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CHAPTER OUTLINE

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CHAPTER OBJECTIVES

The objectives of this chapter are to provide a brief understanding of the following:

1. Clinical evaluation of infectious diseases and altered immune disorders, including physical examination and laboratory studies
2. Various infectious disease processes, including etiology, pathogenesis, clinical presentation, and management
3. Commonly encountered altered immune disorders, including etiology, clinical presentation, and management
4. Precautions and guidelines that a physical therapist should implement when treating a patient with an infectious disease process or altered immunity

PREFERRED PRACTICE PATTERNS

The most relevant practice patterns for the diagnoses discussed in this chapter, based on the American Physical Therapy Association's *Guide to Physical Therapist Practice*, second edition, are as follows:

- Health Care–Associated or Nosocomial Infections (*Escherichia coli*, *Staphylococcus aureus*, *Enterococcus faecalis*, *Pseudomonas aeruginosa*, *Candida albicans*, and Coagulase-Negative Staphylococci): 6B, 7A
- Antibiotic-Resistant Infections: Methicillin-Resistant *Staphylococcus aureus*, Vancomycin-Resistant Enterococci, Multi-Drug Resistant *Acinetobacter baumannii*: 6B, 7A
- Upper Respiratory Tract Infections (Rhinitis, Sinusitis, Influenza, Pertussis): 6B, 6F, 6G
- Lower Respiratory Tract Infections (Tuberculosis, Histoplasmosis, Legionellosis, Severe Acute Respiratory Syndrome [SARS]): 6B, 7C, 7D
- Cardiac Infections: Pericarditis, Myocarditis, Left-Sided Endocarditis, Acute Rheumatic Fever, Rheumatic Heart Disease. See Chapter 3: 6B, 6D
- Neurological Diseases: Poliomyelitis, Postpoliomyelitis Syndrome, Meningitis, and Encephalitis: 4A, 5C, 5D, 5G, 6E, 5H, 7A
- Musculoskeletal Infections: Osteomyelitis and Its Variations: 4G, 4H, 5H
- Skin Infections: Cellulitis, groups A and G Streptococcus, and *Staphylococcus aureus*: 4E, 6H, 7B, 7C, 7D, 7E
- Gastrointestinal Infections: Gastroenteritis, *Escherichia coli*, *Shigella*, *Clostridium difficile*, *Salmonella*, Rotavirus, Norovirus, Adenovirus, and Astrovirus: Please refer to Chapter 8
- Immune System Infections: HIV, Mononucleosis, Cytomegalovirus Infection, and Toxoplasmosis: 4C, 6B
- Sepsis: Bacteremia, Septicemia, and Shock Syndrome (or Septic Shock): 5C, 6F, 6H

Please refer to Appendix A for a complete list of the preferred practice patterns, as individual patient conditions are highly variable and other practice patterns may be applicable.

A patient may be admitted to the hospital setting with an infectious disease process acquired in the community or may develop one as a complication from the hospital environment. The current terminology is to call this type of infection a health care–associated infection (HAI). In 2002, the estimated number of HAIs in U.S. hospitals was 1.7 million, resulting in about 99,000 deaths.¹ The major source of HAI is likely the patient's endogenous flora, but up to 40% of HAIs can be caused by cross infection via the hands of health care workers.² An infectious disease process generally has a primary site of origin; however, it may result in diffuse

systemic effects that may limit the patient's functional mobility and activity tolerance. Therefore a basic understanding of these infectious disease processes is useful in designing, implementing, and modifying physical therapy treatment programs. The physical therapist may also provide treatment for patients who have disorders resulting from altered immunity. These disorders are mentioned in this chapter because immune system reactions can be similar to those of infectious disease processes (see Appendix 13-A for a discussion of four common disorders of altered immunity: systemic lupus erythematosus, sarcoidosis, amyloidosis, and rheumatoid arthritis).

Definition of Terms

To facilitate the understanding of infectious disease processes, terminology that is commonly used when referring to these processes is presented in Table 13-1.³⁻⁶

Body Structure and Function

A person's immune system is composed of many complex, yet synergistic, components that defend against pathogens (Table 13-2).³ Any defect in this system may lead to the development of active infection. Patients in the acute care setting often present with acquired factors that can create some or most of

these defects, which can ultimately affect their immune system (Box 13-1).⁴ Congenital factors such as lymphocyte deficiency occur rarely.

Evaluation

When an infectious disease process is suspected, a thorough patient interview (history) and physical examination are performed to serve as a screening tool for the differential diagnosis and to help determine which laboratory tests are further required to identify a specific pathogen.⁷

History

Potential contributing factors of the infection are sought out, such as immunocompromise, immunosuppression, recent exposure to infectious individuals, or recent travel to foreign countries. Also, a qualitative description of the symptomatology is discerned, such as onset or nature of symptoms (e.g., a nonproductive versus productive cough over the past days or weeks).

Physical Examination

Observation

Clinical presentation of infectious diseases is highly variable according to the specific system that is involved. However,

TABLE 13-1 Terminology Associated with Infectious Disease Processes

Term	Definition
Antibody	A highly specific protein that is manufactured in response to antigens and defends against subsequent infection.
Antigen (immunogen)	An agent that is capable of producing antibodies when introduced into the body of a susceptible person.
Carrier	A person who harbors an infectious agent that can cause a specific disease but who demonstrates no evidence of the disease.
Colonization	The process of a group of organisms living together; the host can carry the microorganism without being symptomatic (no signs of infection).
Communicable	The ability of an infective organism to be transmitted from person to person, either directly or indirectly.
Disseminated host	Distributed over a considerable area. The person whom the infectious agent invades and from whom it gathers its nourishment.
Health care–associated infection (HAI)	Localized or systemic condition resulting from an adverse reaction to the presence of an infectious agent(s) or its toxin(s); there must be no evidence that the infection was present or incubating at the time of admission to the acute care setting.
Immunocompromised	An immune system that is incapable of a normal response to pathogenic organisms and tissue damage.
Immunodeficiency	Decreased or compromised ability to respond to antigenic stimuli by appropriate cellular immunity reaction.
Immunosuppression	The prevention or diminution of the immune response, as by drugs or radiation.
Nosocomial infection	Infection acquired in the hospital setting; note that this has been replaced by HAI (see above).
Opportunistic	An infectious process that develops in immunosuppressed individuals. (Opportunistic infections normally do not develop in individuals with intact immune systems.)
Pathogen	An organism capable of producing a disease.
Subclinical infection	A disease or condition that does not produce clinical symptoms, or the time period before the appearance of disease-specific symptoms.

TABLE 13-2 Components of the Immune System

Lines of Defense	Components	Description
First line of defense	Skin, conjunctivae, mucous membranes	Physical barriers to pathogens.
Second line of defense	Inflammatory response	Inflammatory response acts to (1) contain pathogens and (2) bring immune cells to antigens by releasing histamine, kinins, and prostaglandins that cause vasodilation and vascular permeability.
Third line of defense	Immune response Humoral immunity (B cells)* Cellular immunity (T cells)*	Specific immune response to pathogens. B cells produce antibodies. T cells: (1) Augment production of antibodies. (2) Directly kill antigens. (3) Turn off immune system.

Data from NS Rote: Immunity. In SE Heuther, KL McCance, editors: Understanding pathophysiology, ed 2, St Louis, 2000, Mosby, pp 125-150; Marieb EN, editor: Human anatomy and physiology, ed 2, Redwood City, CA, 1992, Benjamin Cummings, pp 690-723; Guyton AC, Hall JE: Textbook of medical physiology, ed 9, Philadelphia, 1996, Saunders, pp 445-455.

*B cells and T cells can also be referred to as B lymphocytes and T lymphocytes, respectively.

BOX 13-1 Factors Affecting the Immune System

- Pregnancy
- Preexisting infections
- Malignancies (Hodgkin's disease, acute or chronic leukemia, nonlymphoid malignancy, or myeloma)
- Stress (emotional or surgical—anesthesia)
- Malnutrition (insufficiency of calories, protein, iron, and zinc)
- Age
- Chronic diseases (diabetes, alcoholic cirrhosis, sickle cell anemia)
- Lymph node dissection
- Immunosuppressive treatment (corticosteroids, chemotherapy, or radiation therapy)
- Indwelling lines and tubes

Data from Rote NS, Heuther SE, McCance KL: Hypersensitivities, infection, and immunodeficiencies. In Heuther SE, McCance KL, editors: Understanding pathophysiology, ed 2, St Louis, 2000, Mosby, pp 204-208.

common physical findings that occur with infection include sweating and inflammation, both of which are related to the metabolic response of the body to the antigen. The classic signs of inflammation (redness [rubor], and swelling [tumor]) in certain areas of the body can help delineate the source, location(s), or both of infection. Delineating the source of infection is crucial to the diagnostic process.

Palpation

The presence of warmth (calor) and possible pain (dolor) or tenderness is another typical classic sign of inflammation that may be consistent with active infection. Lymphoid organs (lymph nodes and spleen) can also be swollen and tender with infection, because lymphocytes (processed in these organs) are multiplying in response to the antigen. Inflammation and tenderness in these or other areas of the body can further help to delineate the infectious process.

Vital Signs

Heart Rate, Blood Pressure, and Respiratory Rate. Measurement of vital signs helps in determining whether an infectious process is occurring. (Infections result in an increased metabolic rate, which presents as an increased heart rate and

respiratory rate.) Blood pressure may also be elevated when metabolism is increased, or blood pressure can be decreased secondary to vasodilation from inflammatory responses in the body.

Temperature. Monitoring the patient's temperature over time (both throughout the day and daily) provides information regarding the progression (a rise in temperature) or a regression (a fall in temperature) of the infectious process. With an infectious process, some of the bacteria and extracts from normal leukocytes are pyrogenic, causing the thermostat in the hypothalamus to rise, resulting in an elevated body temperature.⁸ A fall in body temperature from a relatively elevated temperature may also signify a response to a medication.



CLINICAL TIP

An afebrile status is not always indicative of the absence of infection. If a patient is on antipyretics, the fever symptoms may be controlled. Check the medication list and ask about the administration schedule. A patient must be afebrile for at least 24 hours before being discharged from an inpatient setting.

Auscultation

Heart and lung sounds determine whether infectious processes are a direct result from these areas or are indirectly affecting these areas. Refer to Chapters 3 and 4, respectively, for more information on heart and lung auscultation.

Laboratory Studies

Most of the evaluation process for diagnosing an infectious disease is based on laboratory studies. These studies are performed to (1) isolate the microorganisms from various body fluids or sites; (2) directly examine specimens by microscopic, immunologic, or genetic techniques; or (3) assess specific antibody responses to the pathogen.⁹ This diagnostic process is essential to prescribing the most specific medical regimen possible for the patient.

Hematology

During hematologic studies, a sample of blood is taken and analyzed to assist in determining the presence of an infectious

process or organism. Hematologic procedures used to diagnose infection include leukocyte count, differential white blood cell (WBC) count, and antibody measurement.¹⁰

Leukocyte Count. Leukocyte, or WBC, count is measured to determine whether an infectious process is present and should range between 5000 and 10,000 cells/mm³.³ An increase in the number of WBCs, termed *leukocytosis*, is required for phagocytosis (cellular destruction of microorganisms) and can indicate the presence of an acute infectious process.¹¹ Leukocytosis can also be present with inflammation and may occur after a surgery with postoperative inflammation.⁸ A decreased WBC count from baseline, termed *leukopenia*, can indicate altered immunity or the presence of an infection that exhausts supplies of certain WBCs.¹¹ A decreased WBC count relative to a previously high count (i.e., becoming more within normal limits) may indicate the resolution of an infectious process.¹¹

Differential White Blood Cell Count. Five types of WBCs exist: lymphocytes, monocytes, neutrophils, basophils, and eosinophils. Specific types of infectious processes can trigger alterations in the values of one or more of these cells. Detection of these changes can assist in identification of the type of infection present. For example, an infection caused by bacteria can result in a higher percentage of neutrophils, which have a normal range of 2.0 to 7.5×10^9 /liter. In contrast, a parasitic infection will result in increased eosinophils, which have a normal count of 0.0 to 0.45×10^9 /liter.¹¹

Antibody Measurement. Antibodies develop in response to the invasion of antigens from new infectious agents. Identifying the presence and concentration of specific antibodies helps in determining past and present exposure to infectious organisms.¹²

Microbiology

In microbiology studies, specimens from suspected sources of infection (e.g., sputum, urine, feces, wounds, and cerebrospinal fluid) are collected by sterile technique and analyzed by staining, culture, or sensitivity or resistance testing, or a combination of all of these.

Staining. Staining allows for morphologic examination of organisms under a microscope. Two types of staining techniques are available: simple staining and the more advanced differential staining. Many types of each technique exist, but the differential Gram's stain is the most common.¹²

Gram's stain is used to differentiate similar organisms by categorizing them as gram-positive or gram-negative. This separation assists in determining subsequent measures to be taken for eventual identification of the organism. A specimen is placed on a microscope slide, and a series of steps are performed.¹³ A red specimen at completion indicates a gram-negative organism, whereas a violet specimen indicates a gram-positive organism.¹³

Culture. The purpose of a culture is to identify and produce isolated colonies of organisms found within a collected specimen. Cells of the organism are isolated and mixed with specific media that provide the proper nourishment and environment (e.g., pH level, oxygen content) needed for the organism to reproduce into colonies. Once this has taken place, the resultant

infectious agent is observed for size, shape, elevation, texture, marginal appearance, and color to assist with identification.¹³

Sensitivity and Resistance. When an organism has been isolated from a specimen, its sensitivity (susceptibility) to antimicrobial agents or antibiotics is tested. An infectious agent is sensitive to an antibiotic when the organism's growth is inhibited under safe dose concentrations. Conversely, an agent is resistant to an antibiotic when its growth is not inhibited by safe dose concentrations. Because of a number of factors, such as mutations, an organism's sensitivity, resistance, or both to antibiotics are constantly changing.¹⁴

Cytology

Cytology is a complex method of studying cellular structures, functions, origins, and formations. Cytology assists in differentiating between an infectious process and a malignancy and in determining the type and severity of a present infectious process by examining cellular characteristics.^{12,15} It is beyond the scope of this book, however, to describe all of the processes involved in studying cellular structure dysfunction.

Body Fluid Examination

Pleural Tap. A pleural tap, or thoracentesis, is the process by which a needle is inserted through the chest wall into the pleural cavity to collect pleural fluid for examination of possible malignancy, infection, inflammation, or any combination of these. A thoracentesis may also be performed to drain excessive pleural fluid in large pleural effusions.¹⁶

Pericardiocentesis. Pericardiocentesis is a procedure that involves accessing the pericardial space around the heart with a needle or cannula to aspirate fluid for drainage, analysis, or both. It is primarily used to assist in diagnosing infections, inflammation, and malignancies and to relieve effusions built up by these disorders.¹⁷

Synovial Fluid Analysis. Synovial fluid analysis, or arthrocentesis, involves aspirating synovial fluid from a joint capsule. The fluid is then analyzed and used to assist in diagnosing infections, rheumatic diseases, and osteoarthritis, all of which can produce increased fluid production within the joint.¹⁸

Gastric Lavage. A gastric lavage is the suctioning of gastric contents through a nasogastric tube to examine the contents for the presence of sputum in patients suspected of having tuberculosis. The assumption is that patients swallow sputum while they sleep. If sputum is found in the gastric contents, the appropriate sputum analysis should be performed to help confirm the diagnosis of tuberculosis.^{16,19} Historically, gastric lavage has also been administered as a medical intervention to prevent absorption of ingested toxins in the acutely poisoned patient, although its use for this purpose is now rarely recommended.²⁰

Peritoneal Fluid Analysis. Peritoneal fluid analysis, or paracentesis, is the aspiration of peritoneal fluid with a needle. It is performed to (1) drain excess fluid, or ascites, from the peritoneal cavity, which can be caused by infectious diseases, such as tuberculosis; (2) assist in the diagnosis of hepatic or systemic malfunctions, diseases, infection such as spontaneous bacterial peritonitis (SBP), or malignancies; and (3) help detect the presence of abdominal trauma.^{16,19,21}

Other Studies

Imaging with plain x-rays, computed tomography scans, positron emission tomography, and magnetic resonance imaging scans can also help identify areas with infectious lesions.^{22,23} Minuscule amounts of pathogens can be detected by using the molecular biology techniques of enzyme-linked immunosorbent assay (ELISA), radioimmunoassay (RIA), and polymerase chain reaction (PCR).^{24,25} In addition, the following diagnostic studies can also be performed to help with the differential diagnosis of the infectious process. For a description of these studies, refer to the sections and chapters indicated below:

- Sputum analysis (see Chapter 4)
- Cerebrospinal fluid (see Chapter 6)
- Urinalysis (see Chapter 9)
- Wound cultures (Chapter 12)

Health Conditions

Various infectious disease processes, which are commonly encountered in the acute care setting, are described in the following sections. Certain disease processes that are not included in this section are described in other chapters. Please consult the index for assistance.

Health Care–Associated or Nosocomial Infections

Nosocomial infection is an older general term that refers to an infection that is acquired in the hospital setting. Since 2008 the Centers for Disease Control and Prevention (CDC) has used the generic term *health care–associated infections* instead of *nosocomial*.⁶ Many pathogens can cause an HAI, but the most commonly reported bacteria in past years have been *Escherichia coli*, *Staphylococcus aureus*, *Enterococcus faecalis*, *Pseudomonas aeruginosa*, *Candida albicans*, and coagulase-negative staphylococci.^{26,27} Patients who are at risk for developing HAIs are those who present with²⁸:

1. Age: the very young or the very old
2. Immunodeficiency: chronic diseases (cancer, chronic renal disease, chronic obstructive pulmonary disease, diabetes, or acquired immunodeficiency syndrome [AIDS])
3. Immunosuppression: chemotherapy, radiation therapy, or corticosteroids
4. Misuse of antibiotics: overprescription of antibiotics or use of broad-spectrum antibiotics, leading to the elimination of a patient’s normal flora, which allows for the colonization of pathogens and development of drug-resistant organisms
5. Use of invasive diagnostic and therapeutic procedures: indwelling urinary catheters, monitoring devices, intravenous (IV) catheters, and mechanical ventilation with intubation
6. Agitation: Resulting in removal of medical equipment such as central venous catheters or self-extubation of artificial airways
7. Surgery: incisions provide access to pathogens
8. Burns: disrupt the first line of defense
9. Length of hospitalization: increases the exposure to pathogens and medical intervention

The mode of transmission for pathogens that cause HAIs can vary from contact to airborne. Pathogens can also become opportunistic in patients who are immunocompromised or immunosuppressed. Common sites for HAIs are in the urinary tract, surgical wounds, joints, and the lower respiratory tract (e.g., pneumonia). Clinical manifestations and management of HAIs vary according to the type of pathogen and the organ system involved. However, the primary management strategy for HAIs is prevention by following the standard and specific precautions outlined in [Table 13-3](#).^{9,26,29,30}

 **CLINICAL TIP**

Prevention or minimizing the risk of developing a pneumonia in patients who have been on bed rest and/or on mechanical intervention can be achieved through chest physical therapy and increased mobility. (Refer to Table 4-12, Dean’s Hierarchy for Treatment of Patients with Impaired Oxygen Transport.)

TABLE 13-3 Summary of Precautions to Prevent Infection

Precaution	Description
Standard	Treat all patient situations as potentially infectious. Wash hands before and after each patient contact. Wear a different set of gloves with each patient. If splashing of body fluids is likely, wear a mask or face shield, or both, and a gown.
Airborne*	A mask is required in situations where contagious pathogens can be transmitted by airborne droplet nuclei, as in the case of measles, varicella (chickenpox), or tuberculosis.
Droplet*	A mask or face shield, or both, are required when large-particle droplet transmission (usually 3 ft or less) is likely. Droplet transmission involves contact of the conjunctivae or the mucous membranes of the nose or mouth with large-particle droplets (larger than 5 µm in size) generated from coughing, sneezing, talking, and certain procedures, such as suctioning and bronchoscopy. Examples of pathogens requiring droplet precautions are <i>Haemophilus influenzae</i> , <i>Neisseria meningitidis</i> , mycoplasmal pneumonia, streptococcal pneumonia, mumps, and rubella.
Contact*	Gown and gloves are required when pathogens are transmitted by direct person-to-person contact or person-to-object contact. Examples of these pathogens include <i>Acinetobacter baumannii</i> , <i>Clostridium difficile</i> , <i>Escherichia coli</i> , herpes simplex virus, herpes zoster, methicillin-resistant <i>Staphylococcus aureus</i> , and vancomycin-resistant <i>Enterococcus</i> .

Data from Rice D, Eckstein EC: Inflammation and infection. In Phipps WJ, Sands JK, Marek JF, editors: Medical-surgical nursing, concepts and clinical practice, ed 6, St Louis, 1999, Mosby, pp 237-245; Anderson KN, editor: Mosby’s medical, nursing, and allied health dictionary, ed 5, St Louis, 1998, Mosby, p 2BA5.

*These precautions are in addition to practicing Standard Precautions.

Antibiotic-Resistant Infections

The number of antibiotic-resistance infections is growing in health care facilities. Approximately 50% of antibiotic use in hospitals is unnecessary or inappropriate. In response to this problem, the CDC has launched a program called “Get Smart for Healthcare” whose goals include reducing unnecessary antibiotic use (resulting in less antimicrobial resistance), decreasing health care costs, and improving patient outcomes in hospitals and long-term care facilities.³¹

Microbial experts from the European Centre for Disease Prevention and Control and in the United States from the CDC have recently developed interim standard terminology to describe this resistance.³² They developed three major definitions for resistance: multidrug-resistant (MDR), extensively drug-resistant (XDR), and pandrug-resistant (PDR) bacteria. The agreed-on definitions are MDR as acquired nonsusceptibility to at least one agent in three or more antimicrobial categories, XDR as nonsusceptibility to at least one agent in all but two or fewer antimicrobial categories (i.e., remaining susceptible to only one or two categories), and PDR as nonsusceptibility to all agents in all antimicrobial categories.

Methicillin-Resistant *Staphylococcus aureus* Infection.

Methicillin-resistant *S. aureus* (MRSA) is a strain of *Staphylococcus* that is resistant to methicillin or similar agents, such as oxacillin and nafcillin. Methicillin is a synthetic form of penicillin and was developed because *S. aureus* developed resistance to penicillin, which was originally the treatment choice for *S. aureus* infection. However, since the early 1980s, this particular strain of *S. aureus* has become increasingly resistant to methicillin. The contributing factor that is suggested to have a primary role in the increased incidence of this HAI is the indiscriminate use of antibiotic therapy.^{30,33}

In addition, patients who are at risk for developing MRSA infection in the hospital are patients who³³⁻³⁵:

- Are debilitated, elderly, or both
- Are hospitalized for prolonged time periods
- Have multiple surgical or invasive procedures, an indwelling cannula, or both
- Are taking multiple antibiotics, antimicrobial treatments, or both
- Are undergoing treatment in critical care units

MRSA is generally transmitted by person-to-person contact or person-to-object-to-person contact. MRSA can survive for prolonged periods of time on inanimate objects, such as telephones, bed rails, and tray tables, unless such objects are properly sanitized. Hospital personnel can be primary carriers of MRSA, as the bacterium can be colonized in healthy adults. MRSA infections can be diagnosed via nasal swabs.³⁶

Management of MRSA is difficult and may consist of combining local and systemic antibiotics, increasing antibiotic dosages, and applying whole-body antiseptic solutions. In recent years, vancomycin has become the treatment of choice for MRSA; however, evidence has shown that patients with this strain of *S. aureus* are also developing resistance to vancomycin (vancomycin intermediate *S. aureus*—VISA).³⁰ Therefore prevention of MRSA infection is the primary treatment strategy and includes the following^{26,33-35}:

- Placing patients with MRSA infection on isolation or contact precautions
- Strict hand-washing regulations before and after patient care using proper disinfecting agent
- Use of gloves, gowns (if soiling is likely), or both
- Disinfection of all contaminated objects

Vancomycin-Resistant Enterococci Infection. Vancomycin-resistant enterococci (VRE) infection is another HAI that has become resistant to vancomycin, aminoglycosides, and ampicillin. The infection can develop as endogenous enterococci (normally found in the gastrointestinal or the female reproductive tract) become opportunistic in patient populations similar to those mentioned earlier with MRSA. VRE infections can be diagnosed via rectal swab.^{26,30,37,38}

Transmission of the infection can also occur by (1) direct patient-to-patient contact, (2) indirect contact through asymptomatic hospital personnel who can carry the opportunistic strain of the microorganism, or (3) contact with contaminated equipment or environmental surfaces.

Management of VRE infection is difficult, as the enterococcus can withstand harsh environments and easily survive on the hands of health care workers and on hospital objects. Treatment options are very limited for patients with VRE, and the best intervention plan is to prevent the spread of the infectious process.³⁰ Strategies for preventing VRE infections include the following³⁷:

- The controlled use of vancomycin
- Timely communication between the microbiology laboratory and appropriate personnel to initiate contact precautions as soon as VRE is detected
- Implementation of screening procedures to detect VRE infection in hospitals where VRE has not yet been detected (i.e., randomly culturing potentially infected items or patients)
- Preventing the transmission of VRE by placing patients in isolation or grouping patients with VRE together, wearing gown and gloves (which need to be removed inside the patient’s room), and washing hands immediately after working with an infected patient
- Designating commonly used items, such as stethoscopes and rectal thermometers, to be used only with VRE patients
- Disinfecting any item that has been in contact with VRE patients with the hospital’s approved cleaning agent

Multidrug-Resistant *Acinetobacter baumannii*. Over the past decade *Acinetobacter baumannii* (AB) has become one of the most difficult pathogens to effectively treat because it easily acquires a wide spectrum of antimicrobial resistance, resulting in the commonly found MDR and the much more serious but fortunately rarer PDR forms. It is a gram-negative coccobacillus that has become one of the most important pathogens, particularly in the intensive care unit (ICU). AB infections in the hospital can cause serious complications such as ventilator-associated pneumonia (VAP), bloodstream infection, wound infections, and nosocomial meningitis.^{39,40}

AB is remarkable in that it is ubiquitous, exists in diverse habitats (e.g., human skin), can survive for long periods of time on dry inanimate surfaces (e.g., hospital bed rails) and as already

mentioned can acquire antimicrobial resistance extremely rapidly. These factors combined, especially the latter two, greatly facilitate MDR-AB outbreaks in the ICU, in physical therapy wound clinics and even multi-facility outbreaks.^{41,42} Fortunately, strict infection-control measures (e.g., contact isolation precautions outlined in Table 13-3 and in guidelines for physical therapy intervention at the end of the chapter) can decrease health care staff and environmental colonization and/or contamination.⁴³ MDR-AB and PDR-AB infections can also be prevented by following the previously mentioned guidelines effective against MRSA and VRE.

CLINICAL TIP

Equipment used during physical therapy treatments for patients with antibiotic-resistant bacteria (e.g., MRSA, VRE, or MDR-AB), such as assistive devices, gait belts, cuff weights, or goniometers, should be left in the patient's room and not be taken out until the infection is resolved. If there is an equipment shortage, thorough cleaning of the equipment is necessary before using the equipment with other patients. Linens, hospital curtains, and laboratory coats also need to be properly cleaned to avoid transmission of infection.

Respiratory Tract Infections

Infections of the respiratory tract can be categorized as upper or lower respiratory tract infections. Upper respiratory tract infections that are discussed in this section consist of allergic and viral rhinitis, sinusitis, influenza, and pertussis. Lower respiratory tract infections that are discussed in this section consist of tuberculosis, histoplasmosis, legionellosis, and severe acute respiratory syndrome. Pneumonia is the most common lower respiratory tract infection and is discussed under Health Conditions in Chapter 4.

Upper Respiratory Tract Infections

Rhinitis. Rhinitis is the inflammation of the nasal mucous membranes and can result from an allergic reaction or viral infection. Allergic rhinitis is commonly a seasonal reaction from allergens, such as pollen, or a perennial reaction from environmental triggers, such as pet dander or smoke. Viral rhinitis, sometimes referred to as the common cold, is caused by a wide variety of viruses that can be transmitted by airborne particles or by contact.

Clinical manifestations of allergic and viral rhinitis include nasal congestion; sneezing; watery, itchy eyes and nose; altered sense of smell; and thin, watery nasal discharge. In addition to these, clinical manifestations of viral rhinitis include fever, malaise, headache, and thicker nasal discharge.

Management of allergic rhinitis includes antihistamines, decongestants, nasal corticosteroid sprays, and allergen avoidance. Management of viral rhinitis includes rest, fluids, antipyretics, and analgesics.⁴⁴⁻⁴⁶

Sinusitis. Sinusitis is the inflammation or hypertrophy of the mucosal lining of any or all of the facial sinuses (frontal,

ethmoid, sphenoid, and maxillary). This inflammation can result from bacterial, viral, or fungal infection.

Clinical manifestations of sinusitis include pain over the affected sinus, purulent nasal drainage, nasal obstruction, congestion, fever, and malaise.

Management of sinusitis includes antibiotics (as appropriate), decongestants or expectorants, and nasal corticosteroids.⁴⁵

CLINICAL TIP

Despite the benign nature of rhinitis and sinusitis, the manifestations (especially nasal drainage and sinus pain) of these infections can be very disturbing to the patient and therapist during the therapy session and may lower the tolerance of the patient for a given activity. The therapist should be sympathetic to the patient's symptoms and adjust the activity accordingly.

Influenza. Influenza (the flu) is caused by any of the influenza viruses (A, B, or C and their mutagenic strains) that are transmitted by aerosolized mucous droplets. These viruses have the ability to change over time and are the reason why a great number of patients are at risk for developing this infection. Influenza B is the most likely virus to cause an outbreak within a community. Health care workers should be vaccinated against the influenza virus to decrease the risk of transmission.

Clinical manifestations of influenza include (1) a severe cough, (2) abrupt onset of fever and chills, (3) headache, (4) backache, (5) myalgia, (6) prostration (exhaustion), (7) coryza (nasal inflammation with profuse discharge), and (8) mild sore throat. Gastrointestinal signs and symptoms of nausea, vomiting, abdominal pain, and diarrhea can also present in certain cases. The disease is usually self-limiting in uncomplicated cases, with symptoms resolving in 7 to 10 days. A complication of influenza infection is pneumonia, especially in the elderly and chronically diseased individuals.^{3,4,16,45}

CLINICAL TIP

A rapid flu nasal swab can diagnose influenza. If results have not come back or they are positive, wear a simple face mask to prevent transmission.

If management of influenza is necessary, it may include the following^{3,4,16,45}:

- Antiinfective agents
- Antipyretic agents
- Adrenergic agents
- Antitussive agents
- Active immunization by vaccines
- Supportive care with IV fluids and supplemental oxygen, as needed

Pertussis. Pertussis, or whooping cough, is an acute bacterial infection of the mucous membranes of the tracheobronchial tree, and recently the number of cases has been increasing in the United States.⁴⁷ It occurs most commonly in children younger than 1 year and in children and adults of lower socioeconomic populations. The defining characteristics are violent

cough spasms that end with an inspiratory “whoop,” followed by the expulsion of clear tenacious secretions. Symptoms may last 1 to 2 months. Pertussis is transmitted through airborne particles and is highly contagious.⁴⁸

Management of pertussis may include any of the following^{16,48}:

- Antiinfective and antiinflammatory medications
- Bronchopulmonary hygiene with endotracheal suctioning, as needed
- Supplemental oxygen, assisted ventilation, or both
- Fluid and electrolyte replacement
- Active immunization by vaccines
- Respiratory isolation for 3 weeks after the onset of coughing spasms or 7 days after antimicrobial therapy

Lower Respiratory Tract Infections

Tuberculosis. Tuberculosis (TB) is a chronic pulmonary and extrapulmonary infectious disease caused by the tubercle bacillus. It is transmitted through airborne *Mycobacterium tuberculosis* particles, which are expelled into the air when an individual with pulmonary or laryngeal TB coughs or sneezes.⁴⁹ When *M. tuberculosis* reaches the alveolar surface of a new host, it is attacked by macrophages, and one of two outcomes can result: Macrophages kill the particles, terminating the infectious process, or the particles multiply within the WBCs, eventually causing them to burst. This cycle is then repeated for a variable time frame between 2 and 12 weeks, after which time the individual is considered to be infected with TB and will test positive on tuberculin skin tests, such as the Mantoux test, which uses tuberculin-purified protein derivative,* or the multiple puncture test, which uses tuberculin. At this point, the infection enters a latent period (most common) or develops into active TB.^{49,50}

A six-category classification system has been devised by the American Thoracic Society and the Centers for Disease Control and Prevention (CDC) to describe the TB status of an individual.^{49,51}

1. No TB exposure, not infected
2. TB exposure, no evidence of infection
3. Latent TB infection, no disease
4. TB, clinically active
5. TB, not clinically active
6. TB suspect (diagnosis pending)

CLINICAL TIP

Patients with TB are placed, if available, in negative-pressure isolation rooms. This results in air flowing into, but not out, of the isolation room, thus preventing the escape of contaminated air into the rest of the building. Patients who are suspected of TB, but have not been diagnosed with it, are generally placed on “rule-out TB” protocol, in which case respiratory precautions should be observed.

*A person who has been exposed to the tubercle bacillus will demonstrate a raised and reddened area 2 to 3 days after being injected with the protein derivative of the bacilli.

Populations at high risk for acquiring TB include (1) the elderly; (2) Native Americans, Eskimos, and African-Americans (in particular if they are homeless or economically disadvantaged); (3) incarcerated individuals; (4) immigrants from Southeast Asia, Ethiopia, Mexico, and Latin America; (5) malnourished individuals; (6) infants and children younger than 5 years of age; (7) those with decreased immunity (e.g., from AIDS or leukemia, or after chemotherapy); (8) those with diabetes mellitus, end-stage renal disease, or both; (9) those with silicosis; and (10) those in close contact with individuals with active TB.^{5,49}

Persons with normal immune function do not normally develop active TB after acquisition and are therefore not considered contagious. Risk factors for the development of active TB after infection include age (children younger than 8 years and adolescents are at greatest risk), low weight, and immunosuppression.⁵²

When active TB does develop, its associated signs and symptoms include (1) fever, (2) an initial nonproductive cough, (3) mucopurulent secretions that present later, and (4) hemoptysis, dyspnea at rest or with exertion, adventitious breath sounds at lung apices, pleuritic chest pain, hoarseness, and dysphagia, all of which may occur in the later stages. Chest films also show abnormalities, such as atelectasis or cavitation involving the apical and posterior segments of the right upper lobe, the apical-posterior segment of the left upper lobe, or both.⁴⁹

Extrapulmonary TB occurs with less frequency than pulmonary TB but affects up to 70% of human immunodeficiency virus (HIV)-positive individuals diagnosed with TB.⁵³ Organs affected include the meninges, brain, blood vessels, kidneys, bones, joints, larynx, skin, intestines, lymph nodes, peritoneum, and eyes. When multiple organ systems are affected, the term *disseminated*, or *miliary*, TB is used.⁵³ Signs and symptoms that manifest are dependent on the particular organ system or systems involved.

Because of the high prevalence of TB in HIV-positive individuals (up to 60% in some states),⁵³ it should be noted that the areas of involvement and clinical features of the disease in this population differ from those normally seen, particularly in cases of advanced immunosuppression. Brain abscesses, lymph node involvement, lower lung involvement, pericarditis, gastric TB, and scrotal TB are all more common in HIV-positive individuals. HIV also increases the likelihood that TB infection will progress to active TB by impairing the body’s ability to suppress new and latent infections.⁵³

Management of TB may include the following^{3,4,16}:

- Antiinfective agents (see Chapter 19, Table 19-36, Antitubercular Agents)
- Corticosteroids
- Surgical intervention to remove cavitory lesions (rare) and areas of the lung with extensive disease or to correct hemoptysis, spontaneous pneumothorax, abscesses, intestinal obstruction, ureteral stricture, or any combination of these
- Respiratory isolation until antimicrobial therapy is initiated
- Blood and body fluid precautions if extrapulmonary disease is present

- Skin testing (i.e., Mantoux test and multiple puncture test)
- Vaccination for prevention

In recent years, new strains of *M. tuberculosis* that are resistant to antitubercular drugs (e.g., isoniazid, rifampin, and pyrazinamide) have emerged. These multidrug-resistant TB strains are associated with fatality rates as high as 89% and are common in HIV-infected individuals. Treatment includes the use of direct observational therapy (DOT) and direct observational therapy, short-course (DOTS). These programs designate health care workers to observe individuals to ensure that they take their medications for the entire treatment regimen or for a brief period, respectively, in hopes of minimizing resistance.⁵³

CLINICAL TIP

Facilities should provide health care workers personal protective equipment (PPE) effective against TB such as either specialized masks (e.g., N-95) or powered air-purifying respirators (PAPR) to wear around patients on respiratory precautions. These types of PPE are protective against the airborne TB mycobacterium. Always verify with the nursing staff or physician before working with these patients to determine which type of PPE to wear.

Histoplasmosis. Histoplasmosis is a pulmonary and systemic infection that is caused by infective spores (fungi), most commonly found in the soil of the central and eastern United States. Histoplasmosis is transmitted by inhalation of dust from the soil or bird and bat feces. The spores form lesions within the lung parenchyma that can be spread to other tissues. The incidence of fungal infection is rising, particularly in immunocompromised, immunosuppressed, and chronically debilitated individuals who may also be receiving corticosteroid, antineoplastic, and multiple antibiotic therapy.^{54,55}

Different clinical forms of histoplasmosis are (1) acute, benign respiratory disease, which results in flulike illness and pneumonia; (2) acute disseminated disease, which can result in septic-type fever; (3) chronic disseminated disease, which involves lesions in the bone marrow, spleen, and lungs and can result in immunodeficiency; and (4) chronic pulmonary disease, which manifests as progressive emphysema.

Management of histoplasmosis may include the following^{16,54,56,57}:

- Antiinfective agents
- Corticosteroids
- Antihistamines
- Antifungal therapy (see Chapter 19, Table 19-35, Antifungal Agents)
- Supportive care appropriate for affected areas in the various forms of histoplasmosis

Legionellosis. *Legionellosis* is commonly referred to as *Legionnaire's disease* after a pneumonia outbreak in people who attended an American Legion Convention in Philadelphia in 1976. It is an acute bacterial infection primarily resulting in high fever and pneumonia (patchy or confluent consolidation). *Legionella pneumophila* causes more than 80% of all cases of

legionellosis. However, organs beside the lungs may also become involved, especially in the immunocompromised patient. Other risk factors include underlying chronic pulmonary disease, smoking history, and age greater than 50 years. Legionellosis is transmitted by inhalation of aerosolized organisms from infected water sources, such as air-conditioning cooling towers for large buildings including hospitals. Additional examples of infected hospital water sources have included shower heads, tap water from respiratory devices, ice machines, decorative fountains, and even distilled water.^{3,58-60}

Primary clinical manifestations include high fever, pneumonia, malaise, myalgia, headache, and nonproductive cough. Other manifestations can also include diarrhea, confusion and other gastrointestinal symptoms. The disease is rapidly progressive during the first 4 to 6 days of illness, with complications that may include renal failure, bacteremic shock, and respiratory failure.^{3,59}

Management of legionellosis may consist of the following³:

- Antiinfective agents
- Supplemental oxygen with or without assisted ventilation
- Temporary renal dialysis
- IV fluid and electrolyte replacement

Severe Acute Respiratory Syndrome. The single-stranded RNA coronavirus is responsible for severe acute respiratory syndrome (SARS), which affects the epithelial cells of the lower respiratory tract. Pathogenesis is not limited to the lungs but often includes mucosal cells of the intestines, tubular epithelial cells of the kidneys, and brain neurons. This new disease was first identified in China in late 2002, and then spread into the rest of the world in the spring and summer of 2003, resulting in the first pandemic of the twenty-first century. Of the approximately 8000 worldwide cases that occurred during this pandemic, about 25% of patients required mechanical ventilation in the ICU and about 10% of infected patients died.

SARS has flulike symptoms of fever, chills, cough, and malaise along with frequent shortness of breath. A common cause of death during this pandemic was diffuse alveolar damage (DAD). In addition, SARS typically compromises the immune response, which increases lung injury.

The 2003-2004 SARS pandemic showed that a prompt, coordinated worldwide response could help contain the disease. Although SARS was rapidly spread throughout the world by international air travelers, the virus itself was not transmitted through the air. Thus adherence to the basic infection control practice of thorough hand washing, implemented with droplet precautions, was able to ultimately stop this particular SARS pandemic.^{61,62}

Cardiac Infections

Infections of the cardiac system can involve any layer of the heart (endocardium, myocardium, or pericardium) and generally result in acute or chronic depression of the patient's cardiac output. Infections that result in chronic cardiomyopathy most likely require cardiac transplantation. Refer to Chapters 3 and 14 for a discussion of cardiomyopathy and cardiac transplantation, respectively. This section focuses on rheumatic fever and resultant rheumatic heart disease.

Acute rheumatic fever is a clinical sequela occurring in up to 3% of patients with group A and β -streptococcal infection of the upper respiratory tract. It occurs primarily in children who are between the ages of 6 and 15 years. Rheumatic fever is characterized by nonsuppurative inflammatory lesions occurring in any or all of the connective tissues of the heart, joints, subcutaneous tissues, and central nervous system. An altered immune reaction to the infection is suspected as the cause of resultant damage to these areas, but the definitive etiology is unknown. *Rheumatic heart disease* is the term used to describe the resultant damage to the heart from the inflammatory process of rheumatic fever.^{16,34,63,64}

Cardiac manifestations can include pericarditis, myocarditis, left-sided endocarditis, and valvular stenosis and insufficiency with resultant organic heart murmurs, as well as congestive heart failure. If not managed properly, all of these conditions can lead to significant morbidity or death.^{16,34,63}

Management of rheumatic fever follows the treatment for streptococcal infection. The secondary complications mentioned previously are then managed specifically. The general intervention scheme may include the following^{16,34,63}:

- Prevention of streptococcal infection
- Antiinfective agents
- Antipyretic agents
- Corticosteroids
- Bed rest
- IV fluids (as needed)

Neurologic Infections

Poliomyelitis

Poliomyelitis is an acute systemic viral disease that affects the central nervous system and fortunately is in rapid decline, with global eradication a distinct possibility.⁶⁵ Polioviruses are a type of enterovirus that multiply in the oropharynx and intestinal tract.^{16,66}

Poliomyelitis is usually transmitted directly by the fecal-oral route from person to person but can also be transmitted indirectly by consumption of contaminated water sources.⁶⁶

Clinical presentation can range from subclinical infection, to afebrile illness (24 to 36 hours), to aseptic meningitis, to paralysis (after 4 days), and, possibly, to death. If paralysis does occur, it is generally associated with fever and muscle pain. The paralysis is usually asymmetric and involves muscles of respiration, swallowing, and the lower extremities. Paralysis can resolve completely, leave residual deficits, or be fatal.^{16,66}

Management of poliomyelitis primarily consists of prevention with inactivated poliovirus vaccine (IPV) given as four doses to children from the ages of 2 to 6 years of age.⁶⁶ If a patient does develop active poliomyelitis, then other management strategies may include the following¹⁶:

- Analgesics and antipyretics
- Bronchopulmonary hygiene
- Bed rest with contracture prevention with positioning and range of motion

Postpoliomyelitis Syndrome. Postpoliomyelitis syndrome, also known as postpolio syndrome, occurs 30 to 40 years

after an episode of childhood paralytic poliomyelitis. The syndrome results from overuse or premature aging of motor units that were originally affected by the polio virus. It results in muscle fatigue, pain, and decreased endurance. Muscle atrophy and fasciculations may also be present. Patients who are older or critically ill, who have had a previous diagnosis of paralytic poliomyelitis, and who are female are at greater risk for development of this syndrome.⁶⁶⁻⁶⁸

Meningitis

Meningitis is an inflammation of the meninges that cover the brain and spinal cord, which results from acute infection by bacteria, viruses, fungi, or parasitic worms, or from chemical irritation. The route of transmission is primarily inhalation of infected airborne mucus droplets released by infected individuals, or through the bloodstream via open wounds or invasive procedures.^{69,70}

The more common types of meningitis are (1) meningococcal meningitis, which is bacterial in origin and occurs in epidemic form; (2) *Haemophilus* meningitis, which is the most common form of bacterial meningitis; (3) pneumococcal meningitis, which occurs as an extension of a primary bacterial upper respiratory tract infection; and (4) viral (aseptic or serous) meningitis, which is generally benign and self-limiting.

Bacterial meningitis is more severe than viral meningitis and affects the pia mater, arachnoid and subarachnoid space, ventricular system, and cerebrospinal fluid. The primary complications of bacterial meningitis include an increase in intracranial pressure, resulting in hydrocephalus. This process frequently results in severe headache and nuchal rigidity (resistance to neck flexion). Other complications of meningitis include arthritis, myocarditis, pericarditis, neuromotor and intellectual deficits, and blindness and deafness from cranial nerve (III, IV, VI, VII, or VIII) dysfunction.^{69,70}

Management of any form of meningitis may include the following^{16,69,71}:

- Antimicrobial therapy, antiinfective agents, or immunologic agents
- Analgesics
- Mechanical ventilation (as needed)
- Blood pressure maintenance with IV fluids and vasopressors (e.g., dopamine)
- Intracranial pressure control

Encephalitis

Encephalitis is an inflammation of the tissues of the brain and spinal cord, commonly resulting from viral or amebic infection. Types of encephalitis include infectious viral encephalitis, mosquito-borne viral encephalitis, and amebic meningoencephalitis.

Infectious viral encephalitis is transmitted by direct contact with droplets from respiratory passages or other infected excretions and is most commonly associated with the herpes simplex type 1 virus. Viral encephalitis can also occur as a complication of systemic viral infections, such as poliomyelitis, rabies, mononucleosis, measles, mumps, rubella, and chickenpox. Manifestations of viral encephalitis can be mild to severe, with herpes

simplex virus encephalitis having the highest mortality rate among all types of encephalitides.^{16,69,70}

Mosquito-borne viral encephalitis is transmitted by infectious mosquito bites and cannot be transmitted from person to person. The incidence of this type of encephalitis can be epidemic and typically varies according to geographic regions and seasons.^{16,69,70}

Amebic meningoencephalitis is transmitted in water and can enter a person's nasal passages while he or she is swimming. Amebic meningoencephalitis cannot be transmitted from person to person.

General clinical presentation of encephalitis may include the following^{16,69,70}:

- Fever
- Signs of meningeal irritation from increased intracranial pressure (e.g., severe frontal headache, nausea, vomiting, dizziness, nuchal rigidity)
- Altered level of consciousness, irritability, bizarre behaviors (if the temporal lobe is involved)
- Seizures (mostly in infants)
- Aphasia
- Focal neurologic signs
- Weakness
- Altered deep tendon reflexes
- Ataxia, spasticity, tremors, or flaccidity
- Hyperthermia
- Alteration in antidiuretic hormone secretion

Management of encephalitis may include the following¹⁶:

- Antiinfective agents
- Intracranial pressure management
- Mechanical ventilation, with or without tracheostomy (as indicated)
- Sedation
- IV fluids and electrolyte replacement
- Nasogastric tube feedings

Musculoskeletal Infections

Osteomyelitis is an acute infection of the bone that can occur from direct or indirect invasion by a pathogen. Direct invasion is also referred to as exogenous or acute contagious osteomyelitis and can occur any time there is an open wound in the body. Indirect invasion is also referred to as endogenous or acute hematogenous osteomyelitis and usually occurs from the spread of systemic infection. Both of these types can potentially progress to subacute and chronic osteomyelitis. Acute osteomyelitis typically refers to an infection of less than 1 month's duration, whereas chronic osteomyelitis refers to infection that lasts longer than 4 weeks.^{72,73}

Acute contagious osteomyelitis is an extension of the concurrent infection in adjacent soft tissues to the bony area. Trauma resulting in compound fractures and tissue infections is a common example. Prolonged orthopedic surgery, wound drainage, and chronic illnesses, such as diabetes or alcoholism, also predispose patients to acute contagious osteomyelitis.^{73,74}

Acute hematogenous osteomyelitis is a blood-borne infection that generally results from *S. aureus* infection (80%)³ and occurs mostly in infants; children (in the metaphysis of growing long

bones); or patients undergoing long-term IV therapy, hyperalimentation, hemodialysis, or corticosteroid or antibiotic therapy. Patients who are malnourished, obese, or diabetic, or who have chronic joint disease, are also susceptible to acute hematogenous osteomyelitis.^{72,73}

Clinical presentation of both types of acute osteomyelitis includes (1) delayed onset of pain, (2) tenderness, (3) swelling, and (4) warmth in the affected area. Fever is present with hematogenous osteomyelitis. The general treatment course for acute osteomyelitis is early and aggressive administration of the appropriate antibiotics to prevent or limit bone destruction.^{3,56,72,73}

Chronic osteomyelitis is an extension of the acute cases just discussed. It results in marked bone destruction, draining sinus tracts, pain, deformity, and the potential for limb loss. Chronic osteomyelitis can also result from infected surgical prostheses or infected fractures. Debridement of dense formations (sequestra) may be a necessary adjunct to the antibiotic therapy. If the infection has spread to the surrounding soft tissue and skin regions, then grafting, after debridement, may be necessary. Good treatment results have also been shown with hyperbaric oxygen therapy for chronic osteomyelitis.^{72,73}



CLINICAL TIP

Clarify weight-bearing orders with the physician when performing gait training with patients who have any form of osteomyelitis. Both upper and lower extremities can be involved; therefore choosing the appropriate assistive device is essential to preventing pathologic fracture.

Skin Infections

Cellulitis, or erysipelas, is an infection of the dermis and the subcutaneous tissue that can remain localized or be disseminated into the bloodstream, resulting in bacteremia (rare). Cellulitis occurs most commonly on the face, neck, and legs and is associated with an increased incidence of lymphedema.⁷⁵

Groups A and G *Streptococcus* and *Staphylococcus aureus* are the usual causative agents for cellulitis and generally gain entry into the skin layers when there are open wounds (surgical or ulcers). Patients who are at most risk for developing cellulitis include those who are postsurgical and immunocompromised from chronic diseases or medical treatment.

The primary manifestations of cellulitis are fever with an abrupt onset of hot, stinging, and itchy skin and painful, red, thickened lesions that have firm, raised palpable borders in the affected areas. Identifying the causative agent is often difficult through blood cultures; therefore localized cultures, if possible collected from open wounds, may be more sensitive in helping to delineate the appropriate antibiotic treatment.^{74,76,77}

Gastrointestinal Infections

Gastroenteritis is a global term used for the inflammation of the digestive tract that is typically a result of infection. Bacterial sources of gastroenteritis are often caused by *Escherichia coli*, *Shigella* (which causes bacterial dysentery), *Clostridium difficile*,

or *Salmonella*. However, most cases of gastroenteritis are caused by viruses. Rotavirus and norovirus are by far the most frequent cause of gastroenteritis; adenovirus and astrovirus also commonly cause gastroenteritis, especially in children. Transmission of both bacterial and viral gastroenteritis is usually through the ingestion of contaminated food, water, or both or by direct and indirect fecal-oral transmission.



CLINICAL TIP

Strict contact and enteric precautions should be observed with patients who have a diagnosis of *C. difficile* (whose spores can persist on fomites and environmental surfaces for months) and norovirus infection because these pathogens are relatively resistant to waterless alcohol-based antiseptics, and they have been associated with frequent surface contamination in hospital rooms and the hands of health care workers.

Of these aforementioned organisms, rotavirus (a double-stranded RNA virus) infection is the most important cause of severe diarrheal disease in young children. Historically, rotavirus has caused 500,000 childhood deaths annually in the world in less-developed countries. In the United States, 50% of gastroenteritis pediatric cases requiring hospitalization or emergency room visits are caused by rotavirus, and the total health and societal costs of rotavirus infections are estimated to exceed \$1 billion per year. Fortunately, the annual pediatric death rate in the United States is relatively low (20 to 60 deaths). Rotavirus is very contagious in that the virus can survive on dry surfaces for up to 10 days and on human hands for up to 4 hours. It also has a low infectious dose (10 or fewer particles) and the infected stool can contain up to 10^{11} particles per gram that are present before and up to 2 weeks after the onset of symptoms. Because of its highly contagious nature, it is estimated that for every 4 children admitted to the hospital with a rotavirus infection, 1 additional child acquires it as an HAI. Rotavirus infections also may be transmitted to adults who are around infected children, immunocompromised individuals, and older adults in nursing homes. Fortunately, the newly developed second-generation rotavirus vaccines have proven to be effective and have fewer serious side effects (e.g., intussusception [intestinal invagination]).⁷⁸⁻⁸¹

Norovirus (formally known as Norwalk virus, calicivirus, or small round-structured viruses) is a single-stranded positive sense RNA virus and is the most common cause of nonbacterial gastroenteritis worldwide. These outbreaks occur where groups of individuals gather, including nursing homes, hospitals, restaurants, and cruise ships. Like the rotavirus, norovirus is very contagious (<10 particles can cause infection) and can survive for up to 4 weeks in a dried state at room temperature. In



CLINICAL TIP

Norovirus and rotavirus can be transmitted through aerosolization, so health care workers should wear a mask when disposing of infected vomit and feces.

hospitals the most common contaminated sites include toilet tops, door handles, and telephone receivers, and contaminated fingers can spread the norovirus to up to seven clean surfaces.

Research has shown that 1 minute of hand washing with soap and water followed by rinsing the hands for 20 seconds, then drying them with a disposable towel completely removes norovirus from hands contaminated with infected stools. Unlike for rotavirus, there is no fully developed vaccine for norovirus, although vaccines for norovirus are in early stages of development.⁸²⁻⁸⁴

The primary manifestations of any form of gastroenteritis are crampy abdominal pain, nausea, vomiting, and diarrhea, all of which vary in severity and duration according to the type of infection. Gastroenteritis is generally a self-limiting infection, with resolution occurring in 3 to 4 days. However, patients in the hospital setting with reduced immunity can have longer periods of recovery, with dehydration being a primary concern.^{16,56,85}

Management of acute gastroenteritis may include the following^{16,56}:

- Antiinfective agents
- IV fluid and electrolyte replacement
- Antiemetic agents (if nausea and vomiting occur)

Immune System Infections

Human Immunodeficiency Virus Infection

Two types of HIV exist: HIV-1 and HIV-2, with HIV-1 being the more prevalent and the one discussed here. It is a retrovirus, occurring in pandemic proportions, that primarily affects the function of the immune system. Eventually, however, all systems of the body become affected directly, such as the immune system, or indirectly, as in the cardiac system, or through both methods, as occurs in the nervous system. The virus is transmitted in blood, semen, vaginal secretions, and breast milk through sexual, perinatal, and blood or blood-product contact. Proteins on the surface of the virus attach to CD4+ receptors, found primarily on T4 lymphocytes.⁸⁶ Other types of cells found to house the virus include monocytes, macrophages, uterine cervical cells, epithelial cells of the gastrointestinal tract, and microglia cells.⁸⁶

On entering the cell, the viral and cellular DNA combine, making the virus a part of the cell. The exact pathogenesis of cellular destruction caused by HIV is not completely understood, and several methods of destruction may be entailed. It is known that immediately after initial infection, HIV enters a latent period, or asymptomatic stage, in which viral replication is minimal, but CD4+ T cell counts begin to decline.⁸⁶ Continued reduction results in decreasing immunity, eventually leading to symptomatic HIV, in which diseases associated with the virus begin to appear.⁸⁶ This eventually leads to the onset of AIDS, which the CDC defines as occurring when the CD4+ T-lymphocyte count falls below 200 cells/ μ l (reference = 1000 cells/ μ l) or below 14%; when 1 of 26 specific AIDS-defining disorders is contracted, most of which are opportunistic infections; or a combination of these factors.^{87,88}

Six laboratory tests are available to detect HIV infection⁸⁹⁻⁹²:

1. ELISA or enzyme immunoassay test. This procedure tests for the presence of antibodies to HIV proteins in the patient's serum. A sample of the patient's blood is exposed to HIV antigens in the test reagent. If HIV antibodies are identified, it is inferred that the virus is present within the patient.
2. Western blot test. This test detects the presence of antibodies in the blood of two types of HIV viral proteins and is therefore a more specific HIV test. It is an expensive test to perform and is used as a confirmatory tool for a positive ELISA test.
3. Immunofluorescence assay. In this test, the patient's blood is diluted and placed on a slide containing HIV antigens. The slide is then treated with anti-human globulin mixed with a fluorescent dye that will bind to antigen-antibody complexes. If fluorescence is visible when the specimen is placed under a microscope, then HIV antibodies are present in the patient's blood.
4. p24 antigen assay. This test analyzes blood cells for the presence of the p24 antigen located on HIV virions. It can be used to diagnose acute infection, to screen blood for HIV antigens, to determine HIV infection in difficult diagnostic cases, or to evaluate the treatment effects of antiviral agents.
5. PCR for HIV nucleic acid. This highly specific and extremely sensitive test detects viral DNA molecule in lymphocyte nuclei by amplifying the viral DNA. It is used to detect HIV in neonates and when antibody tests are inconclusive.
6. Rapid HIV testing. This highly sensitive and specific test requires a sample of blood, serum, plasma, or oral fluid to detect HIV antibodies. This test can be complete in 20 minutes.

CLINICAL TIP

Any clinician who sustains a needle-stick injury when working with a patient with a suspected HIV infection should have an HIV test. A false-negative HIV test can occur if an individual has not yet developed HIV antibodies. If an individual has had exposure to HIV, he or she should have a repeat HIV test to ensure a true negative result.⁹³

Once HIV has been detected, it can be classified in a number of ways. The Walter Reed staging system has six categories grouped according to the quantity of helper T cells and characteristic signs, such as the presence of an HIV antigen or antibody.⁹⁴ However, a more commonly used classification system was devised by the CDC and was last updated in 1993. In this system, infection is divided into three categories, depending on CD4+ T-lymphocyte counts:

1. Category 1 consists of CD4+ T-lymphocyte counts greater than or equal to 500 cells/ μ l.
 2. Category 2 consists of counts ranging between 200 and 499 cells/ μ l.
 3. Category 3 contains cell counts less than 200 cells/ μ l.
- These groups are then subdivided into A, B, and C, according to the presence of specific diseases.⁸⁷

A major advancement in the medical treatment of HIV has been antiretroviral therapy. This therapy consists of four classes of medications (see Chapter 19, Table 19-37)⁹⁴:

1. Nucleoside analog reverse transcriptase inhibitors, otherwise known as nucleoside analogs
2. Protease inhibitors
3. Nonnucleoside reverse transcriptase inhibitors
4. Fusion inhibitor

Each of these therapies assists in limiting HIV progression by helping to prevent viral replication. This prevention is further increased when the drugs are used in combination in a treatment technique termed *highly active antiretroviral therapy* or HAART.⁹⁴

There is a significant need for more effective and cost-efficient preventions for HIV. The HIV Vaccine Trials Network (HVTN) is an international collaboration working to develop HIV preventive vaccines.⁹⁵

As HIV progresses and immunity decreases, the risk for and severity of infections not normally seen in healthy immune systems increase. These opportunistic infections, combined with disorders that result directly from the virus, often result in multiple diagnoses and medically complex patients. These manifestations of HIV can affect every system of the body and present with a wide array of signs and symptoms, many of which are appropriate for physical therapy intervention. Table 13-4 lists common manifestations and complications of HIV and AIDS and the medications generally used in their management.

Disorders affecting the nervous system include HIV-associated dementia complex, progressive multifocal leukoencephalopathy, primary central nervous system lymphoma, toxoplasmosis, and neuropathies. These manifestations may cause paresis, decreased sensation, ataxia, aphagia, spasticity, altered mental status, and visual deficits.⁹⁶ In the pulmonary system, TB, cytomegalovirus (CMV) and pneumonia can result in cough, dyspnea, sputum production, and wheezing.⁹⁷ In the cardiac system, cardiomyopathy, arrhythmias, and congestive heart failure can cause chest pain, dyspnea, tachycardia, tachypnea, hypotension, fatigue, peripheral edema, syncope, dizziness, and palpitations.⁹⁸

Physical therapy intervention can assist in minimizing the effect of these deficits on functional ability, therefore helping to maximize the independence and quality of life of the individual. However, the course of rehabilitation in HIV-affected individuals can often be difficult owing to coinciding opportunistic infections, an often-rapid downhill disease course, low energy states, and frequent hospitalizations.

Mononucleosis

Mononucleosis is an acute viral disease that has been primarily linked to the Epstein-Barr virus and less commonly to CMV. Mononucleosis is transmitted generally through saliva from symptomatic or asymptomatic carriers (the Epstein-Barr virus can remain infective for 18 months in the saliva).^{16,99}

The disease is characterized by fever, lymphadenopathy (lymph node hyperplasia), and exudative pharyngitis. Splenomegaly, hepatitis, pneumonitis, and central nervous system

TABLE 13-4 Common Complications from HIV and AIDS, and Associated Medical Treatment

Complication	Medication
Cardiomyopathy	May be reversed with reduction or discontinuation of interleukin-2, adriamycin, α 2-interferon, ifosfamide, and foscarnet
Cerebral toxoplasmosis	Trimethoprim-sulfamethoxazole
Coccidioidomycosis	Amphotericin B, fluconazole, or itraconazole
Congestive heart failure	Removal of all nonessential drugs followed by administration of furosemide (Lasix); digoxin; angiotensin-converting enzyme inhibition
Cryptococcal meningitis	Amphotericin B or fluconazole
Cytomegalovirus	Ganciclovir, foscarnet, cidofovir
Distal symmetric polyneuropathy	Pain management using tricyclic antidepressants, gabapentin, and narcotics for severe cases
Herpes simplex	Acyclovir, famciclovir, valacyclovir
Herpes zoster (shingles)	Acyclovir, valacyclovir, famciclovir, foscarnet
HIV-associated dementia complex	Antiretroviral therapy combining at least three drugs, two of which penetrate the blood-brain barrier
Histoplasmosis	Amphotericin B or itraconazole
Kaposi's sarcoma	Radiotherapy, cryotherapy with liquid nitrogen, daunorubicin hydrochloride, or doxorubicin hydrochloride injections
Lymphomas	Chemotherapy: cyclophosphamide, doxorubicin, vincristine, bleomycin, methotrexate, leucovorin
<i>Mycobacterium avium</i> complex	Clarithromycin, rifabutin, ciprofloxacin, ethambutol
Oral hairy leukoplakia	Acyclovir if symptoms present
<i>Pneumocystis jirovecii</i> pneumonia	Trimethoprim-sulfamethoxazole, dapsone, clindamycin, pentamidine isethionate
Progressive multifocal leukoencephalopathy	Antiretroviral therapy, acyclovir, IV cytosine, adenosine-araboside, interferon-alphas
Pulmonary hypertension	Low-flow O ₂ if hypoxia present, vasodilators, including nitroglycerin, hydralazine, nifedipine, lisinopril, and prostaglandin E
Toxic neuronal neuropathy: neuropathy caused by certain medications	May be reversed with discontinuation or reduction in the following: zalcitabine, didanosine, and stavudine
Tuberculosis	Four-drug regimen: isoniazid, rifampin, pyrazinamide, and ethambutol

Data from Zwolski K: Viral infections. In Kirton CA, Talotta D, Zwolski K, editors: Handbook of HIV/AIDS nursing, St Louis, 2001, Mosby, pp 303, 310-311, 313, 315; Cheitlin MD: Cardiovascular complications of HIV infection. In Sande MA, Volberding PA, editors: The medical management of AIDS, Philadelphia, 1999, Saunders, pp 278, 280; Boss BJ, Farley JA: Alterations in neurologic function. In Heuther SE, McCance KL, editors: Understanding pathophysiology, ed 2, St Louis, 2000, Mosby, pp 403-406; Smith L: Management of bacterial meningitis: new guidelines from the IDSA, Am Fam Physician 71(10), 2005; Rhuda SC: Nursing management, musculoskeletal problems. In Lewis SM, Heitkemper MM, Dirksen SR, editors: Medical-surgical nursing, assessment and management of clinical problems, ed 5, St Louis, 2000, Mosby, pp 1795-1798; McCance KL, Mourad LA: Alterations in musculoskeletal function. In Heuther SE, McCance KL, editors: Understanding pathophysiology, ed 2, St Louis, 2000, Mosby, pp 1046-1048; Rowland BM: Cellulitis. In Boyden K, Olendorf D, editors: Gale encyclopedia of medicine, Farmington Hills, MI, 1999, Gale Group, p 616.

AIDS, Acquired immunodeficiency syndrome; HIV, human immunodeficiency virus; IV, intravenous.

involvement may occur as rare complications from mononucleosis. The infection is generally self-limiting in healthy individuals, with resolution in approximately 3 weeks without any specific treatment.^{16,99}

If management of mononucleosis is necessary, it may include the following^{16,99,100}:

- Corticosteroids in cases of severe pharyngitis
- Adequate hydration
- Bed rest during the acute stage
- Saline throat gargle
- Aspirin or acetaminophen for sore throat and fever

Cytomegalovirus Infection

CMV is a member of the herpesvirus group that can be found in all body secretions, including saliva, blood, urine, feces, semen, cervical secretions, and breast milk. CMV

infection is a common viral infection that is asymptomatic or symptomatic. CMV infection can remain latent after the initial introduction into the body and can become opportunistic at a later point.

If CMV infection is symptomatic, clinical presentation may be a relatively benign mononucleosis in adults, or in patients with HIV infection, manifestations such as pneumonia, hepatitis, encephalitis, esophagitis, colitis, and retinitis can occur.

CMV is usually transmitted by prolonged contact with infected body secretions, as well as congenitally or perinatally.^{16,101}

Management of CMV infection may include the following^{16,101}:

- Antiviral agents
- Corticosteroids

- Immune globulins
- Blood transfusions for anemia or thrombocytopenia
- Antipyretics

Toxoplasmosis

Toxoplasmosis is a systemic protozoan infection caused by the parasite *Toxoplasma gondii*, which is primarily found in cat feces. Transmission can occur from three mechanisms: (1) eating raw or inadequately cooked infected meat or eating uncooked foods that have come in contact with contaminated meat; (2) inadvertently ingesting oocysts that cats have passed in their feces, either in a cat litter box or outdoors in soil (e.g., soil from gardening or unwashed fruits or vegetables); and (3) transmission of the infection from a woman to her unborn fetus. Fetal transmission of *T. gondii* can result in mental retardation, blindness, and epilepsy.¹⁰²

Clinical manifestations can range from subclinical infection to severe generalized infection, particularly in immunocompromised individuals, and to death.

The primary way to treat toxoplasmosis is through prevention by safe eating habits (thoroughly cooking meats, peeling and washing fruits and vegetables) and minimizing contact with cat feces when pregnant, along with keeping the cat indoors to prevent contamination.¹⁰²

Sepsis

Sepsis is a general term that describes three progressive infectious conditions: bacteremia, septicemia, and shock syndrome (or septic shock).¹⁶

Bacteremia is a generally asymptomatic condition that results from bacterial invasion of blood from contaminated needles, catheters, monitoring transducers, or perfusion fluid. Bacteremia can also occur from a preexisting infection from another body site. Patients with prosthetic heart valves may need to take prophylactic antibiotics for dental surgery because the bacteremia may progress to endocarditis. Bacteremia can resolve spontaneously or progress to septicemia.

Septicemia is a symptomatic extension of bacteremia throughout the body, with clinical presentations that are representative of the infective pathogen and the organ system(s) involved. Sites commonly affected are the brain, endocardium, kidneys, bones, and joints. Renal failure and endocarditis may also occur.

Shock syndrome is a critical condition of systemic tissue hypoperfusion that results from microcirculatory failure (i.e., decreased blood pressure or perfusion). Bacterial damage of the peripheral vascular system is the primary cause of the tissue hypoperfusion.

Management of sepsis may include any of the following¹⁶:

- Removal of suspected infective sources (e.g., lines or tubes)
- Antiinfective agents
- Blood pressure maintenance with adrenergic agents and corticosteroids
- IV fluids
- Blood transfusions

- Cardiac glycosides
- Supplemental oxygen, mechanical ventilation, or both
- Anticoagulation

Management

Medical Intervention

Management of the various infectious diseases discussed in this chapter is described in the specific sections of respective disorders. Chapter 19 (Table 19-34, Antibiotics; Table 19-35, Antifungal Agents; Table 19-36, Antitubercular Agents; Table 19-37, Antiretroviral Medications; and Table 19-38, Antiviral Medications) also lists common antiinfective agents used in treating infectious diseases.

Lifestyle Management

The critical importance of encouraging healthy lifestyles to combat disease is indicated by the National Prevention and Health Promotion Strategy that was announced in the summer of 2011. The objective of this strategy is to “move the nation away from a health care system focused on sickness and disease to one focused on wellness and prevention.”¹⁰³ The American Physical Therapy Association’s president has encouraged physical therapists to support this national prevention initiative by “expanding quality preventative services in both clinical and community settings, empowering people to make healthful choices, and eliminating health disparities” and to become “leaders in their communities to advance these directions and priorities.”¹⁰⁴ Although the physical therapist may have limited treatment options emphasizing prevention and wellness during the acute care stay, he or she has greater opportunities to effect meaningful lifestyle change in other settings such as in nursing homes and home health. At a minimum, the physical therapist can play a key role in all health care settings in helping patients understand the link between lifestyle and infectious disease. These important links, which may be poorly understood by the typical patient, are discussed in this section.

Many of the same lifestyle and nutrition factors that can delay wound healing (see Chapter 12) also affect the immune system and the infection rate. To have an optimally functioning immune system, one should eat plenty of fresh fruits and vegetables as well as foods rich in fiber. Also, it is important to obtain adequate amounts of the micronutrients zinc, selenium, iron, copper, vitamins A, C, E, and B₆, and folic acid. Vitamin D, which is produced by exposure to sunlight, is known to activate one’s innate immunity (i.e., regulatory T cells) by the production of antimicrobial peptides. Excess sugar also decreases the ability of white blood cells to destroy bacteria (leukocytic phagocytosis). Moreover, a healthy immune system is promoted by not only eating proper foods, but also by staying well hydrated, which is a key consideration in combating septic shock.¹⁰⁵⁻¹¹¹

Exposure to fresh and unpolluted air benefits the immune system. It is a well-studied fact that the higher the ventilation rate (amount of outdoor air circulated per unit time), the lower

the infection rate of airborne diseases such as measles, TB, influenza, and SARS. The cross-infection problem of the 2002-2003 SARS epidemic was particularly evident where people congregated, such as in airplanes, buses, and hospitals. This would imply a strong benefit for exposing patients to as much fresh air as medically prudent, which is reminiscent of the philosophy behind the “open-air treatment” TB hospitals of the last century.^{112,113}

When one obtains the proper balance of exercise and rest, it helps the immune system fight off infection. Exercise and adequate rest are key factors in promoting a healthy psychological state, which also reduces the negative effects of stress (e.g., high levels of cortisol) on the immune system. However, it should be mentioned that the beneficial effects of resistive exercise (as opposed to cardiovascular exercise) on immunity are less clear, though excessive cardiovascular exercise may lead to immunosuppression. The key role that the crucial rest-promoting hormone melatonin plays in influencing our circadian rhythm (sleep-wake cycle) and immune system (specifically T cell populations) is still being unraveled. Melatonin’s peak production occurs at night, which is the inverse of another key immunoregulatory hormone, vitamin D. In order to maximize melatonin levels, which promote sleep efficiency and restfulness, one should exercise regularly, be exposed to natural light (sunlight) especially early in the day, minimize exposure to artificial light at night, and go to bed 2 to 3 hours before midnight in a completely dark room. It is important to follow this advice every day because melatonin has a short half-life and thus must be produced every 24 hours.^{110,114-119}

Alcohol exposure has a well-known immunosuppression effect, which includes negative impacts on lymphocyte activation, cytokine production by macrophages and T cells, and neutrophil function. This results in increased susceptibility to infection and reduces the body’s ability to heal after injury. People who smoke and those who are exposed (especially children) to passive or environmental tobacco smoke (ETS) are at greater risk of impairing their immune system, which can cause infections such as influenza and TB for adults and serious respiratory tract infection and pneumonia for children. Caffeine is largely antiinflammatory in nature and thus has an overall negative effect on the immune system. Specifically, caffeine suppresses lymphocyte function, antibody production, and neutrophil and monocyte chemotaxis. Illicit drug users also have well-documented higher infection rates involving bacteria, viruses, fungi, and protozoans. These rates are even higher in injection drug users.¹²⁰⁻¹²³

Finally, obesity has now been associated with increased infection risk. Increased infection has been observed in obese patients with conditions as diverse as urinary tract infection (UTI), influenza, hepatitis C, and a history of total hip arthroplasty. With obesity rates increasing throughout the world, the exact mechanism of the link between obesity and infection warrants more study.¹²⁴⁻¹²⁷

Physical Therapy Intervention

The following are general physical therapy goals and guidelines to be used when working with patients who have infectious

disease processes, as well as disorders of altered immunity. These guidelines should be adapted to a patient’s specific needs.

Goals

The primary physical therapy goals in this patient population are similar to those of patient populations in the acute care setting: (1) to optimize the patient’s functional mobility, (2) to maximize the patient’s tolerance and endurance, (3) to maximize ventilation and gas exchange in the patient who has pulmonary involvement, and, when appropriate, (4) to educate the patient in proper lifestyle management (see previous section).

Guidelines for Physical Therapy Intervention

General physical therapy guidelines include, but are not limited to, the following:

1. The best modes of preventing the transmission of infectious diseases are to adhere to the standard precautions established by the CDC and to follow proper hand-washing techniques (Box 13-2).
 - a. Facilities’ warning or labeling systems for biohazards and infectious materials may vary slightly.
 - b. Be sure to check the patient’s medical record or signs posted on doors and doorways for indicated precautions.
 - c. Table 13-3 provides an outline of the types of personal protective equipment that should be worn with specific precautions.
2. Personally follow and also educate patients concerning proper coughing and sneezing hygiene etiquette in order to prevent the spread of disease and illness.
 - a. Cover the mouth and nose with a tissue when coughing or sneezing.
 - b. Put the used tissue in a waste basket.

BOX 13-2 Proper Hand-Washing Technique

Hand washing with soap and water is the best method to remove pathogens, including highly contagious pathogens (e.g., norovirus, *Clostridium difficile* spores), from your hands.

1. Wet your hands with clean running water (warm or cold) and apply soap.
2. Rub your hands together to make a lather and scrub them well. Be sure to scrub the backs of your hands, between your fingers, and under your nails.
3. Continue rubbing your hands for at least 20 seconds (as previously mentioned, some pathogens such as norovirus require a longer time of at least 60 seconds to remove stool contamination from hands).
4. Rinse your hands well under running water (stool-contaminated norovirus hands should be rinsed for at least 20 seconds).
5. Dry your hands using a clean disposable towel or air dry.

If soap and water are not available, use an alcohol-based hand sanitizer that contains at least 60% alcohol (continue to rub the sanitizer over all hand and finger surfaces until dry). Alcohol-based hand sanitizers can quickly reduce the number of pathogens, but do not remove all pathogen types (e.g., norovirus, *Clostridium difficile* spores).

- c. If no tissue is available, cough or sneeze into the upper sleeve and not into the hands.
 - d. Wash the hands after coughing or sneezing (see Box 13-2 on previous page).
3. The danger of pathogen aerosolization during common hygiene activities and physical therapy treatment is often not fully recognized.
 - a. Flushing the toilet (even with the lid down) causes aerosolization of pathogens that is greatest with the first flush, and then diminishes with each subsequent flush.
 - b. Aerosolization danger is greatest when the patient has diarrhea (as contrasted to a normal stool) and/or vomits into the toilet.
 - c. Gastroenteritis viral pathogens are especially easy to spread in the foregoing manner because each gram of feces can contain up to 10^{11} virus particles.
 - d. Pulsatile lavage is a common wound physical therapy modality that can cause aerosolization of pathogens.
 - e. In addition to the therapist wearing appropriate PPE during pulsatile lavage, patients receiving treatment should wear surgical masks, and all IV lines and other wounds should be covered during treatment. The procedure should be performed in a private room, with minimal equipment and supplies. The room should be thoroughly cleaned and disinfected after each procedure.
 4. Patients who have infectious processes have an elevated metabolic rate, which will most likely manifest itself as a high resting heart rate. As a result, the activity intensity level should be modified, or more frequent rest periods should be incorporated during physical therapy treatment to enhance activity tolerance.
 - a. Patients with infectious processes will also be prone to orthostatic hypotension, hypotension with functional activities, or both as a result of the vasodilation occurring from the inflammation associated with infection.
 - b. Therefore slow changes in positions, especially from recumbent to upright positions, and frequent blood pressure monitoring are essential to promoting tolerance for functional activities.
 5. Monitoring the temperature curve and WBC count of patients with infectious processes helps to determine the appropriateness of physical therapy intervention.
 - a. During an exacerbation or progression of an infection process, rest may be indicated.
 - b. Clarification with the physician or nurse regarding the type of intended physical therapy intervention is helpful in making this decision.

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APPENDIX 13A DISORDERS OF ALTERED IMMUNITY

Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disease with strong genetic predisposition. There is also evidence suggesting risk factors that can trigger the onset of this disease, such as physical or emotional stress, pregnancy, sulfa antibiotics, and environmental factors, such as sun exposure. Women who are African-American, Asian, or Native American, ages 20 to 40 years, are more susceptible than men in acquiring this disease. SLE is characterized by a systemic, remitting-and-relapsing clinical presentation.¹⁻⁴

The primary laboratory test for diagnosis of SLE is as antinuclear antibody titer.⁵ Diagnosis of SLE is confirmed if a patient has 4 of the following 11 manifestations of SLE: malar rash, discoid rash (individual round lesions), photosensitivity, oral ulcers, arthritis, serositis, renal disorder, neurologic disorder, hematologic disorder, immunologic disorder, and the presence of antinuclear antibodies.³

Prognosis for 10-year survival after diagnosis is 90%. The most common cause of death in SLE is renal failure, and the second most common is central nervous system dysfunction.¹⁻³

Clinical presentation of SLE may include the following¹⁻⁴:

- Arthritis or arthralgias (stiffness and pain in hands, feet, and large joints)
- Red, warm, and tender joints
- Butterfly (malar) rash on face
- Fever, fatigue, anorexia, and weight loss
- Pleurisy, pericarditis
- Headache, seizures
- Hemolytic anemia, thrombocytopenia, leukopenia
- Renal disease or failure

Management of SLE may consist of nonsteroidal antiinflammatory drugs; glucocorticoids; immunosuppressive agents

(cyclophosphamide); dialysis; and renal transplantation in severe cases.^{1,2,4,6}

Sarcoidosis

Sarcoidosis is a systemic granulomatous disorder that primarily affects women and nonwhite adults in the third decade of their life. The definitive etiology is unknown, although an autoimmune process that is environmentally triggered is the generally agreed-on hypothesis. Sarcoidosis may present as acute or chronic and have periods of progression and remission.⁶⁻¹⁰

Multiple body systems may be affected by sarcoidosis. The lungs are the primary organs affected, with dyspnea, dry cough, and chest pain being common symptoms. Pulmonary involvement can be staged according to the following radiographic evidence^{7,8}:

- Stage 0: No radiographic abnormalities
- Stage I: Bilateral hilar lymphadenopathy
- Stage II: Bilateral hilar adenopathy and parenchymal infiltration
- Stage III: Parenchymal infiltration without hilar adenopathy
- Stage IV: Advanced fibrosis with evidence of honeycombing, hilar retraction, bullae, cysts, and emphysema

Other systems of the body can be affected as well, with symptoms including the following:

- Eye and skin lesions
- Fever, fatigue, and weight loss
- Hepatosplenomegaly
- Hypercalcemia, anemia, and leukopenia
- Arthralgia, arthritis

Management of sarcoidosis usually consists of corticosteroid therapy, ranging from topical to oral administration. In addition, cytotoxic agents (methotrexate and azathioprine),

antimalarial agents (chloroquine and hydroxychloroquine), and nonsteroidal antiinflammatory drugs may be used. In severe cases of pulmonary disease, single and double lung transplantation may be performed.⁶⁻⁸

Amyloidosis

Amyloidosis is a group of disorders characterized by deposition of amyloid (a type of protein) in various tissues and organs. Amyloidosis is classified according to protein type and tissue distribution and affects men more than women, between the ages of 60 and 70 years.¹¹ Clinical signs and symptoms are representative of the affected areas, with common manifestations including¹¹:

- Fatigue
- Shortness of breath
- Edema
- Paresthesia
- Weight loss
- Diarrhea
- Peripheral neuropathy

In general, the deposition of protein in these areas will result in firmer, less distensible tissues that compromise organ function. Management of amyloidosis consists of controlling any primary disease process that may promote deposition of amyloid into the tissues.¹¹

Rheumatoid Arthritis

Rheumatoid arthritis (RA) is an autoimmune disease characterized by uncontrolled proliferation of synovial tissue.¹² It is a chronic disease involving systemic inflammation, with periods of exacerbation and remission.¹³ The etiology is not fully understood, but it is believed there is a correlation between environmental and genetic factors. Females have an increased risk of

developing RA, as do individuals with a positive family history, silicate exposure, or smoking history.¹²

There are two forms of RA: juvenile idiopathic arthritis (JIA) and adult RA. JIA occurs most often during the toddler and early adolescent developmental phases, whereas adult RA has a peak onset in the third and fourth decades of life. Both forms of RA have an inflammatory component in disease development and have similar medical management strategies.¹³

With RA, an interaction between autoantibodies (rheumatoid factors) and immunoglobulins initiates the inflammatory process, which involves an increased infiltration of leukocytes from the peripheral circulation into the synovial joint. Pannus—a destructive granulation tissue that dissolves periarticular tissues—can develop, ultimately leading to joint destruction.¹³

Clinical manifestations of RA can include articular and extra-articular symptoms. Most body systems may be involved, including pulmonary, cardiovascular, neurologic, and gastrointestinal.¹³ The joints most commonly affected by RA are those with the highest ratio of synovium compared to articular cartilage, including the wrist, proximal interphalangeal, and metacarpophalangeal joints.¹² Common deformities associated with RA are ulnar drift, swan-neck, and boutonniere deformities.¹³ Other symptoms include anorexia, low-grade fever, fatigue, and malaise.¹² Osteoarthritis (OA), also called degenerative joint disease, may result in joint changes and deformation. [Table 13A-1](#) compares OA and RA.¹³

Diagnosis of rheumatoid arthritis can be made through the following seven criteria established by the American Rheumatism Association¹²:

1. Morning stiffness
2. Arthritis of three or more joint areas
3. Hand joint involvement
4. Symmetric arthritis
5. Rheumatoid nodules
6. Serum rheumatoid factor positive
7. Radiographic changes of the wrist and hand joints

TABLE 13A-1 Comparison of Osteoarthritis and Rheumatoid Arthritis

	Osteoarthritis	Rheumatoid Arthritis
Onset	Majority of adult age > 65 Gradual onset	Adult: ages 25-50 Sudden onset
Incidence	12% U.S. adults	1%-2% U.S. adults
Gender	Age < 45: predominantly males Age > 45: predominantly females	Female: male ratio 3:1
Etiology	Inflammatory response Genetic, environmental factors	Autoimmune process with multifactorial components
Manifestations	Unilateral joint involvement Affects: spine, hips, knees, feet, hands Inflammation present in 10% of cases Brief morning stiffness	Symmetric, bilateral joint involvement Affects any joint, predominantly upper-extremity joints Inflammation present in most cases Prolonged morning stiffness
Systemic symptoms	None	Fatigue, malaise, weight loss, fever
Lab values	ESR: mildly-moderately increased Rheumatoid factor: absent	ESR: increased during inflammatory process (exacerbation) Rheumatoid factor: present but not diagnostic for disease

ESR, Erythrocyte sedimentation rate.

Laboratory diagnostic tests include a complete blood cell count with differential, rheumatoid factor, and erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP).¹¹

Management of RA may include the following^{12,13}:

- Nonsteroidal antiinflammatory drugs
- Glucocorticoids
- Disease-modifying antirheumatic drugs (DMARDs) (Chapter 19, Table 19-13)
- Pain management

- Joint protection
- Control of systemic complications

CLINICAL TIP

Modification of an assistive device may be necessary based on the patient's wrist and hand function. For example, a platform walker may be more appropriate than a standard walker, and Lofstrand crutches may be more appropriate than axillary crutches.

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