virus (VZV)	Type 3
Epstein-Barr infectious mononucleosis virus (EBV)	Type 4
Cytomegalovirus (CMV)	Type 5
Roseola (exanthema subitum) human herpes virus (HHV)	Type 6
Herpes virus serologically associated with roseola, human herpes virus (HHV)	Type 7
Human herpes virus associated with Kaposi's sarcoma (HHV)	Type 8

Both strains can infect any visceral organ or mucocutaneous site, and HSV-1 can be transmitted to the genital area during oral sex. HSV creates a significant health risk since infection with these viruses increases the risk of infection with HIV and increases production of the HIV virus once infected. Seroprevalence for both agents increases with age and with sexual activity for HSV-2.

Intermittent, asymptomatic shedding is common and is the typical time of transmission, usually during the period immediately preceding appearance of sores. Sexual contact during asymptomatic periods is less likely to result in transmission of the virus than when sores are present. However, since people with genital herpes are more likely to engage in sexual contact when they are free of sores, the rate of asymptomatic transmission is still significant.

Infants born to women with genital herpes can be infected with HSV when they pass through an infected birth canal. The virus can also be passed to other regions of the body by hand contact, particularly in people who are immunosuppressed (e.g., older adults, transplant recipients, people with cancer undergoing chemotherapy, and anyone with HIV or other conditions that weaken the immune system).

Pathogenesis. Even though HSV-1 and -2 are the two most closely related herpesviruses and share antigenic cross-reactivity, these two agents are genetically and serologically distinct and produce different clinical symptoms. HSV-1 and -2 primarily affect the oral mucocutaneous (cold sores and mouth sores) and genital areas (genital herpes), respectively. Primary infection occurs through a break in the mucous membranes of the mouth, throat, eye, or genitals or via minor abrasions in the skin. Initial infection can be asymptomatic, although minor localized vesicular lesions may be evident.

Local multiplication occurs, followed by viremia and systemic infection with a subsequent lifelong latent infection and periodic reactivation of the virus. During primary infection, the virus enters peripheral sensory nerves and migrates along axons to sensory nerve ganglia in the CNS, allowing the virus to escape immune detection and response.

During latent infection of nerve cells, viral DNA is maintained and not integrated into surrounding cellular structures, thus maintaining true latency. Various disturbances such as physical or psychologic stress can disrupt the delicate balance of latency, and reactivation of the latent virus occurs. The virus travels back down sensory nerves to the surface of the body and replicates, forming new lesions. Although painful, most recurrent infections resolve spontaneously, recurring at a later time.

Clinical Manifestations. Primary HSV-1 (first episode) typically affects the mouth and oral cavity, causing vesicles in the mouth, throat, and around the lips. Vesicles typically open to form moist ulcers after several days. Systemic symptoms can accompany the lesions such as fever, myalgias, and malaise. Symptoms and lesions resolve within 3 to 14 days.

Herpetic whitlows (herpetic infection of the fingers) can result from inoculation of the finger from a herpes

Table 8-7 Most Common STIs*

Infection	Incidence [†]	Transmission	Clinical Manifestations	Treatment [‡]
HPV (genital warts)	6.2 million new cases/year	Unprotected sexual contact; condoms do not provide 100% protection since the virus can be spread by contact with an infected part of the genitals not covered by a condom; vertical transmission from mother to newborn with vaginal delivery (rare)	Often asymptomatic; warts on the vulva, anal region, vagina, cervix, mouth, penis, scrotum, or groin 1–6 months after sexual contact with infected person; <i>in women:</i> abnormal Pap smear; HPV can cause cervical cancer	Can be removed using topically applied chemicals, cryotherapy, or surgical therapies; recurrence is not uncommon
Chlamydia	1 million new cases/year	Unprotected vaginal or anal intercourse; infection transmitted from infected mother to infant during delivery	<i>In men:</i> none or urethritis with discharge or burning with urination <i>In women:</i> none or vaginal discharge with pus or mucus; pain; burning during urination; can cause PID and infertility if untreated; eye infections and respiratory tract infections in newborn	Can be cured with antibiotics; partner must be treated as well; PID may require additional treatment
HSV type 2 (genital herpes)	1 million new cases/year, 45 million carriers	Oral, genital, or anal sex; kissing or touching an infected area where there is a break in the skin; can be spread by asymptomatic person; transmission from mother to child during vaginal birth	None or vesicular (blisterlike) lesions on the genitals, vagina, cervix, anal region, mouth, or throat; can cause serious complications if untreated	Cannot be cured but healing can be accelerated and recurrence of outbreaks can be reduced with antivirals; partner must be informed
Gonorrhea (the "clap")	330,000 new cases/year	Unprotected oral, vaginal, or anal sex; transmission to baby during delivery	<i>In men:</i> urethritis with discharge, frequent urge to urinate and pain during urination; may be asymptomatic <i>In women:</i> none or slight vaginal discharge and difficulty or pain during urination; pelvic pain; vaginal bleeding between periods; PID <i>Both:</i> arthritis (if untreated)	Can be cured with antibiotics, although some strains are drug resistant
HBV	73,000 new cases/year	Infected blood, sexual contact, occupational needlesticks, sharing needles, newborn infected during delivery	May be asymptomatic; jaundice, arthralgias, dark urine, anorexia, nausea, abdominal pain, cirrhosis, liver failure, liver cancer, clay-colored stools, fever	Can be prevented with HBV vaccine; in unvaccinated people, HBIG and HBV vaccine given as PEP; antiviral agents are used but relapse on cessation of treatment is common
Syphilis (secondary)	See Syphilis, primary below		Flulike symptoms, lymphadenopathy, mucocutaneous lesions and rash occurring 6–12 weeks to 1–2 years after infection (but has a wide range of clinical symptoms)	

Continued.

Table 8-7 Most Common STIs*—cont'd

Infection	Incidence [†]	Transmission	Clinical Manifestations	Treatment [‡]
Syphilis (latent)			None; asymptomatic	
Syphilis (late; can occur up to 20 years after second stage)			Cardiovascular and CNS damage	
HIV/AIDS	42,000 new cases/year; half caused by sexual contact	Exposure to blood or blood products; exposure to body fluids (blood, semen, vaginal secretions, breast milk); sexual contact; shared needles in injection drug users; transmission from mother to child during vaginal delivery or breast feeding	Widespread illness due to immune system decline; may not develop symptoms for 10 years or more after infection	Cannot be cured but combined antiviral therapy can prolong life for many people
Syphilis, primary	8000 new cases/year (primary and secondary combined); overall incidence has been increasing since 2000	Unprotected sexual contact; sexual contact with exudates of skin and mucous membranes of infected person; transplacental infection of fetus if mother is infected; can be transmitted through blood transfusions [‡]	Painless sore at site of infection (genitals, mouth) occurring 3-8 weeks after infection	Can be cured with antibiotics in primary, secondary, and latent stages; late-stage disease may cause irreversible damage

PEP, Postexposure prophylaxis.

Modified from Centers for Disease Control and Prevention: *Sexually transmitted disease surveillance, 2004*, Atlanta, GA, 2005, U.S. Department of Health and Human Services.

*Listed in descending order by incidence.

[†]All sexually transmitted diseases can be prevented by sexual abstinence and mutually monogamous sex between two uninfected partners. The CDC has come under criticism by the medical community for not stressing this point in their prevention programs for young people.

[‡]Centers for Disease Control and Prevention: *Control of communicable diseases manual*, ed 17, Atlanta, GA, 2000, U.S. Department of Health and Human Services.

lesion (Fig. 8-5). Prior to implementation of standard glove precautions HSV-1 was the most common cause of herpetic whitlow. Now, HSV-2 has been implicated more often than HSV-1. HSV-1 can also infect the genitourinary system, causing signs and symptoms similar to HSV-2.

Primary infection is often asymptomatic. Recurrences are usually milder, involve fewer lesions, are of shorter duration, and in immunocompetent hosts are confined to the lips (herpes labialis). Recurrent genital HSV-1 is milder and less frequent than HSV-2. Recurrences are most commonly induced by stress, fever, sunlight, infection, or other factors.

HSV-2 is most often acquired through sexual contact. Primary HSV-2 causes vesicles to form in the genitourinary tract. Lesions are usually painful, small, grouped, and vesicular, with possible burning and itching. The blisterlike lesions break and weep after a few days, leaving ulcerlike sores that usually crust over and heal in 1 to 3 weeks.

Genital ulcers may occur on the genital area, cervix, buttocks, rectum, urethra, or bladder, causing vaginal and urethral discharge, dysuria, cervicitis, proctitis, and tender

inguinal adenopathy. Systemic symptoms occasionally noted include headache, malaise, myalgias, and fever. Primary infection can be asymptomatic. Genital HSV-2 reactivation may be associated with a prodrome such as tingling or pain.

First symptomatic episodes may not be the initial infection. Known as nonprimary infections, individuals may have been previously exposed to HSV-1 and produced antibodies but not developed symptoms until exposed to HSV-2 or vice versa. In these cases, the initial symptomatic nonprimary infection has fewer symptoms and complications as compared to a first episode without previous exposure.⁸⁰

HSV can be responsible for other infections. Viral meningitis from HSV is caused by inflammation of the meninges surrounding the brain. This occurs more commonly from HSV-2 than HSV-1. Aseptic meningitis may develop 3 to 12 days following the appearance of lesions. Typical symptoms are headache, nausea, stiff neck, and fever. The prognosis is good for immunocompetent hosts.³³ An association between HSV-1 and Bell's palsy has also been established.



Figure 8-5

Herpetic whitlow. Herpetic whitlow is an intense, painful infection of the hand involving one or more fingers and typically affecting the terminal phalanx. HSV-1 is the cause in approximately 60% of cases of herpetic whitlow, and HSV-2 is the cause in the remaining 40%. **A**, Herpes simplex infection of the finger in a child. **B**, Herpetic whitlow of the thumb in an adult. (Reprinted from Callen JP: *Color atlas of dermatology*, ed 2, Philadelphia, 2000, W.B. Saunders.)

Herpes encephalitis (an infection of the brain tissue), although rare, is a more serious infection and accounts for 10% to 20% of all cases of acute, sporadic viral encephalitis in the United States. In children and young adults, primary infection is the main cause. Adults may have reactivation as the principal source. Presenting symptoms include fever, headache, behavioral and speech disturbances, and seizures. Abnormalities caused by HSV can be seen on MRI. Encephalitis carries high morbidity and mortality rates, and permanent neurologic sequelae often result even with treatment.

Herpetic keratitis (ulceration of the cornea due to infection) is the most common cause for corneal blindness in the United States. Onset is acute, accompanied by blurred vision, conjunctivitis, and pain. Despite treatment, recurrences are common and cause scarring, making

this a chronic disease. Prophylactic acyclovir may reduce recurrences and long-term scarring.¹¹² Severe scarring is an indication for corneal grafting.

Recurrences of HSV-1 or -2 increase during pregnancy but do not appear to affect the fetus. Primary infection with HSV during pregnancy can occasionally cause visceral dissemination in the mother and possible transmission to the fetus. Neonatal herpes may also occur from unknown shedding in the mother's genital tract at the time of delivery. If untreated, babies develop visceral dissemination or infection of the CNS, with an 80% mortality rate.¹⁷ Cesarean section reduces the risk of neonatal herpes in mothers known to be shedding the virus.

HSV can also disseminate to visceral organs, causing severe consequences such as hepatitis, thrombocytopenia, arthritis, and pneumonitis. Disseminated infection typically occurs in pregnant women (primary genital HSV) or immunocompromised persons (primary or recurrent HSV). HSV esophagitis is seen in immunocompromised hosts but rarely noted in immunocompetent persons.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PREVENTION. Clinical diagnosis of herpes is often insensitive. Up to 30% of first-episode genital herpes are caused by HSV-1, which has a low recurrence rate compared to HSV-2,³⁴ making distinction between the two types important.

Viral cultures of vesicular fluid are the standard laboratory test. The sample must be collected during the first few days the lesion is present in order for the results to be accurate. Type-specific serologic tests, enzyme-linked immunosorbent assay (ELISA), or immunofluorescent assay (IFA) provide a more rapid diagnosis while culture results are pending. PCR tests are also available and are the test of choice for detection of HSV in spinal fluid. While PCR is very sensitive, it is expensive and only available in certain laboratories.

An HSV vaccine that showed promise in animals ultimately failed human testing. Although no immunization against HSV infection is available, antiviral drugs can be used to treat initial cases, reduce the frequency and degree of viral shedding, and suppress recurrences.

The CDC has released recommendations and guidelines for the treatment of HSV-2.¹⁸⁴ Acyclovir, famciclovir, and valacyclovir are approved for the treatment and suppression of HSV-2. Famciclovir can be used to treat recurrent mucocutaneous HSV in individuals infected with HIV; valacyclovir is approved for the treatment of cold sores. These medications do not eradicate the virus and, once discontinued, there is no change in frequency, duration, or severity of recurrences.

Counseling regarding transmission and education on how to recognize symptoms and defer sex are essential in preventing new cases. Daily suppressive use of valacyclovir along with safe sex can reduce transmission of HSV-2. Proper use of condoms can also reduce the risk of acquiring HSV-2.¹⁷³ This is particularly helpful for couples where one is seropositive and the other seronegative for HSV.

SPECIAL IMPLICATIONS FOR THE THERAPIST 8-7

Herpes Simplex Virus**PREFERRED PRACTICE PATTERNS**

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

5C, 5D: Acquired Neuromuscular Involvement (meningitis)

Recurrent disease is best treated with acyclovir, and recurrent genital disease requires barrier precautions during sexual activity in addition to medication. Although herpes simplex is contagious, nosocomial transmission is rare. However, it has been reported in some high-risk areas such as nurseries, intensive care units, burn units, and other areas where immunocompromised individuals might be placed.

Nosocomial transmission of HSV occurs primarily through contact with lesions or with virus-containing secretions such as saliva, vaginal secretions, or amniotic fluid. Exposed areas of skin, particularly when minor cuts, abrasions, or other skin lesions are present, are the most likely sites of viral entry. The incubation period of HSV is 2 to 14 days.

HCWs may acquire a herpetic infection of the fingers (herpetic whitlow or paronychia) from exposure to contaminated oral secretions. Such exposures are a distinct risk for HCWs who have direct contact with either oral or respiratory secretions from clients.

HCWs can protect themselves from acquiring HSV by adhering to standard precautions and handwashing before all client contact and by the use of appropriate barriers such as a mask, gloves, or gauze dressing to prevent hand contact with the lesion. Some work restrictions may be appropriate for affected HCWs when active lesions are present (e.g., herpetic whitlow) (see Table 8-6). No reports are evident that HCWs with genital HSV infections have transmitted HSV to clients, so no work restriction for people with HSV-2 is indicated.¹⁶⁹

During the prodromal stage of herpes simplex, the levator scapulae becomes vulnerable to activation of its trigger points by mechanical stresses that are usually well within its tolerance. However, a stiff neck syndrome can develop a day or two before the fully developed symptoms of herpes simplex.¹⁴⁷

Careful questioning regarding previous history of herpes, presence of prodromal symptoms, and observation for the development of a new outbreak of sores during the episode of care will help the therapist in making an accurate assessment of the client's presentation.

Approximately 10% to 20% of the population develops the secondary, or reactivation, form of VZV, resulting in herpes zoster or shingles. Approximately 300,000 cases of shingles occur in the United States every year and cause significant pain and disability. Adults older than 50 years and anyone who is immunocompromised (e.g., HIV infection, chemotherapy, corticosteroid therapy, or cancer) are at greatest risk. Young adults such as college students living in dormitories are at increased risk for VZV as either chickenpox (first time) or shingles (recurrence).

Pathogenesis. Like other herpesviruses, VZV has the capacity to persist in the body (in sensory nerve ganglia) as a latent infection after the primary infection. VZV is acquired from contact with infected airborne droplets (from coughing or sneezing) into the respiratory tract or by direct contact with vesicular fluid to the respiratory tract or eye.

The virus is believed to initially multiply at the site of entry, with subsequent viremia occurring 4 to 6 days after infection. The virus then disseminates to other organs such as the liver, spleen, and sensory ganglia and further replicates in the viscera, followed by a secondary viremia with viral infection of the skin and mucosa (mouth, respiratory tract, or eye). Viral infection of the skin and mucosa produces vesicles filled with high titers of infectious virus, which then shed more viruses. The incubation period is from 14 to 16 days from exposure with a range of 10 to 21 days. This may be prolonged in immunocompromised people. VZV is present in white blood cells up to 5 days before the rash is present, and individuals can be contagious a day or two prior to the appearance of the rash. Individuals remain contagious until the lesions have crusted.⁷

The exact mechanism for the reactivation of VZV remains unknown, although shingles occurs more often in immunocompromised adults such as older adults, those with hematologic malignancies, especially leukemia and lymphoma, and people with HIV.

Clinical Manifestations. Disease manifestations are either chickenpox (varicella) or shingles (herpes zoster). (See Chapter 10 for discussion of clinical manifestations of herpes zoster.) Primary VZV is virtually always symptomatic. Second episodes of chickenpox are uncommon unless the child is younger than 1 year at the time of the first episode. A mild prodrome consisting of fever and malaise may precede the onset of the rash in adults, while in children the rash is often the first sign of disease.

The rash is classically described as a "dewdrop on a rose petal," with a vesicle on an erythematous base. The lesions begin as macules that quickly progress to papules, vesicles, and then pustules before crusting. VZV usually appears first on the scalp and moves to the trunk and then the extremities. Successive crops appear over several days, with lesions present in several stages of evolution at any one time.⁷

The generalized pattern of eruption without specific dermatome distribution distinguishes varicella from herpes zoster (Fig. 8-6). Shingles in the adult present as blisterlike lesions that erupt along dermatomes, with the highest concentration of lesions on the trunk corresponding with dermatomes from T3 to L3 (Fig. 8-7). Pain and

Varicella Zoster Virus (Herpesvirus Type 3)

Incidence. VZV is HHV-3 and is known as *chickenpox* or *shingles* (see the section on Viral Infections: Herpes Zoster, in Chapter 10). Prior to the availability of the varicella vaccine, primary or first-infection VZV accounted for about 3 to 4 million cases of chickenpox per year in the United States.

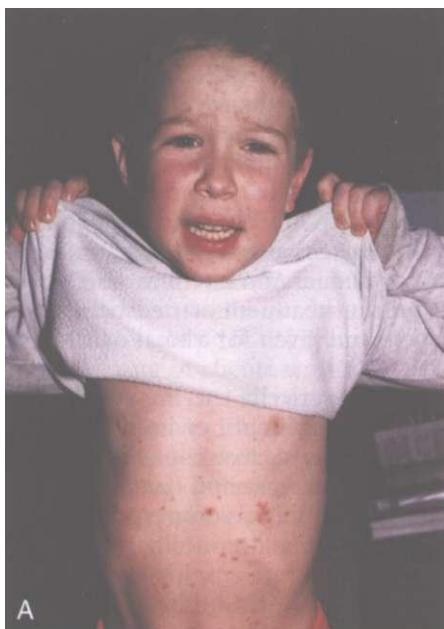


Figure 8-6

A, Early onset of varicella (chickenpox) in a young child. Painful itching can cause severe distress. Note the lesions on face and trunk. **B**, Varicella (chickenpox) with the more characteristic rash classically described as a "dewdrop on a rose petal," with a vesicle on an erythematous base. (A, Courtesy Catherine Goodman. B, Reprinted from Callen JP: *Color atlas of dermatology*, ed 2, Philadelphia, 2000, W.B. Saunders.)

itching are common symptoms during the eruption of the vesicles.

Complications of varicella occur more often in adults, infants, and the immunocompromised. Adults are more likely to develop pneumonitis and CNS involvement (cerebellar ataxia and encephalitis) than are healthy children. The immunocompromised, especially those with leukemia or lymphoma who are receiving continuous chemotherapy, can develop disseminated disease with severe visceral involvement, including pneumonitis and encephalitis. The most common complication among persons affected with VZV is secondary bacterial skin infections.¹⁷⁶

Shingles also can lead to chronic, often debilitating nerve pain called *postherpetic neuralgia (PHN)*, lasting years or even a lifetime and often resulting in significant

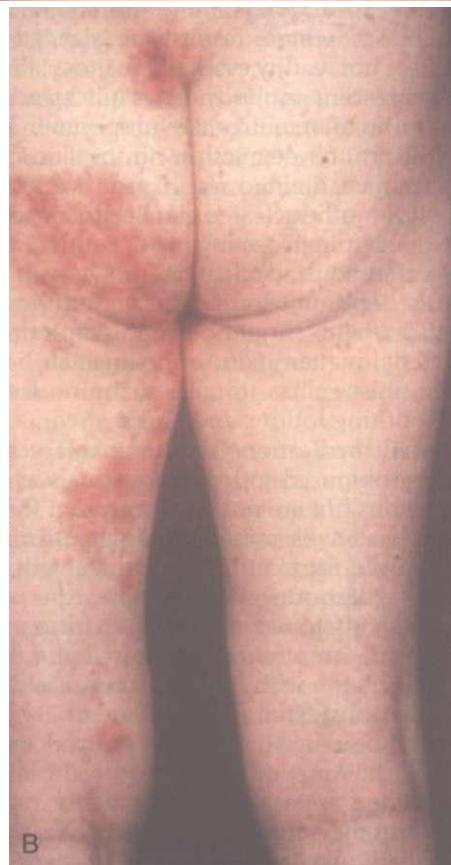


Figure 8-7

Herpes zoster (shingles). Small grouped vesicles occur along the cutaneous sensory nerve, forming pustules that crust over. Reactivation of VZV, the dormant chickenpox virus, is the underlying cause of this condition. **A**, Commonly seen on the trunk, these outbreaks can occur anywhere along the dermatome of the affected nerve. **B**, Lesions appear unilaterally and do not cross the midline. Usually external, these lesions can occur internally as well. Pain is often severe and can become chronic, a condition called *postherpetic neuralgia*. (A, Reprinted from Hurwitz S: *Clinical pediatric dermatology: a textbook of skin disorders of childhood and adolescence*, ed 2, Philadelphia, 1993, W.B. Saunders. B, Courtesy Mary Lou Galantino, Richard Stockton College of New Jersey, Pomona, NJ.)

morbidity and reduction in quality of life. Pain, hyperalgesia, and allodynia are typical of PHN.¹⁷⁶ Examples of allodynia include pain from the touch of clothing (touch allodynia) or pain that occurs from a draft of warm or cold air on the skin (thermal allodynia).

When contracted during the first or second trimesters of pregnancy, varicella carries a low risk of congenital malformations. Yet if a mother develops varicella within 5 days before delivery to 2 days after delivery, the newborn is at risk of serious disseminated disease.

MEDICAL MANAGEMENT

DIAGNOSIS AND TREATMENT. Diagnosis is usually made based on clinical symptoms; however, with the advent of the varicella vaccine, the number of atypical cases has increased, making laboratory diagnosis more relevant. VZV may be cultured from vesicular fluid but requires time.

Real-time PCR for VZV is the test of choice for severe or atypical cases where results are needed quickly, although this is not readily available in most laboratories. The direct fluorescent antibody test is quick and found in most laboratories but requires careful specimen handling. Vesicular fluid from a new lesion on the skin is the best source of specimen. Stained smears from vesicular scrapings may reveal multinucleated giant cells consistent with VZV infection. Serologic testing is not routinely used but may be useful in adult vaccination programs.

Bed rest is important until the fever has gone down, and the person's skin should be kept clean to avoid secondary bacterial contamination. Itching can be relieved with oral antihistamines, topical calamine lotion, and other skin-soothing lotions and baths.

The antiviral medications acyclovir, valacyclovir, and famciclovir can be used to treat individuals at high risk for complications but are not recommended for children with uncomplicated disease. Valacyclovir and famciclovir are only approved for treatment of adults with varicella. Oral acyclovir is recommended by some experts for pregnant women in their second or third trimesters while children who are immunocompromised should receive IV acyclovir. Persons with chronic lung or skin disease should also be considered for treatment.

Treatment should begin within 24 hours of the appearance of the rash. Antivirals may reduce the number of days new lesions appear and the severity of systemic symptoms, but they have not been shown to reduce transmission risk or reduce complications. Secondary bacterial infections of lesions are treated with antibacterial ointment or oral antibiotics if severe. Recovery from varicella infection usually results in lifetime immunity.

There is no cure for PHN. Treatment is often with nonopioid analgesics, tricyclic antidepressants, anticonvulsants, or topical local anesthetics based on the type of pain experienced (e.g., antidepressants for diffused pain, paroxysmal and local pain treated by anticonvulsants). Acyclovir has been shown to reduce the incidence of PHN; famciclovir reduces the duration of PHN. Evidence is lacking that oral corticosteroids given during the acute phase of the illness reduce the incidence or severity of PHN.⁷⁰

For individuals who are exposed but are without immunity to varicella, the varicella vaccine can be used up to 3 days postexposure (particularly in outbreaks) to aid in modifying symptoms or preventing the infection. For those individuals at high risk for severe complications and for whom the vaccine is contraindicated (such as

immunocompromised persons, pregnant women, or in neonatal situations), there are other methods of PEP.

VZV immune globulin (VZIG) is a product that contains high amounts of the VZV antibody. It should be administered within 96 hours of exposure to the virus to modify or prevent complications. In December 2005 the manufacturer of VZIG discontinued production and availability is limited. If VZIG is not available, IV immune globulin can be used, although data supporting its efficacy are not available. Acyclovir has also been suggested as a postexposure treatment started between day 7 and 10 of exposure and given for a total of 10 days.

PREVENTION. The varicella vaccine is recommended for all adults who lack evidence of immunity, especially in those persons who have close contact with individuals at high risk for severe disease and complications.

Adults who are at high risk for exposure and transmission should also receive the vaccine (such as teachers of young children, child care employees, and residents and staff at medical facilities). Children between the ages of 12 and 18 months should routinely receive the vaccine. Currently, the varicella vaccine is available with the measles, mumps, and rubella (MMR) vaccine.

It is also recommended that all children who have not developed immunity by the age of 13 years should be vaccinated (see Table 8-4).⁷ Because the vaccine is a live attenuated vaccine, it is contraindicated in pregnant women, those who may become pregnant within 4 weeks of receiving the vaccine, and individuals with HIV or other immunosuppressed states.

Since the varicella vaccine became available in 1995, there has been a progressive decline in the incidence of chickenpox and hospitalizations from complications.^{37,39,48} In 2004, there was an 80% to 90% decrease in acute varicella cases in active surveillance areas compared with 1995. The vaccine provides long-lasting (but not lifelong) immunity, with an 80% to 85% efficacy. Vaccine breakthrough cases are common but mild. Persons present with fewer lesions (usually less than 50) and lack systemic symptoms (such as fever).

The first shingles vaccine (Zostavax; zoster vaccine live) has been approved for adults aged 60 years and older. Zostavax has been shown to reduce the incidence of shingles by 51% and the incidence of PHN by 67% in adults aged 60 years and older. Among people who get shingles despite being vaccinated, it can reduce the disease's severity. Approval of this drug for use in adults ages 50 to 59 years is pending more evidence to support its safety and effectiveness in this age group.¹¹³

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-8

Varicella Zoster Virus

See Special Implications for the Therapist: Herpes Zoster in Chapters 10 and 39.

PREFERRED PRACTICE PATTERNS

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

Varicella is highly contagious. The period of communicability extends from 1 to 2 days before the onset of the rash through the first 4 to 5 days or until all lesions have formed crusts. Immunocompromised individuals with progressive varicella are probably contagious during the entire period new lesions continue to appear.

Nosocomial transmission of VZV is well known. Sources for nosocomial exposures include clients or residents, HCWs, and visitors, including children or HCWs, with either varicella or zoster. It is generally advisable to allow only HCWs who are immune to varicella to take care of clients with VZV.

Because of the possibility of transmission to and development of severe illness in high-risk clients, HCWs with localized zoster should not take care of such clients until all lesions are dry and crusted. However, they may take care of others if they cover their lesions (see the section on Herpes Zoster in Chapter 10).

Because airborne transmission of VZV occurs, affected individuals in a hospital setting should be isolated in negative pressure rooms until crusts have dried. HCWs who do take care of VZV clients should use contact precautions, including careful use of barriers such as gloves, gowns, and masks whenever in contact with active lesions. If serologic immunity of the HCW cannot be verified, varicella vaccine is recommended (see Table 8-4).

When unvaccinated, susceptible HCWs are exposed to varicella, they are potentially infectious 10 to 21 days after exposure, and exclusion from duty is indicated from day 10 through day 21 after the last exposure or until all lesions are dry and crusted.

When vaccinated HCWs are exposed to varicella, serologic testing for antibodies may be done, and exclusion from duty can occur if they are seronegative or develop varicella symptoms. VZIG and acyclovir are not routinely recommended postexposure for healthy HCWs (exceptions may be made for pregnant or immunocompromised HCWs).⁷

Any patient or client suspected of having herpes zoster (shingles) requires immediate medical attention. Reports of prodromal pain, symptoms, or onset of rash are red flags to warrant immediate diagnosis and early treatment. Early intervention can reduce morbidity.

Infectious Mononucleosis (Herpesvirus Type 4)

Overview. Infectious mononucleosis is an acute infectious disease caused by EBV, a member of the herpesvirus family. Although it may be seen at any age, it primarily affects young adults and children. In children, it is usually so mild that its presence often goes unnoticed.

Incidence, Etiologic Factors, and Risk Factors. Infection with EBV is common in the United States, with 95% of people between the ages of 35 and 40 years having been infected. When an adolescent or young adult becomes infected with EBV, 35% to 50% of the time they

will develop infectious mononucleosis. Both genders are affected equally. Incidence varies seasonally among college students but not among the general population. The reservoir of EBV is limited to human beings, and transmission is through contact with oral secretions, blood, or transplanted organs infected with the virus. Since about 80% of people carry EBV in the throat during the acute infection and for an indefinite period afterward, it is sometimes called the "kissing disease."⁸

Pathogenesis and Clinical Manifestations. EBV causes lymphoid proliferation in the blood, lymph nodes, and spleen. Characteristically, the virus produces fever, sore throat, and tender cervical lymphadenopathy; headache, malaise, and abdominal pain (from splenic enlargement or hepatitis) may also be present. The incubation period is about 4 to 6 weeks.

Temperature fluctuations occur throughout the day, peaking in the evening. There is often an increase in the white blood cell count, with an elevation in atypical lymphocytes. Hepatomegaly (accompanied by elevated liver enzymes), palatal petechiae, and splenomegaly are manifested in more than 10% of cases. The spleen may enlarge to two to three times its normal size, causing left upper quadrant pain with possible referral to the left shoulder and left upper trapezius region. Affected individuals are at risk for splenic rupture, and care should be taken to avoid trauma. Both the peripheral nervous system and CNS can be involved.

Overall, major complications are rare but may include splenic rupture, aseptic meningitis, encephalitis, hemolytic anemia, aplastic anemia, idiopathic thrombocytopenia, myocarditis, and Guillain-Barre syndrome. Symptoms subside about 6 to 10 days after onset of the disease but may persist for weeks. Symptoms from EBV-related infectious mononucleosis rarely last longer than 4 months.

Studies support an association between infectious mononucleosis and the subsequent development of multiple sclerosis for both adults and children.^{6,40,95} Young people who have had a strong immune response to the EBV are twice as likely to develop multiple sclerosis in adulthood. Scientists suspect this strong immune response could cross-react with brain substances, causing the brain to attack its own myelin in genetically susceptible individuals rather than the idea that the virus actually enters the brain.^{35,40}

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Diagnosis is based on clinical examination, laboratory tests, and a positive heterophil (Monospot) test. Heterophil antibodies (agglutinins for sheep red blood cells) in serum drawn during the acute illness and at 3- to 4-week intervals rise to four times normal. Rising levels of antibodies to EBV were once thought to be the cause of chronic fatigue syndrome but are now considered a result of chronic fatigue syndrome (see the section on Chronic Fatigue and Immune Dysfunction Syndrome in Chapter 7).

EBV may have a pathogenic role in causation of cancers such as Burkitt's lymphoma in Africa, nasopharyngeal carcinoma, Hodgkin's disease, and lymphoproliferative disorders in immunosuppressed and posttransplant clients.¹³⁸ Oral hairy leukoplakia, lymphoid interstitial

pneumonitis, and non-Hodgkin's lymphoma are diseases that may be linked with EBV in HIV-positive individuals.¹²

The prognosis is excellent with rest and supportive care. No other specific intervention alters or shortens the disease process. If given ampicillin, clients often develop a maculopapular rash. Since the virus can live indefinitely in B lymphocytes and the oropharynx, reactivation of the virus frequently occurs. The virus is commonly found in the saliva, although most often without symptoms.

SPECIAL IMPLICATIONS FOR THE THERAPIST 8-9

Infectious Mononucleosis

PREFERRED PRACTICE PATTERNS

6B: Impaired Aerobic Capacity/Endurance Associated with Deconditioning

Infectious mononucleosis is probably contagious before symptoms develop until the fever subsides and the oral and pharyngeal lesions disappear. Although infectious mononucleosis appears to be only mildly contagious, adherence to standard precautions, especially good handwashing and avoidance of shared dishware or food items with other people, is essential in preventing the HCW from contracting this condition.

The person with infectious mononucleosis should be cautioned against engaging in excessive activity, especially contact sports, which could result in splenic rupture or lowered resistance to infection. Usually this guideline is appropriate for a period of at least 1 month.

Any sign of splenic rupture (e.g., abdominal or upper quadrant pain, Kehr's sign, sudden left shoulder pain, or shock) requires immediate medical evaluation. Any soft tissue mobilization or myofascial techniques necessary in the left upper quadrant, especially up and under the rib cage, must take into consideration the enlarged liver and/or spleen; indirect techniques away from the spleen are indicated.

In rare cases mononucleosis impairs the CNS. Any change in neurologic status must be evaluated and reported to the physician. Changes in respiration or signs and symptoms of airway obstruction may require emergency intervention.

Cytomegalovirus (Herpesvirus Type 5)

Overview and Incidence. CMV (herpesvirus type 5) is a commonly occurring DNA herpesvirus. It increases in frequency with age. One percent of newborns have it, and four out of five adults older than 35 years have CMV (usually contracted during childhood or early adulthood) and are seropositive. For the majority of people who are infected with the virus after birth, there are few symptoms or complications. However, for unborn babies or the immunocompromised (posttransplant or with HIV disease), the consequences can be severe or life threatening.

Etiologic and Risk Factors. CMV is transmitted by human contact with infected secretions, such as urine, breast milk, feces, blood, semen, and vaginal and cervical secretions. It may also be transmitted through the placenta. The virus can be acquired from transplanted organs and rarely via blood transfusions. As with other herpesviruses, CMV can remain dormant to evade detection and persists in multiple organs. There is frequent intermittent reactivation with asymptomatic shedding of virus.

Pathogenesis and Clinical Manifestations. CMV probably spreads through the body via lymphocytes or mononuclear cells to the lungs (CMV pneumonitis), liver (CMV hepatitis), GI tract (CMV gastroenteritis), eyes (CMV retinitis), and CNS, where it produces inflammatory reactions. Complications include diffuse interstitial pneumonitis, leading to respiratory distress syndrome, hepatitis, adrenalitis, intestinal ulcerations, and calcifications around ventricles in neonatal CNS infections.

In normal adolescents or adults, the infection is usually asymptomatic or presents as an infectious mononucleosis-like illness with a self-limiting course.¹⁶ Unlike infectious mononucleosis from EBV, CMV rarely causes pharyngitis or adenopathy. In about 1% to 3% of women who have their first or primary infection during pregnancy, 40% of the babies become infected.

The course of the illness for the fetus ranges from mild splenomegaly or hepatitis to disseminated disease. Approximately 10% to 15% of those infected are born with the complications of hearing loss, vision impairment, or varying degrees of mental retardation, and the infection is deadly for 20% to 30% of affected neonates. Even up to 10% of infected babies born without symptoms go on to demonstrate varying degrees of hearing, mental, or coordination problems during the first few years of life.

In immunosuppressed people, particularly transplant recipients and those with HIV, various syndromes develop with CMV infection. Primary CMV infection can be more serious, but reactivation of the virus is more common in this group.¹³²

Fever, splenomegaly, hepatitis, pneumonitis, esophagitis, gastritis, colitis, encephalitis, or retinitis may occur in individuals who are immunocompromised. The specific transplanted organ is particularly susceptible to disease (e.g., hepatitis in liver transplants). Transplant recipients are most at risk the first 100 days following transplantation (see Chapter 21). With improved treatment of HIV using highly active antiretroviral therapy, CMV retinitis has significantly decreased but remains an important cause of blindness in advanced HIV disease.¹⁶

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Diagnosis is made either by culture (blood, sputum [from bronchoalveolar lavage], urine, throat swabs, or tissue samples) or by serologic identification of virus antigens. Positive results from urine or saliva samples do not necessarily indicate an acute infection since the virus may be shed for months to years. Traditional viral culture requires time; however, expedited results can be obtained with a tissue culture method (shell viral assay).

ELISA is the most common serologic test available and is able to distinguish between previous or active infection or, in the case of newborns, to detect passive maternal antibodies. Paired samples must be drawn at least 2 weeks apart to demonstrate increasing titers of antibodies and an active infection. PCR of viral DNA from blood or tissue is rapid but still undergoing refining. In seropositive transplant recipients, studies are underway to determine if prophylaxis or preemptive treatment is most beneficial posttransplant.¹⁶¹

In immunocompromised clients, pharmacologic treatment with ganciclovir and valganciclovir has proven effective. Foscarnet and cidofovir can be used in cases of resistance to ganciclovir but have more significant side effects. The prognosis for people with transplanted organs or who are immunocompromised is poor, as they may have fatal disseminated infections with multiple organ involvement.¹⁶² Vaccines are currently in development.¹³⁷

SPECIAL IMPLICATIONS FOR THE THERAPIST 8-10

Cytomegalovirus

PRACTICE PATTERNS

6B: Impaired Aerobic Capacity/Endurance Associated with Deconditioning

Other practice patterns depend on organ systems involved and clinical presentation.

The two principal reservoirs of CMV in health care institutions are (1) infants and young children and (2) immunocompromised individuals. However, HCWs who provide care to these high-risk populations have a rate of primary CMV that is no higher than among personnel without such client contact.

CMV transmission appears to occur directly, either through close, intimate contact with contaminated secretions or through excretions, especially saliva or urine.¹⁶³ Transmission by the hands of HCWs or individuals with the virus has also been suggested. The incubation period for person-to-person transmission is not known, and although CMV can survive on environmental surfaces or objects for a very short time, no evidence of fomite transmission exists.¹⁶⁴

Pregnant women and immunosuppressed people should avoid exposure to confirmed or suspected CMV infection. Pregnant women or women of childbearing age need to be counseled regarding the risks and prevention of transmission of CMV, but no data show that HCWs can be protected from infection by transfer to areas with less contact with individuals who have been diagnosed with CMV. Work restrictions for HCWs who contract CMV are not necessary since the risk of transmission of CMV can be reduced by careful adherence to handwashing and standard precautions.¹⁶⁵

Clients with CMV infection should be encouraged to wash their hands thoroughly and frequently to prevent spreading it. It is especially important to impress this on young children. As difficult as it may be, the child should not be allowed to kiss others, and parents and others should also avoid kissing the affected child.

Herpesviruses Types 6, 7, and 8

HHV-6 is a B-cell lymphotropic virus that is the principal cause of exanthema subitum (roseola infantum, or sixth disease).¹⁶⁶ Primary HHV-6 is common in children, with 90% infected by the age of 2 years. Although classically described as 3 to 5 days of high fever followed by a macular rash on the neck and trunk (roseola), children more commonly develop a fever, runny nose, and fussiness.¹⁶⁷ Its occurrence in adults is more complicated and associated with immunocompromised states such as AIDS and lymphoma. It has been associated with graft rejection and bone marrow suppression in transplant recipients and has been associated with multiple sclerosis.

HHV-7 is a T-cell lymphotropic virus that has also been serologically associated with roseola.¹⁶⁸ HHV-8 is associated with Kaposi's sarcoma in AIDS and other immune-related diseases (e.g., body cavity lymphoma).^{163, 169} (See the section on Kaposi's Sarcoma in Chapter 10.)

Viral Respiratory Infections

Viral respiratory infections (influenza, respiratory syncytial [RSV] virus) are common problems in health care settings. Many viral pathogens can cause respiratory infections, but influenza and RSV are associated with significant morbidity and mortality rates.

Influenza

Each year in the United States influenza viruses cause serious illness and even death, especially in young children with chronic diseases; immunocompromised adults; and the frail elderly. Influenza is caused by influenza viruses A or B and occurs in epidemics each winter between December and March. The mode of transmission is from person to person by inhalation of aerosolized virus or direct contact. Nosocomial transmission of influenza has been reported in acute and long-term health care facilities and has occurred from clients to HCWs, from HCWs to clients, and among HCWs. The incubation period is usually 1 to 4 days (average of 2 days).

Since 1997 influenza avian infections have been recognized. In 2005, an avian influenza A virus made the news for its ability to cause severe symptoms with fatal outcomes in humans. The subtype is H5N1 and was originally identified in Asia. Currently transmission is from infected birds to people in close contact with the birds, with rare person-to-person transmission.¹⁷⁰

Because of the severity of illness, with nearly 50% mortality, there is significant concern that the virus will undergo a mutation that will more readily allow person-to-person transmission.¹⁷¹ Zanamivir and oseltamivir may be beneficial in the treatment of this virus and studies are underway to develop and test an effective vaccine.

Influenza A and B resemble some other respiratory illnesses such as parainfluenza, RSV, and adenovirus. The onset is usually abrupt, with high fever, chills, malaise, muscular aching, headache, sore throat, nasal congestion, and nonproductive cough. The fever lasts about 1 to 7 days (usually 3 to 5). Children often manifest nausea, vomiting, and otitis media. The infection can progress

rapidly in the first few days, causing pneumonia and respiratory failure, particularly in high-risk groups. Secondary bacterial pneumonia may also develop, usually 5 to 10 days after the onset of viral symptoms, particularly in the older adult.

MEDICAL MANAGEMENT

PREVENTION. Vaccination is recommended before the beginning of each influenza season for people over age 50; people with chronic heart or lung disease, diabetes, renal dysfunction, or immunosuppression (including from HIV), pregnant women; nursing home residents; employees of medical or long-term care facilities; and HCWs (see Table 8-4).

In April 2000 the Advisory Committee on Immunization Practices (ACIP) lowered the age by 15 years from 65 and older to 50 and older. This policy changed because under the old guidelines, many people at risk for complications from influenza were being missed. About 25% of people between the ages of 50 and 64 have chronic medical conditions that place them at risk for influenza-related hospitalizations and possible death. The new plan recognizes that mass immunization programs based on age have been more successful than those targeting people with chronic diseases.

Vaccination against influenza is associated with reduced hospitalization rates and shorter hospital stays for pneumonia, diabetes, heart disease, and stroke in adults age 65 and older who are immunized. Mortality rates are also lower for all causes during influenza season in older adults who are immunized.^{108,109} A live attenuated influenza vaccine is also available. It is given intranasally to healthy persons between the ages of 5 and 49 years, who are not in contact with immunosuppressed individuals and do not have chronic medical problems.¹²⁵

DIAGNOSIS. The virus may be isolated from nasal washes, nasopharyngeal swabs or aspirates, or throat swabs. These can be sent for a rapid detection of antigen (ELISA or IFA) and culture. With the advent of improved anti-influenza medications requiring initiation within 48 hours, the antigen detection method is a mainstay in many laboratories. Culture requires 3 to 10 days but should be performed if the rapid detection test is negative. Like other antigen detection methods, culture provides information regarding the subtype of Influenza virus yet provides a definitive diagnosis.

Genetic mutations in the influenza virus create hundreds of variations within the two main types; being immune to one variant does not ensure immunity to another. Trivalent influenza virus vaccine provides partial immunity (about 85% efficacy) for a few months to 1 year. The CDC updates the vaccine annually to include the most current influenza A and B virus strains.

TREATMENT. Influenza antiviral agents can be given in conjunction with vaccine during institutional outbreaks of influenza. Amantadine and rimantadine are effective prophylaxis and treatment against influenza A, while oseltamivir can be effective prophylaxis and treatment of influenza A and B. Zanamivir is approved for treatment but not prophylaxis of influenza A and B.¹⁸¹

Antiviral agents used to treat influenza help decrease the duration and severity of signs and symptoms. Treatment must be initiated within the first 2 days of the illness and benefits those at high risk for complications. Resistance to these antivirals does occur and the Centers for Disease Control and Prevention monitors and recommends specific, effective treatment for each season.⁶⁸

Many people with influenza prefer to rest in bed; analgesics and a cough medicine mixture are often used. Droplet precautions (see Table 8-4) are imperative for all diagnosed and suspected cases of influenza. Antibacterial antibiotics are used only for treatment of bacterial complications.

PROGNOSIS. The duration of the uncomplicated illness is 3 to 7 days, and the prognosis is usually very good in previously healthy people; Reye's syndrome is a rare (almost eradicated) and severe complication of influenza and other viral diseases, especially in young children (to avoid Reye's syndrome, acetaminophen should be used for fever instead of aspirin in children).

Most fatalities related to influenza are due to bacterial and viral pneumonia.⁶⁶ The mortality rate is low except in debilitated individuals. People at greatest risk for influenza-related complications are (1) individuals older than 65 years, (2) residents of chronic health care facilities such as nursing homes, (3) people with chronic pulmonary or cardiovascular disease, and (4) people with diabetes mellitus.¹⁶⁹

Respiratory Syncytial Virus

RSV causes annual outbreaks of pneumonia, bronchitis, and tracheobronchitis in infants and very young children and is the main cause of hospitalization for a respiratory illness in this group.⁶⁴

In adults and older children, reinfection is common and manifests itself as mild upper respiratory tract infection and tracheobronchitis. Serious pulmonary RSV infections have been described in older adults⁴⁷ and immunocompromised individuals, and there is a high mortality rate in bone marrow and solid organ transplant recipients.^{53,90} In addition, infants with congenital heart disease, intensive care unit clients, those with cystic fibrosis, and older adults are at high risk for serious and complicated RSV.

Clients with HIV tend to have a less-severe course of the illness than transplant clients and although hospitalization for RSV is high, it rarely causes death. Annual epidemics occur in winter and spring. The incubation period is between 3 and 8 days. Inoculation occurs through the eyes or nose but rarely the mouth.

Nosocomial transmission of RSV occurs among clients, visitors, and HCWs. RSV is present in large numbers in the respiratory secretions of children with symptomatic RSV infections. It can be transmitted through large droplets (although not aerosolized) during close contact with such individuals or indirectly by hands or fomites that are contaminated with RSV. Hands can become contaminated through touching or handling of fomites or respiratory secretions and can transmit RSV by touching the nose or eyes.

Usually people shed the virus for 3 to 8 days, but young infants may shed the virus for as long as 3 to 4 weeks. Signs include low-grade fever, tachypnea, and wheezing. Hyperinflated lungs, decreased gas exchange, and increased work of breathing are also often present, and otitis media is a common complication.

Rapid diagnosis of RSV may be made by viral antigen identification of nasal washings using an ELISA or IFA. Culture of nasopharyngeal secretions is the standard for definitive diagnosis but requires 4 to 15 days. PCR for RNA of the virus is becoming more available but is not currently used in diagnosis except in research and large medical centers. These tests may have a decreased sensitivity in the older adult; therefore it is recommended that more than one be performed.

Treatment consists of hydration, humidification of inspired air, and ventilatory support as needed. Aerosolized ribavirin (an antiviral agent used in chronic HCV therapy) is FDA approved for the treatment of RSV in children, but close monitoring must be provided. Pregnant women should avoid ribavirin exposure since it is associated with fetal malformation or fetal death.

Palivizumab, a humanized monoclonal antibody (IgG), has been approved for prevention of serious lower respiratory tract illness in infants and young children who are at high risk of serious RSV, but it is expensive and must be administered intramuscularly. Growing amounts of data suggest a link with recurrent infections and the development of asthma later in life.

Avoidance of exposure to tobacco smoke, cold air, and air pollutants is also beneficial to long-term recovery from RSV bronchiolitis. A number of vaccines to prevent this infection are currently being studied, but because the immune response is neither durable nor complete it has been a difficult task.^{64,81}

SPECIAL IMPLICATIONS FOR THE THERAPIST 8-11

Viral Respiratory Infections

Influenza

HCWs must follow the guidelines in Tables 8-3 and 8-4 regarding prevention of transmission of influenza, both for themselves and their clients. Recommendations for immunization must be reviewed and acted on individually. Since the immunization for influenza does not provide immunity for the entire year or for all strains of influenza, common sense must prevail in the case of a HCW who suspects he or she has early signs and symptoms of influenza.

An early diagnosis can result in the use of antivirals to minimize intensity and duration of symptoms, especially in those at high risk for complications. HCWs must be aware of their responsibility to avoid transmission of infectious diseases such as influenza and either use personal protective equipment or practice self-isolation by staying home. This is especially important for the therapist in a setting with aged, immunocompromised, or chronically ill individuals.

Influenza can cause substantial morbidity and mortality among persons aged 65 years and older and among adults aged 50 years and older who have

chronic illnesses. Anyone in these two groups is vulnerable to the serious complications of influenza. Routine influenza vaccination has been associated with reductions in influenza-associated and all-case mortality during influenza season.¹⁰⁸ Despite the benefits from vaccination, utilization remains below target rates. Physical therapists can be instrumental in reducing morbidity and mortality rates by encouraging clients in these two groups to get a flu shot each year and to get one themselves.

Respiratory Syncytial Virus

Nosocomial RSV infections disseminate quickly, requiring rapid diagnosis, droplet and contact precautions (see Table 8-3), handwashing, and sometimes passive immunization to achieve prevention in hospitals. Because viral respiratory infections are so common during the winter and spring months, it is difficult for health care facilities to restrict workers with the virus from all client care duties. However, restricting HCWs with acute viral respiratory infections from care of high-risk clients may be necessary during community outbreaks of RSV and influenza.

MISCELLANEOUS INFECTIOUS DISEASES

Prostheses and Implant Infections

Any device implanted into the body of any synthetic material (e.g., titanium, cobalt, silicone)¹⁴² can give rise to serious life-threatening infections. Bioprostheses, implanted in large numbers in the 1970s and early 1980s, have now gone into the second decade of life since implantation, a time when biodegradation becomes more common. Multiple reoperations carry a higher risk of infection.

Likewise, as the population ages, an increasing number of primary and revision arthroplasties are being done. Early detection of infection or other problems can reduce complications and morbidity associated with these devices. Anyone with implants of any kind with onset of increasing musculoskeletal symptoms (especially in the area of the surgery) must be screened for the possibility of infection.

Normal radiographs and negative needle aspirates can delay medical diagnosis of infection. Knowing the risk factors for developing an antibiotic-resistant infection (e.g., multiple surgical procedures, previous *S. aureus* infection, multiple antibiotics) and recognizing red flag symptoms of infection can help the therapist recognize the need for persistence in obtaining follow-up medical care.

See Chapter 25 for complete discussion of this topic.

Lyme Disease

Definition and Overview

Lyme disease is an infectious multisystemic disorder caused by the tick-borne spirochete *Borrelia burgdorferi*. It

was first recognized in 1976 when a group of children in Lyme, CT, developed an unusual type of arthritis and a bull's-eye rash.¹⁵⁸ Some of these children also had a history of tick bites. Not until 1983 was the organism recovered from affected individuals and tick vectors established the relationship between the spirochete and the infection.^{19,157}

In the United States, the disease is only transmitted to human beings by certain ticks of the *Ixodes* species: *Ixodes scapularis* (formerly called *I. dammini*), known as the deer or black-legged tick in the Northeast (from Massachusetts to Maryland) and North Central United States (Wisconsin and Minnesota), and *I. pacificus*, the Western black-legged tick found on the western coast of northern California and Oregon. The ticks are extremely small, measuring approximately 1 to 2 mm. Several more genospecies of *Borrelia* are known to cause the disease in Europe, Asia, and Australia.⁷⁶

Incidence

Lyme disease has become the most prevalent vectorborne infectious disease in the United States.^{71,76} In 1982, when the CDC began national surveillance, only 491 cases were reported,¹¹³ while in 2002 a total of 23,763 cases were brought to medical attention.¹⁰⁴ This increased frequency is most likely multifactorial, a result of both a heightened awareness of the illness in endemic areas and an increase in the number of infected vectors.^{104,156}

Most cases (more than 90%) have been reported from the mid-Atlantic, Northeastern, and North Central regions of the country. It has been reported in 49 states and the District of Columbia. In the United States, Lyme disease is often seen in the late spring and summer months when the tick nymphs are most active and human outdoor activities are greatest.

Pathogenesis

I. scapularis exists in larval, nymphal, and adult stages. Larvae contract *B. burgdorferi* by feeding on infected rodents. The bacteria are then passed to nymphs and then to adults. The host of the adult is the white-tailed deer, which is required for survival of the ticks.

Human beings generally acquire the infection from nymphs when they attach to the skin to feed. The tick becomes engorged with blood and turns a grayish color. After approximately 36 hours, the bacteria from an infected tick are passed into the host when a tick injects spirochete-laden saliva into the host.¹²⁰ Most commonly, however, the tick falls off or is removed before the bacteria are injected into the host's bloodstream.

After incubating for 3 to 32 days, the spirochetes cause an inflammatory response, resulting in characteristic skin lesions at the site of the tick bite (see Clinical Manifestations below). The bacteria then disseminate to other organs via the bloodstream or lymphatic system.¹⁵²

The human host activates an immune response, producing cytokines and antibodies against the bacteria. Despite the host's response and if untreated, *B. burgdorferi* can survive for years in certain areas of the body by genetically adapting and inhibiting host immune responses.¹⁵⁴

Clinical Manifestations

Like syphilis, Lyme disease can be described as an imitator since its signs and symptoms mimic those of many other diseases. Symptoms vary widely and may not develop for as long as 1 month after a bite; in some cases, symptoms do not develop at all. Clinical manifestations of the infection occur in three stages.

Stage 1, the early, localized stage, usually occurs within days following a tick bite. About 80% of affected individuals will have a red, slowly expanding rash called *erythema migrans* (Fig. 8-8).¹⁵³ Not all people with the disease develop the telltale rash, and because early symptoms are often mild, some people may remain undiagnosed and untreated. Erythema migrans resolves spontaneously without treatment within an average of 4 weeks. Flu-like symptoms suggestive of early dissemination such as fatigue, chills, fever, headache, lethargy, myalgias, or arthralgias may also develop early in the course of the infection and may be the presenting symptoms for anyone without a rash.

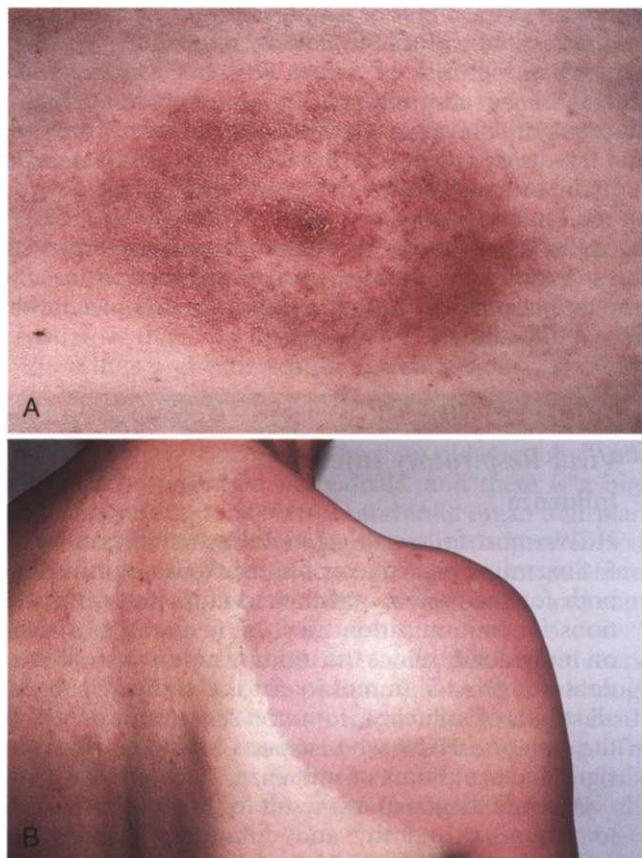


Figure 8-8

Examples of erythema migrans associated with Lyme disease. **A**, Many sources describe a characteristic bulls-eye rash with Lyme disease. **B**, However, there is a wide range of skin reactions labeled as erythema migrans possible with Lyme disease, as shown. Some skin rashes may be so minor as to be ignored or go unnoticed by the affected individual. (A, From Swartz MH: *Textbook of physical diagnosis: history and examination*, ed 4, Philadelphia, 2002, W.B. Saunders. B, Reprinted from Mandel GL: *Principles and practice of infectious diseases*, ed 6, Edinburgh, 2005, Churchill Livingstone.)

Box 8-10**NEUROLOGIC MANIFESTATIONS OF LYME DISEASE**

- Facial nerve palsy (Bell's palsy)
- Cognitive impairment (e.g., forgetfulness, decreased concentration, personality changes)
- Inflammation of the brain, spinal cord, or nerves
 - Cranial neuritis
 - Encephalitis
 - Encephalomyelitis
 - Encephalopathy
 - Meningitis
 - Radiculoneuropathy

Stage 2, disseminated infection, occurs within days to weeks after the spirochete spreads, particularly to the nervous system, heart, and joints. Neurologic symptoms may be the first to arise and occur in 15% of all cases,¹⁵³ most commonly manifested as aseptic meningitis with mild headache, stiff neck, and difficulty with mentation; cranial neuropathies, particularly Bell's palsy; and radiculopathies (Box 8-10). Even in anyone who remains untreated, neurologic symptoms may improve or resolve.¹⁵⁴

About half of those diagnosed may go on to develop painful Lyme arthritis characterized by unilateral inflammation and swelling in the large joints, especially the knees.¹⁵⁵ Migratory musculoskeletal pain in joints, bursae, tendons, muscle, and bone may occur in one or a few locations at a time, often lasting only hours or days in a given location. Weeks to months later, after the development of a marked cellular and humoral response to the spirochete, untreated people often have intermittent or chronic monoarticular (one joint) or oligoarticular (affecting only a few joints) arthritis.

Some people (4% to 10%) experience cardiac signs and symptoms, including myocarditis and various types of heart block and dysrhythmias, which can result in irregular, rapid, or slowed pulses; dizziness; fainting; and shortness of breath. Involvement of the eye is rare but can cause serious damage to the eye.

Stage 3, late persistent infection, may become apparent weeks to months after the initial infection. In the United States about 60% of individuals left untreated develop stage 3 symptoms characterized by intermittent arthritis associated with marked pain and swelling, especially in the large joints (Fig. 8-9). Rarely, affected individuals may go on to develop erosions or permanent joint abnormalities.¹⁵⁶ In approximately 5% of untreated individuals, chronic neurological symptoms occur, including spinal radicular pain, distal paresthesias, and a mild encephalopathy with subtle cognitive disturbances.¹⁵⁴

Postinfection syndromes. Several syndromes have been reported describing persistent symptoms despite antibiotic treatment. One such syndrome, called the post-Lyme syndrome or chronic Lyme disease, resembles fibromyalgia or chronic fatigue syndrome. Affected individuals describe disabling fatigue, severe headache, diffuse muscle or joint pain, cognitive difficulties, and sleep abnormalities.¹⁵⁵ Symptoms may begin with the infection or emerge

**Figure 8-9**

Swollen knee of a youth with Lyme arthritis. (Reprinted from National Institutes of Health: *Lyme disease: the facts, the challenge*, NIH publication no. 92-3193, Bethesda, MD, 1992, U.S. Department of Health and Human Services, p 12.)

soon after treatment and persist for months to years. Debate continues as to whether these patients ever had an active infection with *B. burgdorferi*.^{145,156} Of those with documented, treated disease it is hypothesized that the bacteria may trigger a neurohormonal or immunologic process that causes symptoms despite eradication of the spirochete.¹⁵⁶

About 10% of the people who have Lyme arthritis will continue to have joint symptoms for months to years following treatment. Although evidence of the spirochete exists in the synovial fluid prior to treatment, posttreatment joint fluid is often negative for infection. This again may be immune rather than infection related.¹⁵⁶

MEDICAL MANAGEMENT

PREVENTION. Prevention is the key to avoiding Lyme disease.^{65,172} Lyme disease is most common during the late spring and summer months in the United States when nymphal ticks are most active and human populations are frequently outdoors and most exposed. People who live or work in residential areas surrounded by woods or overgrown brush infested by ticks or favored by white-tailed deer and live in the endemic geographic areas are at risk. In addition, people who participate in outdoor recreational activities in tick habitat are also at risk for Lyme disease.

In December 1998 the FDA licensed the vaccine LYMERix to aid in the prevention of Lyme disease. However, in February 2002 the vaccine was withdrawn from the market reportedly for poor sales. Drawbacks surrounding the vaccine included cost and multiple, repeated doses for efficacy. Prophylactic treatment is

Box 8-11**PREVENTION OF LYME DISEASE**

Precautions for people living in tick-infested areas:

- Avoid tick-infested areas, especially in May, June, and July (check with local health departments or park services for the seasonal and geographic distribution in your area).
- Walk along cleared or paved surfaces rather than through tall grass or wooded areas.
- Wear long-sleeved shirts, long pants tucked into socks, and closed shoes (no part of foot exposed).
- Wear light-colored clothing to make it easy to detect ticks.
- Always check for ticks after being outdoors. If ticks are removed within 36 hours of attachment, the risk of infection decreases significantly.
- Shower as soon as possible after being outdoors. Ticks take several hours to attach themselves to the skin and can be washed away first.
- Wash clothing worn outdoors immediately and use a dryer (heat kills the ticks). If no access to laundry facilities is available, the clothing should not be stored in the bedroom; if camping, the clothing should not be stored in the same area where people are sleeping.
- If bitten by a tick, remove the tick immediately by grasping it as close to the skin as possible with tweezers and tugging gently. Do not twist or turn the tweezers; pull straight away from the skin. Do not use petroleum jelly, fingernail polish, or a hot match to remove ticks.
- To lessen the chance of contact with the bacterium, do not crush the tick's body or handle the tick with bare hands. Clean the bite area thoroughly with soap and water, then swab the area with an antiseptic to prevent bacterial infection.
- Whenever possible, save the tick in a glass jar for identification should symptoms develop.
- If living in an area in which deer ticks are common, keep the weeds and grass around the house mowed. Consider using wood chips where lawns meet forested areas. Ticks are less able to survive in a dry environment.
- Use flea and tick collars on pets; brush and examine them carefully after they have been outdoors. People can use insecticides such as permethrin or insect repellents containing diethyltoluamide (DEET).*

*The use of such chemicals may be objectionable to some people because they may cause neurotoxicity in children. Alternative methods are available.

controversial, but a single dose of doxycycline for ticks attached to the skin between 36 and 72 hours may be helpful.¹⁰⁵ Box 8-11 provides specific strategies available for Lyme disease prevention.

DIAGNOSIS. The CDC suggests a two-step process in diagnosing Lyme disease. The first step is to test the blood for antibodies to the spirochete. This is done with a sensitive enzyme immunoassay (EIA) or IFA. If this test is positive or equivocal, a Western immunoblot should be performed to confirm the diagnosis.²² Since antibodies are not present for 1 to 2 weeks following infection, these tests may not be positive early in the infection.

If erythema migrans is present, treatment can begin without serologic tests for Lyme disease. For those who present in the summer months with symptoms of Lyme disease without erythema migrans, empiric treatment

with doxycycline can be considered. Since EIA may become negative with treatment, acute and convalescent blood samples can be collected for antibody titers to confirm infection. These tests are very sensitive in diagnosing infection in later stages.

The PCR uses gene segment amplification techniques to detect the DNA of the bacterium itself. PCR has detected bacterium DNA in synovial fluid, CSF, skin, blood, and urine. Synovial fluid may be tested to distinguish Lyme arthritis from acute septic arthritis. PCR is not routinely used in the diagnosis of Lyme disease and often is only available at specialized centers or for research purposes.

TREATMENT. Early Lyme disease is treated with oral antibiotics, typically doxycycline or amoxicillin for pregnant women or children. Antibiotics are given for 14 to 21 days. If third-degree heart block or neurologic symptoms are present, other than isolated facial palsy, IV antibiotics are required for 14 to 28 days, typically with ceftriaxone. Lyme arthritis can be treated with oral or IV antibiotics, although oral therapy is easier and equally effective.

For anyone with continued arthritis despite recommended treatment, another 4-week oral or 2-week IV treatment may be given. If symptoms persist despite the second treatment, data do not support the continued use of antibiotics. Nonsteroidal medications, disease-modifying antirheumatic drugs, and arthroscopic synovectomy are used for arthritis pain. Guidelines for fibromyalgia or chronic fatigue syndrome are followed in anyone with post-Lyme syndrome who has negative PCR results.¹⁵⁶

PROGNOSIS. Early treatment is vital for the prevention of long-term complications, but even so, complications involving the heart, joints, and nervous system occur in about 15% of people who do undergo early treatment. For most people, Lyme disease is curable with standard antibiotic therapy, and the effects of Lyme disease resolve completely within a few weeks or months of treatment. Unfortunately, no natural immunity develops from exposure to Lyme disease, and anyone can be reinfected. Although Lyme disease is rarely fatal, heart complications may cause life-threatening cardiac arrhythmias.

Great concern came about in the past regarding potential fetal infection and teratogenicity from Lyme disease contracted during pregnancy. However, in recent prospective studies no cases of congenital infection have been documented.^{93,150,183}

SPECIAL IMPLICATIONS FOR THE THERAPIST

8-12

Lyme Disease**PREFERRED PRACTICE PATTERNS****Stage 1**

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

Stages 2 and 3

4D: Musculoskeletal Impairment Associated with Connective Tissue Disorder (rheumatoid arthritis)

5C, 5D: Impaired Neuromuscular Function with Acquired Nonprogressive Disorder of the CNS

6D: Impaired Aerobic Capacity/Endurance Associated with Cardiovascular Pump Dysfunction (cardiac arrhythmias)

Chronic arthritis is the most widely recognized result of untreated Lyme disease in the United States. Unlike other forms of rheumatoid arthritis, Lyme arthritis does not affect the joints bilaterally, though both sides may be affected alternately.

The condition has been called chronic because episodes can last months, occurring intermittently over a period of 1 to 3 years. Permanent joint damage and cartilage destruction can occur if excessive use occurs during the inflammatory period. Range-of-motion and strengthening exercises are important but must be carried out carefully and without overexertion.

Nervous system abnormalities can develop weeks, months, or even years following an untreated infection. These symptoms often last for weeks or months and may recur. The therapist may treat such a client at any time during the course of symptomatic presentation.

For anyone with known Lyme disease, frequent assessment of the person's neurologic function and level of consciousness is important. Any signs of cardiac abnormality or increased intracranial pressure and cranial nerve involvement (e.g., ptosis, strabismus, diplopia) must be reported to the physician immediately. Both upper- and lower-extremity peripheral nerve problems can occur and are managed as any neuropathy from other causes.

It has been hypothesized that people who present with symptoms of multiple sclerosis but respond to antibiotics may have been bitten by ticks years ago. Along the same lines, the question has been raised whether Lyme disease triggers fibromyalgia since symptoms consistent with fibromyalgia and chronic fatigue syndrome develop in individuals with clear-cut Lyme disease, even after adequate treatment. To date no biologic relationship has been proven between these conditions and Lyme disease.¹⁰²

time low in the United States, chlamydia and human papillomavirus (HPV) remain significant health problems. Chlamydia is the most common reportable STD in the United States, with almost 1 million cases reported to the CDC in 2004.¹⁰⁶

HPV, which causes genital warts and can lead to cervical cancer, most likely infects over 6 million men and women per year. Additionally, more than 45 million people have chronic genital herpes, with perhaps 1 million new cases diagnosed every year. There were 73,000 new cases of HBV reported in 2003, with 1.25 million people chronically infected despite the availability of a preventative vaccine. In 2004, an estimated 1 to 1.1 million Americans were living with the HIV/AIDS virus, with an additional 42,000 cases newly reported.

STDs and STIs are spread primarily through sexual contact, but some cases may also be spread by sharing infected needles or by transmission from mother to child during vaginal childbirth. Many STDs and STIs are easily treated and cured, but others remain chronic. More than 50 different STDs and STIs have been described; only the most common ones are included here (Table 8-7).

Etiologic and Risk Factors

All groups of people are potentially at risk for STDs and STIs, but women, teens, men who have sex with men, and minorities have been disproportionately affected. Young people under the age of 18 years are considered at greatest risk for getting a sexually transmitted disease but, in fact, the over-50 population is also at risk.

Although 25% of all STDs and STIs occur in people younger than 25 years, numerous surveys of healthy adults have verified that older people are sexually active and less likely to practice safe sex. Risk factors vary but most often include multiple sex partners, a partner with a known risk factor, a history of a blood transfusion between 1977 and 1984, failure to use a condom (or use it properly) during sexual intercourse, and sharing needles during illicit drug use. The presence of STDs and STIs is a risk factor itself for facilitating the transmission of HIV. In fact, persons with an STD are two to five times more likely than a person without an STD of sexually acquiring HIV.¹⁷⁴

Pathogenesis and Clinical Manifestations

STDs and STIs are caused by bacteria, viruses and, occasionally, parasites and may have a considerable latency period when the infectious organism lies dormant before triggering symptomatic presentation (Fig. 8-10).

Clinical manifestations vary according to the STD present (Figs. 8-11 and 8-12; see also Table 8-7). STDs and STIs may be completely asymptomatic and therefore are less likely to be diagnosed until serious problems develop. Complications of STDs and STIs are more severe and more frequent among women than men. Once infected, women are more susceptible to reproductive cancers, infertility, and contracting other STDs and STIs.

MEDICAL MANAGEMENT

PREVENTION. Prevention is the most important key to managing STDs and STIs. The only prevention that is 100% effective is abstinence and/or a mutually monogamous

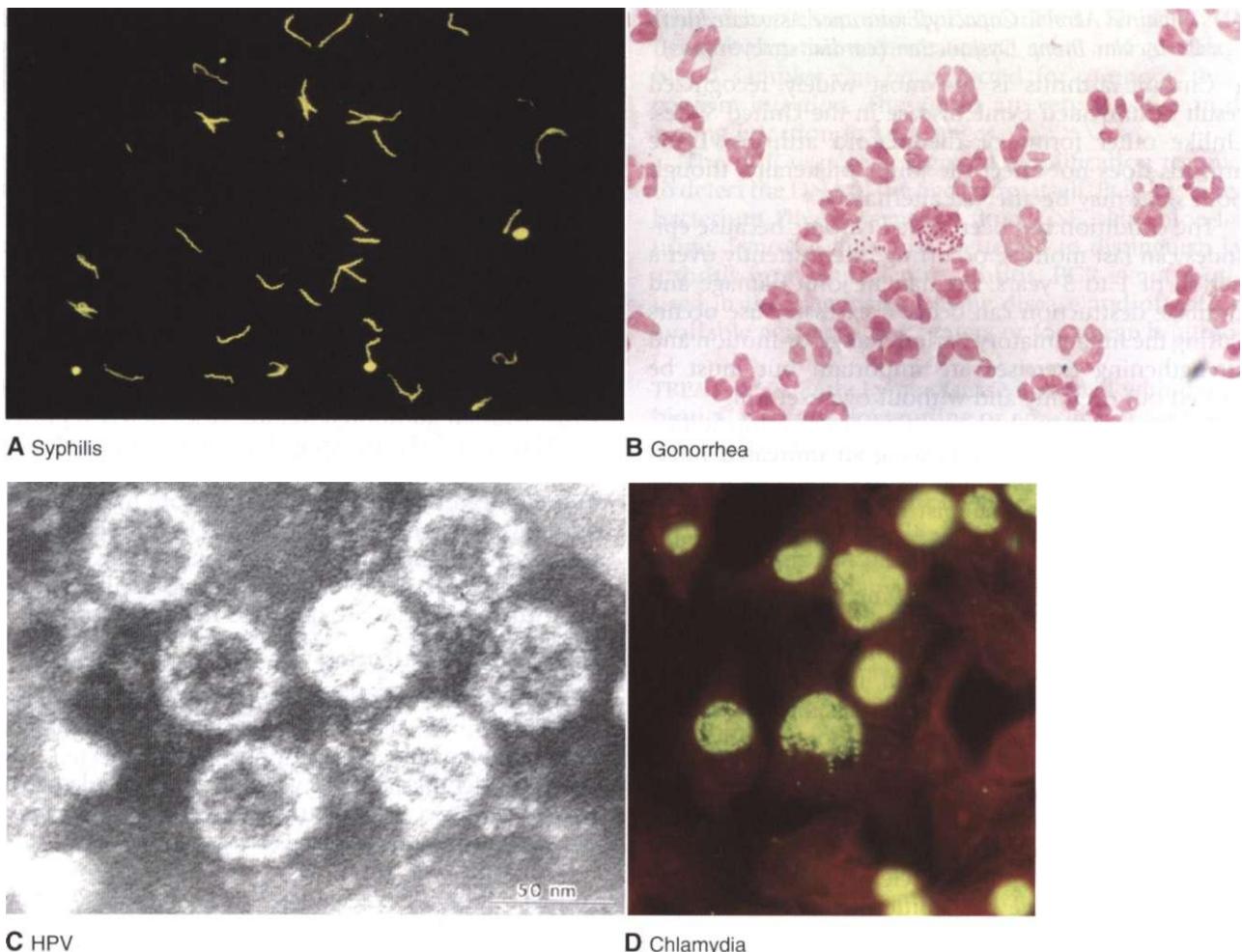
Sexually Transmitted Infections

Overview and Incidence

Each year 19 million Americans contract a sexually transmitted disease (STD) or infection (STI), and it is estimated that at least one out of every four sexually active people (56 million Americans) is carrying an infection other than HIV. It is likely that the incidence of STDs and STIs is underreported for several reasons.

Physicians fail to report STD cases to local health departments despite being mandated to do so. Physicians also rely on patients to notify their sexual partners, who may or may not be tested and/or treated.¹¹ A lack of free screening or reimbursement for screening may be a contributing factor.⁸²

Syphilis, once thought to be trending toward elimination, has steadily increased since 2000, particularly in black men and men who have sex with men.^{28,104,129} Although the incidence of gonorrhea has reached an all-

**Figure 8-10**

Sexually transmitted infections. **A**, *Syphilis* mimics so many diseases it is called “the great imitator.” Dark-field microscopy showing several spirochetes in scrapings from the base of a syphilitic chancre. **B**, *Gonorrhea*, called the “preventer of life,” can cause sterility. Gram-stained smear of urethral discharge showing intracellular gram-negative diplococci characteristic of gonorrhea. **C**, *HPV*, also known as genital herpes, is the most common cause of cervical and other reproductive cancers. **D**, *Chlamydia* is the most common STD reported in the United States. (A, Reprinted from Kumar V: *Robbins and Cotran: pathologic basis of disease*, ed 7, Philadelphia, 2005, W.B. Saunders, courtesy Paul Southern, Department of Pathology, University of Texas Southwestern Medical School, Dallas. B, Reprinted from Mandell GL: *Principles and practice of infectious diseases*, ed 6, Philadelphia, 2005, Churchill Livingstone. C, Reprinted from Kumar V: *Robbins and Cotran: pathologic basis of disease*, ed 7, Philadelphia, 2005, W.B. Saunders, courtesy Ian Frazer, Princess Alexandra Hospital, University of Queensland, Australia. D, Reprinted from Mandell GL: *Principles and practice of infectious diseases*, ed 6, Philadelphia, 2005, Churchill Livingstone, courtesy Robert Suchland, Seattle, WA.)

sexual relationship (single partner) between two uninfected people. For those who are sexually active, condoms, properly used, are able to reduce the transmission of sexually transmitted diseases spread by mucosal fluid (e.g., gonorrhea, chlamydia, and HIV).

However, condoms do not cover all surfaces and only protect the skin they cover, and are therefore less likely to protect against diseases acquired from skin-to-skin contact such as syphilis, HPV, and HSV.¹⁸⁴ Genital warts are contagious; avoid touching them. Spermicides with nonoxynol-9 may not be effective against gonorrhea, chlamydia, or HIV infection; the CDC does not recommend the use of nonoxynol-9 with or without condoms for STD or HIV protection.¹⁸⁴

Pregnant women should have blood tests for syphilis and HBV. Early syphilis, if untreated in pregnant women, can cause fetal death in 40% of cases. Even if syphilis is acquired up to 4 years before pregnancy, infection of the fetus will occur in 70% of cases.²⁶ A vaccine is available to protect anyone, especially women of childbearing age, against HBV (see Table 8-4).

Pregnant women should also be tested for gonorrhea, chlamydia, HPV, and HIV. Those with recurrent genital herpes and open sores benefit from cesarean section delivery to protect the child. Anyone with a STD or known possibility of infection must inform a physician and all partners to obtain appropriate treatment and prevent spreading the disease to others.

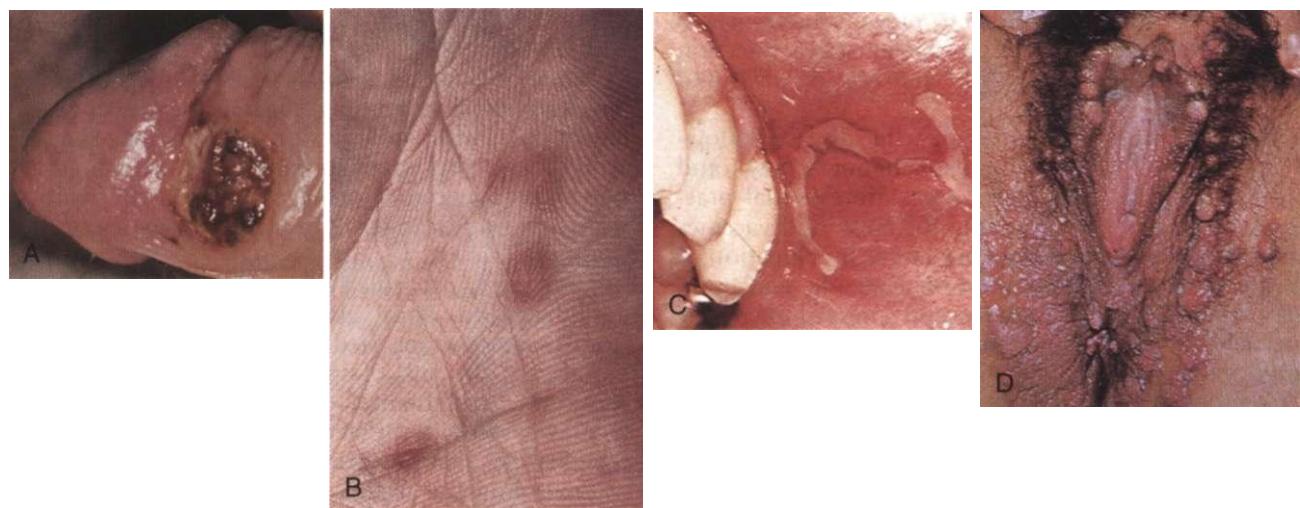


Figure 8-11

Clinical manifestations of syphilis. Many STIs present with lesions of the skin and/or genitals. Each one presents differently based on the stage of the disease. **A**, Chancre in primary syphilis on the penis. **B**, Palmar lesions of a coppery color in secondary syphilis. **C**, Mucous patch of the mouth in secondary syphilis. **D**, Genital lesions called condylomata lata in a female (secondary syphilis). (A, C, and D, Reprinted from Forbes CD, Jackson WF: *Color atlas and text of clinical medicine*, London, 2003, Mosby. B, Reprinted from Habif TP: *Skin disease: diagnosis and treatment*, St Louis, 2001, Mosby.)

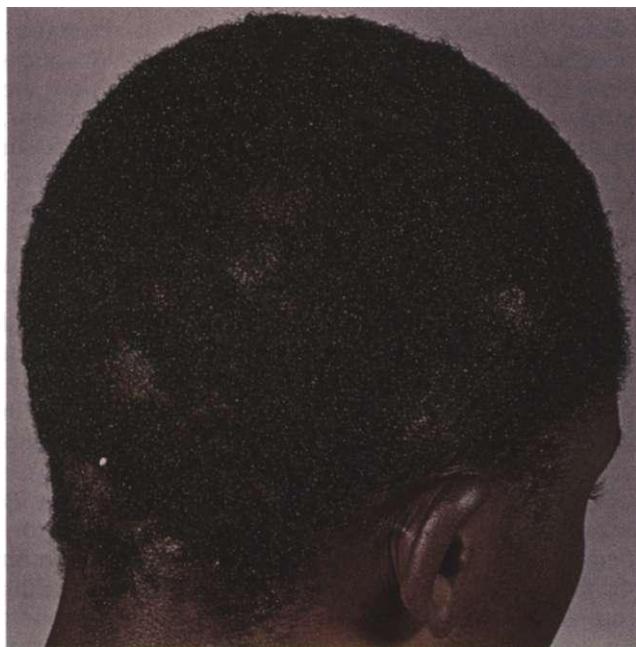


Figure 8-12

Alopecia of the scalp (balding) associated with secondary syphilis. This is a temporary, irregular presentation of alopecia sometimes referred to as "moth-eaten" alopecia. (Reprinted from Habif TP: *Clinical dermatology*, ed 4, St Louis, Mosby, courtesy Subhash K. Hira.)

Drug users, especially injection-drug users, can prevent transmission of disease best by discontinuance of drugs. However, in most cases this is not immediately realistic. Programs have been set up to help reduce needle sharing by providing needle exchange centers and street education programs aimed at teaching more sterile practices.

SCREENING, DIAGNOSIS, TREATMENT, AND PROGNOSIS.

STDs and STIs can often be identified by the clinical manifestations, but various screening tests are also available. Experts recommend that sexually active adolescents and women aged 25 years and younger, pregnant women aged 25 years and younger, and older women at high risk be screened for chlamydia. The interval of screening should take into account changes in partners, but women at high risk with a history of previous infection may benefit from screening every 6 to 12 months.¹⁰⁷ With the advent of urine-based testing, more frequent screening has been successful in both women and men.

HPV infection is another prevalent STD that has been found to cause cervical cancer in women. Since HPV slowly creates cellular changes prior to the development of cancer, recommendations for screening have been published (see Table 20-2). Liquid-based cytology is a newer technique that allows the sampled cells to be concentrated and better preserved for visual evaluation. The liquid-based samples can also be used for HPV DNA testing.

Evidence supporting the specific use of liquid-based cytology and HPV DNA testing is not currently available because of their relative newness; however, recommendations by multiple medical societies have been issued as guidelines. Although HPV is most often acquired in younger clients, older women continue to be at risk, so testing should take into consideration risk factors.⁶² Various immunoassays, serologic techniques, and culture methods are used to diagnose HIV, syphilis, gonorrhea, herpes, hepatitis, and other STDs and STIs.

Antibiotics can cure some STDs and STIs (see Table 8-7), although some may be drug resistant (e.g., gonorrhea resistance to quinolones). Limiting the number of sexual partners, practicing abstinence, and safe sex (proper use

of condoms) are recommended to prevent the transmission of disease. Intercourse during an active infection dramatically increases the risk of transmitting STDs and STIs.

When working with clients with active disease, following contact precautions, frequent handwashing, and avoiding touching the affected areas are essential practices. A vaccine is now available for HBV, and vaccines against HPV, HIV, and herpes are under investigation. A new vaccine for HPV may soon be available, and questions are being raised as to the receptiveness of the public (especially adolescents and young adults) and how it may change sexual habits.¹⁶⁰

The prognosis varies with each STD, but with treatment symptoms can be minimized and complications prevented. Without treatment, serious complications can occur such as infertility, chronic pelvic pain, ectopic pregnancy and miscarriage, cardiovascular disease, CNS impairment, blindness, cervical cancer, and even death.

SPECIAL IMPLICATIONS FOR THE THERAPIST 8-13

Sexually Transmitted Diseases and Infections

PREFERRED PRACTICE PATTERNS

These are variable depending on specific disease entity and clinical presentation.

Any therapist treating men or women with clinical presentation of pelvic, buttock, hip, or groin pain of apparent unknown cause must be prepared to ask the client about past history of STDs and STIs, sexual activity, changes in sexual function, and presence of urogenital signs or symptoms (e.g., discharge from penis or vagina, painful urination, difficulty initiating or continuing a stream of urine). Any suspicion that the clinical manifestations may be correlated to an STD must be further evaluated by a physician.

Clostridial spores may occur when adulterants are cut with the heroin.

IV use of black-tar heroin causes sclerosis of the veins and leads to "skin popping," or injecting the drug subcutaneously. Continued injection into the same site creates a necrotic environment suitable for clostridial germination and toxin production. Necrotizing fasciitis with toxic shock syndrome can result from the spread of a clostridial infection.¹⁰³

Drug users, particularly IV drug users, vary the site of administration. Local abscess formation or infections from hematogenous seeding are seen in unusual places because of the site of injection (the femoral vein, or "groin hit," and the neck, or "pocket shot"). Osteomyelitis may develop in the sternoclavicular, sacroiliac, or vertebral spine. Septic arthritis is often seen in the knees.

Environmental factors frequently contribute to infections. Some users may lick the skin or needle prior to injecting, leading to polymicrobial infections. Others crush tablets between their teeth or blow clots out of needles before reusing. Sharing of needles and paraphernalia is also common.⁶¹ Because of these habits, drug users are more likely to develop certain types of bacterial infections with specific organisms.

The four types of infections most often seen are of the skin or soft tissue, endovascular infections, respiratory infections, and musculoskeletal infections.⁶¹ *S. aureus* and streptococcal species (flora from the user's skin) are the most common pathogens in drug-related infections. Drug users are more likely to be colonized with MRSA in the nares and skin than non-drug users; this most likely occurs because of tissue damage from inhaling or injecting drugs.

Abscess formation is common. More serious infections such as endocarditis, septic thrombophlebitis, mycotic aneurysms, and sepsis occur from hematogenous spread of organisms. In injection drug users *S. aureus* is the most common organism causing endocarditis. Although polymicrobial endocarditis is rare, it is most often seen in injection drug users. Complications of endocarditis include brain, lung, and splenic abscesses.

Drug users are more likely to develop a respiratory tract infection compared with nonusers, and respiratory infections, particularly pneumonia, are the most common infection in drug users. Damage to cells from inhaling drugs and chronic cigarette use (many drug users also smoke) may lead to inability to clear secretions. Aspiration may occur because of decreased mental alertness.

Clients with HIV may present with atypical features and radiographs, so a good history and physical examination are important. Pulmonary tuberculosis (and drug-resistant tuberculosis) is encountered more frequently in drug users who practice "shotgunning" (inhaling cocaine and blowing smoke into the mouth of another person), live in crowded spaces, delay diagnosis, or have HIV.

Musculoskeletal infections may occur in unusual places, as discussed above. Flora from the skin is the most common pathogen, although polymicrobial infections are seen, especially if saliva contaminates the skin, drugs, or needles. The infection may be subtle, with mild fever and pain.⁶¹

Infections in Drug Users

Drug use in the United States continues to be a significant health problem, with over 19.5 million people, or 8.2% of the population, found to be using drugs in 2003.¹⁶⁸ Serious illnesses such as HIV and hepatitis are transmitted with injection drug use. Drug users as a whole also have a higher incidence of bacterial infections because of the various drugs used, the route and sites of administration, and preparation of the drug. Each of these factors determines risk for infection and the likelihood of specific bacterial infections.⁶¹

Drugs themselves may be contaminated or become contaminated when "cut" (diluted) with adulterants (dextrose or dyed paper). Black-tar heroin, produced in Mexico, has been associated with outbreaks of wound botulism. Since clean needles and skin are not protective, the black-tar heroin is most likely the cause of the infection. Contamination with *Clostridium tetani* or other

SPECIAL IMPLICATIONS FOR THE THERAPIST 8-14***Infections in Drug Addicts***

See Special Implications for the Therapist: Substance Abuse in Chapter 3.

Being aware of the signs of substance abuse or drug addiction and the patterns of infection associated with drug addiction may assist the therapist in recognizing early signs of infection requiring medical evaluation and treatment. Any therapist involved in wound care management, needle electromyography, or other high-risk practice techniques who has not already been immunized against HBV should be vaccinated.

References

To enhance this text and add value for the reader, all references are included on the companion Evolve site that accompanies this textbook. The reader can view the reference source and access it on-line whenever possible. There are a total of 185 cited references and other general references for this chapter.

CHAPTER 9

Oncology

CATHERINE C. GOODMAN

Cancer is a term that refers to a large group of diseases characterized by uncontrolled cell proliferation and spread of abnormal cells. Other terms used interchangeably for cancer are malignant neoplasm, tumor, malignancy, and carcinoma. According to the American Cancer Society (ACS), about 5% of cancer is genetic, whereas 95% is related to other (often modifiable) factors. Only oncologic concepts are presented in this chapter; individual cancers are discussed in the chapters devoted to the affected system. Since cancer and cancer treatment can affect multiple systems, the reader is encouraged to read this chapter along with Chapter 5 for a more complete understanding of its potentially wide-ranging systemic effects.

DEFINITIONS

Differentiation

Normal tissue contains cells of uniform size, shape, maturity, and nuclear structure. Differentiation is the process by which normal cells undergo physical and structural changes as they develop to form different tissues of the body. Differentiated cells specialize in different physiologic functions.

In malignant cells, differentiation is altered and may be lost completely so that the malignant cell may not be recognizable in relationship to its parent cell. When a tumor has completely lost identity with the parent tissue, it is considered to be undifferentiated (anaplastic). In this case, it may become difficult or impossible to identify the malignant cell's tissue of origin. In general, the less differentiated a tumor becomes, the faster the metastasis (spread) and the worse the prognosis.

Dysplasia

A variety of other tissue changes can occur in the body. Some of these changes are benign, whereas others denote a malignant or premalignant state. Dysplasia is a general category that indicates a disorganization of cells in which an adult cell varies from its normal size, shape, or organization. This is often caused by chronic irritation such as is seen with changes in cervical (uterine) epithelium as a result of long-standing irritation of the cervix. Dysplasia may reverse itself or may progress to cancer.

Metaplasia

Metaplasia is the first level of dysplasia (early dysplasia). It is a reversible and benign but abnormal change in which one adult cell changes from one type to another. For example, the most common type of epithelial metaplasia is the change of columnar epithelium of the respiratory tract to squamous epithelium.

Another example of metaplasia is Barrett's esophagus (also called Barrett's epithelium) in which squamous epithelium of the esophagus is replaced by the glandular epithelium of the stomach. Although metaplasia usually gives rise to an orderly arrangement of cells, it may sometimes produce disorderly cellular patterns (i.e., cells varying in size, shape, and orientation to one another). Anaplasia (loss of cellular differentiation) is the most advanced form of metaplasia and is a characteristic of malignant cells only.

Hyperplasia

Hyperplasia refers to an increase in the number of cells in tissue, resulting in increased tissue mass. This type of change can be a normal consequence of physiologic alterations (physiologic hyperplasia) such as increased breast mass during pregnancy, wound healing, or bone callus formation. Neoplastic hyperplasia, however, is the increase in cell mass because of tumor formation and is an abnormal process. The presence of these types of hyperplastic breast tissue increases the risk of later development of breast cancer.¹⁴¹

Tumors

Tumors, or neoplasms, are defined as abnormal growths of new tissue that serve no useful purpose and may harm the host organism by competing for vital blood supply and nutrients. These new growths may be benign or malignant (see the next discussion) and primary or secondary.

A primary tumor arises from cells that are normally local to the given structure, whereas a secondary tumor arises from cells that have metastasized from another part of the body. For example, a primary neoplasm of bone arises from within the bone structure itself, whereas a

Table 9-1 Classification of Neoplasms by Cell Type of Origin

Tissue of Origin	Benign	Malignant
Epithelial Tissue		
Surface epithelium (skin) and mucous membrane	Papilloma	Squamous cell, basal cell, and transitional cell carcinoma
Epithelial lining of glands or ducts	Adenoma	Adenocarcinoma
Pigmented cells (melanocytes of basal layer)	Nevus (mole)	Malignant melanoma
Connective Tissue and Muscle		
Fibrous tissue	Fibroma	Fibrosarcoma
Adipose	Lipoma	Liposarcoma
Cartilage	Chondroma	Chondrosarcoma
Bone	Osteoma	Osteosarcoma
Blood vessels	Hemangioma	Hemangiosarcoma
Smooth muscle	Leiomyoma	Leiomyosarcoma
Striated muscle	Rhabdomyoma	Rhabdomyosarcoma
Nerve Tissue		
Nerve cells	Neuroma	—
Glia	—	Glioma or neuroglioma
Ganglion cells	Ganglioneuroma	Neuroblastoma
Nerve sheaths	Neurilemoma	Neurilemma sarcoma
Meninges	Meningioma	Meningeal sarcoma
Retina	—	Retinoblastoma
Lymphoid Tissue		
Lymph nodes	—	Lymphoma
Spleen	—	—
Intestinal lining	—	—
Hematopoietic Tissue		
Bone marrow	—	Leukemias, myelodysplasia, and myeloproliferative syndromes
Plasma cells	—	Multiple myeloma

secondary neoplasm occurs in bone as a result of metastasized cancer cells from another (primary) site.

Carcinoma *in situ* refers to preinvasive, premalignant epithelial tumors of glandular or squamous cell origin. These tumors have not broken through basement membranes of the squamous cells and occur in the cervix, skin, oral cavity, esophagus, bronchus, and breast. Carcinoma *in situ* that affects glandular epithelium occurs most commonly in the cervix, breast, stomach, endometrium, large bowel, and prostate gland (prostate intraepithelial neoplasia). How long the characteristic cell disorganization and atypical changes last before becoming invasive is variable for different cancers.

CLASSIFICATIONS OF NEOPLASM

A neoplasm can be classified on the basis of cell type, tissue of origin, degree of differentiation, anatomic site, or whether it is benign or malignant. A benign growth is usually considered harmless and does not spread to or invade other tissue. Certain benign growths, recognized clinically as tumors, are not truly neoplastic but rather represent overgrowth of normal tissue elements (e.g., vocal cord polyps, skin tags, hyperplastic polyps of the colon). However, benign growths can become large enough to distend, compress, or obstruct normal tissues

and to impair normal body functions, as in the case of benign central nervous system (CNS) tumors. These tumors can cause disability and even death.

When tumors (benign or malignant) are classified by cell type, they are named according to the tissue from which they arise (Table 9-1). The five major classifications of normal body tissue are epithelial, connective and muscle, nerve, lymphoid, and hematopoietic tissue. Not all tissue types fit into one of these five categories, thus requiring a miscellaneous category for other tissues (not included in Table 9-1) such as the tissues of the reproductive glands, placenta, and thymus.

Epithelium covers all external body surfaces and lines all internal spaces and cavities. The skin, mucous membranes, gastrointestinal tract, and lining of the bladder are examples of epithelial tissue. The functions of epithelial tissues are to protect, excrete, and absorb. Cancer originating in any of these epithelial tissues is called a *carcinoma*. Tumors derived from glandular tissues are called *adenocarcinomas*.

Connective tissue consists of elastic, fibrous, and collagenous tissues such as bone, cartilage, and fat. Cancers originating in connective tissue and muscle (mesenchymal origin) are called *sarcomas*. Nerve tissue includes the brain, spinal cord, and nerves and consists of neurons, nerve fibers, dendrites, and a supporting tissue composed of glial cells.

Tumors arising in *nerve tissue* are named for the type of cell involved. For example, tumors arising from astrocytes, a type of glial cell thought to form the blood-brain barrier, are called *astrocytomas*. Tumors arising in nerve tissue are often benign, but because of their critical location they are more likely to be harmful than benign tumors in other sites.

Malignancies originating in *lymphoid tissues* are called *lymphomas*. Lymphomas can arise in many parts of the body, wherever lymphoid tissue is present (see Fig. 7-8). The most common sites to find lymphoid malignancies are the lymph nodes and spleen. However, lymphomas can appear in other parts of the body such as the skin, CNS, stomach, small bowel, bone, and tonsils.³⁰

Hematopoietic malignancies include leukemias, multiple myeloma, myelodysplasia, and the myeloproliferative syndromes.

Staging and Grading

Staging is the process of describing the extent of disease at the time of diagnosis to aid treatment planning, predict clinical outcome (prognosis), and compare the results of different treatment approaches. The stage of disease at the time of diagnosis reflects the rate of growth, the extent of the neoplasm, and the prognosis. A simplified way to stage cancer is as follows:

Stage 0: Carcinoma in situ (premalignant, preinvasive)

Stage I: Early stage, local cancer.

Stage II: Increased risk of spread because of tumor size.

Stage III: Local cancer has spread but may not be disseminated to distant regions.

Stage IV: Cancer has spread and disseminated to distant sites.

In some cases, cancer may be staged as II or III depending on the spread of the specific type of cancer. For example, in Hodgkin's disease, stage II indicates lymph nodes are affected on one side of the diaphragm. Stage III indicates affected lymph nodes above and below the diaphragm.²⁰⁸

Systems of Staging

Staging systems are specific for each type of cancer. For example, cervical cancer is staged using the International Federation of Gynecology and Obstetrics (FIGO) System of Staging, which is based on clinical examination, rather than surgical findings (see Box 20-3). Lymphomas are staged using the Ann Arbor staging (Box 9-1).

The TNM (tumor, node, metastases) system is used most often for solid tumors and has been adapted for other types of tumor. Some cancers do not have a staging system (e.g., brain cancer) and some can be staged using more than one system. The International Union Against Cancer (UICC) is the universally accepted staging system, which incorporated the TNM classification of malignant tumors the American Joint Committee on Cancer (AJCC) system previously used. The National Cancer Institute provides more detailed information about staging.¹³⁰

It is important to realize the TNM staging system is simply an anatomic staging system that describes the

Box 9-1

ANN ARBOR STAGING

Stage I: Local cancer in one area such as one lymph node and the local surrounding area; usually there are no other systemic or clinical symptoms.

Stage II: Cancer is located in two separate regions on one side of the diaphragm (above or below the diaphragm); two separate regions refers to an affected lymph node or organ within the lymphatic system and a second affected area.

Stage III: Cancer has spread to both sides of the diaphragm (above and below); includes one organ or area near the lymph nodes or the spleen.

Stage IV: Diffuse or disseminated spread to one or more extralymphatic organ or area near the lymph nodes or the spleen; liver, bone marrow, or nodular involvement of the lungs is possible.

Ann Arbor Staging is used for lymphomas (Hodgkin's disease and non-Hodgkin's lymphoma).

Letters, such as A, B, E, and X, may be used to modify or append the stage.

A = absence of constitutional symptoms.

B = presence of constitutional symptoms.

E = extranodal (not in the lymph nodes or has spread from the lymph nodes to adjacent tissue).

X = used to describe mass larger than 10 cm or mediastinum wider than 1/3 of the chest on a chest x-ray.

Data from Wikipedia: Ann Arbor staging. Available at <http://en.wikipedia.org>. Accessed January 3, 2007.

anatomic extent of the primary tumor, as well as the involvement of regional lymph nodes and distant metastases. Revisions to the TNM staging system are made as the understanding of the natural history of tumors at various sites improves with advancing technology. In the TNM classification scheme, tumors are staged according to the following basic components (Box 9-2):

- Tumor (T) refers to the primary tumor and carries a number from 0 to 4.
- Regional lymph nodes (N) represents regional lymph node involvement; also ranked from 0 to 4.
- Metastasis (M) is zero (0) if no metastasis has occurred or 1 if metastases are present.

Numbers are used with each component to denote extent of involvement; for example, T0 indicates undetectable, and T1, T2, T3, and T4 indicate a progressive increase in size or involvement.¹³⁵

In the TNM system, clinical stage is denoted by a small "c" before the stage (e.g., cT2N1M0) or by a small "p" to indicate the pathologic stage (e.g., pT2N0). Clinical staging is made indirectly by observation of the person before the tumor is treated or removed. The pathologic stage is determined by direct examination of the tumor by the pathologist once it has been removed and is considered a more accurate reflection of the tumor and its spread. Not all tumors are resected or excised so pathologic staging is not always available. Pathologic staging may underestimate the true stage for individuals who received adjuvant treatment (radiation or chemotherapy) before surgery.

Pathologic staging may also be in error if the slice of tissue examined is normal; oversight of tumor burden