CHAPTER 12 Nursing Care of Clients with Infections

LEARNING OUTCOMES

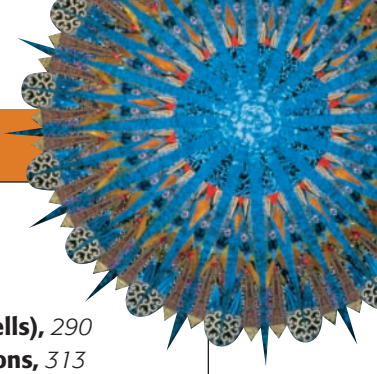
- Discuss the components and functions of the immune system and the immune response.
- Compare antibody-mediated and cell-mediated immune responses.
- Describe the pathophysiology of wound healing, inflammation, and infection.
- Identify factors responsible for nosocomial infections.
- Use the nursing process as a framework to provide individualized care to clients with inflammation and infection.

CLINICAL COMPETENCIES

- Apply universal precautions, particularly hand washing, to prevent the spread of infection within the client, to other clients in the facility, and to members of the interdisciplinary team and visitors.
- Assess for signs and symptoms of inflammation or infection.
- Determine priority nursing diagnosis, based on assessment data, to select and implement individualized nursing interventions for clients with infections.
- Integrate interdisciplinary care into care of clients with infection.
- Promote therapeutic levels of anti-inflammatory and anti-infective medication through prompt administration and client and family teaching.
- Be alert for hypersensitivities to anti-infectives prior to administering and during administration.
- Provide teaching for clients with inflammation or an infection and their families.
- Revise plan of care as needed to provide effective care to promote, maintain, or restore functional health patterns to clients with infections.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone



KEY TERMS

acquired immunity, *298*
active immunity, *298*
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antibodies, *291*
antibody-mediated (humoral) immune response, *291*
antigen, *290*
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The human body is continually threatened by foreign substances, infectious agents, and abnormal cells. The immune system is the body's major defense mechanism against infectious organisms and abnormal or damaged cells. Recent years have seen the emergence of resistant microorganisms such as methicillin-resistant *Staphylococcus aureus* and altered strains of familiar diseases, such as multiple-drug-resistant tuberculosis. New diseases have also emerged, such as Lyme disease, *Clostridium difficile,* and human immunodeficiency virus (HIV).

A thorough knowledge of the immune system increases understanding of the local and systemic inflammatory response, resistance to infectious disease, and the importance of immunization. This foundation can help the nurse teach clients and families to follow recommended treatment regimens, to promote and maintain health, and to prevent disease. In addition, the nurse can prescribe appropriate rehabilitative measures, such as increased rest and attention to optimal nutrition.

OVERVIEW OF THE IMMUNE SYSTEM

The immune system is a complex and intricate network of specialized cells, tissues, and organs. Cells of the immune system seek out and destroy damaged cells and foreign tissue, yet recognize and preserve host cells (Porth, 2005). The immune system performs the following functions:

- Defends and protects the body from infection by bacteria, viruses, fungi, and parasites.
- Removes and destroys damaged or dead cells.
- Identifies and destroys malignant cells, thereby preventing their further development into tumors.

The immune system is activated by minor injuries, such as small lacerations or bruises, or by major injuries, such as burns, surgeries, and systemic diseases (e.g., pneumonia). The response of the immune system may be nonspecific or specific. Nonspecific responses prevent or limit the entry of invaders into the body, thereby limiting the extent of tissue damage and reducing the workload of the immune system. Inflammation is a nonspecific response activated by both minor and major injuries. When the inflammatory process is unable to destroy invading organisms or toxins, a more specific response, called the immune response, is activated.

Immune System Components

The immune system consists of molecules, cells, and organs that produce the immune response (Table 12–1). These components may be involved in the nonspecific inflammatory response, the specific immunologic response, or both.

Leukocytes

Leukocytes, or white blood cells (WBCs), are the primary cells involved in both nonspecific and specific immune system responses. Like all blood cells, leukocytes derive from stem cells, the hemocytoblasts, in the bone marrow (Figure 12–1 ■). Unlike red blood cells (RBCs), which are confined to the circulation, leukocytes use the circulation to transport themselves to the site of an inflammatory or immune response. As the mobile units of the immune system, leukocytes detect, attack, and destroy anything that is recognized as "foreign." They are able to move through tissue spaces, locating damaged tissue and infection by responding to chemicals released by other leukocytes and damaged tissue.

The normal number of circulating leukocytes is 4,500 to 10,000 cells per cubic millimeter (mm^3) of blood. Many more leukocytes are marginated; that is, they adhere to vascular epithelial cells along the vessel walls, in other tissue spaces, or in the lymph system. In the presence of an attack such as an infection, additional WBCs are released from the bone marrow, leading to **leukocytosis**, a WBC count of greater than 10,000/mm^3. As WBCs move out of the bone marrow into the blood, the bone marrow increases its production of additional leukocytes. A decrease in the number of circulating leukocytes, known as **leukopenia**, occurs when bone marrow activity is suppressed or when leukocyte destruction increases.

Leukocytes are divided into three major groups: granulocytes, monocytes, and lymphocytes. The granulocytes and monocytes derive from the myeloid stem cells of the bone marrow and are instrumental in the inflammatory response. Lymphocytes derive from the lymphoid stem cells of the bone marrow and are the primary cells involved in the specific immune response. In laboratory tests, the WBC count indicates the total number of circulating leukocytes. The WBC differential identifies the portion of the total represented by each type of leukocyte.

TABLE 12–1 **Cells and Tissues of the Immune System**

COMPONENT	LOCATION	FUNCTION
Leukocytes		
Granulocytes		
Neutrophils	Circulation	Phagocytosis and chemotaxis
Eosinophils	Circulation, respiratory tract, and gastrointestinal tract	Phagocytosis Protection against parasites Involved in allergic response
Basophils	Circulation	Release of chemotactic substances
Monocytes and macrophages	Circulation (monocytes) and body tissue, such as skin (histocytes), liver (Kupffer cells), alveoli, spleen, tonsils, lymph nodes, bone marrow, brain	Trapping and phagocytizing of foreign substances and cellular debris Secretion of interleukin-1 to stimulate lymphocyte growth
Lymphocytes		
T cells (mature in thymus gland)	Circulation, lymph system, tissues	Activation of T and B cells Control of viral infections and destruction of cancer cells Involved in hypersensitivity reactions and graft tissue rejection
B cells (mature in bone marrow)	Circulation, spleen	Production of antibodies (immunoglobulins) to specific antigens
NK (natural killer) cells	Circulation	Cytotoxic; killing of tumor cells, fungi, viral-infected cells, and foreign tissue
Lymphoid Tissues		
Primary or central lymphoid structures	Bone marrow and thymus gland	Production of immune cells; sites for cell maturation
Secondary or peripheral lymphoid structures	Lymph nodes, spleen, tonsils, intestinal lymphoid tissue, lymphoid tissue in other organs	Sites for activation of immune cells by antigens

GRANULOCYTES Granulocytes constitute 60% to 80% of the total number of normal blood leukocytes. Their cytoplasm has a granular appearance, and their nuclei are distinctively multilobular (see Figure 12–1). Granulocytes have a short life span, measured in hours to days, compared to the life span of monocytes, which is measured in months to years. Granulocytes play a key role in protecting the body from harmful microorganisms during acute inflammation and infection. There are three types of granulocytes: neutrophils, eosinophils, and basophils.

Neutrophils, also called polymorphonuclear leukocytes (PMNs or polys), are the most plentiful of the granulocytes, constituting 55% to 70% of the total number of circulating leukocytes. Neutrophils are *phagocytic* cells, responsible for engulfing and destroying foreign agents, particularly bacteria and small particles. Neutrophils are the first phagocytic cells to arrive at the site of invasion, drawn by chemicals released by damaged tissue and invading organisms.

Neutrophils are produced in the bone marrow and released into the circulation when they mature. Segmented neutrophils (or segs) are mature forms, and usually account for about 55% of total leukocytes. *Bands* are immature neutrophils and usually comprise 5% of leukocytes. As neutrophils mature, their nucleus changes from round to kidney bean shaped (banded) and then the nucleus separates into small, attached segments, thus the designations "banded" versus "segmented" neutrophils. It takes about 10 days for a neutrophil to mature and be released into the circulation. Once released, neutrophils have a circulating half-life of 6 to 10 hours. They cannot replicate and must be replaced constantly to maintain adequate numbers in the circulation. They do not return to the bone marrow.

Eosinophils account for 1% to 4% of the total number of circulating leukocytes. They mature in the bone marrow in 3 to 6 days before being released into the circulation. Eosinophils have a circulating half-life of 30 minutes and a tissue half-life of 12 days. They too are phagocytic cells, but are less efficient at this process than neutrophils. Eosinophils are found in large numbers in the respiratory and gastrointestinal tracts, where they are thought to be responsible for protecting the body from parasitic worms, including tapeworms, flukes, pinworms, and hookworms. Eosinophils surround the parasite and release toxic enzymes from their cytoplasmic granules. The parasite, although too large to be phagocytized, is destroyed. Eosinophils are also involved in a hypersensitivity response, inactivating some of the inflammatory chemicals released during the inflammatory response.

Basophils constitute about 0.5% to 1% of the circulating leukocytes. These cells are not phagocytic. Granules within basophils contain proteins and chemicals such as heparin, histamine, bradykinin, serotonin, and a slow-reacting substance of anaphylaxis (leukotrienes). These substances are released into the bloodstream during an acute hypersensitivity reaction or stress response.

MONOCYTES, MACROPHAGES, AND DENDRITIC CELLS These cells are the mediators of immunity. They recognize foreign matter (from molecules to cells) and initiate immune responses. *Monocytes* are the largest of the leukocytes and constitute 2% to 3% of circulating leukocytes. After their release from the bone marrow,

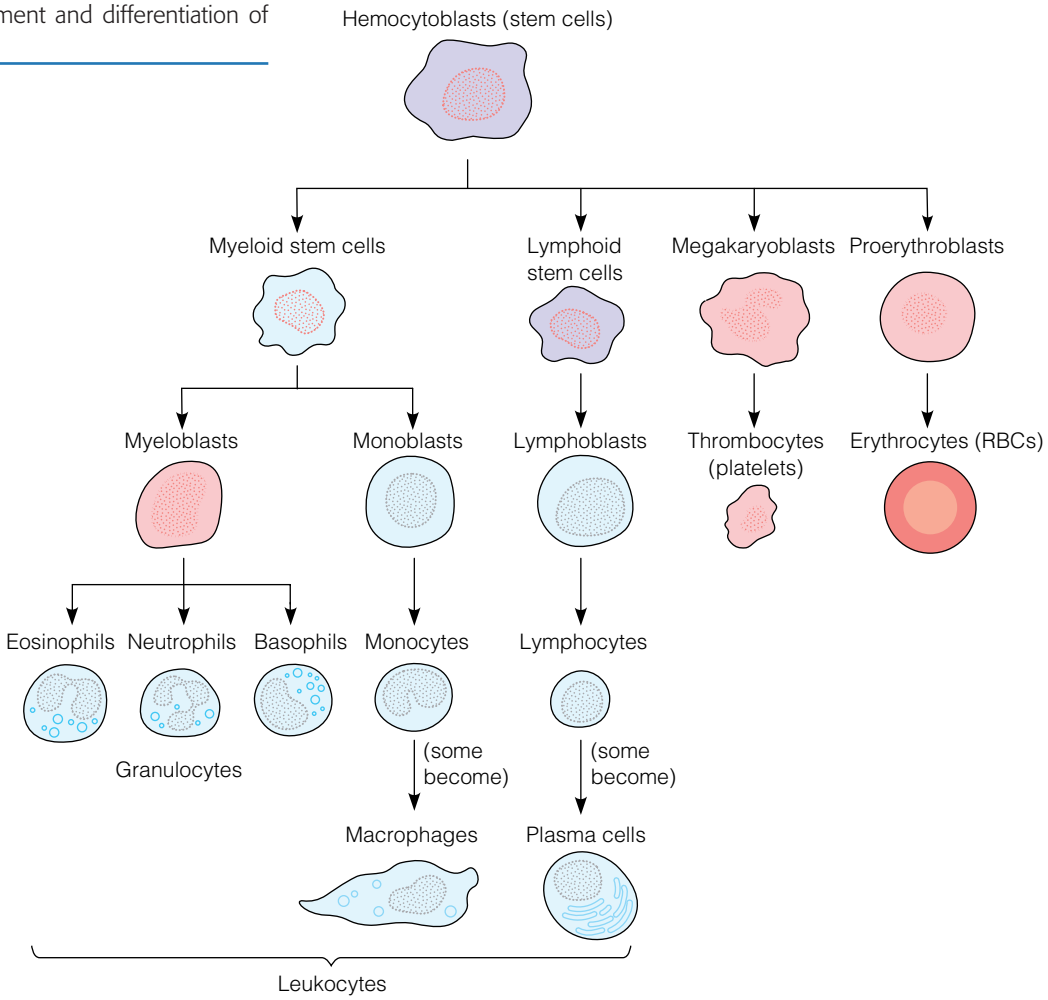


Figure 12–1 ■ The development and differentiation of leukocytes from hemocytoblasts.

monocytes circulate in the serum for 1 to 2 days. They then migrate to various tissues throughout the body, attaching themselves to the tissues, where they remain for months or even years until they are activated. Monocytes mature into **macrophages** after settling into the tissues. Once they have migrated and matured, macrophages are differentiated by the tissues in which they reside. *Histiocytes* are tissue macrophages in loose connective tissue, *Kupffer cells* are found in the liver, *alveolar macrophages* in the lungs, and *microglia* in the brain. Tissue macrophages are also found in the spleen, tonsils, lymph nodes, and bone marrow. Dendritic cells are star-shaped cells that originate in both the myeloid and the lymphoid cell lines. Langerhans cells are specialized dendritic cells in the skin. Monocytes, macrophages, and dendritic cells are antigen-presenting cells (APCs), which activate immune responses in both B and T lymphocytes.

Monocytes, macrophages, and dendritic cells are actively phagocytic, with the capacity to phagocytize large foreign particles and cell debris. Once they are in the tissue, macrophages can multiply to encapsulate and trap foreign matter that cannot be phagocytized. Dendritic cells have long processes that can capture antigens and migrate to lymphoid tissue. They serve as sentinels for antigens in most organs including heart, lungs, liver, kidney, and gastrointestinal tract (Goldsby et al., 2003). Like neutrophils, macrophages are drawn to an inflamed area by chemicals released from damaged tissue, a process known as chemotaxis. Monocytes and macrophages activate the immune response against chronic infections such as tuberculosis, viral infections, and certain intracellular parasitic infections; dendritic cells activate T cells against cancer, assist B lymphocytes to produce antibodies, and downregulate the immune system (DeMeyer & Buchsel, 2005).

LYMPHOCYTES Small and nondescript cells, the **lymphocytes** account for 20% to 40% of circulating leukocytes. Lymphocytes are the principal effector and regulator cells of specific immune responses to protect the body from microorganisms, foreign tissue, and cell mutations or alterations. Through a process known as immune surveillance, lymphocytes monitor the body for cancerous cells and eliminate or destroy them.

Like other leukocytes, lymphocytes derive from the stem cells in the bone marrow (Figure 12–2 ■). Lymphocytes have "homing" patterns: They constantly circulate, then return to concentrate in lymphoid tissues (the lymph nodes, spleen, thymus, tonsils, Peyer's patches in the submucosa of the distal ileum, and the appendix). On contact with an antigen, B lymphocytes are activated and mature into either plasma cells, which secrete antibodies, or memory cells. On contact with APCs, T lymphocytes mature into active T_{helper} cells, cytotoxic T cells, or memory T cells. Memory cells stay inactive, sometimes for years, but activate immediately with subsequent

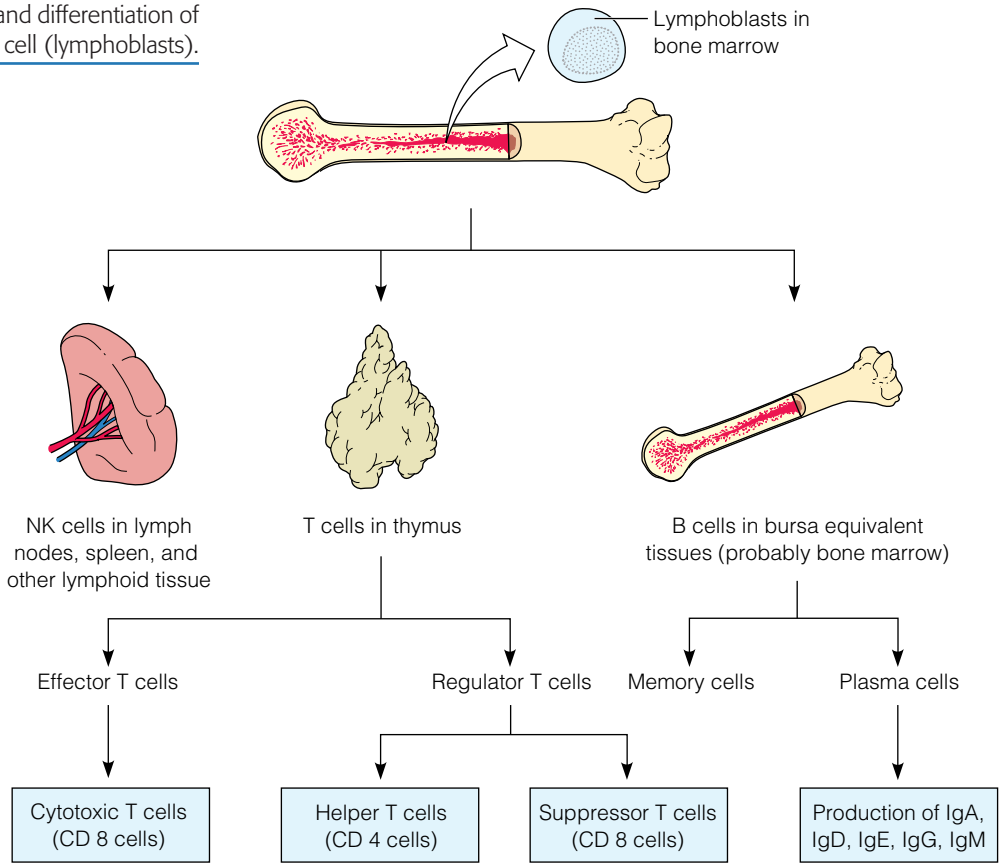


Figure 12–2 ■ The development and differentiation of lymphocytes from the lymphoid stem cell (lymphoblasts).

exposure to the same antigen. They then proliferate rapidly, producing an intense immune response. Memory cells are responsible for providing acquired immunity.

Lymphocyte types are difficult to distinguish by appearance. They have distinct differences in how and where they mature, and in life cycle, surface characteristics, and function.

The three types of lymphocytes are **T lymphocytes (T cells)**, **B lymphocytes (B cells)**, and **natural killer cells (NK cells** or **null cells)**. None of these cells acts independently. Their functions are closely interrelated.

T cells mature in the thymus gland, whereas B cells complete their maturation in the bone marrow. T cells and B cells are integral to the specific immune response and are discussed further in that section of this chapter.

NK cells are large, granular cells found in the spleen, lymph nodes, bone marrow, and blood. They constitute 15% of circulating lymphocytes. NK cells provide immune surveillance and resistance to infection, and they play an important role in the destruction of early malignant cells. Like B cells and T cells, NK cells are cytotoxic, but unlike T cells do not require connection with an APC to become activated and kill cancer cells, virus-infected cells, and cells infected with microbes (Porth, 2005). Fortunately, NK cells are inhibited when contact is made with normal host cells.

ANTIGENS Substances that are recognized as foreign or "nonself" are called **antigens**; they provoke a specific immune response when introduced into the body. Typically, antigens are large protein molecules, although polysaccharides, polypeptides, and nucleic acids may also be antigenic. Many antigens are proteins found on the cell membrane or cell wall of microorganisms or tissues such as transplanted tissue or organs, incompatible blood cells, vaccines, pollen, egg white, and insect or snake venom.

Complete antigens, known as immunogens, have two characteristics:

- *Immunogenicity* is the ability to stimulate a specific immune response.
- *Specific reactivity* is the stimulation of specific immune system components.

The portion of an antigen that incites a specific immune response is called its antigenic determinant site (*epitope*). Complete antigens typically are large molecules with multiple antigenic sites; examples include proteins and certain polysaccharides. Small molecules (e.g., chemical toxins, drugs, and dust) that cannot evoke an antigenic response alone may link to proteins to function as complete antigens. These proteins to which they link are known as haptens.

When an antigen is encountered in the body, generation of an effective immune response involves two major groups of cells: lymphocytes and APCs. Dendritic cells are APCs; two specific types of dendritic cells develop from pluripotent stem cells in the bone marrow. DC1s arise from monocytes, myeloid-type immune cells, and DC2s derive from lymphocyte precursors (DeMeyer & Buchsel, 2005). DC1s activate T cells against cancer cells. DC2s assist B lymphocytes to produce antibodies and to downregulate the immune system (Kimball, 2005). Downregulation is very important to avoid autoimmune diseases.

Antigen-presenting cells are recognized by a specific receptor on a lymphocyte, and an immune response is generated by the lymphocytes. Two separate but overlapping immune responses may occur, depending on the antigen itself and the type of immune cell activated by contact with the antigen. The B cell or humoral branch of the immune system mainly eliminates extracellular antigens such as bacteria, bacterial toxins, and free viruses through the production of **antibodies**, molecules that bind with the antigen and inactivate it. There are five classes of antibodies: IgG, IgA, IgM, IgD, and IgE. These proteins make up the **antibody-mediated (humoral) immune response**. Intracellular pathogens, such as viral-infected cells, cancer cells, and foreign tissue, activate T lymphocytes, which are the primary agents of the **cell-mediated (cellular) immune response**. In this immune response, the lymphocytes themselves, in the form of helper T cells, cytotoxic T cells, and NK cells, inactivate the antigen, either directly or indirectly.

Lymphoid System

The *lymphoid system* consists of the lymph nodes, spleen, thymus, tonsils, lymphoid tissue scattered in connective tissues and mucosa, and the bone marrow. The thymus and bone marrow, in which T cells and B cells mature, are considered central lymphoid organs. The spleen, lymph nodes, tonsils, and other peripheral lymphoid tissue are peripheral lymphoid organs (Figure 12–3 ■). This system exists to recover proteins such as albumin for the vascular system and to protect the bloodstream from invading organisms. Cells of the immune system such as neutrophils, macrophages, and dendritic cells carry antigens from interstitial space to lymph nodes for immune surveillance in the lymphatic circulation. Unlike the vascular tree, which has tight epithelial junctions, lymphatic epithelium is replete with open junctions that promote lymphocyte access and effectively protect the bloodstream from antigen entry.

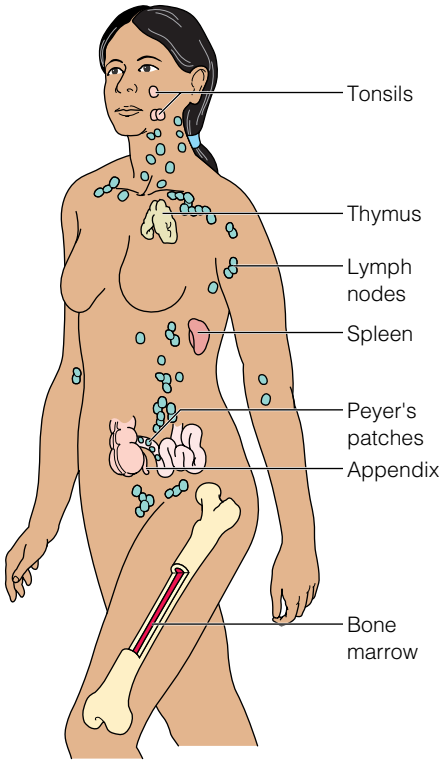


Figure 12–3 ■ The lymphoid system: the central organs of the thymus and bone marrow, and the peripheral organs, including the spleen, tonsils, lymph nodes, and Peyer's patches.

Lymph nodes, the most numerous elements of the lymphoid system, are small, round, or bean-shaped encapsulated bodies that vary in size from 1 mm to 2 cm. Distributed throughout the body, lymph nodes generally occur in groups at the junction of the lymphatic vessels. They can be found in the neck, axillae, abdomen, and groin.

Lymph nodes have two functions: (1) to filter foreign products or antigens from the lymph and (2) to house and support proliferation of lymphocytes and macrophages. Lymph, a clear, protein-containing fluid transported within lymph vessels, enters the node through afferent lymphatic vessels. Inside the node, the lymph flows through sinuses in the cortex of the lymph node where T and B lymphocytes and macrophages are abundant, then through sinuses of the medulla of the lymph node, which contains macrophages and plasma cells. The presence of a foreign antigen stimulates lymphocytes and macrophages to proliferate in the lymph nodes. Macrophages destroy the antigen by phagocytosis. Immune cells and lymph then leave the lymph node through efferent vessels. An abundant blood supply to the node also facilitates lymphocyte movement.

The *spleen* is the largest lymphoid organ in the body and the only lymphoid organ that can filter blood. The spleen is located in the upper left quadrant of the abdomen. The spleen has two kinds of tissue, white pulp and red pulp. White pulp is lymphoid tissue that serves as a site for lymphocyte proliferation and immune surveillance. B cells predominate in the white pulp. Blood filtration occurs in the red pulp. In blood-filled venous sinuses, phagocytic cells dispose of damaged or aged RBCs and platelets. Other debris and foreign matter, such as bacteria, viruses, and toxins, are also removed from the blood. The spleen also stores blood and the breakdown products of RBCs for future use. The spleen is not essential for life. If it is removed because of disease or trauma, the liver and the bone marrow assume its functions.

The *thymus gland* is located in the superior anterior mediastinal cavity beneath the sternum. It reaches its maximum size at puberty, then begins to atrophy slowly. By adulthood, it is difficult to differentiate from surrounding adipose tissue even though it remains active. In the elderly, the vast majority of thymus tissue has been replaced by adipose and fibrous connective tissue. During fetal life and childhood, the thymus serves as a site for the maturation and differentiation of thymic lymphoid cells, the T cells. Thymosin, an immunoregulatory hormone of the thymus, stimulates lymphopoiesis, the formation of lymphocytes or lymphoid tissue.

Bone marrow is soft organic tissue found in the hollow cavity of the long bones, particularly the femur and humerus, as well as the flat bones of the pelvis, ribs, and sternum. Bone marrow produces and stores hematopoietic stem cells, from which all cellular components of the blood are derived (see Figure 12–1).

Lymphoid tissues are also located at key sites of potential invasion by microorganisms: the submucosa of the genitourinary, respiratory, and gastrointestinal tracts and the skin. Plasma cells in these lymphoid tissues defend the body against bacterial

invasion at areas exposed to the external environment. In general, these tissues are known as *mucosa-associated lymphoid tissue* (MALT). Diffuse collections of lymphocytes, plasma cells, and phagocytes are scattered throughout the respiratory tract, concentrating at bifurcations of the bronchi and bronchioles. Peyer's patches, or gut-associated lymphoid tissue (GALT), comprises the largest collection of immune cells in the body (Bourlioux et al., 2003). Ingestion and absorption of solid foodstuffs and liquids continually expose the lining of the gut to resident microflora and infectious pathogens. Unlike peripheral lymph nodes, which respond to pathogens with acute inflammatory responses, GALT processes common intestinal antigens without producing acute inflammation. Collections of immune cells make up the GALT. Intraepithelial lymphocytes fill the spaces between mucosal epithelial cells. Beneath the basement membrane of gut epithelium lie abundant T cells and mature plasma cells, which are sources of IgA. Peyer's patches hold dense collections of lymphocytes in lymphoid nodules. As naïve B and T cells migrate through Peyer's patches, they are sensitized to specific antigens. In mesenteric lymph nodes these sensitized cells proliferate and circulate throughout the vascular tree where they produce secretory IgA. Secretory IgA coats mucosal cells and prevents attachment of intraluminal bacteria in the intestine, upper respiratory tract, the bronchi, mammary ducts, and salivary glands. Thus the GALT collection of immune cells effectively protects mucosa throughout the body that is exposed to resident and foreign pathogens.

Tonsils and adenoids protect the body from inhaled or ingested foreign agents. Skin-associated lymphoid tissue contains lymphocytes and dendritic cells such as Langerhans cells in the epidermis, which transport antigens to regional lymph nodes for destruction and development of specific immunity to the antigen.

MEDIALINK Inflammatory Response Animation

Nonspecific Inflammatory Response

Barrier protection is the body's first line of defense against infection. The skin is the primary barrier. When intact, it prevents invasion by external organisms. When the skin is damaged or lost (e.g., as a result of injury, surgery, or burns), infection is much more likely. The membranes lining inner surfaces of the body are protected by a barrier of mucus, which traps microorganisms and other foreign substances. These can then be removed by other protective mechanisms, such as ciliary movement or the washing action of tears or urine. In addition, many body fluids contain bactericidal substances that provide barrier protection. These include acid in gastric fluid, zinc in prostatic fluid, and lysozyme in tears, nasal secretions, saliva, and sweat (Porth, 2005; Rink & Gabriel, 2000).

When these first-line defenses are breached, resulting tissue damage or foreign material entering the body induces a nonspecific immune response known as inflammation. **Inflammation** is an adaptive response to injury that brings fluid, dissolved substances, and blood cells into the interstitial tissues where the invasion or damage has occurred. The response is called *nonspecific* because the same events occur regardless of cause of the inflammatory process. Through the inflammatory reaction, the invader is neutralized and eliminated, destroyed tissue removed, and the process of healing and repair initiated.

The inflammatory response has three stages: (1) a vascular response characterized by vasodilation and increased permeability of blood vessels, (2) a cellular response and phagocytosis, and (3) healing (tissue repair).

Vascular Response

After tissue cells are damaged, local blood vessels briefly constrict. Vasodilation follows almost immediately as inflammatory mediators such as histamine and kinins are released from damaged tissue (Box 12–1). Increased blood flow causes vasocongestion at the injury site with resultant redness and heat. The congestion also increases local hydrostatic pressure. This, along with increased vessel permeability that results from chemical mediators, moves fluid out of the capillaries and into the interstitial spaces of the tissue. The escaping fluid, called fluid exudate, contains large amounts of protein and causes local edema. Fluid exudate has three functions: (1) It provides protection to the injured tissue by bringing certain nutrients needed for tissue healing;

BOX 12–1 Inflammatory Mediators

Many of the manifestations of inflammation are produced by *inflammatory mediators,* which are chemicals released as a result of immunologic processes or tissue injury or damage. These inflammatory mediators are broadly classified as follows:

- Vasoactive substances produce smooth muscle constriction, postcapillary vasodilation, and increased capillary permeability.
- Chemotactic factors attract leukocytes to the damaged tissue.
- Plasma enzymes activate the clotting cascade, plasminogen system, and complement system.
- Miscellaneous cell products (e.g., oxygen metabolites and lysosomal enzymes) damage surrounding tissue.

Many of the outward manifestations of inflammation result from vasoactive substances such as *histamine, serotonin,* and *leukotrienes* (formerly known as slow-reacting substance of anaphylaxis, or SRS-A). Stored in mast cells, basophils, and platelets, histamine is released when an injury occurs or with stimulation by the immune system. An important component of the early inflammatory response, histamine causes vasodilation and vascular permeability in the affected area. Histamine is also a key factor in many hypersensitivity reactions. Serotonin is released from platelets and produces effects similar to those of histamine. The leukotrienes play a significant vasoactive role in the later stages of the inflammatory response.

Prostaglandins are chemotactic substances that draw leukocytes to the inflamed tissue. In addition, they play a vasoactive role and are pain and fever inducers. Aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs) as well as the glucocorticoids inhibit prostaglandin synthesis, thereby reducing fever, pain, and inflammation.

Plasma factors such as Hageman factor activate the clotting cascade, plasminogen system (involved in the lysis of clots), and complement system. With activation of the clotting cascade, bacteria and other foreign substances are trapped in the area of tissue damage. Fibrin, which has vasoactive by-products, is also released. (See Chapter 34 ∞ for a full description of the clotting process.) The complement system serves a chemotactic role and facilitates the phagocytic process.

Major chemical mediators of inflammation are summarized in Table 12–7 on page 304.

(2) it dilutes bacterial toxins; and (3) it transports cells needed for phagocytosis. Mild tissue damage such as a blister produces a *serous* exudate of primarily plasma fluid and a few proteins. With moderate to severe tissue damage, fluid exudate is *sanguineous* or *hemorrhagic,* containing large amounts of RBCs. A mixture of RBCs and serum is referred to as *serosanguineous* exudate. *Fibrinous* exudate forms a thick, sticky meshwork of fibrinogen, in effect "walling off" inflamed tissues and preventing the spread of infection (Porth, 2005). In more severe or acute inflammation, the fluid contains fibrin, RBCs, and dead and live bacteria. This type of exudate, called *purulent* exudate, has an odor and color characteristic of the bacteria present.

The vascular response localizes invading bacteria and keeps them from spreading. Increased capillary permeability enhances the release of clotting factors such as fibrinogen, which converts to fibrin threads, entrapping the bacteria and walling them off from contact with the rest of the body.

Cellular Response

The cellular stage of the inflammatory process begins within less than an hour after the injury. This stage is marked by the margination and emigration of leukocytes into the damaged tissue, chemotaxis, and phagocytosis (Porth, 2005).

As serous fluid escapes the capillaries, the viscosity of blood in the area increases and its flow becomes more sluggish. Leukocytes marginate, moving to the edges of the blood vessels, and begin to adhere to the capillary endothelium. This process is known as *pavementing*. After margination and pavementing, leukocytes emigrate from the blood vessel into the tissue spaces (Figure 12–4 ■). Within hours, millions of leukocytes emigrate into the area of inflammation (Price & Wilson, 2003).

Once leukocytes have emigrated, they are drawn to the damaged or inflamed tissues by chemotactic signals. Infectious agents, damaged tissues, and activated plasma substances such as complement fractions provide chemotaxic signals that attract an army of neutrophils, monocytes, and macrophages to the injury site.

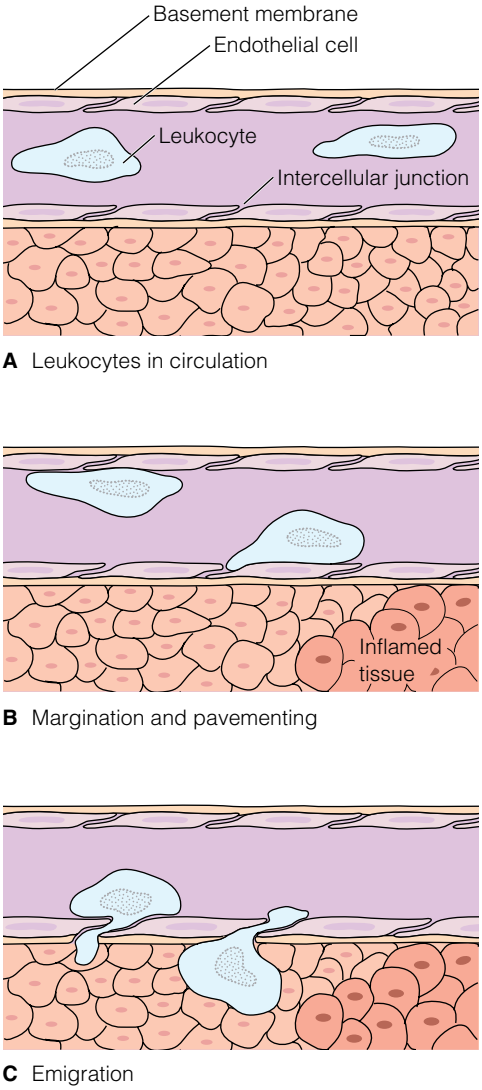
The number of neutrophils around the site increases to about 15,000 to 25,000/mm^3, and they begin their role in phagocytosis within a few hours. Monocytes become transient macrophages to augment the activity of the fixed macrophages and dendritic cells; together they engulf dead cells, damaged tissue, nonfunctioning neutrophils, and invading bacteria.

Phagocytosis

Phagocytosis is a process by which a foreign agent or target cell is engulfed, destroyed, and digested. Neutrophils, macrophages, and dendritic cells, known as *phagocytes,* are the primary cells involved in phagocytosis. Once attracted to the inflammatory site, phagocytes select and engulf foreign material.

The following factors or processes help phagocytes differentiate foreign tissue from normal cells:

- *Smooth surface.* Normal tissue has a smooth surface that is resistant to phagocytosis, whereas the rough surface of a foreign agent or target cell promotes phagocytosis.
- *Surface charge.* Healthy body cells present an electronegative surface charge that repels phagocytes. Cellular debris and foreign agents, by contrast, have an electropositive charge that attracts them.



A Leukocytes in circulation

B Margination and pavementing

C Emigration

Figure 12–4 ■ The process of leukocyte emigration at the site of inflammation. *A,* Normal blood flow with free movement of formed elements. *B,* As blood flow slows, leukocytes move toward the periphery of stream and begin to cling to capillary endothelium, a process known as margination and pavementing. *C,* Leukocytes emigrate from the vessel into inflamed tissues.

- *Opsonization.* This immune system process coats the surface of bacteria or target cells with a substance (an opsonin) as in the complement system (Box 12–2). Opsonization enables the phagocyte to bind tightly with the foreign tissue, facilitating phagocytosis (Figure 12–5A ■).

Phagocytes engulf the foreign agent or target cell by projecting pseudopodia ("false feet") in all directions around it (Figure 12–5B). This produces a chamber called a *phagosome* containing the antigen, which is ingested into the cytoplasm (Figure 12–5C). Once the phagosome has been engulfed, lysosomes fuse with the phagosome, killing any live organism and releasing digestive enzymes, which destroy the antigen (Figure 12–5D).

Phagocytes—in particular, neutrophils and macrophages—contain bactericidal agents that kill most of the bacteria they ingest before the bacteria can multiply and destroy the phagocyte itself. The phagocyte kills bacteria in a number of ways; for

BOX 12–2 **The Complement System**

The *complement system* consists of approximately 20 complex plasma proteins that are activated by a tissue injury or antigen–antibody reaction. The complement system is involved in both nonspecific and specific immune responses. Its activation results in the production of effector molecules that are involved in the processes of inflammation, phagocytosis, and cell lysis or destruction (Goldsby et al., 2003; Porth, 2005). Specifically, complement activation leads to the following:

- *Mediation of the inflammatory response.* When the complement system is activated, chemical mediators such as histamine are released from mast cells and basophils, leading to smooth muscle contraction, increased vascular permeability and edema, and the attraction of leukocytes.
- *Opsonization (or coating) of antigen–antibody complexes to facilitate phagocytosis.*
- *Alteration of the cell membrane or viral capsule.* When the cell surface is altered, lysis results. Bacteria and viruses are destroyed; certain normal cells, such as RBCs, platelets, and lymphocytes, that are damaged or old may also be destroyed through this process.

The complement system has three "arms," or pathways, of protein and enzyme reactions. The *classic pathway* is activated by antibody-containing immunoglobulins and other substances such as DNA and C-reactive protein. The *alternate and lectin pathways* do not use antibodies, are activated by tissue injury, polysaccharides, or enzymes, and are part of the nonspecific immune system (Porth, 2005). When either pathway is activated, the result is mediation of the inflammatory process, attraction of phagocytes, facilitation of phagocytosis, and lysis of microbes.

example, it alters the intracellular pH and produces bactericidal agents. Oxidizing agents, such as superoxide, hydrogen peroxide, and hydroxyl ions, are bactericidal. Two lysosomal substances that kill bacteria are lysozyme and phagocytin.

Some antigens, such as the tubercle bacterium, have coats or secrete substances that are resistant to lysosomal and bactericidal agents. To destroy such antigens, lysosomes release digestive enzymes into the phagosome. The lysosomes of neutrophils and macrophages contain an abundance of proteolytic (protein-destroying) enzymes that digest bacteria and other foreign protein components. The macrophage's lysosomes also contain lipases (fat-splitting enzymes) capable of digesting the thick lipid membranes of such bacteria as *Mycobacterium tuberculosis* and *Mycobacterium leprae.*

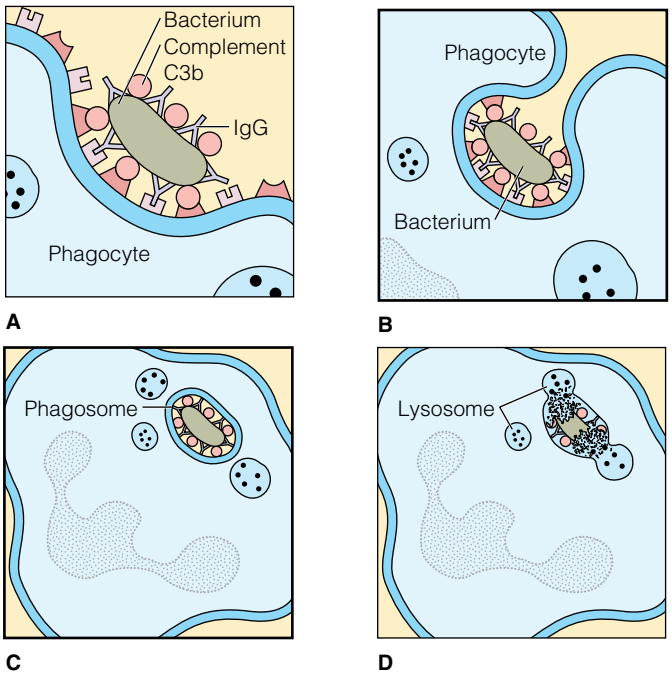


Figure 12–5 ■ The process of phagocytosis. *A,* Opsonization coats the surface of the bacterium with IgG (an antibody) and complement. *B,* The bacterium is bound to and engulfed by the phagocyte. *C,* The phagosome is ingested into the cytoplasm of the phagocyte. *D,* Lysosomes fuse with the phagosome, releasing digestive enzymes and destroying the antigen.

Once neutrophils have ingested toxic substances to their capacity, they in turn are killed. Neutrophils have the capacity to phagocytize 5 to 20 bacteria before they become inactive. Macrophages then digest the dead neutrophils. Monocytes or macrophages are capable of phagocytizing up to 100 bacteria. Because of their size, they can ingest larger particles than neutrophils can ingest, such as whole RBCs, necrotic tissue, cell fragments, malarial parasites, and dead neutrophils. Dendritic cells are also phagocytic and secrete Il-12, which is an important cytokine in the maturation of T_{helper} cells (Kimball, 2005). Macrophages have the ability to extrude (release) the toxic substances and lysosomal enzymes within their phagosomes. As a result, they can continue to function for months and even years.

Healing

Inflammation is the first phase of the healing process. During the inflammatory process, particulate matter, bacteria, damaged cells, and inflammatory exudate are removed by phagocytosis. This process, called *debridement*, prepares the wound for healing. Adequate nutrition is essential for inflammation and healing to proceed. Protein, glucose, and oxygen are needed by leukocytes for chemotaxis, phagocytosis, and intercellular killings. Persons with diabetes are at risk for poor healing of wounds. Probable causes for this risk may be small vessel disease, which impairs microcirculation and increased affinity for oxygen common to glycosylated hemoglobin. Oxygen circulation is impaired as well as release of oxygen to the cells (McCance & Huether, 2002).

The second phase of the healing process, known as *reconstruction,* may overlap the inflammatory phase. The ideal result of the healing process is *resolution,* the restoration of the original structure and function of the damaged tissue. Simple resolution occurs when there is no destruction of the normal tissue and the body is able to neutralize and remove the offending agent through the inflammatory process.

Resolution may also occur when the damaged tissue is capable of regeneration. The ability to regenerate, or replace lost *parenchyma* (functional tissue) with new, functional cells varies by tissue and cell type.

- *Labile cells* continue to regenerate throughout life. These cells are found in tissues where there is a daily turnover of cells—namely, bone marrow and the epithelial cells of the skin, mucous membranes, cervix, gastrointestinal tract, and genitourinary tract.
- *Stable cells* normally stop replicating when growth ceases, but are capable of regeneration when stimulated by an injury. Osteocytes (which are found in bone) and parenchymal cells of the kidneys, liver, and pancreas are stable cells.
- *Permanent* or *fixed cells* are unable to regenerate. When these cells are destroyed, they are replaced by fibrous scar tissue. Nerve cells, skeletal muscle cells, and cardiac muscle cells are fixed cells (Porth, 2005). Researchers are searching for signaling molecules that will stimulate repair of permanent cells, allowing regeneration. Paralysis from spinal cord injury is often the stimulus for this research focus. When regeneration and complete resolution are not possible, healing occurs by replacement of the destroyed tissue with collagen scar tissue. This process is known as *repair.* Although tissue that has undergone repair lacks the physiologic function of the destroyed tissue, the scar fills the lesion and provides tensile tissue strength. The healing process is discussed further in Chapter 4 ∞.

Specific Immune Response

The introduction of antigens into the body causes a more specific reaction than the nonspecific inflammatory response. On the first exposure to an antigen, a change occurs in the host, resulting in a specific and rapid response following subsequent exposures. This specific response is known as the *immune response.*

The immune response to an antigen has the following distinctive properties:

- The immune response typically is directed against materials recognized as foreign (i.e., from outside the body) and is not usually directed against the self (i.e., cells or structures produced by the body). This property is known as *self-recognition*.
- The immune response is *specific.* It is initiated by and directed against particular antigens (such as a specific virus, bacterium, or transplanted tissue).
- Unlike a localized inflammatory response, the immune response is systemic. Immunity is generalized; it is not restricted to the initial site of infection or entry of foreign tissue.
- The immune response has memory. Repeated exposures to an antigen produce a more rapid response.

A client whose immune system is able to identify antigens and effectively destroy or remove them is said to be **immuno-competent**. Health problems may occur when the immune response is altered (see Chapter 13 ∞ for further discussion).

Antibody-Mediated Immune Response

The antibody-mediated (humoral) immune response is produced by B lymphocytes (B cells). B cells are constantly replaced through cell division and proliferation in the bone marrow. It is believed that B cells mature in the bone marrow and then migrate to the spleen to await activation. They normally constitute 10% to 15% of circulating lymphocytes.

B cells are activated by contact with an antigen and by T cells (discussed in the next section). Each B cell has receptor sites for a specific antigen or antigens. When the antigen is encountered, the activated B cell proliferates and differentiates into antibody-producing plasma cells and memory cells (Figure 12–6 ■). Plasma cells are short lived, lasting only about 1 day. While alive, however, they can produce thousands of antibody molecules per second. Memory cells retain antibody-producing information, allowing a rapid response if the antigen is again encountered.

An antibody is an **immunoglobulin (Ig)** molecule with the ability to bind to and inactivate a specific antigen. Immunoglobulins comprise the gamma globulin portion of the blood proteins. The immune system produces numerous antibodies, each active against a specific antigen. As mentioned earlier, antibodies fall into five classes of immunoglobulins: IgG, IgA, IgM, IgD, and IgE. Each has a slightly different structure and function. Their roles are summarized in Table 12–2.

TABLE 12–2 **Immunoglobulin Characteristics and Functions**

CLASS	PERCENTAGE OF TOTAL	CHARACTERISTICS AND FUNCTION
IgG	75%	Most abundant Ig; also known as gamma globulin; found in blood, lymph, and intestines Active against bacteria, bacterial toxins, and viruses Activates complement The only Ig to cross the placenta, providing immune protection to neonate
IgA	10% to 15%	Found in saliva, tears, and bronchial, gastrointestinal, prostatic, and vaginal secretions, as well as blood and lymph Provides local protection on exposed mucous membrane surfaces and potent antiviral activity by preventing binding of the virus to cells of the respiratory and gastrointestinal tracts Levels decrease during stress
IgM	5% to 10%	Found in blood and lymph First antibody produced with primary immune response High concentrations early in infection, decreases within about a week Mediates cytotoxic response and activates complement
IgD	<1%	Found in blood, lymph, and surfaces of B cells Exact function unknown; may be receptor-binding antigens to B-cell surface
IgE	<0.1%	Found on mast cells and basophils Involved in release of chemical mediators responsible for immediate hypersensitivity (allergic and anaphylactic) response

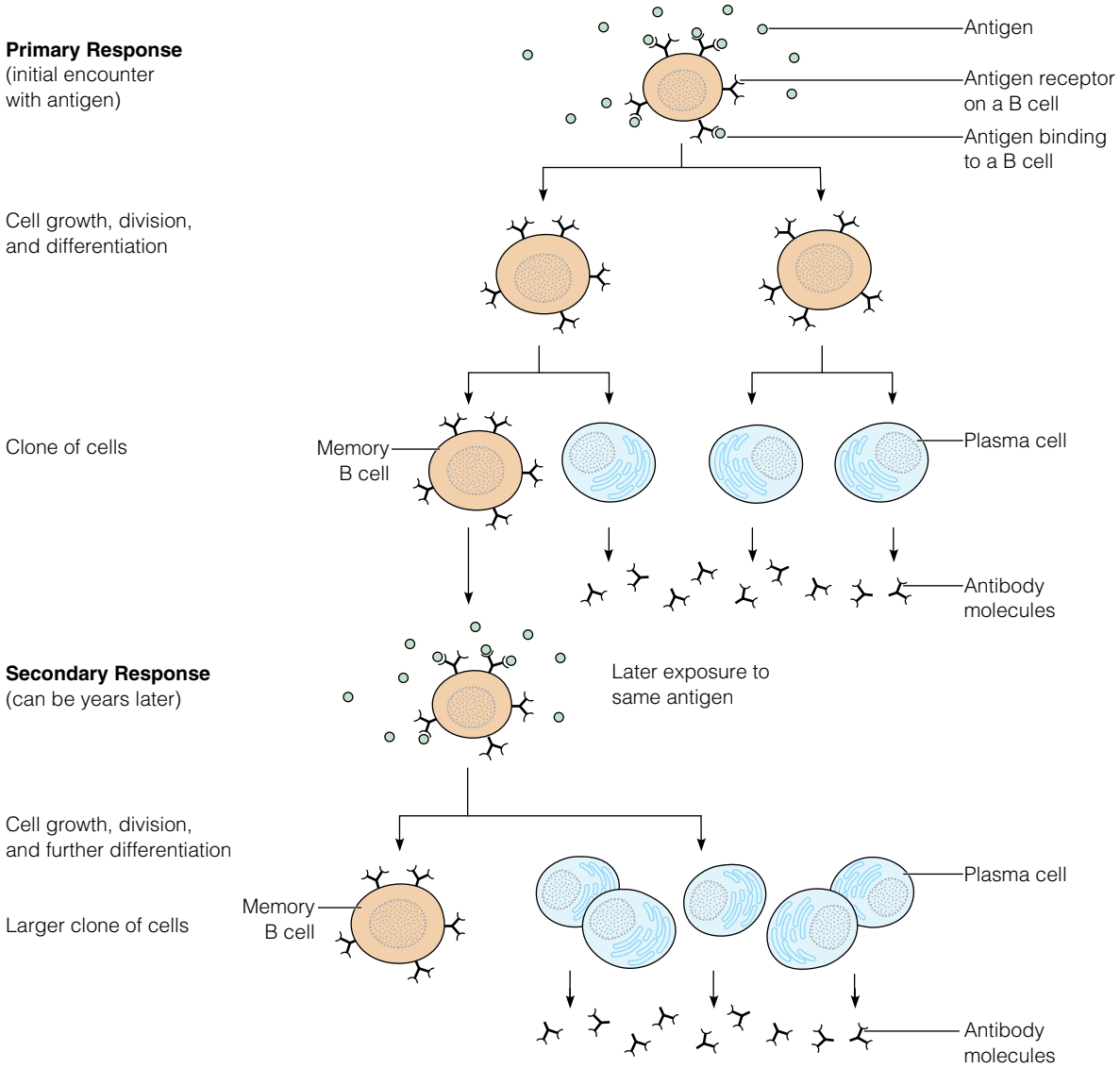


Figure 12–6 ■ Antibody-mediated (humoral) immunity. On initial exposure to the antigen, B cells with appropriate receptor sites are activated to become plasma cells and produce antibodies or memory cells. This is known as the primary response. With subsequent exposures, memory cells respond rapidly with antibody production. This is known as the secondary response.

Antibodies are Y-shaped molecules with two light and two heavy polypeptide chains (Figure 12–7 ■). The top portion of the Y, called the *Fab* or *antigen-binding fragment,* is chemically variable and specific to the antigen. The lower portion, the F_c, or *crystallized fragment,* is constant for its class of immunoglobulin and directs the biologic activity of the immunoglobulin (the manner in which it functions). For example, the lower portion of immunoglobulin molecules produced against hepatitis A and hepatitis B are the same (IgG), but the upper portion is different and specific to the virus.

The antibodies produced by B cells (see Figure 12–7) link with the antigen (Figure 12–8 ■) and inactivate it through one of the following processes:

- Promoting phagocytosis of the antigen by neutrophils
- Precipitation: combining with soluble antigens to form an insoluble complex or precipitate
- Neutralization: combining with a toxin to neutralize its effects; the antigen–antibody complex is then destroyed by the process of phagocytosis
- Lysis of the antigen cell membrane caused by combination with antibodies and complement proteins
- Agglutination (clumping) of antigens to form a noninvasive aggregate
- Opsonization: coating of the antigen with antibodies and complement, making them more susceptible to phagocytosis.

The complete antibody-mediated response occurs in two phases. With initial exposure to an antigen, the primary response develops. B cells are activated to proliferate and begin producing antibodies. There is a latency period of 3 to 6 days before antibodies become detectable in the blood. Levels then continue to rise, peaking at 10 to 14 days after the initial exposure. With many illnesses (e.g., chickenpox), this peak correlates with recovery.

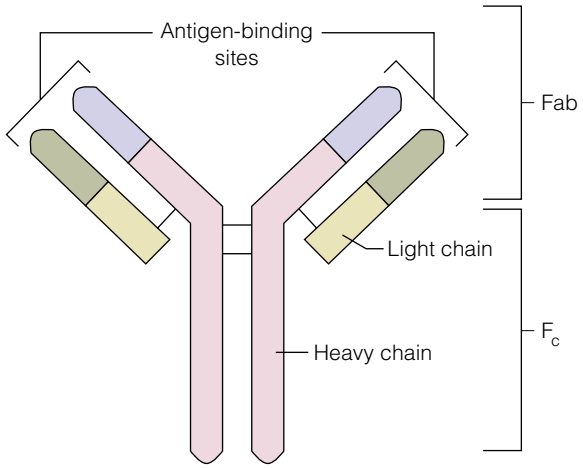


Figure 12–7 ■ An antibody molecule. The Fab section is unique, providing an antigen-specific binding site. The F_c section is common to each class of immunoglobulin (IgG, IgA, IgM, IgD, IgE).

Subsequent exposure to the same antigen elicits a secondary response. Memory cells (see Figure 12–6) formed during the primary response stimulate the production of plasma cells, and an almost immediate rise in antibody levels occurs (Figure 12–9 ■). This rapid secondary response is the basis of acquired immunity and is instrumental in preventing disease. It is also the mechanism through which vaccines provide protection from disease.

Cell-Mediated Immune Response

Many antigens cannot stimulate the antibody-mediated response or are "hidden" from it because they live inside the body's cells (viruses and mycobacteria are examples of such antigens). The immune response providing protection against these antigens is the cell-mediated immune response, also called *cellular immunity.* T lymphocytes (T cells) initiate this type of immune response.

Approximately 70% to 80% of circulating lymphocytes are T cells. T cells migrate to the thymus during fetal and early life,

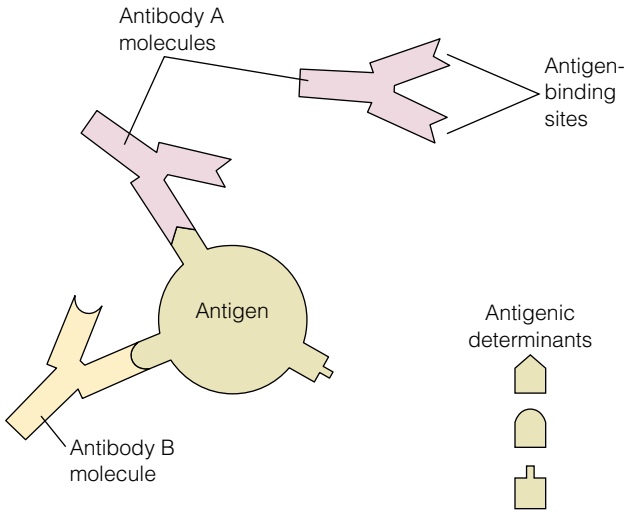


Figure 12–8 ■ Antigen–antibody binding. The unique Fab site on the antibody binds with specific receptor sites on the antigen. As shown, more than one kind of antibody may be produced to an antigen.

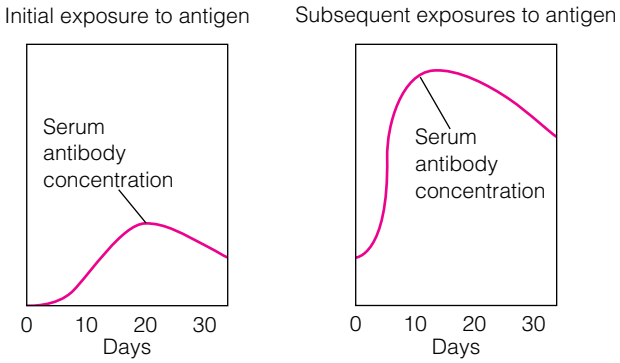


Figure 12–9 ■ Antibody production in the primary and secondary responses of the antibody-mediated immune response. Note the more rapid and effective production following subsequent exposure.

establishing the lifetime pool of cells. T cells have a life span measured in years, maintaining their numbers through proliferation, primarily in the lymph nodes.

T cells are much more complex than B cells. There are two major classes of T cells, *effector cells* and *regulator cells.* The main effector T cell is the *cytotoxic cell,* also called the *killer T cell.* Regulator T cells are further classified into two groups: *helper T cells* and *suppressor T cells.*

T cells are antigen specific; that is, each subset is activated by a particular antigen. The antigens that activate T cells must be presented on another cell surface, such as pieces of virus presented on the surface of an infected cell, or the histocompatibility locus antigen on a cell of transplanted tissue. When activated, T cells divide and proliferate, forming antigen-specific *clones* (Figure 12–10 ■). A clone is an exact copy of another cell.

Cytotoxic T cells bind with cell surface antigens on virus-infected or foreign cells. Killer T cells destroy the identified cell by combining with it and then either destroying its cell membrane or releasing cytotoxic substances into the cell. They are vital in the control of viral and bacterial infections.

Regulator T cells play a key role in controlling the immune response. The majority of regulator T cells are helper T cells. They stimulate the proliferation of other T cells, amplify the cytotoxic activity of killer T cells, and activate B cells to proliferate and differentiate. They interact directly with B cells to promote their multiplication and conversion into plasma cells capable of producing antibodies. The other regulatory T-cell group, suppressor T cells, provides negative feedback, making the immune response a self-limiting process.

On activation, both effector and regulator T cells synthesize and release lymphokines, a type of soluble protein. Lymphokines are a subgroup of nonspecific defense mechanisms known as **cytokines** (Box 12–3). Lymphokines secreted by cytotoxic and helper T cells are important in amplifying the immune response and the nonspecific inflammatory response. They stimulate the following:

- B cells to become plasma cells and produce antibodies
- Macrophages to become activated macrophages (the most aggressive phagocyte)
- Proliferation of killer T cells.

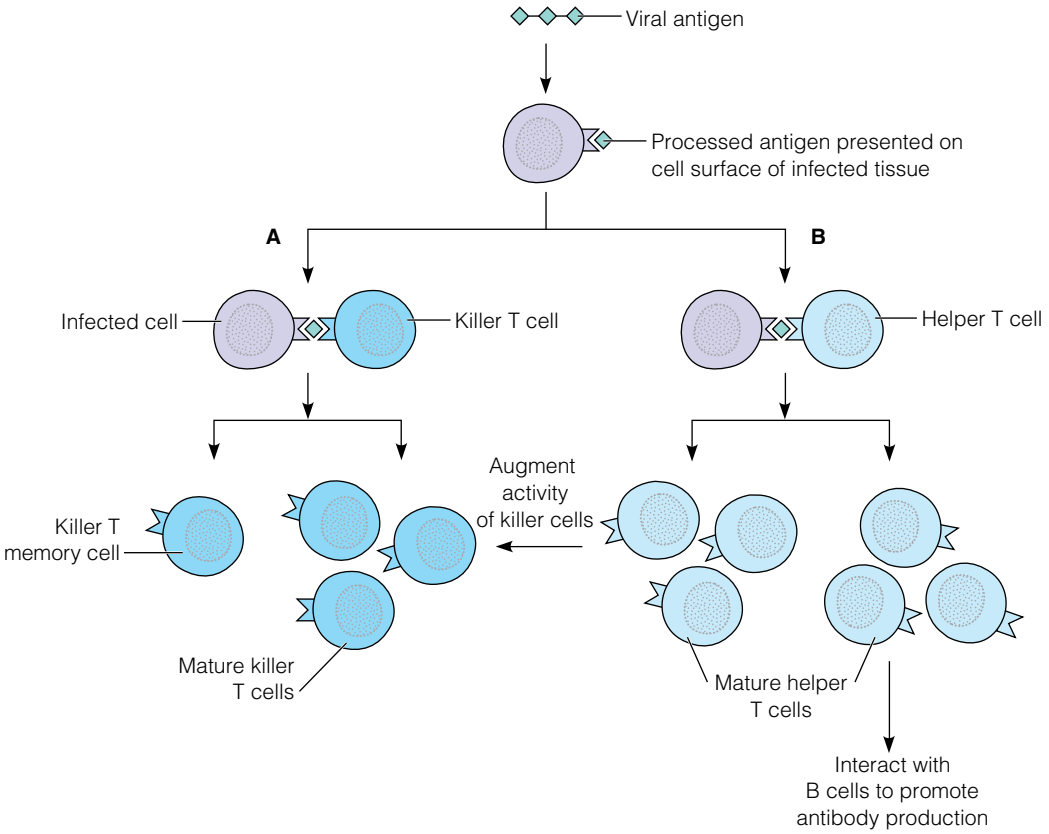


Figure 12–10 ■ Cellular immune response. *A,* An infected cell, abnormal cell, or phagocyte presents antigen on its surface that binds with a receptor site on a killer T cell or a helper T cell. The killer T cell is activated to proliferate into memory cells or mature cytotoxic cells. *B,* The helper T cell is activated to augment the cytotoxic response and stimulate the antibody-mediated immune response.

Suppressor T cells release lymphokines, which inhibit the activity of other T cells and B cells.

Although T cells can be activated only by specific antigens, much of the resulting effect is nonspecific—in other words, an enhanced inflammatory response. Like the antibody-mediated response, the cell-mediated response has memory. Subsequent exposures to an antigen result in a more rapid and effective inflammatory response and more effective phagocytosis by macrophages. This memory provides the basis for skin testing. A client previously exposed to tuberculosis, for example, develops a more pronounced inflammatory response when minute amounts are injected under the skin.

The Client with Natural or Acquired Immunity

Immunity refers to the protection of the body from disease. Immunity to disease may be either natural or acquired, active or passive.

Immunity develops from the activation of the body's immune response. Depending on the antigen, antibody-mediated or cell-mediated responses are activated. The immune response typically involves components of both. In the immunocompetent client, these responses inactivate and remove the antigen, allowing recovery to occur or preventing the development of disease. Clients with suppressed or impaired immune function are more susceptible to disease and require protection from exposure to environmental elements. Isolation techniques are employed to prevent the spread of disease and to protect immune-suppressed clients.

Pathophysiology

The processes of antibody-mediated and cell-mediated immunity result in the development of **acquired immunity** or **active immunity**. Active immunity occurs when the body produces antibodies or develops immune lymphocytes against specific antigens. Memory cells, which can produce an immediate immune response on reexposure to the antigen, provide long-term immunity.

Active immunity can be naturally acquired, resulting from contact with the disease-producing antigen and subsequent development of the disease. Naturally acquired immunity is common for diseases such as chickenpox and hepatitis A, making the risk of developing the disease a second time very low.

For many diseases, the potential consequences of a single disease episode on the individual and society make prevention desirable, especially for highly contagious diseases capable of causing epidemics. In these instances, immunization or vaccination is used to provide artificially acquired immunity. The purpose of vaccination is to establish adequate levels of antibody and/or memory cells to provide effective immunity (Goldsby et al., 2003). Vaccination introduces the

BOX 12–3 **Cytokines**

Cytokines are hormone-like polypeptides produced primarily by cells of the immune system. Cytokines are also produced in small quantities in many different tissues throughout the body. Cytokines act as messengers of the immune system, facilitating communication between the cells to adjust or vary the inflammatory reaction or to initiate immune cell proliferation and differentiation. Cytokines are an essential component of an adequate immune response. The major cytokines and their functions are summarized in Table12–3.

Interferons are a class of cytokine with broad antiviral and anticancer effects. A number of different forms of interferon exist, broadly grouped as alpha, beta, and gamma interferons. Interferon is synthesized by cells infected with a virus and secreted into extracellular fluid. It then binds to specific receptors on uninfected neighboring cells, protecting them from infection. The spread of the virus is thus inhibited, and recovery from infection enhanced. It appears that interferons also moderate the activity of NK cells and may be involved in preventing the spread of abnormal malignant cells.

disease-producing antigen into the body in a manner that will stimulate the immune system to form antibodies and memory cells but will not produce disease. Vaccines may be made of killed organisms or of live organisms that have been attenuated or modified to reduce their disease-producing capability. Typhoid is an example of a killed organism vaccine; measles-mumps-rubella (MMR) vaccine, by contrast, is made from attenuated organisms. Many newer vaccines use subunits of the antigen; these are portions of the organism that have antigenic properties but are unable to produce disease.

Passive immunity provides temporary protection against disease-producing antigens. Passive immunity is provided by antibodies produced by other people or animals. These acquired antibodies are used up; they either combine with the antigen or are naturally degraded by the body, and their protection is gradually lost. Naturally acquired passive immunity is provided by the transfer of maternal antibodies via the placenta and breast milk to the infant. Rabies human immune globulin and hepatitis B immune globulin (HBIG) are examples of immunizations used to provide artificially acquired passive immunity. The types of active and passive immunity are summarized in Table 12–4.

INTERDISCIPLINARY CARE

Collaborative care focuses primarily on assessing the client's immune status and ensuring acquired immunity to prevent disease.

Diagnosis

A number of diagnostic tests can be performed to assess the client's immune status.

TABLE 12–3 **Major Cytokines and Their Functions**

CYTOKINE	WHERE PRODUCED	PRIMARY FUNCTIONS
Interleukin-1 (IL-1)	Monocytes and macrophages; other cells	Activates T and B cells Induces fever and tissue catabolism Enhances NK activity Attracts neutrophils, macrophages, and lymphocytes Stimulates endothelial cell growth, collagen, and collagenases
Interleukin-2 (IL-2)	Helper T cells	Stimulates T and B cell proliferation Activates killer T and NK cells
Interleukin-3 (IL-3)	T cells	Stimulates growth and differentiation of bone marrow stem cells
Interleukin-4 (IL-4)	Activated helper T cells	Stimulates proliferation of T and B cells Increases IgE secretion by B cells
Interleukin-5 (IL-5)	T cells and activated mast cells	Promotes differentiation of B cells and eosinophils Stimulates production of IgA
Gamma interferon	T and NK cells	Stimulates phagocytosis by neutrophils and macrocytes Activates NK cells Augments B cell proliferation, enhancing both cellular and humoral immune responses
Alpha and beta interferons	Virus-infected cells; macrophages	Activate macrophages and endothelial cells Augment NK cell activity Act at gene level to protect neighboring cells from invasion by intracellular parasites, such as viruses, rickettsia, malaria
Tumor necrosis factor (TNF)	Activated macrophages, T cells, and NK cells	Major chemical mediator of inflammatory response Stimulates T-cell activation, antibody production, and accumulation of leukocytes at inflammatory site Directly cytotoxic to some tumor cells Induces fever

TABLE 12–4 **Types of Acquired Immunity**

TYPE OF IMMUNITY		HOW DEVELOPED	EXAMPLES
Active Immunity	Natural	Acquired by infection with an antigen, resulting in the production of antibodies	Chickenpox, hepatitis A
	Artificial	Acquired by immunization with an antigen, such as attenuated live virus vaccine	MMR, polio, DPT, hepatitis B vaccines
Passive Immunity	Natural	Acquired by transfer of maternal antibodies to the fetus or neonate via the placenta or breast milk	Neonate initially protected against MMR if mother immune
	Artificial	Acquired by administration of antibodies or antitoxins in immune globulin	Gamma globulin injection following hepatitis A exposure

- *Serum protein* measures the total protein in the blood including albumin and globulins. Normal levels for the adult are 6 to 8 g/dL; albumin is approximately 60% (3.2 to 4.5 g/dL) of the total serum protein, and globulins are normally 2.3 to 3.4 g/dL. Total protein levels, albumin, and globulin are decreased in malnutrition and liver disease. Decreased globulin levels are noted with immunologic deficiencies.
- *Protein electrophoresis* analyzes protein content especially for albumin and gamma globulin and is used to assess immune function. Gamma globulins subjected to further electrophoresis separate into immunoglobulins: IgA, IgD, IgE, IgG, and IgM (see Table 12–2). Analysis of specific levels of each provides clues about the immune status of the client. IgG levels are increased during acute infection. Decreased levels of IgG, IgA, and IgM are found in malignancies.
- *Antibody testing* is ordered to determine if a client has developed antibodies in response to an infection or immunization. Antibodies for hepatitis, HIV, rubella, toxoplasmosis, and *Treponema pallidum* (the organism causing syphilis) can be identified. An elevated titer level for hepatitis and rubella indicates immunity. For the other disorders and hepatitis, it may also be used to determine if the client has the disease.
- *Skin testing* can assess cell-mediated immunity. A known antigen such as streptokinase, tuberculin purified protein derivative, or candida is injected intradermally. The site is then observed for induration and erythema, which typically peaks at 24 to 48 hours. An induration of at least 10 mm in diameter is a positive reaction indicating previous exposure and sensitization to the antigen. (See Chapter 13 ∞ for further information on skin testing for hypersensitivity reactions.) No reaction, or **anergy**, indicates depressed cell-mediated immunity.

Immunizations

Vaccines are suspensions of whole or fractionated bacteria or viruses that have been treated to make them nonpathogenic. Vaccines are given to induce an immune response and subsequent immunity. Although vaccine development has been a major factor in improving public health, no vaccine is completely effective or entirely safe. Table 12–5 outlines the vaccines recommended for the adult client to maintain optimal health and immune status.

Adults born before 1956 are generally considered to be immune to measles, mumps, and rubella by prior infection. For persons born after 1956 whose immunologic status is unclear or who are at significant risk of exposure to these diseases (e.g., persons entering healthcare careers), reimmunization is recommended (Centers for Disease Control and Prevention [CDC], 2002c).

Tetanus and diphtheria toxoids are combined in a single immunization. The pediatric form of the vaccine is known as DT; the adult form is Td. The vaccine stimulates active immunity by inducing the production of antibodies and antitoxins. After an initial series of three immunizations, an intramuscular (IM) booster injection of 0.5 mL is recommended every 10 years to maintain protection. Older clients, particularly those who never entered the workforce (e.g., older female adults), may have never received the initial series of DT vaccine.

Hepatitis B (HB) vaccine is given as a series of three immunizations to promote active immunity to hepatitis B. This vaccine is recommended for everyone at high risk for exposure through blood or other body fluids. It is mandated by the Occupational Health and Safety Administration (OSHA) for all healthcare workers at risk. Other high-risk populations include intravenous drug users, sexual partners of infected individuals, clients on hemodialysis, prison guards, and athletic coaches.

Influenza vaccine is recommended for persons at high risk for serious sequelae of influenza, including older adults, persons with lung disease or other chronic illness, and immunosuppressed individuals. The antigenic strain included in influenza vaccine varies each year according to the predicted predominant strains affecting the population. Yearly reimmunization is therefore required.

Pneumococcal vaccine is generally recommended for the same populations as influenza vaccine. A single dose of this vaccine confers lifetime immunity, although repeating immunization every 6 years may be considered for high-risk clients. Pneumococcal vaccine for all senior citizens is a U.S. public health and Medicare goal. The purpose of immunization is to prevent respiratory infections and hospitalizations (CMS, 2003; Quinley & Shih, 2004; Shorr, 2005).

In addition to routine immunizations, people traveling outside the United States and Canada should receive vaccines against diseases that are endemic in certain regions of the world.

Other immunologic substances may be administered as indicated. Immune globulins provide passive immunity as protection against a known or potential exposure to an antigen. Standard immune globulin is given to household contacts of clients with hepatitis A and persons traveling to areas in which

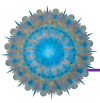
TABLE 12–5 **Recommended Immunizations for Adults**

VACCINE	TYPE	DOSE	INDICATIONS	PRECAUTIONS AND NURSING IMPLICATIONS
Measles-mumps-rubella (MMR)	Live virus	0.5 mL SC	All adults born after 1956, particularly those who are at risk for infection, such as college students and military recruits. Measles and mumps vaccination particularly recommended for males without history of previous infection; rubella vaccination recommended for all seronegative females.	As a live virus vaccine, should not be administered to pregnant women or immunocompromised clients. Do not administer to clients with a history of anaphylactic reaction to egg protein or neomycin.
Tetanus and diphtheria toxoids (Td)	Inactivated toxins	0.5 mL IM	Initial series of 3 injections (2 doses, 4 to 6 weeks apart; third dose 6 to 12 months after dose 2) if never immunized; booster every 10 years; following a major or contaminated wound if more than 5 years since last booster.	Do not give in first trimester of pregnancy or to clients with a history of anaphylactic reaction to horse serum; administer deep IM in deltoid of dominant arm.
Hepatitis B (HB)	Inactive viral antigen	1.0 mL IM	Series of 3 doses: initial and at 1 and 6 months. Recommended for anyone at risk for exposure and for postexposure prophylaxis.	Use with caution in pregnant or lactating females, older clients, and clients with active infection; have epinephrine 1:1000 available on unit in case of anaphylaxis and laryngospasm.
Influenza	Inactivated virus or viral components	0.5 mL IM	Yearly for all clients over age 65 and those at risk for complications, including debilitated clients and clients with chronic disease.	Do not administer to acutely ill clients or clients with history of anaphylactic reaction to egg protein.
Pneumococcal	Bacterial polysaccharides	0.5 mL IM or SC	One dose for clients over age 65 and those at risk for pneumococcal pneumonia, including clients with chronic lung disease or other chronic diseases.	Do not administer to pregnant women.

it is endemic. HBIG contains higher titers of antibody to hepatitis B virus and is used for persons exposed by blood or sexual contact. Following confirmed or suspected contact with a pathogen, selected vaccines may be administered to stimulate an immediate immune response (Table 12–6).

For most vaccines, a sensitivity test should be performed prior to administration to detect sensitivity to substances such as horse serum or eggs. The substance is injected intradermally; if after 20 minutes there is no evidence of a reaction, the selected vaccine can be administered.

Moderate to severe local reactions may occur following administration of an immunization. Common reactions include redness, swelling, tenderness, and muscle ache. Administering the vaccine in the dominant arm of the client helps minimize local reactions, because use and movement of the arm facilitates absorption of the solution. Applying heat to the site is also beneficial. Occasionally local ulcerations occur; when they do, warm, wet pack, or sterile wet-to-dry dressings may be prescribed.



NURSING CARE

Maintaining a population that is fully immunized against common, potentially epidemic, and devastating diseases is a major public health task for nursing. Nurses not only recommend and administer vaccines to individual clients and their families, but also plan and implement preventive care for whole communities.

Although this process may appear to be straightforward, multiple issues affect society's ability to immunize the entire population. For some people, for example, religious beliefs may preclude the use of immunizations to prevent disease. Also, people who are not citizens and the medically indigent population have difficulty accessing immunization services. Lack of immunization not only puts the individual at increased risk for infectious disease, but also increases the cost of medical services and the possibility of exposing immunocompromised people to disease.

Health Promotion

In the public health setting, the nurse looks at the immunization needs and illness risk for an entire community. Communities include not only cities and localities but also groups of people, such as college populations and employees in a workplace. Public education needs may be met through presentations to groups of people, feature articles in newspapers and other local publications, advertising, radio presentations and public service announcements, and one-to-one discussion and teaching.

TABLE 12–6 **Preparations for Postexposure Prophylaxis**

DISEASE	PREPARATION AND DOSE	INDICATIONS
Hepatitis A	Human immune globulin (IG), 0.02 mL/kg IM	Contacts in day care centers, households, custodial institutions; patrons of eating establishments known to have been exposed by infected food worker
Hepatitis B	Hepatitis B immune globulin (HBIG), 0.06 mL/kg IM	Possible percutaneous or sexual contact with blood or body fluids of an infected individual; usually given concurrently with hepatitis B vaccine
Varicella	Varicella-zoster immune globulin (VZIG), 12.5 units/kg; minimum dose 125 units, maximum 625 units	Susceptible adults exposed to varicella virus (e.g., chickenpox or shingles)
Tetanus	Tetanus immune globulin (TIG), 500 to 3000 units IM (part infiltrated around wound)	Clients with major or contaminated wounds who have no history of tetanus immunization or an unclear one that is not up to date; Td usually given as well
Rabies	Human rabies immune globulin (HRIG), 20 IU/kg, half IM, half infiltrated around wound	Persons with a significant exposure to a rabid or potentially rabid animal; followed with 5-dose course of rabies vaccine
Measles	Human immune globulin (IG), 0.25 mL/kg IM	Susceptible close contacts, especially people who are immunosuppressed; postpone immunization with measles vaccine until 3 months after IG
Rubella	Human immune globulin (IG), 0.55 mL/kg IM	Pregnant women exposed in first trimester when termination of pregnancy is not an option; does not ensure protection of fetus

Source: Table adapted from *Harrison's Principles of Internal Medicine* (14th ed.) by A. S. Fauci et al. (Eds.), 1998, New York: McGraw-Hill.

Assessment

Collect the following data through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

- *Health history:* age, medication use (corticosteroids and antibiotics) and blood transfusion, nutrition, allergies, infection, immunizations, autoimmune disorders, chronic diseases such as diabetes mellitus, cancer
- *Physical assessment:* skin lesions or rashes, breath sounds, respiratory rate.

Nursing Diagnoses and Interventions

Nursing care focuses on preventing injury from the immunization and educating the client. See the accompanying Nursing Care Plan on the following page.

Health-Seeking Behaviors: Immunization

For individual clients and their families, nurses promote immunocompetence by assessing immune status, recommending appropriate immunizations, and administering vaccines as ordered or indicated. Once a person reaches adulthood, routine immunizations often become a neglected part of health care.

- Determine knowledge level, understanding, attitudes, and religious beliefs about immunization. *This provides a basis for further education and determines if religious beliefs may contraindicate immunization.*
- Discuss the value and reasons for recommended immunizations. *Understanding promotes adherence.*
- Reinforce positive health-seeking behaviors. *This will help promote future health maintenance activities.*
- Using recommended immunization schedules, develop a plan to attain optimal immunization status. *Adherence with recommended schedules for immunization is important in preventing disease and disability.*
- Do not administer MMR or influenza vaccine if allergic to eggs, or tetanus antitoxin if sensitive to horse serum. *Vaccines prepared from chicken or duck embryos are contraindicated in clients who are allergic to eggs. Tetanus antitoxin is prepared from horse serum. Both will cause a severe allergic reaction.*
- Withhold administration of active immunologic products in the presence of an upper respiratory infection or other infection. *Active immunizations can cause a greater inflammatory reaction in the presence of infections.*
- Do not administer oral polio vaccine, MMR, or any live virus vaccine to immunosuppressed clients or to clients who are in close household contact with an immunosuppressed person. *Live virus vaccines can cause disease in the immunosuppressed client. The virus may be transmitted from close household contacts during the initial postvaccination period.*
- Do not administer live attenuated virus vaccines and passive immunizations such as gamma globulin simultaneously. *Passive antibodies interfere with the response of the live attenuated virus.*
- Prior to administering prescribed vaccine, check the expiration date and manufacturer's instructions. *Outdated vaccines cannot provide adequate immunization protection. Certain injection sites have better absorption than others.*
- Keep epinephrine 1:1000 readily available for subcutaneous injection when administering immunizations. *Epinephrine causes vasoconstriction and reduces laryngospasm; in acute anaphylaxis, it can be lifesaving.*

NURSING CARE PLAN A Client with Acquired Immunity

Terry Adams is a 48-year-old executive who is planning a trip to central Africa. In preparation, he contacts his local healthcare provider to obtain the necessary immunizations. Jane Wong, the registered nurse in the clinic, obtains a nursing history of Mr. Adams.

ASSESSMENT

Mr. Adams's history reveals that he has always been very healthy and active, apart from a mild case of asthma. As an adult, he has had little problem with his asthma "except for those rare occasions on which I am dumb enough to smoke more than one cigarette!" He is divorced and is not currently in a continuing relationship. He has two grown daughters with whom his relationship is good. Since contracting hepatitis A several years ago, he drinks alcohol only rarely, and never more than one or two drinks at any one time. He confesses to little organized exercise but plays golf two or three times a week and states that he is such a hyperactive workaholic that he rarely sits for any length of time. Mr. Adams has not seen a physician since recovering from the hepatitis and is unsure when he last received any immunizations. He does not know if he had all recommended childhood immunizations, but recalls getting both Salk and Sabin polio vaccines when they became available. His physical examination reveals an alert and healthy individual with no abnormalities noted. His vital signs are as follows: T 97.4°F, P 64, R 14, and BP 142/82.

The physician orders the following immunizations for Mr. Adams:

- Measles-mumps-rubella (MMR)
- Combined tetanus and diphtheria toxoids (Td)
- Yellow fever vaccine
- Typhoid vaccine
- Meningococcal meningitis vaccine

DIAGNOSES

- *Health-Seeking Behaviors: Immunization* related to impending international travel
- *Ineffective Health Maintenance* related to apparent lapse in immunization status
- *Risk for Injury* related to adverse response to immunization

EXPECTED OUTCOMES

- Obtain necessary immunizations.
- Verbalize a schedule for maintaining up-to-date immunization status.
- Experience no significant adverse effects from immunization.

PLANNING AND IMPLEMENTATION

- Administer MMR, Td, and meningococcal meningitis vaccines prior to discharge from clinic.
- Observe closely for 30 minutes following immunization for potential adverse responses.
- Schedule return visit in 1 week for typhoid vaccine.
- Provide referral to a registered vaccination center for yellow fever vaccine and documentation of vaccination.
- Provide instructions for comfort measures to relieve local and systemic adverse effects of vaccines. Provide written instructions on manifestations that should be reported to the physician.
- Document immunizations on a permanent record at the clinic and for the client.

EVALUATION

Terry Adams completes his prescribed immunizations without major adverse effects, although he does complain of fever, malaise, and general achiness for several days following the typhoid vaccination. His trip to Africa is successful, and he returns to the United States without contracting any infectious diseases.

CRITICAL THINKING IN THE NURSING PROCESS

1. Explain why it is important for adults to continue receiving immunizations throughout their life span.
2. If a client says to you, "I don't believe in immunizations. I hear they are dangerous," how would you respond?
3. When should a client contact the primary caregiver after receiving an immunization?

See Evaluating Your Responses in Appendix C.

PRACTICE ALERT

Observe the client for 20 to 30 minutes following inoculation to monitor for possible adverse reactions.

Community-Based Care

Educating individual clients, families, and the public about the maintenance of immune status is a significant nursing responsibility. For individual clients and their families, instructions focus on the following areas:

- Appropriate immunizations and recommended schedules for initial vaccination and boosters
- How and where to obtain immunizations
- The need to observe the client for up to 30 minutes following a vaccination for possible adverse reactions
- Possible side effects and adverse effects of the immunization administered
- Self-care measures for side effects and postvaccination discomfort
- Responses to immunization that should be reported immediately to the primary care provider
- Maintenance of a permanent immunization record
- Beneficial resources:
  - County or public health departments
  - Centers for Disease Control and Prevention National Immunization Program
  - National Institute of Allergy and Infectious Diseases

NORMAL IMMUNE RESPONSES

The Client with Tissue Inflammation

As noted, inflammation is a nonspecific response to injury that serves to destroy, dilute, or contain the injurious agent or damaged tissue. Inflammation may be either acute or chronic.

MEDIALINK Community Resources

Acute inflammation is a short-term reaction of the body to all types of tissue damage. It is immediate and aimed at protecting the body and preventing further invasion or injury. Acute inflammation usually lasts less than 1 to 2 weeks. Once the injurious agent is removed, the inflammation subsides. Healing with tissue repair or scar formation occurs, and the body functions in normal or near-normal capacity.

Chronic inflammation is slower in onset and may not have an acute phase. Its clinical manifestations occur over months or years. It involves cell proliferation and is debilitating, with long-term adverse effects. There is increased cellular exudate, necrosis, fibrosis, and sometimes tissue scarring, resulting in severe tissue damage.

Pathophysiology of Tissue Inflammation

The tissue damage that evokes an inflammatory response may be caused by specific or nonspecific agents. These agents may be *exogenous,* from outside the body, or *endogenous,* from within the body. Causes of inflammation include the following:

- Mechanical injuries, such as cuts or surgical incisions
- Physical damage, such as burns
- Chemical injury from toxins or poisons
- Microorganisms, such as bacteria, viruses, or fungi
- Extremes of heat or cold
- Immunologic responses, such as hypersensitivity reactions
- Ischemic damage or trauma, such as a stroke or myocardial infarction.

Acute Inflammation

Regardless of the cause, location, or extent of the injury, the acute inflammatory response follows the previously outlined sequence of vascular response, cellular and phagocytic response, and healing.

Many of the manifestations of inflammation are produced by inflammatory mediators such as histamine and prostaglandins released when tissue is damaged (see Table 12–7 and Box 12–1 on inflammatory mediators).

The cardinal signs of inflammation include the following:

- Erythema (redness)
- Local heat caused by the increased blood flow to the injured area (hyperemia)
- Swelling due to accumulated fluid at the site
- Pain from tissue swelling and chemical irritation of nerve endings
- Loss of function caused by the swelling and pain.

The degree of functional loss depends on the location and extent of the injury. With increased tissue damage, more fluid exudate is formed, resulting in more swelling, pain, and functional impairment. Pain may be immediate or delayed. Prostaglandins intensify and prolong the pain. Kinins cause irritation to the nerve endings and contribute to the pain sensation.

Dead neutrophils, necrotic tissue, and, if the tissue is infected, digested bacteria accumulate as a result of inflammation and phagocytosis, forming *pus*. Pus usually forms and remains until after the infection subsides. Pus may push itself to the surface of the body or become internalized. In the latter case, pus is gradually autolyzed (self-digested) by enzymes over a period of days. The end product is then absorbed by the body. On occasion, pus may remain after the infection is resolved. Pockets of pus, called abscesses, may need to be artificially drained with a procedure called *incision and drainage (I&D).* Ectopic calcifications are another possible result of residual collections of pus.

Systemic responses to inflammation include an increase in the size of lymph nodes due to the proliferation of macrophages within the nodes in response to microorganisms in the lymph. Enlarged lymph nodes are usually noted in the groin, axillae, and neck (Figure 12–11 ■). Fever, often precipitated by inflammatory mediators or bacterial toxins, inhibits the growth of many microorganisms and increases tissue repair functions. Loss of appetite and fatigue may occur in the effort to conserve energy during the inflammatory process. Leukocytosis occurs with increased WBC production to support inflammation and phagocytosis.

Local and systemic manifestations of inflammation are summarized in the box below.

MANIFESTATIONS of Inflammation

Local Manifestations

- Erythema
- Warmth
- Pain
- Edema
- Functional impairment

Systemic Manifestations

- T > 100.4°F (38°C) or <96.8°F (36°C)
- P > 90/min
- R > 20/min (tachypnea)
- WBC > 12,000/mm^3 or >10% bands

TABLE 12–7 **Major Chemical Mediators of Inflammation**

FACTOR	SOURCE	EFFECT
Histamine	Mast cells, basophils, and platelets	Vasodilation and increased capillary permeability, producing tissue redness, warmth, and edema
Kinins (bradykinin and others)	Plasma protein factors	Histamine-like effects; chemotaxis and pain inducers
Prostaglandins	Metabolism of arachidonic acid from cell membranes	Histamine-like effects; chemotaxis, pain, and fever inducers
Leukotrienes	Arachidonic acid metabolism	Smooth muscle constriction (especially bronchoconstriction), increased vascular permeability, chemotaxis

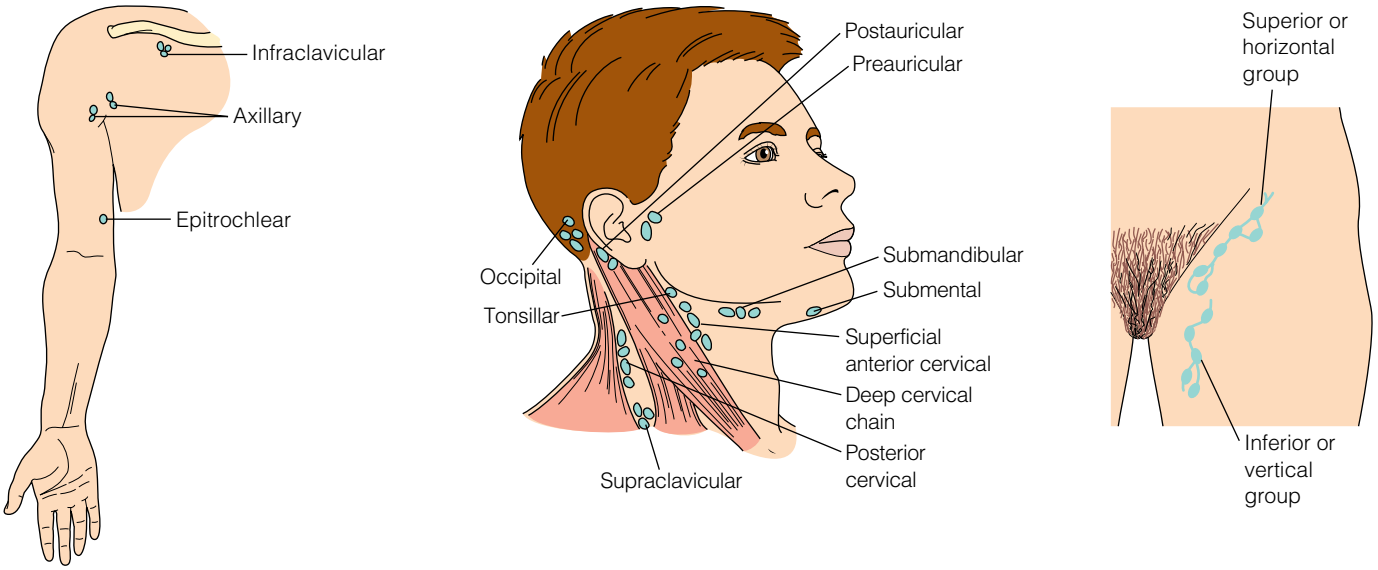


Figure 12–11 ■ Lymph nodes that may be assessed by palpation.

Chronic Inflammation

Whereas acute inflammation is a self-limiting process lasting less than 2 weeks, chronic inflammation tends to be self-perpetuating, lasting weeks to months or years. Chronic inflammation may develop when the acute inflammatory process has been ineffective in removing the offending agent. For example, mycobacteria have cell walls with high lipid and wax content, making them resistant to phagocytosis. Chronic inflammation and granuloma formation is common with *Mycobacterium tuberculosis* infection. Persistent irritation by chemicals, particulate matter, or physical irritants such as talc, asbestos, or silica may also result in chronic inflammation.

The chronic inflammatory process is characterized by a dense infiltration of the site by lymphocytes and macrophages. The macrophages mass or coalesce to form a multinucleated giant cell surrounded by lymphocytes, in a lesion called a *granuloma.* The granuloma is effective in walling off the offending agent, isolating it from the rest of the body; however, the infectious agent or offending irritant may not be destroyed and can survive within the granuloma for a long period of time. The granuloma formed in tuberculosis is called a tubercle. *M. tuberculosis* can survive for many years within the tubercle, emerging when the client's immune system is no longer able to contain it.

Osteoarthritis is another condition hallmarked by chronic inflammation. Inflammation in the joint capsule makes movement of the joint very painful. The capsule is enlarged with inflammatory exudates, which cause stretching of the capsule for movement to occur. This is not an infectious process; it is a disease process that stimulates the inflammatory processes.

Complications

Inflammation and wound healing are highly metabolic processes that may be affected by a number of factors. Without adequate nutrition, blood supply, and oxygenation, tissues cannot effectively complete the process. Impaired inflammatory and immune processes can interfere with phagocytosis and preparation of the wound for healing. Infection prolongs the inflammatory process and delays healing.

Chronic diseases may also impair healing. Diabetes mellitus is a prominent example. With high blood glucose levels associated with poorly controlled diabetes, chemotaxic and phagocytic function are decreased. Collagen formation and tensile strength of the wound are also impaired. Small blood vessel disease is common in people with diabetes, a factor that further impairs the healing process.

Drug therapy, particularly corticosteroid medications, may suppress the immune and inflammatory responses, delaying healing (Porth, 2005). Other external factors, such as exposure to ionizing radiation and wound cleansing agents, can also affect healing. Table 12–8 summarizes major factors that affect the inflammatory process and wound healing.

INTERDISCIPLINARY CARE

Management of the client with inflamed tissue focuses on promoting healing. Care is generally supportive, allowing the client's own physiologic processes to remove foreign matter and damaged cells. Wound care may be minimal, involving only simple cleaning, or extensive, involving irrigations and debridement. The client is encouraged to rest, to increase fluid intake, and to eat a well-balanced, nutritious diet. Anti-inflammatory medications are administered only when the inflammatory process has become problematic. With orthopedic cases, anti-inflammatory medications are avoided because they may retard bone formation and healing (Dahners & Mullis, 2004). Antibiotics may also be prescribed to help eliminate infectious causes of inflammation.

Diagnosis

The following diagnostic tests may be ordered to identify the source and extent of inflammation. An important part of the

TABLE 12–8 Factors That May Impair Healing

FACTOR	EFFECT
Malnutrition	
Protein deficit	Prolongs inflammation and impairs healing process
Carbohydrate and kilocalorie deficit	Impairs metabolic processes and promotes catabolism; proteins are used for energy rather than for healing
Fat deficit	Impairs cell membrane synthesis in tissue repair
Vitamin deficits	
Vitamin A	Limits epithelialization and capillary formation
B-Complex	Inhibits enzymatic reactions that contribute to wound healing
Vitamin C	Impairs collagen synthesis
Tissue hypoxia	Associated with an increased risk of infection and impaired healing, because oxygen is required to support cell function and collagen synthesis
Impaired blood supply	Inadequate delivery of oxygen and nutrients to healing tissues and removal of waste products
Impaired inflammatory and immune processes	Decreased phagocytosis and wound debridement; increased risk of infection; delayed healing

assessment of the client with an inflammation is monitoring the results of these tests.

- *WBC with differential* provides information about the type and extent of inflammatory response. The differential count (the percentage of the total WBC made up by each type of leukocyte) provides further clues about inflammatory processes (Table 12–9).
- *Erythrocyte sedimentation rate* (*ESR* or *sed rate*) is a nonspecific test to detect inflammation. The rate at which RBCs fall to the bottom of a vertical tube is an indicator of inflammation. An increased ESR may indicate acute or chronic inflammation, tuberculosis, autoimmune disorders, some malignancies, and nephritis. Decreased ESR is found in congestive heart failure, sickle cell anemia, and polycythemia vera.
- *C-reactive protein (CRP) test* is used to detect CRP. This abnormal glycoprotein is produced by the liver and is excreted into the bloodstream during the acute phase of an inflammatory process. The expected result of this test is negative for CRP. A positive result indicates an acute or chronic inflammatory process. It may also indicate the client's response to therapy, because it decreases when inflammation subsides.

In addition to the above diagnostic tests, cultures of the blood and other body fluids may be ordered to determine if infection is the cause of inflammation.

Medications

Medications may be prescribed for the client with an inflammatory response to help alleviate distressing symptoms or destroy infectious agents.

Acetaminophen (Tylenol) may be administered to reduce the fever and pain associated with inflammation. Acetaminophen has no anti-inflammatory effect; it will not reduce the inflammatory process but will relieve associated symptoms. Acetaminophen decreases fever by acting directly on the hypothalamus heat-regulating center. It also works on the central nervous system to relieve pain sensations.

Antibiotics may be used either prophylactically to prevent infection from interfering with the healing process of damaged tissue, or therapeutically to treat the infection. If infection is present, the organism and its response or sensitivity to various antibiotics are used to guide therapy. Antibiotic therapy is presented in greater depth in the section of this chapter on infectious diseases.

Although inflammation is a beneficial process to prepare acutely injured tissue for healing, it can have damaging effects as well. When these effects are a concern or the manifestations of inflammation are deleterious to the client, anti-inflammatory medications may be prescribed. Anti-inflammatory medications fall into three broad groups: salicylates, such as aspirin; other nonsteroidal anti-inflammatory drugs (NSAIDs); and corticosteroids.

Aspirin (also called acetylsalicylic acid, or ASA) is an NSAID that has antipyretic, analgesic, and antiplatelet effects. Its beneficial effects are largely dose related. Low doses (as little as 81 mg/day) inhibit platelet aggregation and normal blood clotting. Higher doses (650 to 1000 mg 4 to 5 times per day) are required to accomplish its anti-inflammatory effects. However, 650 mg of aspirin is an effective analgesic and antipyretic dosage. To relieve pain, aspirin acts primarily on peripheral sensory nerves by inhibiting the synthesis of prostaglandins and kinins, which are chemical stimuli of sensory nerves. As an antipyretic, aspirin acts both centrally and peripherally. It inhibits the formation of pyrogenic substances that raise the hypothalamic thermostat. It also dilates peripheral blood vessels and promotes diaphoresis, increasing the dissipation of heat (Wilson, 2006).

In therapeutic doses, aspirin mediates the inflammatory process by inhibiting the synthesis of prostaglandins and acting on the mobility and activation of leukocytes. Inflammation is reduced, along with the swelling, redness, and impaired function that accompanies it.

The other NSAIDs have activity similar to that of aspirin. They inhibit prostaglandin synthesis, reducing the inflammatory and pain response. NSAIDs fall into the following classifications:

- *Salicylates,* which include aspirin and related compounds
- *Acetic acids,* including indomethacin (Indocin), ketorolac (Toradol), sulindac (Clinoril), and tolmetin (Tolectin)
- *Propionic acids,* including ibuprofen (Motrin and numerous nonprescription preparations), fenoprofen (Nalfon), and naproxen (Naprosyn)
- *Fenamates,* including meclofenamate (Meclomen)
- *Pyrazoles,* including phenylbutazone (Butazolidin)
- *Oxicams,* including piroxicam (Feldene).

Each group has a slightly different mode of action for prostaglandin inhibition. Clients may have varying degrees of relief with different NSAIDs; sometimes, several different

TABLE 12–9 **The White Blood Cell Count and Differential**

CELL TYPE AND NORMAL VALUE	INCREASED	DECREASED
Total WBCs: 4000 to 10,000 per mm^3	*Leukocytosis:* Infection or inflammation, leukemia, trauma or stress, tissue necrosis	*Leukopenia:* Bone marrow depression, overwhelming infection, viral infections, immunosuppression, autoimmune disease, dietary deficiency
Neutrophils (segs, PMNs, or polys): 55% to 70%	*Neutrophilia:* Acute infection or stress response, myelocytic leukemia, inflammatory or metabolic disorders	*Neutropenia:* Bone marrow depression, overwhelming bacterial infection, viral infection, Addison's disease
Eosinophils (eos): 1% to 4%	*Eosinophilia:* Parasitic infections, hypersensitivity reactions, autoimmune disorders	*Eosinopenia:* Cushing's syndrome, autoimmune disorders, stress, certain drugs
Basophils (basos): 0.5% to 1%	*Basophilia:* Hypersensitivity responses, chronic myelogenous leukemia, chickenpox or smallpox, splenectomy, hypothyroidism	*Basopenia:* Acute stress or hypersensitivity reactions, hyperthyroidism
Monocytes (monos): 2% to 8%	*Monocytosis:* Chronic inflammatory disorders, tuberculosis, viral infections, leukemia, Hodgkin's disease, multiple myeloma	*Monocytopenia:* Bone marrow depression, corticosteroid therapy
Lymphocytes (lymphs): 20% to 40%	*Lymphocytosis:* Chronic bacterial infection, viral infections, lymphocytic leukemia	*Lymphocytopenia:* Bone marrow depression, immunodeficiency, leukemia, Cushing's syndrome, Hodgkin's disease, renal failure

Source: Data from Corbett, J. V. (2004). Laboratory tests and diagnostic procedures with nursing diagnoses (6th ed.), Upper Saddle River, NJ: Prentice Hall and *Diagnostic and Laboratory Test Reference* (3rd ed.) by K. D. Pagana and T. J. Pagana, 1997, St. Louis, MO: Mosby-Year Book.

agents must be tried before the most effective is identified. Side effects also differ to a certain extent; however, all have a potential cross-sensitivity with aspirin, all irritate the gastrointestinal tract, and all cause some degree of sodium and water retention. They also are more costly than aspirin, but they have a longer duration of action; therefore, fewer daily doses are required to achieve the desired effect. Indomethacin and phenylbutazone are the most toxic of the NSAIDs. Their use is limited to short-term therapy. (See Chapter 9 ∞ for further information on NSAIDs.)

For acute hypersensitivity reactions, such as reactions to poison oak, or for inflammation that cannot be managed by aspirin or NSAID therapy, corticosteroid therapy may be prescribed. The glucocorticoids are hormones produced by the adrenal cortex that have widespread effects on body metabolism and the immune response. Glucocorticoids inhibit inflammation and may be lifesaving in acute fulminating or chronic progressive inflammation. They do not cure disease; they are palliative to manage the inflammatory process.

When glucocorticoids are prescribed to manage inflammation, the following principles are used to guide therapy:

- The smallest possible effective dose is used.
- If a local-acting preparation such as a topical agent or intra-articular injection will be effective, it is prescribed.
- To minimize suppression of adrenal gland activity, an alternate-day dose schedule is used when possible.
- High-dose corticosteroid therapy is rarely stopped abruptly, but tapered, allowing the client's adrenal glands to resume normal function.

The incidence of potentially harmful side effects increases with higher doses and prolonged therapy.

The nursing implications of caring for a client receiving corticosteroid medications are discussed in Chapter 26 ∞.

Nutrition

Healing depends on cell replication, protein synthesis, and the function of specific organs—the liver, heart, and lungs in particular. Weight loss and protein depletion are risk factors for poor healing and wound complications. Even a few days of severely impaired nutritional intake can noticeably affect healing.

The client with an inflammatory process or healing wound requires a well-balanced diet of sufficient kilocalories to meet the metabolic needs of the body (see Table 12–8). Inflammation often produces *catabolism,* a state in which body tissues are broken down. Healing, by contrast, is a process of *anabolism,* or building up. Without sufficient kilocalories and nutrients, catabolism may predominate, impairing healing.

Carbohydrates are important to meet energy demands, as well as to support leukocyte function. However, hyperglycemia experienced in diabetes may impair healing. In diabetes, the glucose molecules bind the oxygen more tightly to the hemoglobin molecule and prevent adequate release of oxygen to the tissues for healing. In diabetes this is at least the partial explanation for impaired healing (McCance & Huether, 2002). Adequate protein is necessary for tissue healing and the production of antibodies and WBCs. Lack of adequate protein increases the risk of infection. Complete protein sources, those that provide the essential amino acids, are preferred. Dietary fats are used in the synthesis of cell membranes.

Vitamins A, B-complex, C, and K are also important to the healing process. Vitamin A is necessary for capillary formation and epithelialization. B-complex vitamins promote wound healing, and vitamin C is necessary for collagen synthesis. Vitamin K provides a vital component for the synthesis of clotting factors in the liver.

Although it has been established that minerals contribute to the inflammatory and healing processes, less is known about

required amounts. Zinc appears to be important for tissue growth, skin integrity, cell-mediated immunity, and other general immune mechanisms (Arnold & Barbul, 2006). However, the use of zinc supplements in smokers is cautioned against and sources in whole foods are recommended over supplements. (See Chapter 21 ∞ for a detailed discussion on nutrition.)

NURSING CARE

Acute inflammation may be self-limiting or extensive and require hospitalization. Nursing care includes teaching clients with acute and chronic inflammatory conditions self-management at home.

Health Promotion

Health promotion activities to prevent inflammation focus on reducing the risk for accidents and exposure to harmful agents that can result in subsequent injury. It is important to educate the public about potential hazards in both the work and home environments. In addition, safety education guidelines such as not drinking and driving, wearing a protective helmet when riding a bicycle, and using a safety belt in the car are important areas for discussion. Because most injuries occur at home, it is also important to discuss ways to make the home safer.

Assessment

The following data are collected through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

- *Health history:* risk factors, nutrition, medication use (anti-inflammatory and corticosteroids), location, duration, and type (redness, heat, pain, swelling, and impaired function) of symptoms
- *Physical assessment:* movement of injured area, circulation, wounds, lymph nodes.

Nursing Diagnoses and Interventions

The nursing care needs of the client with an inflammatory process are related to the manifestations of inflammation (pain in particular) and altered tissue integrity. Priority nursing diagnoses include *Pain, Impaired Tissue Integrity,* and *Risk for Infection.*

Pain

Along with redness, warmth, swelling, and impaired function, pain is one of the cardinal manifestations of inflammation. Depending on the cause, affected area, and degree of inflammation, pain may be acute and immobilizing or chronic and demoralizing. It is important to remember that pain is a subjective experience and that client responses to pain vary. (Refer to Chapter 9 ∞ for more information about pain and pain management.)

- Assess pain using a scale of 0 to 10, with 0 being no pain and 10 being the worst pain; note the character and location of the pain. *Because pain is subjective, the client provides the most accurate information regarding his or her pain experience.*
- Use physical and nonverbal cues to further assess the level of pain. *This intervention is especially important if the client is nonverbal or tends to underreport pain.*
- Administer anti-inflammatory medications as prescribed. *These medications help reduce the pain resulting from acute inflammation.*
- Administer analgesic medications as prescribed. *Although most analgesics do little to reduce inflammation, they provide additional pain relief by reducing the perception of pain.* Chest infections such as pneumonia may compromise breathing. Do not hesitate to administer opioids to ease the pain of breathing. In addition to applying oxygen at appropriate levels (low for those with chronic obstructive pulmonary disease to preserve the oxygen drive to breathe), relieving pain helps increase the depth and slow the rate of respirations. Because opioids can depress respirations, it is important to monitor oxygen saturation and encourage the patient to take deep breaths to keep oxygen saturation adequate.
- Monitor effectiveness of interventions. *Results may call for modifications in the regimen.*
- Provide comfort measures, such as back rubs, position changes, or relaxation techniques. *These measures reduce muscle tension, relieve areas of pressure, and provide distraction.*
- Encourage activities such as reading, watching television, and taking part in social interactions. *Such activities provide distraction from the pain experience.*
- Encourage rest. *Strenuous activity or exercising an inflamed body part may increase discomfort and tissue damage.*
- Provide cold or heat as pain-relief measures, as ordered. *For an acute injury, cold reduces swelling and relieves pain; after the initial stage, heat increases blood flow to the affected tissue and relieves pain and swelling by promoting absorption of edema. Either heat or cold may be contraindicated with some inflammatory processes; for example, if the appendix is acutely inflamed, applying heat to the abdomen may prompt the appendix to rupture, increasing the risk of peritonitis. If unsure, check with the client's primary care provider.*

PRACTICE ALERT

Use heat or cold application cautiously in older clients who have fragile skin and are at risk for tissue injury.

- Elevate the inflamed area if possible. *Elevation promotes venous return and reduces swelling.*
- Teach about the appropriate use and expected effects of anti-inflammatory medications. *If the client's pain continues after the initial doses of anti-inflammatory medication, he or she may become discouraged and stop taking the medication before it becomes fully effective.*

Impaired Tissue Integrity

The inflammatory response can either precipitate or result from an impairment in the integrity of skin, support, or other tissues. Whatever the cause of the tissue alteration, it is vital that the nurse consider this alteration in delivering care.

- Assess general health and nutritional status. *Poor general health or chronic diseases such as diabetes mellitus or renal failure interfere with the healing processes and increase the risk of infection.*

- Assess circulation to the affected area. *Adequate tissue perfusion and oxygenation are necessary for healing.*
- Monitor the skin and surrounding tissue for increased signs of inflammation. *Inflammation can spread to adjacent tissues leading to conditions such as cellulitis.*
- Provide protection and support for inflamed tissue. *This reduces discomfort and decreases the risk of further tissue damage.*
- Clean inflamed tissue gently; if possible, use water, normal saline, or nontoxic wound cleansers such as Comfeel (Coloplast Corporation) only. *Soap and harsh cleansers such as povidone-iodine (Betadine) and hydrogen peroxide can cause further drying and tissue damage. Granulation tissue in a healing wound is fragile and easily damaged.* (See Chapter 4 ∞ for further discussion of wound care.)
- Keep the inflamed area dry, and expose it to air as much as possible. *This promotes healing and helps prevent infection.*
- Balance rest with the tolerable degree of mobility. *Rest decreases metabolic demands and allows for cell regeneration while mobility helps to promote oxygenation and perfusion of the tissues.*
- Provide supplemental oxygen as ordered. *Supplemental oxygen improves tissue oxygenation and reduces hypoxia.*
- Provide a well-balanced diet with adequate kilocalories to meet the body's metabolic and healing needs. If the client is allowed nothing by mouth (NPO), suggest parenteral or enteral nutrition. For the client who is unable to consume an adequate diet, consult with a dietitian for between-meal supplements and/or multivitamin supplements. *Careful attention to diet and nutrient intake is important to provide the nutrients necessary for immune function and healing and to prevent catabolism.*

Risk for Infection

The inflammatory response often indicates that body defense mechanisms have been set in motion to protect against invading microorganisms. Wounds, whether traumatic or surgical in nature, are typically contaminated, as attested to by subsequent wound infections. The client with a healing wound is at particular risk for infection.

- Assess the wound for specific signs of infection, including purulent drainage, odor, and poor healing. *The normal inflammatory response can indicate infection and, on occasion, mask its presence.*
- Evaluate complete blood counts for adequate WBC response. *Leucocytosis may indicate infection or healthy response to injury and protection from infection. Immune-impaired clients may not respond with increased WBCs; signs of inflammation may be diminished in those individuals.*
- Monitor temperature, pulse, and respirations at least every 4 hours. *In response to the inflammatory process the temperature rises, usually in the range of 99°F (37.2°C) to 100.9°F (38.2°C). A temperature of 101.0°F (38.3°C) or above indicates infection. Fever is usually accompanied by increased heart and respiratory rates.*
- Culture purulent or odorous wound drainage. *Wound culture is used to determine the infectious organism and to direct antibiotic therapy.*
- Apply dry or moist heat to the affected area for no longer than 20 minutes several times a day. *Heat increases the circulation of blood to and from the inflamed tissue. Time is limited to prevent burns.*
- Provide fluid intake of 2500 mL/day. *Adequate hydration promotes blood flow and nutrient supply to the tissues and also dilutes and removes waste products from the body.*
- Ensure adequate nutrition. *Adequate nutrition enhances the function and production of T cells and B cells, which are important in the immune response.*
- Use good hand washing techniques. *Hand washing removes transient microorganisms and is the best mechanism to prevent the spread of infection to a susceptible person.*
- Wear sterile gloves when providing wound care. *Using sterile gloves helps prevent further contamination of the wound and the spread of infection to other clients.*

Community-Based Care

Client and family teaching enhances understanding of the inflammatory process, its cause, and its management. Teaching is also important to prevent further compromise that could result in infection.

Instructions, verbal and written, should include the following:

- Increase fluid intake to 2500 mL (approximately 2.5 quarts) per day.
- Eat a well-balanced diet high in vitamins and minerals and with adequate protein and kilocalories for healing.
- Use good hand washing techniques, particularly when caring for wounds or inflamed tissue and after using the bathroom.
- Elevate the inflamed area to reduce swelling and pain.
- Apply heat or cold for no longer than 20 minutes at a time to reduce the risk of tissue damage from burns or frostbite.
- Take all medications as prescribed, notifying the physician if adverse effects or hypersensitivity responses are noted. (See Nursing Research: Evidence-Based Practice on page 310).
- Rest acutely inflamed tissue; do not engage in strenuous activity until the inflammation has subsided.

The Client with an Infection

Microorganisms—including bacteria, viruses, fungi, and parasites—often invade the human body and proliferate if undetected and controlled or eliminated by inflammatory and immune responses. In most cases, contact between humans and microorganisms is incidental and may even be beneficial to both organisms. Resident bacteria of the skin, mucous membranes, and gastrointestinal tract are an important part of the body's defense system. However, many microorganisms are virulent; that is, they have the ability to cause disease. **Pathogens** are virulent organisms rarely found in the absence of disease. Some microorganisms, known as opportunistic pathogens, rarely, if ever, cause harm to persons with intact immune systems, but are capable of producing infectious disease in the immunocompromised host (Porth, 2005).

Infectious disease has been pervasive throughout history. Modern medicine, antibiotic therapy, immunizations, and other public health measures to protect food and water supplies have

NURSING RESEARCH Evidence-Based Practice for Antibiotics and Infection

Nurses discharging clients from outpatient and acute care settings frequently teach clients to take a complete prescribed dose of oral antibiotics to manage acute infectious illness. Ingesting less than complete doses exposes clients to the risk of resistant infections and less than therapeutic outcomes. There are many potential restraining forces to the completion of antibiotic dosing: cost of purchase; difficulty swallowing the pills; multiple, frequent doses; and the potential for adverse, unpleasant side effects.

Because adherence is so important and nurses are client educators, Aronson (2005) studied in depth the experience of 11 clients who had just completed a short-term antibiotic regimen to treat a variety of acute infectious illnesses with various antibiotic regimens. The 11 subjects represent diverse gender and cultural backgrounds. They participated in 30-minute interviews within 2 weeks of completing their antibiotic regimen. The interviews were audiotaped and evaluated for themes. This qualitative study is the first part of a research program to evaluate an intervention to promote adherence to taking antibiotics for a short-term period. The clients' descriptions, views, and experiences are the unique aspect of this research on adherence to antibiotic self-administration. Most studies of adherence have been conducted from the prescribers' rather than the clients' perspective.

Aronson analyzed the client descriptions of their experience taking their prescribed antibiotics by organizing the responses into categories of consistent themes; a second colleague experienced in qualitative research independently analyzed the data and the results were compared until the categories were agreed on. The central theme that emerged was successful antibiotic self-administration. The clients integrated the dosing into their daily schedules and adapted to any unplanned circumstances. The primary categories involved in self-administration were (1) medication-taking behaviors, (2) factors influencing adherence, and (3) attitudes and beliefs about the medication and the value of completing the prescribed dose. Subcategories were identified for each of these main categories.

Clients described methods for remembering to take the medication, methods of dealing with anticipated or experienced side effects, and factors that build trust in their relationship with the prescriber. The severity of the symptoms that led to antibiotic prescription made them more likely to report intention to adhere to the dosing regimen.

IMPLICATIONS FOR NURSING

Nurses teach clients about short-term antibiotic self-administration in outpatient and inpatient settings. The findings from this study can be used to guide educational interactions. Based on the findings in this study, encourage client involvement in the decision to take short-term antibiotic medications to strengthen the relationship with the prescriber. Ask clients to identify the method they will use to remind themselves of each dose; inquire about their knowledge of and plans to manage side effects from the medication.

CRITICAL THINKING IN CLIENT CARE

1. Identify methods that clients can use to remind themselves of dosing schedules.
2. An 86-year-old woman is being discharged to her home following a respiratory infection. Identify the information she will need about short-term antibiotic medication when she is discharged.
3. Discuss potential side effects of short-term antibiotics on the gastrointestinal tract.
4. Discuss the interrelationship between malnutrition and immune system function.

Data from Aronson, B. (2005). Medication management behaviors of adherent short-term antibiotic users. *Clinical Excellence for Nurse Practitioners, 9*(1), 23–30.

significantly reduced the prevalence of infectious diseases in many parts of the world. In spite of these advances, many infections, including malaria, typhoid, and tuberculosis, remain prevalent in developing nations. Sexually transmitted infections rage through modern cities and industrialized populations. New varieties and strains of pathogens, such as HIV, evolve to cause disease.

To a certain extent, modern medicine has contributed to the development of infectious diseases caused by antibiotic-resistant strains of microorganisms. Tuberculosis is on the rise in the United States, partially because organisms have become resistant to standard therapies. Clients receive immunosuppressive therapy following organ or tissue transplant or in the treatment of neoplasms, making them more susceptible to infection. Metal and plastic prosthetic devices are implanted, providing potential sites for colonization by disease-producing organisms (Fauci et al., 1998). It has also become apparent that many diseases long considered unrelated to microorganisms may actually be infectious; for example, colonization of the gastric mucosa with *Helicobacter pylori* is the predominant cause of peptic ulcer disease, and oncogenic viruses have the ability to transform normal cells into malignant cells.

Pathophysiology

Infection occurs when an organism is able to colonize and multiply within a host. The host can be any organism capable of supporting the nutritional and physical growth requirements of the microorganism—for example, humans. When the host experiences injury, pathologic changes, inflammation, or organ dysfunction in response to an infection or from intoxication by cellular poisons produced by a pathogen, the host is said to have an infectious disease.

For a microorganism to cause infection, it must have disease-causing potential (virulence), be transmitted from its reservoir, and gain entry into a susceptible host. This is known as the chain of infection (Figure 12–12 ■).

PATHOGENS Pathogens capable of infecting and causing disease in a susceptible host include bacteria, viruses, mycoplasma, rick-

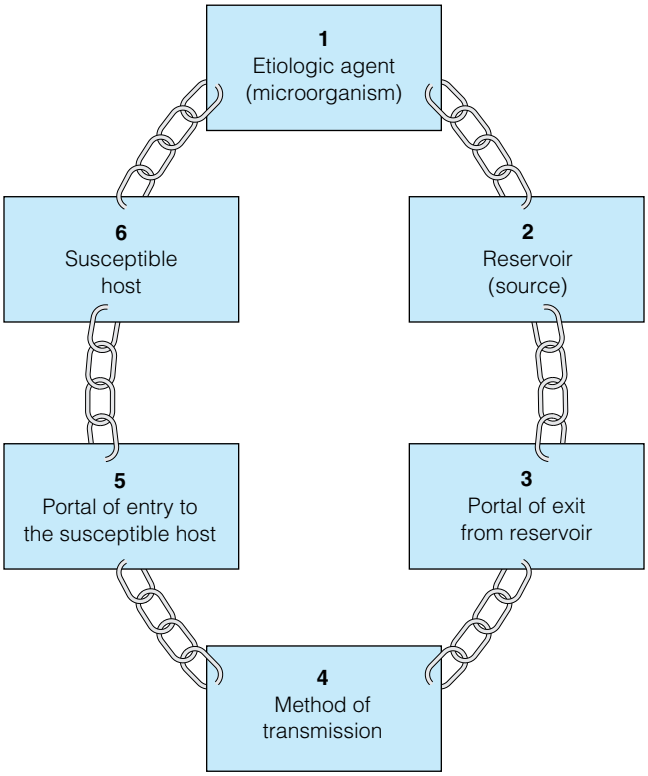


Figure 12–12 ■ The chain of infection.

ettsia, chlamydia, fungi, and parasites such as protozoa, helminths (worms), and arthropods (Box 12–4). Each organism causes a different specific reaction in the host.

A number of different mechanisms have evolved in pathogens to facilitate their transmission and increase their ability to invade the host and cause disease. Factors influencing the transmission of an organism include its resistance to drying and to variations in environmental temperature. For example, spore-forming organisms are extremely resistant to drying.

Many microorganisms are capable of producing toxins or enzymes to facilitate their invasion of the host, increase their resistance to host defenses, and increase their ability to cause disease. Adhesion factors produced by or incorporated into the cell wall or membrane of the pathogen improve its ability to attach and colonize the host. Pathogens may also produce enzymes to enhance their spread to local tissues, chemicals to block specific immune processes or deplete neutrophils and macrophages, or extracellular capsules to discourage phagocytosis.

Pathogens are often capable of producing toxins that alter or destroy the normal function of host cells and promote colonization, proliferation, and invasion by the pathogen. Toxins often increase the disease-producing capability of the pathogen and, in some cases, are totally responsible for it; for example, cholera, tetanus, and botulism result from bacterial toxins, not from the direct effects of the infection. **Exotoxins** are soluble proteins secreted into surrounding tissue by the microorganism.

BOX 12–4 **Pathogenic Organisms**

Bacteria

Bacteria are single-celled organisms capable of autonomous reproduction. Relatively small and simple organisms, they contain a single chromosome. A flexible cell membrane and rigid cell wall surrounds their cytoplasm, giving them a distinctive shape; some also have an extracellular capsule for additional protection. Bacteria have different characteristics and growth requirements: *aerobes* require oxygen for survival, whereas *anaerobes* cannot survive in the presence of oxygen; *gram-positive* bacteria stain purple when subjected to crystal violet stain, whereas *gram-negative* bacteria do not stain with crystal violet but turn red when subjected to safranin stain; the colonies formed by replicating bacteria differ from one another.

Viruses

Viruses are obligate intracellular parasites that are incapable of reproducing outside of a living cell. Viruses consist of a protein coat around a core of either DNA or RNA. Some viruses are shed continuously from infected cell surfaces; others, after inserting their genetic material into that of the infected cell, remain latent until they are stimulated to replicate. Viruses may or may not cause lysis and death of the host cell during replication. Oncogenic viruses are able to transform normal cells into malignant cells.

Mycoplasma

Although similar to bacteria, mycoplasma are smaller and have no cell wall, making them resistant to antibiotics that inhibit cell wall synthesis (e.g., penicillins).

Rickettsia and Chlamydia

As obligate intracellular parasites with a rigid cell wall, rickettsia and *Chlamydia* have some features of both bacteria and viruses. Rather than depending on the host cell for reproduction, they use vitamins, nutrients, or products of metabolism (e.g., ATP) from the host. *Chlamydia* are transmitted by direct contact, whereas many rickettsiae infect the cells of arthropods (e.g., fleas, ticks, and lice) and are transmitted from these vectors to humans.

Fungi

Fungi are prevalent throughout the world, but few are capable of causing disease in humans. Most fungal infections are self-limited, affecting the skin and subcutaneous tissue. Some fungi, such as *Pneumocystis carinii*, can cause life-threatening opportunistic infections in the immunocompromised host.

Parasites

The term *parasite* is typically applied to members of the animal kingdom that infect and cause disease in other animals. Protozoa, helminths, and arthropods are considered parasites. Protozoa are single-celled organisms transmitted via direct or indirect contact or an arthropod vector. Helminths are wormlike parasites: roundworms, tapeworms, and flukes are examples. They gain entry into humans primarily through ingestion of fertilized eggs or penetration of larvae through the skin or mucous membranes. Arthropod parasites, such as scabies (mites), lice, and fleas, typically infest external body surfaces, causing localized tissue damage and inflammation. Transmission is by direct contact with the arthropod or its eggs.

Source: Data summarized from *Pathophysiology: Concepts of Altered Health States* (7th ed.) by C. M. Porth, 2005, Philadelphia: Lippincott Williams & Wilkins.

Exotoxins are highly poisonous, causing cell death or dysfunction. **Endotoxins** are found in the cell wall of gram-negative bacteria and are released only when the cell is disrupted. Endotoxins have less specific effects than exotoxins, but they act as activators of many human regulatory systems, producing fever, inflammation, and potentially clotting, bleeding, or hypotension when released in large quantities.

RESERVOIR AND TRANSMISSION The reservoir or source, where the pathogen lives and multiplies, may be either endogenous or exogenous. Organisms that reside on skin or mucosal surfaces of the host are endogenous. Exogenous sources can include other humans, animals, soil, water, intravenous fluid, or equipment. Infectious diseases are usually transmitted from human sources, that is, persons who have clinical disease or carriers with subclinical infection. Carriers harbor the pathogen without showing evidence of clinical disease. Pathogens exit human hosts via respiratory secretions, body fluids from the gastrointestinal and genitourinary tracts, skin or mucous membrane lesions, the placenta, and blood.

Organisms may be transmitted from the source to the susceptible host by direct or indirect contact, droplet or airborne transmission, or a vector. Direct contact includes person-to-person spread or contact with infected body fluids, as well as transmission from contaminated food or water. Indirect contact occurs when the infectious agent is contracted by use of inanimate objects, such as dirty eating utensils. Sneezing, talking, and coughing allow transmission by droplet contact when the host is within 2 to 3 feet of the source. Smaller respiratory particles that stay suspended in air and are carried via air currents allow airborne transmission. Vectors are insects and animals such as flies, mosquitoes, or rodents that act as intermediate hosts between the source and host. Microorganisms usually first colonize the portal of entry: nonintact skin, wounds, mucous membranes, and the respiratory, gastrointestinal, or genitourinary tracts.

HOST FACTORS The susceptible host is the final link in the chain of infection. Exposure to pathogens does not automatically cause infection or infectious disease. The outcome of contact with a pathogenic microorganism is determined by the balance of microbial virulence and host resistance. Factors that can enable the host to resist infection include the following:

- Physical barriers, such as the skin and mucous membranes
- The hostile environment created by acid stomach secretions, urine, and vaginal secretions
- Antimicrobial factors in saliva, tears, and prostatic fluid
- Respiratory defenses, including humidification, filtration, the mucociliary escalator, cough reflex, and alveolar macrophages
- Specific and nonspecific immune responses to pathogenic invasion.

Stages of the Infectious Process

When infectious disease develops in the host, it typically follows a predictable course with stages based on the progression and intensity of manifestations.

The initial stage is the incubation period, during which the pathogen begins active replication but does not yet cause symptoms. Depending on the organism and host factors, the incubation period may last from hours, as with salmonella, to years, as with HIV infection.

The prodromal stage follows, during which symptoms first begin to appear. At this stage, symptoms are often nonspecific and include general malaise, fever, myalgias, headache, and fatigue.

Maximal impact of the infectious process is felt during the acute phase as the pathogen proliferates and disseminates rapidly. Toxic by-products of microorganism metabolism and cell lysis, along with the immune response, produce tissue damage and inflammation during this stage (Porth, 2005). Manifestations are more pronounced and specific to the infecting organism and site during the acute stage. Fever and chills may be significant during this phase. However, alcoholic clients and the very old may respond to severe infection by becoming hypothermic. The client is often tachycardic and tachypneic because of increased metabolic demands. Localized manifestations include redness, heat, swelling, pain, and impaired function. When the infectious disease affects an internal organ, manifestations are related to inflammatory changes in that organ and surrounding tissue. The client may experience tenderness to palpation over the site or show signs of impaired function, such as the hematuria and proteinuria characteristic of renal infections.

If the infectious process is prolonged, manifestations of the continuing immune response may become apparent. Catabolic and anorexic effects of the infection can lead to loss of body fat and muscle wasting. Immune complexes may be deposited at sites other than the primary infection, resulting in an inflammatory process. Glomerulonephritis (e.g., following strep throat) and vasculitis are possible results. Another possible consequence of prolonged infection and immune response is the triggering of an autoimmune disease process (discussed in Chapter 13 ∞), such as rheumatic cardiomyopathy or celiac disease. Type 1 diabetes mellitus is thought to be the result of such a response (Porth, 2005).

As the infection is contained and the pathogen eliminated, the convalescent stage of the disease occurs. During this stage, affected tissues are repaired and manifestations resolve. Resolution of the infection is total elimination of the pathogen from the body without residual manifestations. If a balance between organism and host factors occurs with neither predominating, chronic disease may develop or the organism may be driven into a protected site, such as an abscess. A carrier state develops when host defenses eliminate the infectious disease but the organism continues to multiply on mucosal sites (Fauci et al., 1998).

Complications

Multiple and varied complications are associated with infectious diseases. They are typically specific to the infecting organism and the body system affected.

Acute invasion of the blood by certain microorganisms or their toxins can result in septicemia and septic shock. Whereas bacteremia, the presence of bacteria in the blood, may not have serious effects, septicemia refers to systemic disease associated with their presence or toxins. Septic shock indicates a state of hypotension and impaired organ perfusion resulting from sep-

sis. Unless treated aggressively, septic shock leads to diffuse cell and tissue injury, and potentially to organ failure. See Chapter 11 ∞ for an in-depth discussion of septic shock, other shock syndromes, and their management.

Nosocomial Infections

Nosocomial infections are acquired in a healthcare setting, such as a hospital or nursing home. Also called healthcare-associated infections (HAIs), nosocomial infections account for an estimated 2 million infections, 90,000 deaths, and $4.5 billion in excess healthcare costs annually. HAIs add hospital days, reduce admissions by occupying available beds, and add to the cost of health care (Stone et al., 2005).

FAST FACTS

- Nosocomial infections typically manifest after 48 hours of hospitalization. Infections that manifest within 48 hours of hospitalization are attributed to community sources.
- Urinary tract infection is the most common type of HAI, leading to the most frequent cause of gram-negative septicemia in hospitalized patients.
- Pneumonia is the second most common hospital-acquired infection and has a mortality rate of 20% to 50%. It is associated with mechanical ventilators, tracheostomies, and endotracheal intubation (Porth, 2005).
- Bacteremia is associated with intravascular and urinary catheters. Because of the risk of infection, insertion of central lines as well as urinary catheters is conducted as a sterile procedure with careful attention to preventing contamination.
- *Clostridium difficile*–associated diarrhea is a frequently acquired nosocomial infection. It is an antibiotic-associated diarrhea and the risk of acquiring it increases with length of hospital stay, especially in an intensive care unit (ICU).

Clients entering hospitals are often the least able to mount immune defenses to infection. Immunologic responses may be compromised and normal defenses impaired in clients with, for example, cancer or chronic diseases, pressure ulcers, or organ transplants (Tierney et al., 2006). Nosocomial infections also occur when antibiotic therapy has altered natural defenses and impaired resistance to harmful microorganisms. Endogenous organisms outside their normal habitats (such as in *Escherichia coli* in the urinary tract) become a threat to the client. Other pharmacologic and therapeutic procedures such as chemotherapy, the use of corticosteroids, or radiation therapy also contribute to nosocomial infections. Gram-negative enteric bacteria and gram-positive *Staphylococcus aureus* are the most common bacteria responsible.

Invasive procedures and altered immune defenses are the main factors contributing to infection. Urinary catheterization is the number one cause; cardiac catheterization, peripheral and central intravenous lines, respiratory care procedures, and surgical procedures are also closely linked to nosocomial infection. Consequently, the urinary tract, surgical wounds, the respiratory tract, and invasive catheter sites on the skin are most often affected by hospital-acquired infection. Hospital-acquired pneumonia is the second most common nosocomial infection, accounting for 15% to 20% of these serious infections. Usually associated with ICU residency and mechanical ventilation, Sopina and Sobria (2005) found hospital-acquired pneumonia in non-ICU clients with severe underlying disease and a hospital stay greater than 5 days. Organisms causing the infection are often resistant to many drugs, not responding to antibiotics usually effective in treating infections acquired outside the hospital.

PRACTICE ALERT

Since October 2002, alcohol-based hand rub has been recommended by the CDC as the preferred method for hand hygiene (CDC, 2002a). Antiseptic soaps and detergents are the next most effective agents and nonantiseptic soaps are the least effective. A soap and water wash is recommended for visibly soiled hands. Wearing gloves does not eliminate the need for hand washing.

Prevention is the most important control measure for nosocomial infections. The pathogens causing these infections are transmitted primarily by contact with hospital personnel and contaminated inanimate objects (Posani, 2004). *Effective hand washing is the single most important measure in infection control.* Although infections may also be transmitted by the airborne route, contaminated equipment, or from the environment, these are less significant causes. Invasive procedures and equipment should be used only when absolutely necessary; for example, it is not appropriate to insert an indwelling catheter when the only indication is incontinence. Peripheral intravenous equipment and sites must be kept clean and changed regularly: intravenous bags and bottles every 24 hours, tubing every 24 to 96 hours, and sites every 2 to 3 days according to agency policy (CDC, 2002b; Evans-Smith, 2005).

Antibiotic-Resistant Microorganisms

Antibiotic-resistant microorganisms are increasing at an alarming rate primarily due to prolonged or inappropriate use of antibiotic therapy. Although antibiotic therapy is expected to eradicate all targeted microorganisms, sometimes a few bacteria survive, leading to bacteria that reproduce with antibiotic resistance already encoded into their genetic makeup (Lehne, 2004). Other bacteria produce enzymes that inactivate drugs, change drug binding sites, or alter their cell membrane to prevent drug absorption.

Some of the current resistant strains include:

- Methicillin-resistant *S. aureus* (MRSA)
- Multidrug-resistant tuberculosis (MDR-TB)
- Penicillin-resistant *Streptococcus pneumoniae* (PRSP)
- Vancomycin-resistant *Enterococci* (VRE)
- Vancomycin intermediate or resistant *S. aureus* (VISA or VRSA)
- Extended-spectrum beta-lactamase (ESBL) (Kjonegaard & Myers, 2005).

MRSA exists not only in hospitals but also is becoming more prevalent in the community setting where young people such as children in day care or amateur and professional athletes share equipment. MRSA colonizes in the nares and skin. Healthcare personnel often transmit *S. aureus* unknowingly on their hands for it is transmitted primarily by direct physical contact, not through respiratory droplets (Kjonegaard & Myers, 2005). Most

S. aureus strains resist treatment by methicillin and other similar drugs, the treatment of choice for *S. aureus* infections. Vancomycin has been the only uniformly effective drug for hospital-acquired MRSA; however, community-acquired MRSA is sensitive to antibiotics such as tetracycline, doxycycline, clindamycin, and sulfamethoxazole-trimethoprim. The community-acquired MRSA is also resistant to cephalexin, dicloxacillin, erythromycin, and quinolones. Soft-tissue infections with MRSA may manifest as abscesses, furuncles, or cellulitis and may be mistaken for a spider bite.

In 1997, a new form of *S. aureus* emerged with resistance to vancomycin, known as vancomycin intermediate or resistant *S. aureus.* Today VISA or VRSA has been identified; both VISA and VRSA are resistant to methicillin. Clients with MRSA, VISA, or VRSA are isolated in a private room using contact precautions.

Enterococci are part of the normal flora of the gastrointestinal and female genital tracts. Frequent use of vancomycin caused *Enterococci* to develop resistance, leading to vancomycin-resistant *Enterococci*. Direct transmission occurs on the hands of healthcare personnel or contact with contaminated equipment. Stringent infection control measures are instituted; care is provided using contact precautions and clients are placed either alone or with other VRE-infected clients.

Streptococcus pneumoniae, the most common cause of community-acquired pneumonia, has developed into its resistant form, penicillin-resistant *S. pneumoniae*. Unlike MRSA and VRE, PRSP is transmitted by droplets from the respiratory tract and requires transmission-based droplet precautions.

Clostridium difficile is an organism that has developed very resistant and highly morbid strains associated with frequent use of broad-spectrum antibiotics in hospitals. A common cause of nosocomial diarrhea, it is usually treated with metronidazole or vancomycin (Rao & Bradley, 2003). An even more virulent strain has been identified that is resistant to both metronidazole and vancomycin (Warny et al., 2005).

Extended-spectrum beta-lactamase–producing microorganisms are resistant to third-generation cephalosporins and include gram-negative *Klebsiella* and *E.coli*. These organisms colonize indwelling urinary catheters or gastrostomies and also mechanical ventilators. They spread by direct and indirect contact.

Universal precautions, most importantly hand washing, and modest use of antibiotics are critical actions for stopping the spread of these diseases. Equipment such as stethoscopes, blood pressure cuffs, and thermometers should be restricted to use by each patient identified with one of these diseases. Personal protective gear used and disposed of appropriately are important safeguards.

Biological Threat Infections

Following the terrorist attacks on September 11, 2001, and the development of anthrax cases in the United States, concern has arisen about the possible use of biological weapons. The most likely pathogens to be used for this purpose include anthrax, smallpox, botulism, pneumonic plague, and viral hemorrhagic fevers. (See Chapter 7 ∞ for more on biological terrorism.)

Infectious Process in Older Adults

Older adults, particularly those over the age of 75 years, are at greater risk of acquiring an infection than younger people. Although the incidence of septicemia in the United States is increasing in all age groups, the greatest increase is among people over the age of 65 years (Baine, Yu, & Summe, 2001). Physiologic changes of aging that put the elderly at increased risk for infection include the following:

- *Cardiovascular changes:* decreased cardiac output, loss of capillaries, and decreased tissue perfusion delaying inflammatory response and healing
- *Respiratory system changes:* decreased mucociliary escalator, decreased elastic recoil, and a diminished cough reflex leading to decreased clearance of respiratory secretions
- *Genitourinary changes:* loss of muscle tone, reduced bladder contractility, altered bladder reflexes, and prostatic hypertrophy in men leading to reduced bladder capacity and incomplete emptying
- *Gastrointestinal system changes:* impaired swallow reflex, decreased gastric acidity, and delayed gastric emptying thus increasing the risk of aspiration
- *Skin and subcutaneous tissue changes:* thinning of skin, decreased cushioning, and decreased sensation leading to increased risk of injury and ulceration
- *Immune changes:* decreased phagocytosis, reduced inflammatory response, slowed or impaired healing processes leading to reduced immunity.

In addition to the previous physiologic changes, other factors that may contribute to the older adult's increased risk for infectious disease are as follows:

- Decreased activity level related to musculoskeletal, neurologic, or balance problems
- Poor nutrition and an increased risk of dehydration
- Chronic diseases, such as diabetes mellitus, cardiac disease, and renal disease
- Chronic medication use
- Lack of recent immunizations against preventable infectious diseases
- Altered mentation and dementias
- Hospitalization or residence in a long-term care facility
- Presence of invasive devices, such as indwelling urinary catheters and gastric tubes.

In addition, the thymus gland atrophies and by age 50 to 60 years thymic hormone levels are undetectable. Although the exact relationship of these events to T-cell function is unclear, some T-cell populations decrease or decline in function as the person ages. The ability of T cells to proliferate following activation also declines with advancing age; in addition, a portion of T cells cannot be activated in the elderly (Porth, 2005). With these changes, cell-mediated immune function declines. The client has reduced resistance to antigens such as *M. tuberculosis,* influenza and varicella-zoster viruses, malignant cells, and tissue grafts.

Immunoglobulin levels remain relatively stable, but primary and secondary antibody responses decline with aging. This diminished antibody production has clinical implications in that immunizations (single-dose and booster) may not produce the expected protective immune response.

The older adult is not only at increased risk for infection, but also may not exhibit the classic manifestations of inflammation and infection. Older adults are likely to take NSAIDs and corticosteroids that interfere with inflammation and healing. The cardinal signs of inflammation—redness, heat, and swelling—tend to be diminished or absent in older adults. The classic signs of infection—fever and chills—may be absent altogether because of age-related changes in the immune system, loss of central temperature control mechanisms, decreased muscle mass, and loss of shivering ability. The older adult may have only subtle signs of sepsis, including changes in mental status, disorientation, and tachypnea (Porth, 2005). Infectious diseases commonly seen in the elderly client are outlined below.

INTERDISCIPLINARY CARE

The goals of care for the client with an infection are to identify the organ system affected by the infection, to identify the causative agent, and to achieve a cure by the least toxic, least expensive, and most effective means. Fortunately, most infectious diseases are self-limiting and will resolve with little or no medical care. However, medical treatment can be lifesaving in an overwhelming infection or immunocompromised host.

The body part or organ system affected by the infection is often obvious from the client's history and presenting signs and symptoms. Identifying the system allows the range of possible infecting organisms to be narrowed to those known to affect that system. The manner of presentation provides further cues as to the diagnosis. For example, pneumococcal pneumonia typically presents with an acute onset of chills, fever, and cough in a previously healthy adult, whereas the client with viral pneumonia relates a gradual onset of symptoms, with systemic manifestations such as muscle aches and headache often predominant. A history of recent activities also provides clues. Family members who all vomit and have diarrhea within 12 hours after a picnic probably do not have the flu.

NURSING CARE OF THE OLDER ADULT Infections

Because immune function declines with aging, older adults are more susceptible to infections. In fact, infections are among the top 10 causes for hospitalization and 1 of the 5 leading causes of death among people over 65 years of age.

Age-related changes may obscure the presentation of infection in older adults. Rather than an elevated temperature to signal an infection, confusion and subtle changes in behavior such as restlessness may be observed. The WBC count may be slightly elevated.

In addition to monitoring for changes in the client's mental status or behavior, the nurse should collect data on the amount of fluids consumed, urinary output, activity levels, complaints of fatigue, and respiratory status. A thorough assessment is necessary to facilitate an early diagnosis and prompt treatment that will improve outcomes for the older adult. Delay in treating infection may prolong the client's immobility and reduce the ability to perform activities of daily living.

Urinary Tract Infections

Urinary tract infection (UTI) is not only the most common infection but also the leading cause of bacteremia and sepsis in older adults. Factors that contribute to UTI include poor hygiene, incomplete bladder emptying, inadequate fluid intake, and long-term indwelling catheters. In addition, chronic conditions and medications may contribute to retention, which can result in urinary tract infection.

Respiratory Tract Infections

The leading causes of pneumonia in older adults include *S. pneumoniae, Haemophilus influenzae, Klebsiella pneumoniae, S. aureus,* and pneumococci. As a result of physiologic changes, the older adult with pneumonia may not present with cough or sputum production. Influenza A and B are prevalent in the aged; however, strain A accounts for greater illness severity and death (Eliopoulos, 2005). Both pneumonia and influenza cause high mortality rates in the older person.

Frail older adults, especially those with chronic respiratory conditions such as emphysema, are at risk for developing pneumonia as a complication of influenza. To prevent this complication, all persons over 65 should receive an annual immunization for influenza. In addition, pneumonia caused by pneumococcus bacteria can be deadly for older adults. Therefore, it is recommended that individuals over 65 also receive the pneumococcal immunization.

Tuberculosis has a relatively high incidence in older adults, especially those living in long-term care facilities. It often occurs as reactivation or secondary tuberculosis when the immune system can no longer contain the bacteria.

Other Infections

Older adults have the highest incidence of gangrene of the appendix and gallbladder. Diverticulitis increases due to chronic constipation and changes in the intestinal wall. Postoperative wound infections and decubitus ulcers are also most prevalent in older adults.

Nosocomial Infections

Nosocomial infections are more common in older adults. Nursing interventions should focus on prevention strategies such as (1) avoiding prolonged bed rest unless the medical condition contraindicates mobilization, (2) encouraging clients to take deep breaths, (3) providing adequate fluids, and (4) providing regular toileting schedules with good hygiene. The nurse must steadfastly adhere to principles of infection control.

Home Care

To prevent infection, the older adult should be taught to:

- Eat a well-balanced diet.
- Drink adequate fluids.
- Get adequate rest.
- Obtain an annual influenza immunization.
- Use good hand washing techniques.

Older adults and families should be taught to seek medical attention if they:

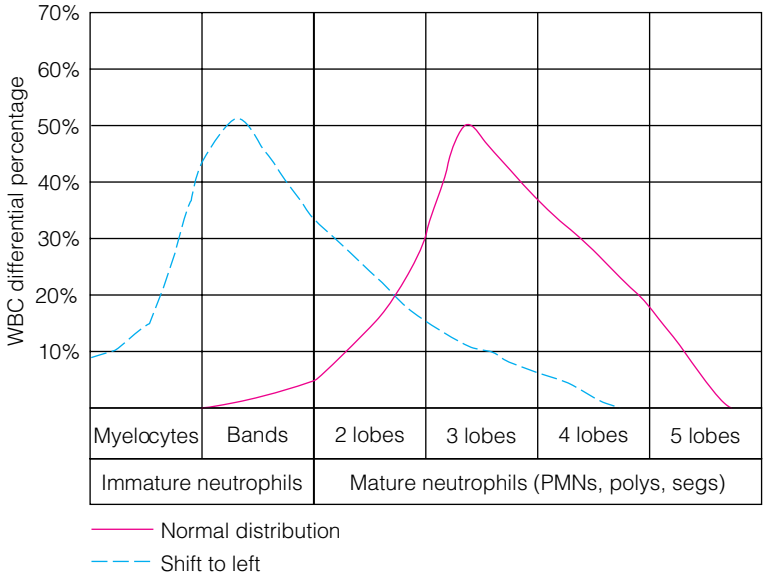
- Develop a fever or other signs and symptoms of infection.
- Exhibit changes in mental status and/or behavior.
- Experience fatigue or changes in activity levels.

Once the infecting agent has been identified, either positively or by probability, therapy can be specifically tailored to the client's needs. Viral infections often resolve without treatment other than supportive care, such as providing rest and fluids. Skin infections may respond to a topical agent, avoiding the potential adverse effects of one administered systemically.

Diagnosis

To assess the client's response to infection, identify the infecting organism, and monitor the progress of therapy, the following diagnostic tests may be ordered:

- *WBC count* provides clues about the infecting organism and the body's immune response to it (see Table 12–9).
- *WBC differential* is also ordered (see Table 12–9). Neutrophilia, increased numbers of circulating neutrophils (or PMNs), is a common response with infection as the bone marrow responds to an increased need for phagocytes. Along with neutrophilia, a shift to the left is common in acute infection. This means that there are more immature neutrophils in circulation than normal (Figure 12–13 ■), indicating an appropriate bone marrow response.
- *Procalcitonin (CTpr)* is a precursor of the hormone calcitonin. Procalcitonin increases dramatically during infection and sepsis and is accepted as both a marker of sepsis and a harmful mediator in lower respiratory tract and systemic infections (Christ-Crain et al., 2004; Müller & Becker, 2001).
- *Cultures of the wound, blood, or other infected body fluids* are used to identify probable microorganisms by their characteristics, such as shape, growth patterns, and Gram-staining qualities. After the organism is cultured, it is subjected to various antibiotics known to be effective against its particular strain to determine which antibiotic is likely to be most effective. This is known as sensitivity testing. Generally 24 to 48 hours are required to grow the organism, potentially delaying the institution of therapy. Because antibiotics (and possibly oxygen therapy) can alter the ability to culture an organism, specimens should be obtained before instituting therapy.
- *Serologic testing* provides an indirect means of identifying infecting agents by detecting antibodies to the suspected organism. When the antibody titer against a specific organism rises during the acute phase of an infectious disease and begins to fall during convalescence, the diagnosis is supported. Although it is not as accurate as culture, serology is particularly useful for organisms that cannot easily be cultured, such as hepatitis B or HIV (Porth, 2005).
- *Direct antigen detection methods* are in the process of being developed. These tests use monoclonal antibodies, which are purified antibody forms, to detect antigens in specimens from the diseased host (Porth, 2005). See Box 12–5. These tests offer rapid and accurate identification of the offending microorganism.
- *Antibiotic peak and trough levels* monitor therapeutic blood levels of the prescribed medication(s). The therapeutic range, that is, the minimum and maximum blood levels at which the drug is effective, is known for a given drug. By measuring blood levels at the predicted peak (1 to 2 hours after oral administration, 1 hour after intramuscular administration, and 30 minutes after intravenous administration) and trough (lowest level, usually a few minutes before the next scheduled dose), healthcare personnel can determine that the client is maintaining a level within the therapeutic range at all times, ensuring maximal effect from the drug. It is also possible to determine whether the drug is reaching a toxic or harmful level during therapy, increasing the likelihood of adverse effects.
- *Radiologic examination of the chest, abdomen, or urinary system* may be ordered to detect organ abnormalities indicating an inflammatory response or tissue damage.



Type of WBC	Normal differential	Shift to left
Myelocytes	0%	Present
Band neutrophils (bands)	3% to 5%	Increased
Segmented neutrophils (segs, polys, PMNs)	50% to 65%	May be stable, increased, or decreased

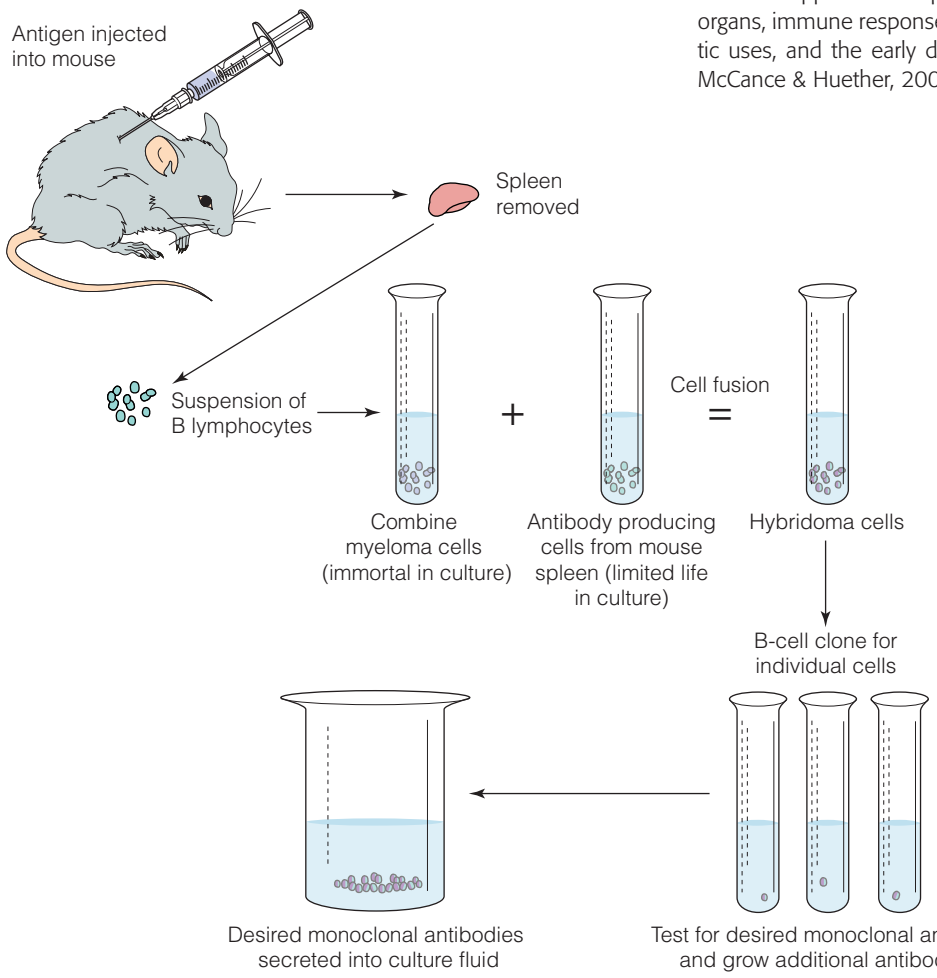
Figure 12–13 ■ Neutrophils by stage of maturity and normal distribution in the blood.

BOX 12–5 **Monoclonal Antibodies**

Antigens typically have numerous antigenic determinant sites, each capable of stimulating a different subset of B cells. Each clone secretes a slightly different antibody from the others. The immunoglobulin produced as a result is therefore *polyclonal,* with multiple different antibodies. In 1975, researchers devised a technique for making a single clone of "immortal" B cells that could be maintained indefinitely in a laboratory and would produce a single antibody to a specific antigen (see figure below). This pure antibody, known as a *monoclonal* antibody, offers the following advantages:

- It can target specific antigens.
- It has a single, constant binding affinity for the antigen.
- It can be diluted to a specific titer or concentration, because it is not mixed with other antibodies.
- It can be purified to avoid adverse responses (McCance & Huether, 2002).

In addition to their use in providing passive protection from disease, multiple other uses are being identified for monoclonal antibodies, including the diagnosis and treatment of cancer, immunosuppression to prevent rejection of transplanted tissue or organs, immune response analysis, imaging techniques for diagnostic uses, and the early detection of viral infections (Lehne, 2004; McCance & Huether, 2002).



Source: Figure adapted from *The World of the Cell* by W. Becker & D. Deane, 1986, Redwood City, CA: Benjamin Cummings; *Textbook of Diagnostic Microbiology* by C. R. Mahon & G. Manuselis Jr., 1995, Philadelphia: W.B. Saunders. Reprinted by permission.

- *Lumbar puncture* is performed to obtain cerebrospinal fluid (CSF) for examination and culture if a central nervous system (CNS) infection, such as meningitis or encephalitis, is suspected.
- *Ultrasonic examination* is a noninvasive diagnostic test to evaluate organ function such as an echocardiogram or renal ultrasonography.

Medications

Once the infecting organism and affected body system have been identified, specific therapy to cure the infectious disease can be instituted. The number of antimicrobial agents available makes choosing the appropriate one seem overwhelming. The perfect anti-infective agent would destroy pathogens while preserving host cells, would be effective against many different organisms while not promoting the development of resistance, would distribute to necessary tissues, and would remain in the body for relatively long periods.

Because no currently available antimicrobial meets all the above criteria, physicians look for the agent that will be effective, has little toxicity, can be administered with relative convenience,

and is cost effective. Characteristics of both the host and the infecting organism are considered in making the selection. The following host factors are considered in choosing an antimicrobial agent:

- *History of hypersensitivity.* Previous hypersensitivity responses to an antimicrobial contraindicate the use of that agent or one of its class.
- *Age and childbearing status of the client.* Sulfonamides and tetracyclines as well as some less common agents are contraindicated for pregnant women due to their possible effects on the fetus. Some drugs may cross into breast milk.
- *Renal function.* Because most antimicrobials are excreted through the kidneys, renal function is important. Impaired renal function may contraindicate a specific drug, such as an aminoglycoside, because of its nephrotoxicity, or it may call for a reduced dosage.
- *Hepatic function.* Because hepatic function may alter the metabolism of a particular antimicrobial, the risk of toxicity increases. Again, certain drugs are avoided with impaired hepatic function; others may dictate a reduced dosage.
- *Site of the infection.* The infection site is critical in choosing both the antimicrobial to be used and the route by which it is administered. Antimicrobials can be applied topically or administered by oral, intramuscular, intravenous, interperitoneal, intrathecal, or intramedullary routes. Oral and intravenous routes are most commonly used.
- *Other host factors.* Chronic diseases or other medications in the treatment regimen are also considered.

Antimicrobial preparations are broadly classified as bacteriostatic or bactericidal. **Bacteriostatic agents** inhibit the growth of the microorganism, leaving its destruction to the host's immune system. These agents are generally not indicated for the immunocompromised host. Tetracyclines, erythromycin, and chloramphenicol are bacteriostatic preparations. **Bactericidal agents** are capable of killing the organism without immune system intervention. These include the penicillins, cephalosporins, and aminoglycoside antibiotics.

The activity of antimicrobial agents on bacteria, fungi, and viruses falls under five basic mechanisms:

- Impairing cell wall synthesis, leading to lysis and cell destruction
- Inhibiting protein synthesis, causing impaired microbial function
- Altering cell membrane permeability, causing intracellular contents to leak
- Inhibiting the synthesis of nucleic acids
- Inhibiting cell metabolism and growth.

Obviously, agents that work on the cell wall will not be effective against organisms that have no cell wall, such as mycoplasma and viruses. The antimicrobial's spectrum of activity is also considered in making a selection. Therapy is often initiated with a broad-spectrum antimicrobial until the specific organism is identified.

Finally, many microorganisms have the ability to develop resistance to an anti-infective agent; that is, the pathogen continues to live and grow in the presence of the anti-infective. Resistance develops as a result of a chance mutation by the pathogen, allowing a subpopulation of cells to survive. The chance of an organism's becoming resistant to an agent is partially related to the dose delivered. Resistance is less likely to occur when a lethal dose is administered; therefore, it is vital that clients understand the need to take all doses of the prescribed drug as ordered.

Antimicrobial medications are generally classified as antibacterial or antibiotic, antiviral, antifungal, and antiparasitic.

ANTIBIOTICS Medications used to treat bacterial infections are generally known as antibiotics. Their development and use began before World War II and has proliferated rapidly since. Most antibiotics are biologic substances, that is, substances produced by other microorganisms. Antibiotics fall into classes of drugs with related chemical structure and activity. Some are effective against only gram-positive bacteria, and others are effective against only gram-negative organisms. Newer broad-spectrum antibiotics have activity against a wide variety of bacteria, including both gram-positive and gram-negative forms. No antibiotic is totally safe. Hypersensitivity responses occur, and some drugs are toxic to organ systems, exhibiting hepatotoxicity, nephrotoxicity, ototoxicity, or bone marrow suppression. Therefore, always check for allergies before administering the first dose. The antibiotics presented in the Medication Administration box are organized according to their antibacterial action.

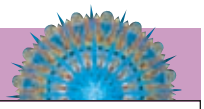
ANTIVIRALS Antiviral therapy is a relatively new phenomenon. Most antibiotics have little effect on viruses, because the virus has no cell wall and no cytoplasm, produces no enzymes, and sequesters itself in a host cell to reproduce. Antiviral agents must be very selective in differentiating normal cellular activity from viral activity. In addition, the immune function of the host is a vital component in fighting viral infections; antiviral therapy may be relatively ineffective in the severely immunocompromised host. Making a timely diagnosis to allow institution of antiviral therapy can be an additional problem, because viruses are less easily identified using laboratory techniques. Antiviral agents in common use are summarized in the Medication Administration box on page 322. Antiretroviral agents used in the management of HIV and AIDS are presented in Chapter 13 ∞.

ANTIFUNGALS Antifungal agents are available in both topical and systemic forms. They act by interfering with the cytoplasmic membrane of the fungus. Topical agents include preparations for cutaneous use to treat candidiasis, tineas, and ringworm. Vaginal preparations to treat vulvovaginal candidiasis are also available, as are several nonprescription topical and vaginal antifungal agents.

Amphotericin B (Fungizone) is a systemic antifungal agent for parenteral administration. It is used to treat severe, life-threatening fungal infections including histoplasmosis, blastomycosis, and candidiasis. Another systemic antifungal in current use is flucytosine (Ancobon). Unlike amphotericin B, flucytosine can be administered orally. It is used to treat severe candidiasis infections such as candida septicemia, endocarditis, pulmonary or urinary tract infections, and *Cryptococcus* meningitis.

Fluconazole (Diflucan) has the broadest use as an antifungal agent. It can be administered either orally or parenterally and is

MEDICATION ADMINISTRATION **Antibiotic Therapy**



MEDIALINK Penicillin Animation

I. Cell Wall Synthesis Inhibitors

PENICILLINS

Penicillin G
Penicillin V
Amoxicillin (Amoxil)
Amoxicillin and potassium clavulanate (Augmentin)
Ampicillin (Polycillin)
Carbenicillin (Geopen, Geocillin)
Dicloxacillin (Dynapen)
Methicillin (Staphcillin)
Mezlocillin (Mezlin)
Nafcillin (Unipen)
Oxacillin (Prostaphlin)
Piperacillin (Pipracil)
Ticarcillin (Ticar)

Penicillins are bactericidal and interfere with cell wall synthesis and the enzymes involved in cell division and synthesis. They are more effective on gram-positive than gram-negative organisms. Resistance is now more common among *Streptococci* and *Staphylococci.* They are considered to be safe, effective, and of low toxicity.

Nursing Responsibilities

- Monitor for hypersensitivity responses such as local erythema and itching at the site of injection, skin rashes, urticaria (hives), itching, fever, chills, and anaphylaxis.
- Observe clients receiving parenteral penicillin for at least 30 minutes.
- Discontinue the drug immediately if any hypersensitivity response occurs. Be prepared to administer antihistamines or corticosteroids for a mild reaction. Anaphylaxis is treated with epinephrine subcutaneously or intravenously and airway support.
- Do not administer penicillin to anyone with a history of a severe allergic reaction to any form of the drug; a cross-reactivity may occur in clients allergic to cephalosporin or carbapenem antibiotics.
- Assess for superinfection (vaginitis, stomatitis, or diarrhea) due to elimination of resident bacteria.
- Monitor for therapeutic response.

Health Education for the Client and Family

- Notify the physician if you see white patches on the oral mucosa or if vaginitis develops. An antifungal drug may be prescribed and the antibiotic continued.
- Consuming yogurt or buttermilk may prevent superinfection. Do not take these products within 1 hour of taking the drug.

CEPHALOSPORINS

1st Generation
Cephalexin (Keflex)
Cefazolin (Ancef)

2nd Generation
Cefotetan (Cefotan)
Cefaclor (Ceclor)
Cefmetazole (Zefazone)

3rd Generation
Cefoperazone (Cefobid)
Ceftazidime (Fortaz)
Ceftriaxone (Rocephin)

4th Generation
Cefepime (Maxapime)

Cephalosporins are structurally similar to the penicillins and also inhibit cell wall synthesis. They are divided into four groups, or generations. First-generation cephalosporins act primarily against gram-positive organisms. Second- and third-generation drugs are more effective against gram-negative organisms than against gram-positive ones. Fourth-generation cephalosporins act effectively against both gram-positive and gram-negative organisms.

Nursing Responsibilities

- Monitor for previous hypersensitivity response to cephalosporins or penicillins.
- Assess intravenous site for phlebitis; intramuscular site may cause local pain.
- Monitor laboratory results for adverse response, such as leukopenia and thrombocytopenia, nephrotoxicity (elevated BUN and serum creatinine), or hepatotoxicity (elevated bilirubin, LDH, ALT, AST, and alkaline phosphatase).
- Assess for signs of superinfections.

Health Education for the Client and Family

- Take the medication on an empty stomach, 1 hour before or 2 hours after meals.
- Avoid alcohol while using cefmetazole, cefoperazone, or cefotetan because alcohol intolerance can develop with these antibiotics. These same drugs intensify bleeding tendencies.
- Space doses of the medication relatively evenly throughout the day and evening hours.
- Increase consumption of buttermilk or yogurt to prevent intestinal superinfection.

CARBAPENEMS

This newer class of antibiotics includes only three drugs and all must be given parenterally. Imipenem has the broadest antimicrobial spectrum of any drug (Lehne, 2004). This makes it especially useful against mixed organism infections. Imipenem, meropenem, and ertapenem cross the meninges and achieve therapeutic doses in CSF; they are effective against MRSA. These antibiotics cause bacterial cell wall lysis and subsequent death of the bacteria. Side effects include nausea and vomiting, diarrhea, hypersensitivity reactions, occasional superinfections with bacteria or fungi, and, rarely, seizures.

Nursing Responsibilities

Ertapenem should not be mixed with dextrose or other drugs containing dextrose. IV infusions should be slow and given over at least 30 minutes. The dosage of all three drugs should be reduced if there is evidence of renal impairment.

- Check for history of hypersensitivity to cephalosporins and penicillins and monitor for signs of reactions.
- Assess for signs of superinfection.
- Monitor laboratory indicators of renal function.

Health Education for the Client and Family

- Instruct client to report any signs or symptoms of allergy such as skin rash, itching, or hives.

VANCOMYCIN

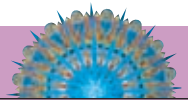
This antibiotic inhibits cell wall synthesis and is used for serious infections. It is only effective against gram-positive bacteria, especially *S. aureus* and *Staphylococcus epidermidis* including the strains resistant to methicillin. *C. difficile* is also susceptible to this antibiotic, but infection with *C. difficile* is often treated first with metronidazole to delay emergence of resistance to vancomycin.

Nursing Responsibilities

- Infuse slowly over 60 minutes or more to avoid "red man" syndrome. The syndrome is characterized by erythematous rash, flushing, tachycardia, and hypotension. Clients may become dizzy and agitated. The occurrence is usually associated with a first dose of vancomycin and is seen within 4 to 6 minutes of the start of a dose or after completion.
- Ototoxicity is a more serious adverse effect of vancomycin because hearing loss may be irreversible. Clients may report a

(continued)

MEDICATION ADMINISTRATION **Antibiotic Therapy (continued)**



sensation of fullness in their ears; this is evidence that ototoxicity is developing.

OXAZOLIDINONES

Linezolid is the first antibiotic in a new class of antibiotics called oxazolidinones. This antibiotic is significant because it is effective against organisms that are resistant to both vancomycin and methicillin. Because of its usefulness against those organisms, these drugs should be reserved for infections caused by VRE and MRSA (Lehne, 2004).

Nursing Responsibilities

This drug is generally well tolerated. Common side effects include nausea, diarrhea, and headache. It can cause reversible myelosuppression if used for longer than 2 weeks. It is a weak inhibitor of monoamine oxidase, creating risk for hypertension. It also prevents serotonin uptake so it increases the risk of serotonin syndrome when taken with selective serotonin reuptake inhibitors.

Health Education for the Client and Family

It can be taken with or without food. Avoid taking ephedrine, pseudoephedrine, methylphenidate, or cocaine with this drug. Hypertension may develop. Large amounts of foods containing tyramine should be avoided because the combination could cause headaches. Tyramine is generally found in foods that have been preserved or aged. Using leftovers with soy sauce or salami that have been refrigerated more than 24 to 48 hours may cause headaches.

B-LACTAMASE INHIBITORS

Clavulanic acid sulbactam

Combination agents

Unasyn
Timentin
Zosyn
Augmentin

II. Bacterial Protein Synthesis Inhibitors

TETRACYCLINES

Tetracycline HCl
Doxycycline (Vibramycin)
Minocycline HCl (Minocin)
Oxytetracycline (Terramycin)

Tetracyclines are active against many gram-positive and gram-negative bacteria, such as *Mycoplasma, Rickettsia*, and *Chlamydia*. They are bacteriostatic, interfering with microbial protein synthesis. Tetracycline binds readily with metal and solid elements in the bowel, limiting its absorption when administered with food; the other preparations are highly soluble in lipids and can be administered with food.

Nursing Responsibilities

- Schedule doses 1 hour before or 2 hours after meals. Do not give with milk or milk products or antacids.
- Monitor for signs of superinfection.
- If the client is taking an anticoagulant, monitor prothrombin time and for signs of bleeding.

Health Education for the Client and Family

- Avoid excessive sun exposure to reduce the risk of photosensitivity reactions.

MACROLIDES

Erythromycin (E-Mycin)
Azithromycin (Zithromax)
Clarithromycin
Erythromycin salts (Ilosone, E.E.S., Erythrocin)

Macrolides are bacteriostatic and act effectively against gram-positive and gram-negative organisms. Erythromycin is used to treat streptococcal pharyngitis in clients who are allergic to penicillin. Azithromycin is ordered more frequently because the client takes the drug for 5 days, increasing client adherence.

Nursing Responsibilities

- Administer erythromycin on an empty stomach or immediately before meals.
- Give the drug with a full glass of water. Do not administer with acidic fruit juice.

Health Education for the Client and Family

- Gastric distress is a common side effect with erythromycin.

AMINOGLYCOSIDES

Amikacin (Amikin)
Kanamycin (Kantrex)
Streptomycin
Gentamicin (Garamycin)
Netilmicin (Netromycin)
Tobramycin (Nebcin)

Aminoglycosides are bactericidal, interfering with protein synthesis in the pathogen. They are especially effective against gram-negative organisms. To provide a broader spectrum of activity, they are often combined with other antibiotics, especially penicillins. Aminoglycosides can be administered in multiple or single daily doses. They are ototoxic and nephrotoxic; the risk is highest for older adults, clients with preexisting renal disease, and persons receiving other ototoxic or nephrotoxic drugs.

Nursing Responsibilities

- Assess renal function before and during aminoglycoside therapy. Monitor intake and output, daily weight, BUN, and serum creatinine.
- Assess for adverse effects on hearing such as loss of perception of high tones, tinnitus, and vertigo.
- Notify the physician if the client is receiving other nephrotoxic or ototoxic drugs such as furosemide (Lasix) and ethacrynic acid (Edecrin).
- Administer intravenous preparations separately from other drugs; flush tubing before and after administration.

Health Education for the Client and Family

- Monitor for a sudden weight gain that may indicate adverse effects on the kidney and report it to the physician.

III. Bacterial Nucleic Acid Inhibitors

FLUOROQUINOLONES

Ciprofloxacin (Cipro)
Levofloxacin (Levaquin)
Gatifloxacin (Tequin)

Fluoroquinolones are bactericidal and especially active against gram-negative and some gram-positive organisms. They are used to manage infections of the respiratory, gastrointestinal, and genitourinary tracts.

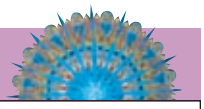
Nursing Responsibilities

- Increase fluid intake to 2000 to 3000 mL/day unless contraindicated to prevent crystalluria.
- Monitor laboratory results for hepatotoxicity (elevated ALT, AST).

Health Education for the Client and Family

- Drink six to eight glasses of water per day.
- Avoid exposure to sunlight while taking these drugs.

MEDICATION ADMINISTRATION **Antibiotic Therapy (continued)**



SULFONAMIDES AND TRIMETHOPRIM

Sulfamethizole (Thiosulfil Forte)
Sulfamethoxazole (Gantanol; in combination with trimethoprim, TMP-SMZ, Bactrim, Septra)
Sulfisoxazole (Gantrisin)

Sulfonamides are bacteriostatic. Trimethoprim is an antibiotic effective against most gram-positive and many gram-negative organisms. It is often combined with sulfamethoxazole to manage urinary tract infections, *P. carinii* pneumonia, and otitis media. Skin rashes and pruritus are the most common hypersensitivity reactions. Severe reactions include exfoliative dermatitis and Stevens-Johnson syndrome.

Nursing Responsibilities

- Assess for history of hypersensitivity to sulfonamides and related medications, such as thiazide diuretics and hypoglycemic preparations.
- Monitor intake and output. Maintain a fluid intake of at least 1500 mL/day.
- Assess for evidence of bleeding, easy bruising, or systemic infection, and monitor blood count for possible bone marrow depression.

Health Education for the Client and Family

- Take medication on an empty stomach with a full glass of water. Maintain a fluid intake of at least 2 quarts per day.
- Protect the skin from excessive sun exposure with clothing and sunscreens to reduce the risk of photosensitivity.

METRONIDAZOLE (FLAGYL)

Metronidazole is effective against anaerobic gram-negative bacteria and protozoan infections caused by amebiasis, giardiasis, and trichomoniasis. It is commonly used to prevent and treat infections following intestinal surgery, and is the drug of first choice with *C. difficile*.

Nursing Responsibilities

- Monitor for CNS effects of dizziness, headache, ataxia, confusion, depression, and peripheral neuropathy.
- Administer with food to minimize gastric distress and metallic taste. Infuse intravenous metronidazole over 60 minutes.
- Discontinue the medication and notify the physician if neurologic reactions occur.
- Increase fluid intake to 2500 mL/day to minimize the risk of nephrotoxicity.

Health Education for the Client and Family

- This medication may turn urine reddish brown; caution client that this is expected and not harmful.
- Discontinue the drug and notify the physician if hypersensitivity reaction or adverse effects occur, such as changes in mentation or coordination, painful or frequent urination, painful or difficult intercourse, impotence.
- Do not drink alcohol while taking this medication; an Antabuse-type reaction (flushing, sweating, headache, vomiting, and abdominal cramps) may occur.
- Maintain a fluid intake of 2.5 to 3 quarts per day.
- When the drug is prescribed for *Trichomonas* infections, treatment of both partners is necessary.
- While taking metronidazole, use condoms to prevent cross-contamination during intercourse.

used to treat candidiasis infections as well as *Cryptococcus* meningitis. It is generally better tolerated than other systemic antifungal medications.

ANTIPARASITICS Drugs used to treat parasitic infections are as varied as the organisms that cause them. Generally, agents classified as antiparasitic are both expensive and likely to be toxic. Quinine was one of the first antiparasitic drugs developed in the treatment of malaria. Quinine is highly toxic, but newer forms such as chloroquine (Aralen, Chlorocon) and hydroxychloroquine (Plaquenil) are widely used as antimalarial drugs. Metronidazole (e.g., Flagyl) is used to treat infections of protozoan parasites (see the Medication Administration box above).

Isolation Techniques

Controlling the spread of infectious diseases in the hospital or long-term care setting is particularly important to preventing nosocomial infection. Hand washing remains the single most important factor in preventing the transmission of infections. Not all infectious diseases spread readily, necessitating special techniques or procedures. However, diseases such as chickenpox (varicella) and pulmonary tuberculosis are highly contagious and are spread by the airborne route, requiring special precautions to protect other hospitalized clients. Two newer airborne threats are SARS and avian flu. These are addressed in Chapter 37 ∞.

In determining the need for isolation precautions, healthcare personnel consider the usual reservoir or source of the microorganism, the mode of transmission, and susceptibility of hospital staff and other clients. For example, clients with *P. carinii* pneumonia do not require isolation, because immunocompetent persons are not susceptible to this infection.

The CDC has published guidelines for isolation precautions to be used in healthcare facilities (Glover, 2000). These guidelines include both *standard precautions* and *category-specific isolation precautions.*

Standard Precautions

Standard Precautions, published by the Hospital Infection Control Practices Advisory Committee of the Centers for Disease Control in 1996, provides guidelines for the handling of blood and other body fluids. These guidelines are used with all clients, regardless of whether they have a known infectious disease. The guidelines were developed in light of the realization that many clients with an infectious disease such as HIV or hepatitis B have no apparent symptoms, but can transmit the disease to others. Standard precautions are used by all healthcare workers who have direct contact with clients or with their body fluids or have indirect contact, such as by emptying trash, changing linens, or cleaning the room.

MEDICATION ADMINISTRATION **Antiviral Agents**

AMANTADINE (SYMMETREL)
Amantadine is used to prevent and treat influenza A. It has been shown to be 55% to 80% or more effective in preventing the disease. When administered within 24 to 72 hours after the onset of symptoms, it reduces common manifestations of influenza. It is generally well tolerated; minimal CNS side effects, such as dizziness, anxiety, insomnia, and difficulty concentrating, may occur.

ACYCLOVIR (ZOVIRAX) AND GANCICLOVIR (CYTOVENE)
Acyclovir and ganciclovir are related compounds used primarily in the treatment of herpes viruses. Acyclovir is prescribed mainly in the treatment of genital herpes simplex infections. Although it does not kill the virus, acyclovir is effective in reducing the severity, duration, and frequency of recurrence of symptoms. Ganciclovir is indicated primarily in the treatment of cytomegalovirus infection. Although acyclovir is generally well tolerated with little toxicity, ganciclovir may profoundly suppress bone marrow function, and its use is therefore limited.

ZIDOVUDINE (AZT, RETROVIR)
Zidovudine inhibits replication of HIV, although it does not kill it. Zidovudine's use is limited to clients with symptomatic HIV infection or CD4 cell counts of less than 200/mm^3. Zidovudine may be administered either orally or parenterally. Many clients are unable to tolerate recommended doses because of the drug's adverse effects, including nausea, anorexia, malaise, severe anemia, and granulocytopenia. Zidovudine is usually administered in combination with other antiretroviral medications.

VIDARABINE (VIRA-A)
Vidarabine inhibits viral DNA synthesis and is effective against many herpesvirus infections. Its primary use is in treatment of herpes simplex encephalitis.

INTERFERONS
Interferons are naturally produced cytokines whose use as antiviral agents is being explored. When administered intranasally, interferons have been shown to be effective in preventing rhinovirus upper respiratory infections. Other uses being explored include treatment of human papillomavirus (genital warts) and preventing or reducing Kaposi's sarcoma in clients with AIDS. They are also used in combination biotherapy regimens for malignant melanoma.

Standard precautions apply to the following:
- Blood
- All body fluids, secretions, and excretions, regardless of whether they contain visible blood
- Nonintact skin
- Mucous membranes.

Barrier protection is used to prevent exposing skin and mucous membrane surfaces to blood and body fluids. Barrier protection involves using gloves for touching or handling body fluids, and adding other protection such as gowns, masks, and goggles if splashing or spraying is likely. Needles and other sharp objects are not recapped or bent, but disposed of in puncture-proof containers to prevent inadvertent percutaneous (needle-stick) exposure. Standard precautions are presented in Appendix A.

Transmission-Based Precautions

In addition to hand washing and standard precautions, the nature and spread of some infectious diseases require that special techniques be used to protect uninfected clients and workers. The CDC identifies three types of transmission-based precautions: airborne, droplet, and contact precautions. Transmission-based precautions may be combined for diseases that have multiple routes of transmission. Indications for the use of transmission-based isolation precautions and the specific measures to be taken are outlined in Table 12–10.

NURSING CARE

Nursing management related to infectious disease has two foci: (1) *prevention* and (2) *health promotion and maintenance.* Prevention focuses on assessing the client's risk for infection based on underlying conditions, immune response, and prophylactic measures such as immunizations.

Health Promotion

Preventing infection requires education of not only healthcare personnel but also the general public. Part of an education program includes understanding the importance of immunizations, the guidelines for using antibiotics to prevent drug-resistant microorganisms, and the ways to prevent the spread of infection. Check immunization records for all family members and encourage them to keep immunizations up to date. Increase public awareness regarding appropriate antibiotic use. Guidelines for preventing the spread of infection to others include the following:
- Avoid crowds and contact with susceptible persons, especially those who are immunosuppressed (e.g., persons who have HIV infection, who are undergoing therapy for cancer, or who have had an organ transplant).
- Use disposable tissues to contain respiratory secretions when coughing or sneezing.
- Use appropriate food-handling precautions for diseases spread via the fecal–oral route, such as hepatitis A.
- Avoid contact with or sharing of body fluids. For example, do not share needles or razors; use a condom during sexual activity, or abstain; have each person clean their own blood spills or wounds if possible.

Assessment

The following data are collected through the health history and physical examination. Further focused assessments are described with nursing interventions in the next section.

TABLE 12–10 **Transmission-Based Precautions**

CATEGORY	INFECTIOUS DISEASES	PURPOSE	PRECAUTIONS
Airborne precautions	Pulmonary tuberculosis, chickenpox (with contact precautions), measles, respiratory infections (pneumonia)	Reduce risk of airborne transmission of infectious agents. Airborne transmission occurs by dissemination of either airborne droplet nuclei or dust particles containing the infectious agent.	Private room with hand washing and toilet facilities, and special ventilation that does not allow air to circulate to general hospital ventilation; mask or special filter respirator for everyone entering room.
Droplet precautions	Meningitis, pertussis	Reduce risk of droplet transmission of infectious agents. Droplet transmission involves contact of conjunctivae of the eyes or mucous membranes of the nose or mouth with large-particle droplets generated during coughing, sneezing, talking, or procedures such as suctioning.	Private room with hand washing and toilet facilities; mask, eye protection, and/or face shields worn by everyone entering room.
Contact precautions	Acute diarrhea, chickenpox (with airborne precautions), respiratory syncytial virus (RSV); skin, wound, or urinary tract infection with multidrug-resistant organisms; *S. aureus* infections	Reduce risk of transmission by direct or indirect contact. Direct contact transmission involves skin-to-skin contact and physical transfer of organisms. It may occur between clients or during direct care activities such as bathing or turning clients. Indirect contact involves contact with a contaminated object.	Private room with hand washing and toilet facilities; gowns and protective apparel to provide barrier protection; disposable supplies or decontamination of all articles leaving room.

- *Health history:* age, medication use (antipyretics and antiinfectives), nutrition, exposure to infectious persons, immunizations, invasive procedures and therapies, chronic diseases such as diabetes mellitus, cancer
- *Physical assessment:* vital signs, body system(s) where infection is suspected, lymph node enlargement, and tenderness.

Nursing Diagnoses and Interventions

Clients with an infection may be managed in the hospital or at home. During the acute phase, nursing care includes administering prescribed antibiotics, implementing and maintaining aseptic technique and infection control measures, and encouraging a balance of rest and activity, good nutritional intake, and other general health measures to support immunologic function and healing. The key nursing diagnoses are *Risk for Infection, Anxiety, Hyperthermia,* and *Pain.*

Risk for Infection

The spread of infection is a risk in any facility that houses many people. It is a particular risk in hospitals, where many clients have at least some degree of immunosuppression and many drug-resistant strains of pathogens are prevalent. It is vital that nurses use good hand washing techniques at all times, employ standard precautions with all clients, and use category-specific isolation techniques as indicated to prevent infectious spread to other clients, themselves, and their families.

- Admit clients with known or suspected infections to a private room. *This is important to minimize the risk to other clients.*
- Wash hands on entering and leaving the client's room, using a 10- to 15-second vigorous scrub with soap or antibacterial scrub solution. *A 10- to 15-second scrub removes transient microorganisms from the skin and helps prevent transmission of infection to or from the client.*
- Use standard precautions and personal protective devices to reduce the risk of transmission. *Gloves, gowns, and masks are to be worn whenever there is a risk of skin or mucous membrane contamination by direct contact with infectious material, airborne spread of organisms, or droplet nuclei.*
- Explain the reasons for and importance of isolation procedures during hospitalization. *Clients with isolation precautions may feel neglected, dirty, or shunned. Explanation of reasons and procedures can enhance the client's and family's understanding and acceptance.*
- Place a mask on the client and/or cover all infectious lesions or wounds completely when transporting the client to other parts of the facility for diagnostic or treatment procedures. *These measures help minimize air contamination and the risk to visitors and personnel.*
- Collect a culture and sensitivity (C&S) specimen as ordered or indicated by purulent drainage, pyuria, or other manifestations of infection. *C&S is performed to determine the presence and type of infectious organisms as well as antibiotics most likely to be effective in eradicating it.*

PRACTICE ALERT
Collect the specimen before the first dose of antibiotics is administered to ensure adequate organisms for culture.

MEDIALINK Care Plan Activity: Postoperative Infection

- Administer prescribed anti-infective agents. *Anti-infectives are used to destroy the invading microorganism.*
- Inform all personnel having contact with the client of the diagnosis. *This is particularly important for a client with a disease requiring category-specific isolation so that personnel can take appropriate precautions.*
- Ensure that visitors don appropriate protective wear before they enter the client's room. *Protective wear reduces their risk of infection.*
- Use appropriate measures for disposing of contaminated tissues, dressings, or other material and for removing soiled linens and equipment from the client's room. *Check hospital policy or published guidelines for category-specific isolation.*
- Teach the importance of complying with prescribed treatment for the entire course of the regimen. *Because anti-infective agents kill only a portion of the pathogen population with each dose, completion of the entire course of therapy is necessary to reduce the risk of relapse and of creating drug-resistant organisms.*

Anxiety

The client with an infectious disease may experience anxiety related to his or her manifestations, treatment measures, the prognosis, and expected outcome of the disease. The diagnosis of an infection can be traumatic, causing feelings of uneasiness, isolation, guilt (e.g., in regard to sexually transmitted infections), apprehension, or depression.

- Assess level of anxiety. *The level of anxiety influences the client's response to and interpretation of the situation and degree of threat it poses.*
- Discuss the infection, treatments, prognosis, and outcomes. *Discussions help to allay fears and misconceptions.*
- Support and enhance the client's coping strategies. *A person uses intrapersonal and interpersonal mechanisms to reduce or relieve anxiety.*
- Include significant others in the plan of care. *Inclusion of the client and family members provides assurance and confidence, and promotes understanding of the unknown.*
- Explain isolation procedures, and answer any concerns. *Isolation may be necessary to prevent the spread of infection but can cause great anxiety for the client and family members.*
- Provide referrals as needed for continuing care, for example, to home health agencies or for dressing changes or periodic assessment. *Referrals are often necessary to provide ongoing interventions and maintain continuity of care.*

Hyperthermia

Hyperthermia is an expected consequence of the infectious disease process. Fever may produce mild, short-term effects or, when prolonged, may cause serious life-threatening effects.

- Monitor temperature especially during episodes of chills; note heart rate and rhythm. *Chills indicate a rising temperature. Hyperthermia can cause dysrhythmias.*

PRACTICE ALERT
Monitor temperature between 5 P.M. and 7 P.M. because the body's daily temperature cycle peaks at this point.

- Administer prescribed antipyretic as indicated for elevated temperature. *Although antipyretics lower the temperature and enhance comfort for the client, this benefit must be weighed against the possible beneficial effect of an elevated temperature in the immune response. Fever increases the motility and activity of WBCs, stimulates the production of interferon, and activates T cells. In addition, temperatures above the normal range inhibit the growth of many microorganisms* (Porth, 2005).
- Promote body cooling through lowering the room temperature. *Rapid cooling stimulates the hypothalamus to increase the body's temperature; this increases both shivering and metabolic rate.*

PRACTICE ALERT
Use ice packs, cool/tepid baths, or hypothermia blanket with caution to prevent unnecessary shivering.

- Monitor fluid loss; encourage increased fluid and electrolyte intake either orally or intravenously. *Hyperthermia causes fluid loss from evaporation and may result in dehydration and electrolyte imbalance.*
- If diaphoretic, bathe and provide dry clothing and bedding. *These measures increase client comfort and decrease further water evaporation.*
- Promote rest periods. *Rest increases energy reserve that is depleted by an increased metabolic, heart, and respiratory rate.*

Pain, Acute

Pain often accompanies infections as part of the inflammatory process or secondary to delayed healing. *Increasing pain in a wound may signal infection, especially if accompanied by erythema or purulence. Keep the wound clean and dry and administer prescribed antibiotics to promote healing and decrease pain.*

Using NANDA, NIC, and NOC

Chart 12–1 shows links between NANDA, NIC, and NOC when caring for the client with an infection.

Community-Based Care

Client and family teaching is directed toward helping the client recover from the infection or disease, preventing its spread to others, and preventing life-threatening complications. Instructions should include the following points:

- Use good hand washing techniques, particularly after touching infected wounds or lesions, coughing, sneezing, blowing the nose, or using the bathroom. Wash hands thoroughly before eating or performing any procedures such as dressing changes. Wash hands with soap and water before and after preparing food or eating and before and after using the toilet or handling diapers. Do not share eating utensils.
- Take all prescribed antibiotics as ordered even after symptoms have subsided. Take the prescription at intervals around the clock as directed.
- Never allow anyone else to use your medications and never use anyone else's prescription even if they appear to be the same.

NANDA, NIC AND NOC LINKAGES

CHART 12–1 **The Client with an Infection**

NANDA

Delayed Surgical Recovery

↓

NIC

Infection Control

Medication Administration

Vital Signs Monitoring

Wound Care

↓

NOC

Infection Severity

Wound Healing: Primary Intention

Post Procedure Recovery Status

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- If instructed to stop taking a medication, destroy any that remains by flushing in toilet or garbage disposal

  Notify your healthcare provider if:
  - Symptoms do not improve within 24 to 48 hours after antibiotic therapy is instituted, or they worsen.
  - Signs of antibiotic allergy (itching, rash, difficulty breathing or swallowing, swelling of the face or tongue) occur. Discontinue medication and contact prescriber.
  - Adverse responses, such as gastrointestinal distress, interfere with completion of the prescription.
  - Signs of superinfection (vaginitis, oral candidiasis, or diarrhea) occur.
  - Manifestations of infection recur after completing prescribed antibiotic.
- Report redness, swelling, or drainage around wounds or persistent high fever.
- Increase fluid intake to at least 2500 mL (2.5 quarts) per day.
- Report any signs of opportunistic infections: loose, watery, and foul-smelling diarrhea; vaginal discharge or itching; fuzzy growth or white plaques in mouth or on tongue; blood in urine, chills, fever, or unusual cough.
- In addition, suggest the following resources:
  - County or public health department
  - Centers for Disease Control and Prevention

EXPLORE MEDIALINK

Prentice Hall Nursing MediaLink DVD-ROM

Audio Glossary
NCLEX-RN® Review

Animations/Video

Inflammatory Response
Penicillin
White Blood Cells

COMPANION WEBSITE www.prenhall.com/lemone

Audio Glossary
NCLEX-RN® Review
Care Plan Activity: Postoperative Infection
Case Study: The Client with an Infection
MediaLink Applications
Antibiotic-Resistant Organisms
Hopsital-Acquired Infections
Links to Resources

CHAPTER HIGHLIGHTS

- The human body has a remarkable capacity to survive in an environment of deadly microorganisms and pathogens that can weaken and kill the body. Both natural barriers and the immune system prevent the invasion and replication of pathogens. The lymphatic system provides conduits for pathogens, isolating them from the bloodstream where they would grow rapidly.
- The adaptability and specificity of immune responses is possible because immune cells are genetically encoded to capture pathogens, move them to lymph nodes, and develop specific immune reactions. However, if the immune system's self-recognition fails, highly damaging autoimmune diseases can develop.

- A revolutionary increase in knowledge and understanding of the immune system has enabled progress in clinical medicine. Through scientific research concerning T and B lymphocytes, cytokines, antibodies, and other elements of the immune system, treatments are evolving for cancer, AIDs, organ transplantation, autoimmune diseases, infectious disease, and vaccines.
- Inflammation is a protective mechanism designed to prevent pathogens from entering the bloodstream and populating functional tissues such as heart, liver, and kidney. Pain acts as a signal that tissue has been damaged and stimulates protective responses such as cleansing wounds and limiting function while healing progresses. Restoration occurs as the inflammatory process isolates the injury and repairs damaged tissue.
- Localized infections may damage tissue and create pain, but systemic infections are life-threatening if they progress to septic shock. Unfortunately, hospitals are hazardous environments populated with collections of pathogens. Hospital-borne infections are often introduced into the body by medical procedures.
- Hygiene, protection from harm, and nutrition support the immune defenses. Antimicrobial medications limit the spread of pathogens, but can lose their effectiveness when microbes mutate and develop resistance.

TEST YOURSELF NCLEX-RN® REVIEW

1 When a client receives gamma globulin following exposure to hepatitis A, the nurse expects the client to develop:
   1. natural passive immunity.
   2. natural active immunity.
   3. acquired passive immunity.
   4. acquired active immunity.

2 When performing a physical assessment of a client, the nurse should expect which of these findings related to a systemic infection?
   1. erythema
   2. enlarged lymph nodes
   3. pain
   4. decreased heart rate

3 Which one of the following medications is known to inhibit prostaglandin synthesis?
   1. acetaminophen (Tylenol)
   2. prednisone
   3. penicillin
   4. aspirin

4 The client with an acute infection shows a shift to the left on the WBC differential count. The nurse recognizes a shift to the left because of which laboratory finding?
   1. increased band neutrophils
   2. increased eosinophils
   3. decreased leukocytes
   4. decreased monocytes

5 A client is admitted with methicillin-resistant *Staphylococcus aureus* in a draining sacral wound. The client should be placed in which type of isolation precautions?
   1. droplet precautions
   2. contact precautions
   3. airborne precautions
   4. protective precautions

6 The T cells of the immune system adapt to kill:
   1. intracellular organisms.
   2. interstitial microbes.
   3. extracellular viruses.
   4. protozoans.

7 Thrombocytosis is best explained as:
   1. increased platelets.
   2. decreased clotting time.
   3. decreased platelets.
   4. average number of platelets.

8 The nursing diagnosis *Risk for Infection* pertains to:
   1. the infected patient only.
   2. healthcare workers in the facility.
   3. patients hospitalized at the same time.
   4. the infected patient, healthcare workers, and other patients.

9 When administering antibiotics, the primary nursing role is monitoring for:
   1. compliance.
   2. therapeutic levels.
   3. oxygen desaturation.
   4. hypersensitivities and teaching.

10 Standard isolation techniques are employed for all hospitalized patients. These include:
   1. hand washing, use of masks, and recapping needles.
   2. use of masks and gowns, and spraying of disinfectant regularly.
   3. use of gloves, gowns, and goggles with contaminated body fluids.
   4. use of alcohol-based hand rub for visibly dirty or blood contaminated hands.

See Test Yourself answers in Appendix C.

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