CHAPTER 13 Nursing Care of Clients with Altered Immunity

LEARNING OUTCOMES

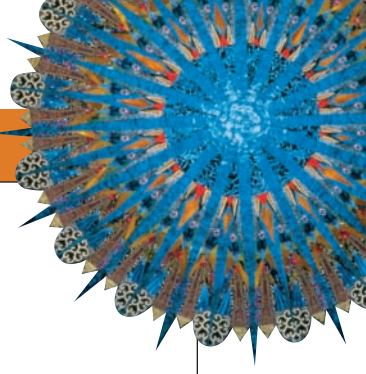
- Review normal anatomy and physiology of the immune system.
- Describe the four types of hypersensitivity reactions.
- Discuss the pathophysiology of autoimmune disorders and tissue transplant rejection.
- Discuss the characteristics of immunodeficiencies.
- Identify laboratory and diagnostic tests used to diagnose and monitor immune response.
- Describe pharmacologic and other collaborative therapies used in treating clients with altered immunity.
- Correlate the pathophysiologic alterations with the manifestations of HIV/AIDS infection.
- Use the nursing process as a framework to provide individualized care to clients with altered immune responses.

CLINICAL COMPETENCIES

- Assess functional health status of clients with altered immunity and monitor, document, and report abnormal manifestations.
- Use evidence-based practice to plan and implement nursing care for clients with AIDS.
- Assess for hypersensitivities and anticipate treatment if signs and symptoms develop.
- Provide client teaching about hypersensitivities, avoidance of sensitizing agents, and prophylactic treatment.
- Determine priority nursing diagnoses, based on assessment data, to select and implement individualized nursing interventions and teaching for clients with altered immunity.
- Protect clients who are immune suppressed.
- Recognize manifestations of developing anaphylaxis.
- Recognize manifestations of infection and minimize nosocomial exposure.
- Utilize universal precautions to protect self and clients from HIV exposure.
- Recognize the burden and benefit of HAART for the client with HIV infection.
- Integrate interdisciplinary care into care of the client with altered immunity.
- Revise plan of care as needed to provide effective interventions to promote, maintain, or restore functional health status to clients with altered immunity.

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 immunodeficiency syndrome (AIDS), *349*
allergy, *331*
allograft, *342*
anaphylaxis, *332*
autograft, *342*
autoimmune disorder, *340*
histocompatibility, *342*
human immunodeficiency virus (HIV), *349*
hypersensitivity, *331*
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Recent years have seen the emergence of new diseases affecting the immune system. These diseases include human immunodeficiency virus (HIV) infection and altered strains of familiar diseases such as multiple-drug-resistant tuberculosis. At the same time, our understanding of the components of the immune system and specific immune responses is increasing. It is therefore vital that today's nurses understand the foundations of the immune system and the immune response.

OVERVIEW OF THE IMMUNE SYSTEM

The immune system functions to protect the body from invasion by foreign antigens, to identify and destroy potentially harmful cells, and to remove cellular debris. These functions are accomplished by the lymphoid organs and specifically designed lymphocytes through the processes of antibody-mediated immune response and cell-mediated immune response.

The effectiveness of the immune system depends on its ability to differentiate normal host tissue from abnormal or foreign tissue. Body cells, tissues, and fluids have unique antigenic properties recognized by the immune system as "self." External agents, such as microorganisms, cells and tissues from other humans or animals, and some inorganic substances, have antigenic properties recognized by the immune system as "non-self."

Each body cell displays specific cell surface characteristics, or markers, that are unique to each person. These are known as human leukocyte antigens (HLAs). A person's HLA characteristics are coded within a large cluster of genes known as the major histocompatibility complex (MHC) located on chromosome 6. Recall that chromosomes are paired; each person inherits one member of the pair from each parent. A chromosome pair contains multiple genes, each carrying instructions for production of one polypeptide chain. The number of genes in the MHC results in a multitude of HLA combinations. As a result, the possibility of two people having the same HLA type is extremely remote. Identical twins may be the exception, and some siblings have very similar HLA patterns. In tissue grafting and organ transplants, matching the HLA type as closely as possible tends to decrease rejection.

Immunocompetent clients have an immune system that identifies antigens and effectively destroys or removes them. When the immune system functions improperly, the result may be an overreaction or deficiency, resulting in health problems. Overreaction of the immune system leads to hypersensitivity disorders, such as allergies. When the immune system loses the ability to recognize self, autoimmune disorders may ensue (Table 13–1). Immunodeficiency diseases or malignancies can develop when the immune system is incompetent or unable to respond effectively, as is the case with acquired immunodeficiency disorder. These alterations in immunity are discussed later in this chapter.

As discussed previously in Chapter 12 ∞, the *antibody-mediated immune response* is accomplished by B lymphocytes (B cells) that are further divided into memory cells and plasma cells. They are activated by contact with an antigen and by T cells. B cells produce antibodies, also known as immunoglobulins (see Table 12–2), and serve to inactivate an invading antigen. One immunoglobulin in particular, IgM, forms natural antibodies, such as those for ABO blood group antigens, and is an important component of the immune system complexes seen in autoimmune disorders. Memory cells "remember" an antigen, and, when exposed to it a second time, immediately initiate the immune response. This action provides the foundation of acquired immunity.

In contrast, *cell-mediated immunity* acts at the cellular level by attacking antigens directly and by activating B cells. T lymphocytes comprise the cell-mediated immune response and are subdivided into effector cells and regulator cells. The cytotoxic cell or killer T cell is the primary effector cell. Regulator T cells are divided into two subsets known as helper T cells and suppressor T cells.

Proteins on the surface of the T cell help define its function and also provide a marker that can be used to identify the cell class. These proteins are known as the cluster of differentiation antigen or *CD antigen.* The two primary CD proteins are CD4 and CD8. Both cytotoxic and suppressor T cells carry the CD8 antigen. Helper T cells have the CD4 antigen and are often called CD4 cells. CD4 cells are the most numerous of the T lymphocytes, making up 70% of the circulating population.

Helper T cells initiate the immune response, whereas suppressor T cells limit it. Helper T cells accomplish their role by promoting growth of additional T cells, by stimulating proliferation of B cells, and by activating killer T cells. It is believed that suppressor T cells are important in preventing autoimmune disorders. Proper immune system function depends on the correct balance between helper and suppressor T cells.

In addition to destroying viruses and bacteria, cytotoxic T lymphocytes also attack malignant cells. They also are responsible for the rejection of transplanted organs and grafted tissues.

TABLE 13–1 Selected Autoimmune Disorders

More organ specific	Hashimoto's thyroiditis	A chronic progressive inflammatory disease of the thyroid with lymphocyte infiltration and gradual destruction of the gland. See Chapter 19.
	Primary myxedema	Thyroid deficiency resulting from destruction of the thyroid gland due to an autoimmune process, often Hashimoto's thyroiditis. See Chapter 19.
	Thyrotoxicosis	Hyperthyroidism resulting from thyroid-stimulating immunoglobulins that stimulate activity of the gland. See Chapter 19.
	Pernicious anemia	Anemia resulting from absence of intrinsic factor associated with loss of parietal cells; most clients have antibodies to parietal cells. See Chapter 34.
	Addison's disease	Atrophy and hypofunction of the adrenal cortex, probably autoimmune in origin. See Chapter 19.
	Myasthenia gravis	A disease characterized by episodic muscle weakness caused by antibodies to the acetylcholine receptor of the neuromuscular junction. See Chapter 46.
	Insulin-dependent diabetes mellitus	Impaired insulin secretion, often the result of islet cell destruction by antibodies directed at the cell surface or cytoplasm. See Chapter 20.
	Goodpasture's syndrome	A type II hypersensitivity disorder with pulmonary hemorrhage and progressive glomerulonephritis characterized by circulating antiglomerular basement membrane antibodies. See Chapter 29.
	Multiple sclerosis	A probable autoimmune process resulting in disseminated patches of demyelination in the brain and spinal cord and varied neurologic manifestations. See Chapter 46.
	Idiopathic thrombocytopenic purpura	A chronic disorder characterized by petechiae, purpura, mucosal bleeding, and antibodies against platelets. See Chapter 34.
	Primary biliary cirrhosis	Inflammation and fibrosis of the bile ducts, probably of autoimmune origin. See Chapter 24.
	Active chronic hepatitis	A serious liver disease often resulting in hepatic failure and/or cirrhosis; may be autoimmune with infiltration by T cells and plasma cells. See Chapter 24.
Less organ specific	Ulcerative colitis	A chronic inflammatory disease of colon mucosa, possibly of autoimmune origin. See Chapter 26.
	Sjögren's syndrome	A systemic inflammatory disorder characterized by dryness of the mouth, eye, and other mucous membranes with lymphocyte infiltration of affected tissues. See Chapter 42.
	Rheumatoid arthritis	A chronic syndrome with inflammation of peripheral joints and generalized manifestations, characterized by infiltration of synovium by lymphocytes and plasma cells. See Chapter 42.
	Scleroderma	Diffuse fibrosis, degenerative changes, and vascular abnormalities of skin, joint structures, and internal organs; probably of autoimmune origin. See Chapter 42.
Non–organ specific	Systemic lupus erythematosus	An inflammatory connective tissue disorder characterized by the presence of antinuclear antibodies. See Chapter 42.

CHANGES IN IMMUNE FUNCTION IN THE OLDER ADULT

Immune function declines with aging, although many of the mechanisms leading to this decline are not clear. External factors, such as nutritional status and the effects of chemical exposure, ultraviolet radiation, and environmental pollution, affect the older adult's immune status. Internal factors affect it as well, including genetics, the function of the neurologic and endocrine systems, chronic and prior illnesses, and individual anatomic and physiologic variations. These myriad influences make it difficult to determine the effect of aging on the immune system. In some older individuals, the immune system is as effective as that of younger persons.

Whereas the antibody response to foreign antigens is diminished, autoantibodies (antibodies that react to the client's own tissues) are more common in older persons. The presence of autoantibodies suggests impaired regulation of the immune system, but it is not associated with an increased incidence of autoimmune disorders (Murasko & Gardner, 2003). The hypersensitivity response is also reduced or delayed.

ASSESSMENT OF ALTERED IMMUNE SYSTEM FUNCTION

Unlike body systems that are composed of a few closely related organs, the immune system is diverse and scattered. Optimal immune function depends on intact skin and mucous membrane barriers, adequate blood cell production and differentiation, a functional system of lymphatics and the spleen, and the ability to differentiate foreign tissue and pathogens from normal body tissue and flora. Because of this diversity of organs and function, assessment of the immune system is often integrated throughout the history and physical examination.

Health History

Prior to interviewing the client, review the biographic data, including age, sex, race, and ethnic background. This information can provide valuable clues about possible immunologic disorders. For example, many autoimmune disorders are more prevalent in women than in men. Family history is also important because there is a genetic component in the etiology of many disorders affecting the immune system.

Many interview questions related to the immune system and disorders that affect it are of a sensitive nature. Be sure to provide for privacy prior to the interview. If family members are present, request that they leave as well. Establish a trusting relationship with the client prior to asking the most sensitive questions (e.g., those related to the use of illicit drugs or sexual activity). Epidemiological data show that social and racial groups have peculiar risks for HIV infection; cultural sensitivity is necessary for effective communication.

Physical Assessment

The techniques of inspection and palpation are especially important in assessing a client's immune system.

- Assess the general appearance. Note whether the client's stated and apparent age coincide. Evident fatigue or weakness may indicate acute or chronic illness or immunodeficiency. Assess height, weight, and body type for apparent weight loss or wasting. Observe ease of movement and note any evident stiffness or difficulty moving. Check vital signs. *An elevated temperature may indicate an infection or inflammatory response.*
- Inspect the mucous membranes of the nose and mouth for color and condition. *Pale, boggy (edematous) nasal mucosa is often associated with chronic allergies. Note petechiae, white patches, or lacy white plaques in the oral mucosa; they may indicate hemolysis or immunodeficiency.*
- Assess skin color, temperature, and moisture. *Pale or jaundiced skin may indicate a hemolytic reaction. Pallor may also indicate bone marrow suppression with accompanying immunodeficiency. Inspect the skin for evidence of rashes or lesions, such as petechiae, numerous bruises, purple or blue patches or lesions indicative of Kaposi's sarcoma, and wounds that are infected, inflamed, or unhealed. Note the location and distribution of any rashes or lesions.*
- Inspect and palpate the cervical lymph nodes for evidence of lymphadenopathy (swelling) or tenderness. *Palpate the nodes of the axillae and groin as well* (see Figure 12–11).
- Assess the musculoskeletal system by inspecting and palpating the joints for redness, swelling, tenderness, or deformity. *Such changes may indicate an autoimmune disorder such as rheumatoid arthritis or systemic lupus erythematosus. Check joint range of motion as well, including that of the spine.*

ALTERED IMMUNE RESPONSES

Considering the complexity of the immune system, it is not surprising that abnormal or harmful responses occur. Altered immune system responses include those characterized by hyperresponsiveness of the immune system and those characterized by an impaired immune response. Allergies, autoimmune disorders, and reactions to organ or tissue transplants are all examples of hyperresponsive immune function. AIDS and other immunodeficiency disorders result from impairment of the immune system.

THE CLIENT WITH A HYPERSENSITIVITY REACTION

Hypersensitivity is an altered immune response to an antigen that results in harm to the client. When the antigen is environmental or exogenous, it is called an **allergy**, and the antigen is referred to as an *allergen.* The tissue response to a hypersensitivity reaction may be simply irritating or bothersome, causing a runny nose or itchy eyes, or it may be life threatening, leading to blood cell hemolysis or laryngospasm.

Hypersensitivity reactions are primarily classified by the type of immune response that occurs on contact with the allergen. They may also be classified as immediate or delayed hypersensitivity responses. Anaphylaxis and transfusion reactions are examples of immediate hypersensitivity reactions; contact dermatitis is a typical delayed response. Allergies are sometimes referred to by the affected organ system (e.g., allergic rhinitis) or the allergen involved, as in hay fever. Classification by immunologic response is the preferred means of studying allergies. Although more than one type of reaction may occur simultaneously, it is practical and insightful to study and treat allergy by classified types (King et al., 2005).

Pathophysiology

In a hypersensitivity reaction, an antigen–antibody or antigen–lymphocyte interaction causes a response that is damaging to body tissues. Antigen–antibody responses characterize types I, II, and III, also known as immediate hypersensitivity responses. Type IV hypersensitivity is an antigen–lymphocyte reaction, resulting in a delayed hypersensitivity response.

Type I IgE-Mediated Hypersensitivity

Common hypersensitivity reactions, such as allergic asthma, allergic rhinitis (hay fever), allergic conjunctivitis, hives, and anaphylactic shock, are typical of type I or IgE-mediated hypersensitivity. This type of hypersensitivity response is triggered when an allergen interacts with IgE bound to mast cells and basophils. The antigen–antibody complex prompts release of histamine and other chemical mediators, complement, acetylcholine, kinins, and chemotactic factors (Figure 13–1 ■).

When a potent allergen such as bee or wasp venom or a drug is injected, resulting in widespread antibody–antigen reaction and response to these chemical mediators, a systemic response such as anaphylaxis, urticaria, or angioedema results.

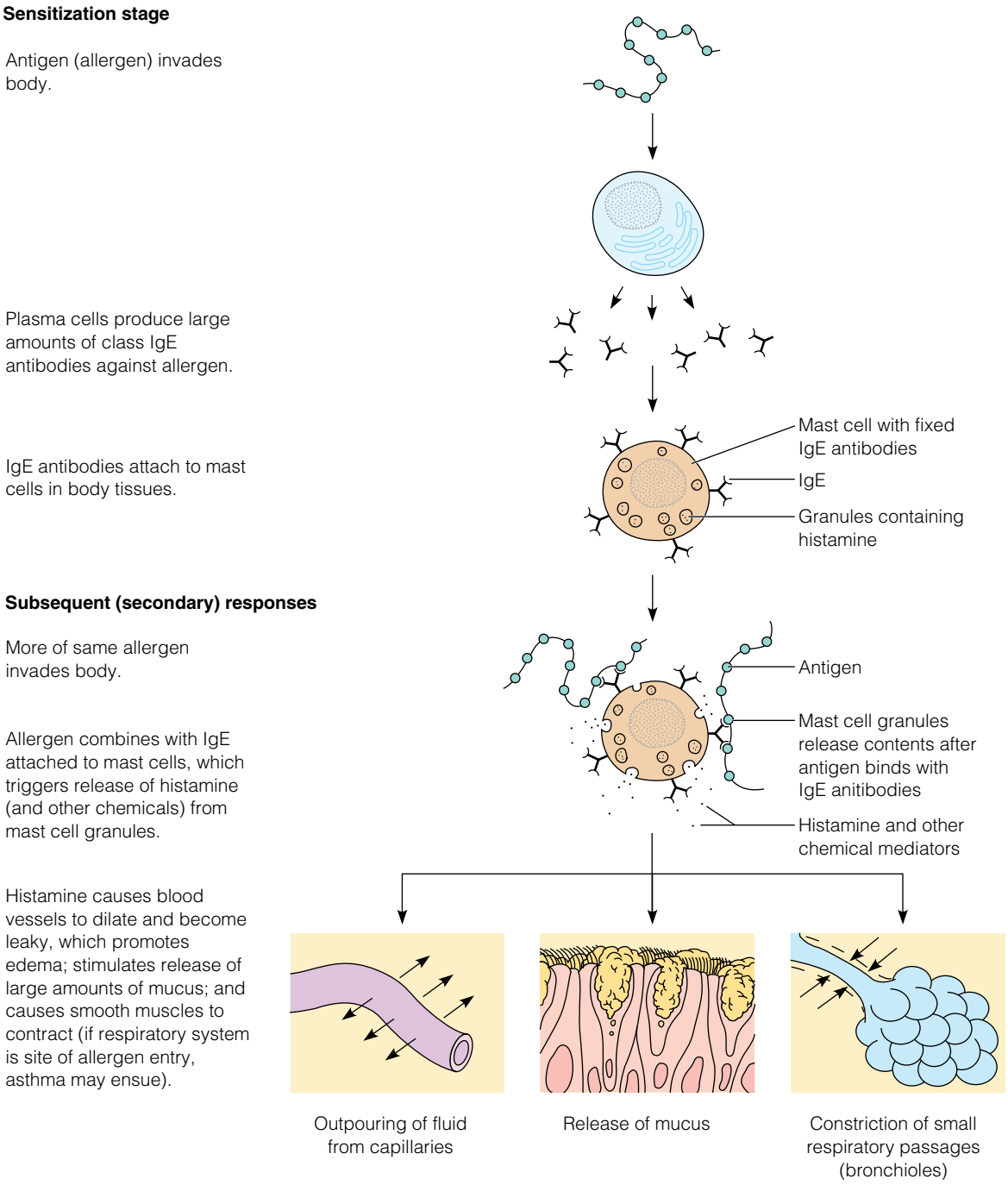


Figure 13–1 ■ Type I IgE-mediated hypersensitivity response.

MEDIALINK Histamine Animation

Anaphylaxis is an acute systemic type I response that occurs in highly sensitive persons following injection of a specific antigen. Substances known to trigger anaphylaxis are summarized in Box 13–1. Anaphylaxis rarely follows oral ingestion, although this is possible. Food allergies are the cause of 150 deaths per year in the United States (Tierney et al., 2005). The reaction begins within minutes of exposure to the allergen and may be almost instantaneous. The release of histamine and other mediators causes vasodilation and increased capillary permeability, smooth muscle contraction, and bronchial constriction. These chemical mediators cause the client to experience the typical manifestations of anaphylaxis. Initially, a sense of foreboding or uneasiness, lightheadedness, and itching palms and scalp may be noted. Hives may develop, along with angioedema (localized tissue swelling) of the eyelids, lips, tongue, hands, feet, and genitals. Swelling can also affect the uvula and larynx, impairing breathing. This is further complicated by bronchial constriction. The client exhibits air hunger, stridor and wheezing, and a barking cough. These respiratory effects can be lethal if the reaction is severe and intervention is not immediately available. Vasodilation and fluid loss from the vascular system can lead to impaired tissue perfusion and hypotension, a condition known as *anaphylactic shock*.

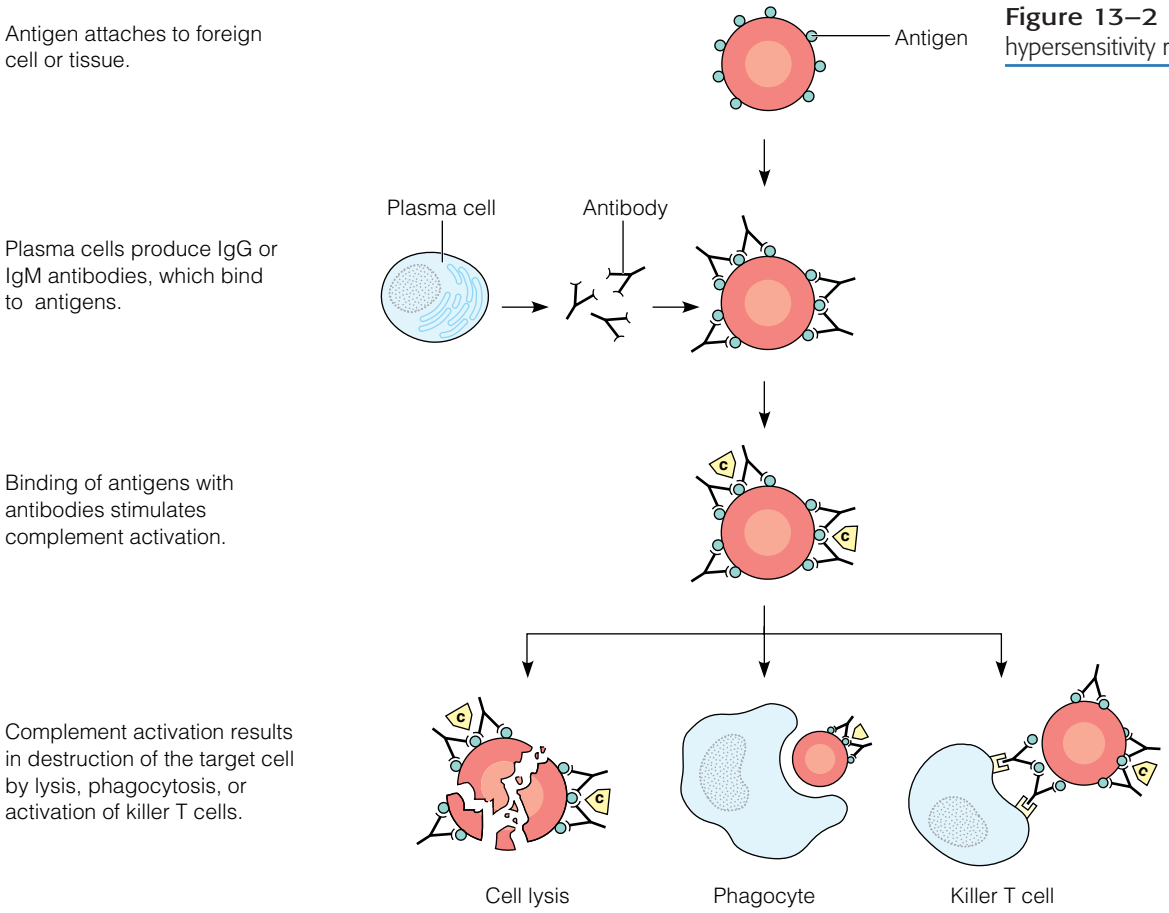


Figure 13–2 ■ Type II cytotoxic hypersensitivity response.

BOX 13–1 Substances Known to Trigger Anaphylaxis in Sensitized Persons

Hormones
- Insulin
- Vasopressin
- Parathormone

Enzymes
- Trypsin
- Chymotrypsin
- Penicillinase

Pollens
- Ragweed
- Grass
- Trees

Foods
- Eggs
- Seafoods
- Nuts
- Grains
- Beans
- Cottonseed oil
- Chocolate

Vitamins
- Thiamine
- Folic acid

Insect Venom
- Yellow jacket
- Hornet
- Paper wasp
- Honey bee

Occupational Agents
- Rubber products
- Industrial chemicals (ethylenes)

Antibiotics
- Penicillins
- Cephalosporins
- Amphotericin B
- Nitrofurantoin

Local Anesthetics
- Procaine
- Lidocaine

Medical Diagnostic Agents
- Sodium dehydrocholate
- Sulfobromophthalein

Antiserum
Antilymphocyte gamma globulin

Fortunately, localized responses are more common manifestations of type I hypersensitivity. These are typically atopic responses; that is, they have a strong genetic predisposition. Atopic reactions are the result of localized, rather than systemic, IgE-mediated responses to an allergen. They are prompted by contact of the allergen with cell-bound IgE in the bronchial tree, nasal mucosa, and conjunctival tissues. Chemical mediators are released locally, producing symptoms such as asthma, allergic rhinitis (hay fever), conjunctivitis, or atopic dermatitis. Allergens commonly associated with atopic reactions of this type include pollens, fungal spores, house dust mites, animal dander, and feathers (Porth, 2005). Food allergens can also cause localized responses such as diarrhea or vomiting. If the gastrointestinal mucosa is altered by a local allergic response, then the allergen may be absorbed, leading to a systemic reaction. Urticaria (hives) is the most common systemic response to food allergies.

Type II Cytotoxic Hypersensitivity

A hemolytic transfusion reaction to blood of an incompatible type is characteristic of a type II or cytotoxic hypersensitivity reaction. IgG or IgM type antibodies are formed to a cell-bound antigen such as the ABO or Rh antigen. When these antibodies bind with the antigen, the complement cascade is activated, resulting in destruction of the target cell (Figure 13–2 ■). Hemolytic disease of the newborn is caused by this type of reaction.

Type II reactions may be stimulated by an exogenous antigen, such as foreign tissue or cells, or a drug reaction, in which

the drug forms an antigenic complex on the surface of a blood cell, stimulating the production of antibodies. The affected cell is then destroyed in the resulting antigen–antibody reaction; for example, hemolytic anemia is sometimes associated with the administration of drugs such as penicillins, cephalosporins, and streptomycin. Withdrawal of the drug stops the reaction and cell destruction (Goldsby et al., 2003).

Endogenous antigens can also stimulate a type II reaction, resulting in an autoimmune disorder such as Goodpasture's syndrome, in which antigens are formed to specific tissues in the lungs and kidneys. Hashimoto's thyroiditis and autoimmune hemolytic anemia are additional examples of autoimmune type II reactions.

Type III Immune Complex–Mediated Hypersensitivity

Type III hypersensitivity reactions result from the formation of IgG or IgM antibody–antigen immune complexes in the circulation. When these complexes are deposited in vessel walls and extravascular tissues, complement is activated and chemical mediators of inflammation such as histamine are released. Chemotactic factors attract neutrophils to the site of inflammation. When neutrophils attempt to phagocytize the immune complexes, lysosomal enzymes are released, increasing tissue damage (Figure 13–3 ■).

Either systemic or local responses may be seen with type III reactions. For example, serum sickness is a systemic response, so named because it was first identified after administration of foreign serum (e.g., horse antitetanus toxin). Although foreign serums are no longer administered, serum sickness still occurs in response to some drugs, such as penicillin and sulfonamides. Immune complexes are deposited in walls of small blood vessels, the kidneys, and joints. Manifestations of serum sickness include fever, urticaria or rash, arthralgias, myalgias, and lymphadenopathy.

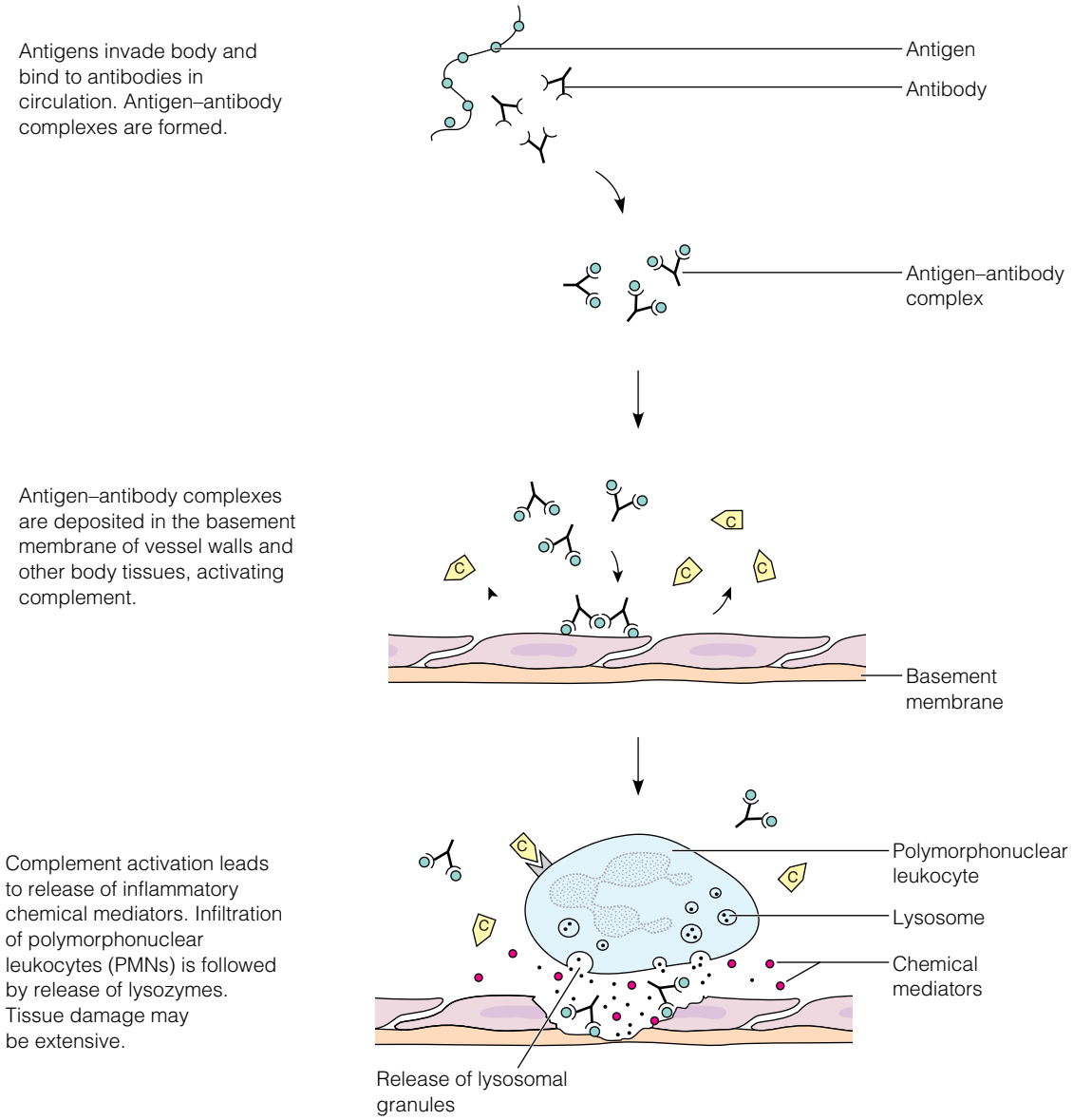


Figure 13–3 ■ Type III immune complex–mediated hypersensitivity response.

Localized responses may occur at a number of different sites. As immune complexes accumulate in the glomerular basement membrane of the kidneys—for example, following a streptococcal infection or with systemic lupus erythematosus—glomerulonephritis develops. When an antigen such as dust from moldy hay is inhaled, an acute alveolar inflammatory response can occur. This condition can develop in agricultural workers.

Type IV Delayed Hypersensitivity

Type IV reactions differ from other hypersensitivity responses in two ways. First, these reactions are cell mediated rather than antibody mediated, involving T cells of the immune system. Second, type IV reactions are delayed rather than immediate, developing 24 to 48 hours after exposure to the antigen. Type IV hypersensitivity responses result from an exaggerated interaction between an antigen and normal cell-mediated mechanisms. This exaggerated interaction results in the release of soluble inflammatory and immune mediators (from the lysozymes within the macrophages) and recruitment of killer T cells, causing local tissue destruction (Figure 13–4 ■).

Contact dermatitis is a classic example of a type IV reaction. Intense redness, itching, and thickening affect the skin in the area exposed to the antigen. Fragile vesicles are often present as well. Many antigens can provoke this response; poison ivy is a prime perpetrator. In the healthcare setting, an allergic response to latex can also produce contact dermatitis. An estimated 8% to 13% of healthcare workers are allergic to latex (National Institute of Occupational Safety and Health [NIOSH], 2005). Other examples of cell-mediated responses include a positive tuberculin test and graft rejection episodes.

Latex Allergy

Although protective against infection, the repetitive use of latex gloves creates a persistent exposure to latex for healthcare workers. When gloves are powdered with cornstarch to facilitate donning and removing gloves, the cornstarch particles aerosolize when the gloves are removed. The cornstarch includes latex particles. This creates a respiratory exposure as well as dermal exposure to latex. In addition, chemicals used in the manufacture of latex products may be irritating. Products such as balloons, condoms, and rubber bands are commonly made of latex.

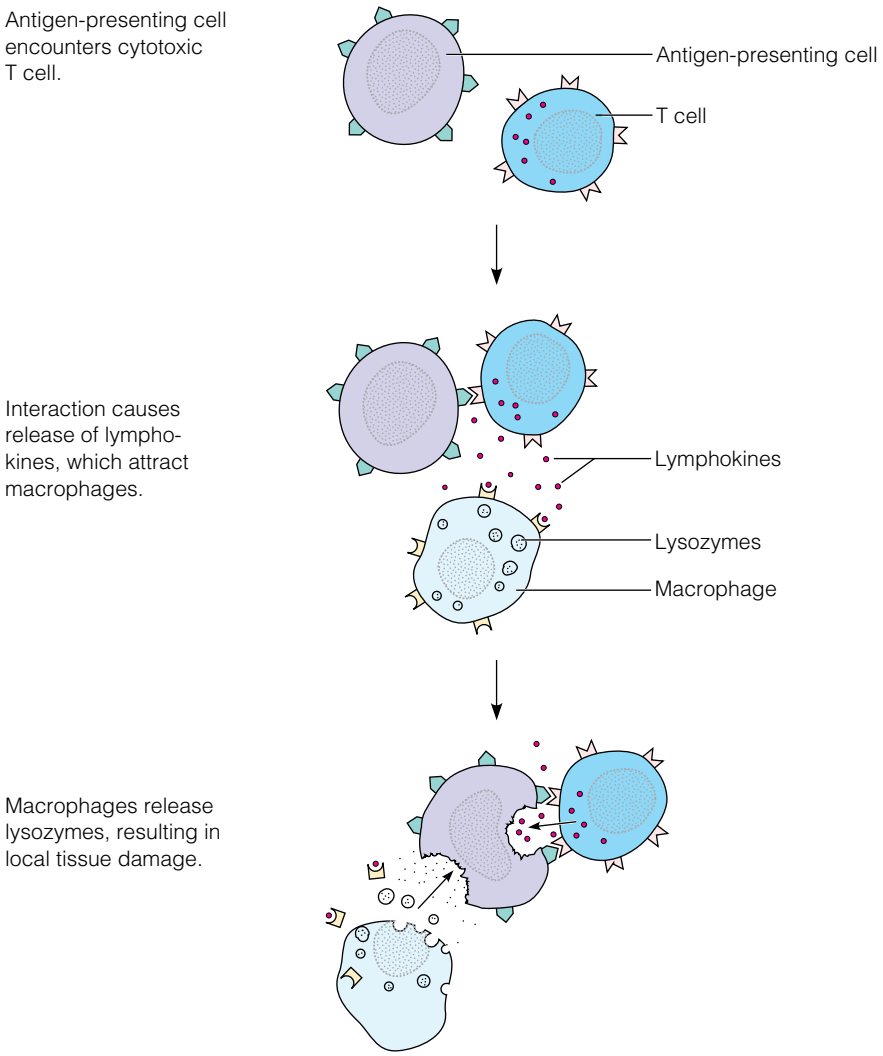


Figure 13–4 ■ Type IV delayed hypersensitivity response.

Sensitivity to latex develops without the user being aware until a rash appears on the hands. Type IV hypersensitivity (contact dermatitis) can progress to type I systemic allergic reactions without previous symptoms signaling an escalation. It is important to protect the client and the healthcare worker who is allergic to latex. Prevention is aided by employers who select products free of latex. Nonlatex gloves are recommended for use where there is no contact with infectious materials or blood. Workers should be screened periodically to detect symptoms of allergy and educated about latex sources. Hand washing after using latex products limits exposure (NIOSH, 2005).

INTERDISCIPLINARY CARE

The focus of care for clients with allergic responses is on the following:

- Minimize exposure to the allergen.
- Prevent a hypersensitivity response.
- Provide prompt, effective interventions for allergic responses when they occur.

Identifying allergens for the individual to reduce the likelihood of exposure is a key aspect of management. A complete history of the client's allergies is obtained, including medications, foods, animals, plants, and other materials. The type of hypersensitivity response is documented, as is its onset, manifestations, and usual treatment.

When a documented or suspected hypersensitivity reaction occurs, the allergen (e.g., intravenous medication or transfusion) is withdrawn immediately. With a type I hypersensitivity response, managing the client's airway takes highest priority, followed by maintaining cardiac output. Type II hypersensitivity responses may necessitate aggressive management of bleeding or renal failure. A type III (immune complex) reaction is treated by removing the offending antigen and interrupting the inflammatory response.

With a hypersensitivity response, supportive care is important to relieve discomfort. This often involves the administration of selected antihistamine or anti-inflammatory medications. Other therapies, such as plasmapheresis, may be prescribed in selected instances.

Diagnosis

To identify possible allergens or hypersensitivity reactions, the following laboratory tests may be ordered:

- *White blood cell (WBC) count with differential* can detect high levels of circulating eosinophils. Normally, eosinophils constitute a very small percentage (1% to 4%) of the total WBCs. Eosinophilia, however, is often present in clients with type I hypersensitivities.
- *Radioallergosorbent test (RAST)* measures the amount of IgE directed toward specific allergens. Test results are compared with control values and used to identify hypersensitivities. RAST poses no risk for an anaphylactic reaction. It is particularly useful in detecting allergies to some occupational chemicals and toxic allergens (Goldsby, 2003).
- *Blood type and crossmatch* are ordered prior to any anticipated transfusions. The client's ABO blood group and Rh status are determined. Two major antigens, designated A and B, may be present on RBCs. Clients with the A antigen are designated as blood type A; those with blood type B have the B antigen. When neither antigen is found on the RBCs, the person is identified as type O. A third major RBC antigen is the Rh antigen. Persons with this antigen are called Rh positive; those without are Rh negative. Because a blood transfusion is actually a transplant of living tissue, antigen matching is vital to prevent significant hypersensitivity reactions. Once blood type is determined, a sample of the client's blood is mixed with a sample of matching donor blood and observed for antigen–antibody reactions in the crossmatch portion of this test. Although this procedure greatly reduces the risk of a hemolytic transfusion reaction (type II hypersensitivity), it does not totally eliminate it.
- *Indirect Coombs' test* detects the presence of circulating antibodies (other than ABO antibodies) against RBCs. The client's serum is mixed with the donor's RBCs. If the client's serum contains antibodies to an RBC antigen, agglutination (clumping together) will occur. This is called a positive response. The normal value is negative, or no agglutination. This test is also part of the crossmatch of a blood "type and crossmatch."
- *Direct Coombs' test* detects antibodies on the client's RBCs that damage and destroy the cells. This is used following a suspected transfusion reaction to detect antibodies coating the transfused RBCs. It can also identify hemolytic anemia when the cause is unknown. In the direct Coombs' test, the client's RBCs are mixed with Coombs' serum, which contains antibodies to IgG and several complement components. Agglutination will occur if the client's RBCs are coated with antibodies, resulting in a positive test. As with the indirect Coombs' test, the normal test result is negative.
- *Immune complex assays* may be performed to detect the presence of circulating immune complexes in suspected type III hypersensitivity responses. The assays are particularly useful in diagnosing suspected autoimmune disorders. Nonspecific assays of IgG-, IgM-, and IgA-containing immune complexes, which do not detect specific antibodies, as well as specific antibody assays may be done. The normal result is a test negative for circulating immune complexes. A negative test does not, however, rule out an immune complex hypersensitivity response. In some cases, a negative result may indicate that the disease process has reached a later stage, in which complexes are no longer circulating but have initiated extensive tissue damage, such as glomerulonephritis (Kasper et al., 2005).
- *Complement assay* is also useful in detecting immune complex disorders. In these disorders, complement is, in effect, used up by the development of antigen–antibody complexes. Decreased levels are seen on examination. Both total complement level and amounts of individual components of the complement cascade can be determined.

Skin tests are also used to determine causes of hypersensitivity reactions. These tests are used to identify specific allergens to which a person may be sensitive. Allergens for testing are selected according to the client's history. Test solutions made from extracts of inhaled, ingested, or injected materials, such as pollens, mites, venoms, or some drugs, are used for the

prick test and intradermal testing. Epicutaneous testing (prick testing) is generally done first to avoid a systemic reaction; it is followed by intradermal testing of allergens with a negative response to prick testing (Tierney et al., 2005). If the large-dose intradermal test were initially made, individuals highly allergic to a substance would be at increased risk for an anaphylactic reaction. Substances that cause a reaction to the prick test should not be tested intradermally.

- *Prick (epicutaneous or puncture) test:* A drop of diluted allergenic extract is placed on the skin, and the skin is then pricked or punctured through the drop. With a positive test, a localized pruritic wheal and erythema occur. The response is maximal at 15 to 20 minutes.
- *Intradermal:* A small amount (just enough to create a wheal) of allergen extract at a 1:500 or 1:1000 dilution is injected on the forearm or intrascapular area. If several allergens are being tested, injections are spaced 0.25 to 0.5 apart. As control measures, plain diluent (negative control) and histamine (positive control) are also injected. If there is no response to a particular allergen at 15 to 20 minutes, the test is negative. The appearance of a wheal and erythema, with a wheal diameter at least 5 mm greater than that produced by the control, indicates a positive response (Figure 13–5 ■).
- *Patch:* A 1-inch patch impregnated with the allergen (e.g., perfume, cosmetics, detergents, or clothing fibers) is applied to the skin for 48 hours. Absence of a response indicates a negative test result. Positive responses are graded from mild (erythema in the exposed area) to severe (erythema, papules, vesicles, or ulceration).
- *Food allergy testing* is performed when a food allergy is suspected but the source or implicated food item has not been clearly identified. Food allergy symptoms are typically demonstrated within hours of eating. Initially, the client is asked to keep a diary of foods consumed and allergic responses for a week. An elimination diet is then prescribed. The diet excludes most common food allergens and all suspected foods for 1 week. Any foods that may contain allergens in combination, such as breads, are also eliminated. If symptoms do not improve, a different variation of the elimination diet is prescribed. If symptoms are relieved, foods are reintroduced to the diet one at a time until symptoms recur, indicating allergy to that food.

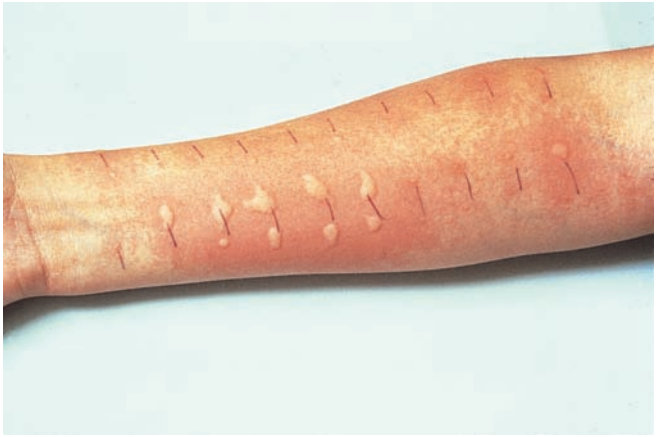


Figure 13–5 ■ Skin testing on the forearm showing induration and erythema typical of a positive response to an antigen.
Source: Southern Illinois University/Photo Researchers, Inc.

Medications

When it is impossible to avoid the offending allergen and allergic manifestations are severe or disrupt the client's activities of daily living (ADLs), pharmacologic intervention is prescribed. *Immunotherapy,* also called hyposensitization or desensitization, consists of injecting an extract of the allergen(s) in gradually increasing doses. Immunotherapy is used primarily for allergic rhinitis or asthma related to inhaled allergens. It has also been shown to be effective in preventing anaphylactic responses to insect venom. With weekly or biweekly subcutaneous injections of the allergen, the client develops IgG antibodies to the allergen that appear to block effectively the allergic IgE-mediated response. Once a therapy plateau is reached, injections are continued indefinitely either monthly or bimonthly.

Antihistamines are the major class of drugs used in treating the symptoms of hypersensitivity responses, type I in particular. They are also useful to some extent in relieving manifestations (such as urticaria) of some type II and type III reactions.

Antihistamines block H_1-histamine receptors, acting as a competitive antagonist to histamine, but they do not affect the production or release of histamine. The prototype antihistamine is diphenhydramine (Benadryl). It and other antihistamines alleviate the systemic effects of histamine such as urticaria and angioedema. They are also useful in relieving allergic rhinitis, although they are not effective in all clients. Antihistamines are available in both prescription and nonprescription preparations. The preferred route of administration is oral, although diphenhydramine and others can be given parenterally, particularly when immediate action is needed, as in anaphylaxis. They also dry respiratory secretions through an anticholinergic effect. Their use is limited by their side effects, especially drowsiness and dry mouth. Antihistamines are not effective in relieving asthmatic responses to allergens and may actually worsen symptoms by their drying effect on respiratory secretions.

Antihistamines are often combined with a sympathomimetic agent such as pseudoephedrine to improve their decongestant activity and counteract their sedative effect. Antihistamines and decongestants are discussed further in Chapter 37 ∞.

The immediate treatment for anaphylaxis is parenteral epinephrine, an adrenergic agonist (sympathomimetic) drug that has both vasoconstricting and bronchodilating effects. These qualities, combined with its rapid action, make epinephrine ideal for treating an anaphylactic reaction. For mild reactions with wheezing, pruritus, urticaria, and angioedema, a subcutaneous injection of 0.3 to 0.5 mL of 1:1000 epinephrine is generally sufficient. For clients with an injected toxin such as a bee sting, an additional amount equivalent to one-half the above may be injected directly into the site of the sting and a tourniquet applied above it to prevent further systemic absorption. Intravenous epinephrine using a 1:100,000 concentration may be used in the client with a more severe anaphylactic reaction.

Clients who have experienced an anaphylactic reaction to insect venom or other potentially unavoidable allergens should

carry a kit (commonly called a bee sting kit) for immediate treatment of future exposures. This kit typically includes a prefilled syringe of epinephrine and an epinephrine nebulizer, allowing prompt self-treatment.

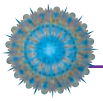
Cromolyn sodium (Intal, NasalCrom) is a drug used to treat allergic rhinitis and asthma. Cromolyn sodium acts by stabilizing the mast cell membrane, thus preventing chemical mediator release (Lehne, 2004). It is most effective when applied directly to involved tissue by inhaler or nasal spray. It has few side effects and a wide margin of safety, making it a good choice for clients in whom it is effective (Tierney et al., 2005).

Glucocorticoids (corticosteroids) are used in both systemic and topical forms for many types of hypersensitivity responses. Their anti-inflammatory effects, rather than their immunosuppressive effects, are of most benefit. A short course of corticosteroid therapy is often used for severe asthma, allergic contact dermatitis, and some immune-complex disorders (Tierney et al., 2005). Corticosteroids in topical forms or delivered by inhaler may be used for longer periods of time with few side effects; however, systemic absorption can occur.

Other Therapies

Other treatments used for hypersensitivity responses are generally dictated by the severity of the response and the organ system affected. Airway management takes highest priority for the client with an acute anaphylactic reaction. Insertion of an endotracheal tube or emergency tracheostomy may be required to maintain airway patency with severe laryngospasm. Because anaphylaxis places the person at risk for vasomotor collapse and significant hypotension, it is necessary to insert an intravenous line and initiate fluid resuscitation with an isotonic solution such as Ringer's lactate.

Plasmapheresis, removal of harmful components in the plasma, may be used to treat immune complex responses such as glomerulonephritis and Goodpasture's syndrome. Plasma and the glomerular-damaging antibody–antigen complexes are removed by passing the client's blood through a blood cell separator. The RBCs are then returned to the client along with an equal amount of albumin or human plasma. This procedure is usually done in a series rather than as a one-time treatment. It is not without risk, and informed consent is required. Potential complications of plasmapheresis include those associated with intravenous catheters, shifts in fluid balance, and alteration of blood clotting.



NURSING CARE

Nursing care related to hypersensitivity reactions is primarily directed toward prevention, early identification, and providing prompt, effective treatment.

Health Promotion

Health promotion activities include helping clients to identify possible allergens that prompt a hypersensitivity response and discussing possible strategies to avoid these allergens. Anyone with severe food allergies may need assistance from a dietitian to discuss necessary dietary changes and ways to continue meeting nutrient needs. It is important that persons with hypersensitivities inform healthcare personnel of all allergens. People who experience anaphylactic reactions should wear a Medic-Alert bracelet or tag at all times to identify the substance(s) that provokes this response.

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:* risk factors, hypersensitivities (medications, household dust, bee stings, etc.), reaction (rash, hives, difficulty breathing), type of treatment for hypersensitivity reactions; allergy skin testing; asthma, hay fever, or dermatitis
- *Physical assessment:* mucous membranes of nose and mouth, skin for lesions or rashes, eyes (tearing and redness), respiratory rate, and adventitious breath sounds.

Nursing Diagnoses and Interventions

Priority nursing diagnoses will vary according to the type of hypersensitivity reaction experienced by the client. Because nurses are most likely to become involved with a client experiencing a type I or type II response, this section focuses on diagnoses for these clients. Airway, breathing, and circulation (the ABCs) are of greatest importance for the client with an anaphylactic reaction. When a hemolytic reaction to an incompatible blood transfusion occurs, the client is at risk for injury.

Ineffective Airway Clearance

PRACTICE ALERT
Placing the client in Fowler's to high-Fowler's position allows optimal lung expansion and ease of breathing.

In anaphylactic reactions, the airway may be obstructed due to facial angioedema, bronchospasm, or laryngeal edema. Establishing and maintaining a patent airway is of highest priority.

- Administer oxygen per nasal cannula at a rate of 2 to 4 L/min. Apply oxygen emergently and obtain a physician order for oxygen administration. *This increases the alveolar oxygen and its availability to cells of the body.*
- Assess respiratory rate and pattern, level of consciousness and anxiety, nasal flaring, use of accessory muscles of respiration, chest wall movement, audible stridor; palpate for respiratory excursion; auscultate lung sounds and any adventitious sounds, such as wheezes. *Extreme anxiety or agitation, nasal flaring, stridor, and diminished lung sounds indicate air hunger and possible airway obstruction, necessitating immediate intervention.*
- Insert a nasopharyngeal or oropharyngeal airway, and arrange for immediate intubation as indicated. *Ensuring an adequate airway is vital to preserve life.*
- Administer subcutaneous epinephrine 1:1000, 0.3 to 0.5 mL, as prescribed. This may be repeated in 20 to 30 minutes if necessary. Administer parenteral diphenhydramine (deep intramuscular or intravenous) as prescribed. *Epinephrine is a potent vasoconstrictor and bronchodilator, counteracting*

the effects of histamine. Diphenhydramine is an antihistamine that blocks histamine receptors and their effect. These medications can be effective in rapidly reversing manifestations of anaphylaxis.

- Provide calm reassurance. *Hypoxemia and air hunger are terrifying for the client. Anxiety can impair the client's ability to cooperate with treatment and can increase the respiratory rate, making breathing less effective.*

Decreased Cardiac Output

Peripheral vasodilation and increased capillary permeability from the release of histamine can significantly impair cardiac output. When it falls to the degree that tissue perfusion becomes impaired and hypoxia results, a state of anaphylactic shock exists.

- Monitor vital signs frequently, noting fall in blood pressure, decreasing pulse pressure, tachycardia, and tachypnea. *These vital sign changes may indicate shock.*
- Assess skin color, temperature, capillary refill, edema, and other indicators of peripheral perfusion. *As cardiac output falls, peripheral vessels constrict and tissue perfusion is impaired.*
- Monitor level of consciousness. *A change in level of consciousness (lethargy, apprehension, or agitation) is often the first indicator of decreased cardiac output.*
- Insert one or more large-bore (18-gauge or larger) intravenous catheters. *It is important to insert intravenous catheters as soon as possible to provide sites for rapid fluid replacement.*
- Administer warmed intravenous solutions of lactated Ringer's or normal saline, as prescribed. *These isotonic solutions help maintain intravascular volume. Warmed solutions are used to prevent hypothermia from the rapid administration of large amounts of fluid at room temperature (about 70°F, or 21.1°C).*
- Insert an indwelling catheter, and monitor urinary output frequently. *As the cardiac output drops, the glomerular filtration rate (GFR) falls. With an output of less than 30 mL/h, the client is at risk for acute renal failure from ischemia.*

PRACTICE ALERT

Aggressive fluid therapy may lead to hypervolemia and pulmonary edema; assess for shortness of breath and crackles in the lungs.

- Place a tourniquet above the site of an injected venom (such as a bee sting), and infiltrate the site with epinephrine as prescribed. *Use of a tourniquet and the vasoconstriction resulting from epinephrine infiltration reduce further absorption of the allergen.*
- Once breathing is established, place the client flat with the legs elevated. *This position enhances perfusion of the central organs, such as the brain, heart, and kidneys.*

Risk for Injury

As noted, the potential for hypersensitivity responses is high in clients subjected to medical treatments. Because a blood transfusion is a transplant of living tissue, the risk for adverse immunologic response and injury is particularly significant.

- Obtain and record a thorough history of previous blood transfusions and any reactions experienced, *no matter how mild.* Alert the physician if previous transfusion reactions have occurred. *The client who has received prior blood transfusions is at increased risk for a hypersensitivity reaction, because antibody production may have been stimulated by prior exposure to antigens.*

PRACTICE ALERT

Begin a blood transfusion within 30 minutes of its delivery from the blood bank to reduce bacterial contamination.

- Check for a signed informed consent to administer blood or blood products. *It is important to obtain informed consent for this invasive and risky procedure.*
- Using two licensed healthcare professionals, double-check patient identity, blood type, Rh factor, crossmatch, and expiration date for all blood and blood components received from the blood bank with the client's data. *This is an important safety measure to reduce the risk of a hemolytic transfusion reaction due to incompatible blood types.*
- Take and record vital signs within 15 minutes prior to initiating the blood infusion. Acetaminophen and diphenhydramine are prescribed and administered prior to beginning a blood transfusion to decrease inflammation and increase client comfort. The medications will not mask serious reactions. *This provides a baseline for evaluating any changes related to the blood transfusion.*
- Infuse blood into a site separate from any other intravenous infusion. Use at least 20-gauge catheter for the infusion to promote flow. *This reduces the risk of damage to the blood cells due to incompatibility with other intravenous solutions or physical trauma. When blood is administered with dextrose solutions (e.g., D_5W, D_5NS), blood cell hemolysis and aggregation occur; administration with lactated Ringer's can cause agglutination of cells. Administer with normal saline to prime intravenous tubing.*
- Administer 50 mL of blood during the first 15 minutes of the transfusion. *Reactions generally occur within the first 15 minutes.*
- During transfusion, monitor for complaints of back or chest pain, an increase in the temperature of more than 1.8°F, chills, tachycardia, tachypnea, wheezing, hypotension, hives, rashes, or cyanosis. *These signs may indicate an adverse reaction to the blood transfusion.*
- Stop the blood transfusion immediately if a reaction occurs, no matter how mild. Remove the blood bag and the tubing with blood in it. Flush new intravenous tubing with normal saline, keeping the intravenous line open. Notify the physician and the blood bank.
- If a reaction is suspected, send the blood and administration set to the laboratory with a freshly drawn blood sample and urine specimen from the client. *These will be used to identify the cause of the reaction as well as its effect on the client.*
- If no adverse reaction occurs, administer the transfusion over 2 to 4 hours. *This time frame is important to limit the risk of bacterial growth.*

Community-Based Care

The vast majority of hypersensitivity responses are appropriately treated by the client or family members with little or no medical intervention. Teaching, therefore, is a vital component of care. If the client is at risk for anaphylaxis, involving the family in teaching is essential because the response may occur with such rapidity that the client will be unable to provide self-care.

Include the following points in teaching the client and family about managing hypersensitivities:

- When and how to use an anaphylaxis kit containing epinephrine and antihistamines in injectable, inhaler, and oral forms
- When to seek medical attention
- Use and adverse reactions of prescription and nonprescription antihistamines and decongestants
- Advantages of autologous blood transfusion if future surgery is scheduled
- Preventing an immune complex reaction such as glomerulonephritis
- Skin care to prevent contact dermatitis, including:
  - Expose affected areas to air and sun as much as possible.
  - Avoid direct contact with people who have an infection.
  - Wear cool, light, nonrestrictive clothing of natural fibers, such as cotton, to avoid irritating affected areas.
  - Avoid exposure to extremes of heat or cold.
  - Use bath oils or plain water instead of soaps and detergents.
  - Take tub baths in cool to lukewarm water rather than showers.
  - To decrease pruritus, maintain a cool environment and avoid exercising.
  - Trim fingernails to reduce the risk of skin damage.
- Helpful resources:
  - ALERT, Inc., Allergy to Latex Education and Resource Team
  - Food Allergy Network

THE CLIENT WITH AN AUTOIMMUNE DISORDER

Maintaining optimal health and preventing disease depend not only on the immune system's ability to recognize and destroy foreign tissues and other antigens, but also on the immune system's ability to recognize self. When this self-recognition is impaired and immune defenses are directed against normal host tissue, the result is an **autoimmune disorder**.

Autoimmune disorders can affect any tissue in the body. Some are tissue or organ specific, affecting particular tissue or a particular organ. Hashimoto's thyroiditis is an example of an organ-specific autoimmune disorder. Circulating antibodies are formed to certain thyroid components, resulting ultimately in destruction of the gland. In other disorders, autoantibodies are formed that are not tissue specific, but tend to accumulate and cause an inflammatory response in certain tissue, for example, the renal glomerulus or the hepatic small bile ductules. Autoimmune disorders may also be systemic, with neither antibodies nor the resulting inflammatory lesions confined to any one organ. Rheumatologic disorders, such as rheumatoid arthritis and systemic lupus erythematosus (SLE), are characteristic of systemic autoimmune disorders (Goldsby et al., 2003). A list of selected autoimmune disorders is included in Table 13–1.

Pathophysiology

The mechanism that causes the immune system to recognize host tissue as a foreign antigen is not clear. The following factors are under study as possible contributors to the development of autoimmune disorders:

- The release of previously "hidden" antigens into the circulation, such as DNA or other components of the cell nucleus, which elicits an immune response
- Chemical, physical, or biologic changes in host tissue that cause self-antigens to stimulate the production of autoantibodies
- The introduction of an antigen, such as a bacteria or virus, whose antigenic properties closely resemble those of host tissue, resulting in the production of antibodies that target not only the foreign antigen but also normal tissue. Heart damage in rheumatic fever and encephalitis following rabies vaccination are examples of the development of antibodies against normal tissue (Goldsby et al., 2003).
- A defect in normal cellular immune function that allows B cells to produce autoantibodies unchecked
- Initiation of the autoimmune response by very slow-growing mycobacteria.

Although the exact mechanism producing autoimmunity is unclear, several characteristics of autoimmune diseases are known. It is apparent that genetics plays a role because a higher incidence is seen in family members of people with autoimmune disorders. Autoimmune disorders are far more prevalent in females than in males. The disorders tend to overlap, so that the client with one autoimmune disorder may develop another or some manifestations of another. The onset of an autoimmune disorder is frequently associated with an abnormal stressor, either physical or psychologic. Autoimmune disorders are frequently progressive relapsing-remissing disorders characterized by periods of exacerbation and remission.

Specific autoimmune disorders are discussed in the sections of this textbook related to the affected organ systems or functional disruption.

INTERDISCIPLINARY CARE

For the most part, the diagnosis of an autoimmune disorder is based on the client's clinical manifestations. Serum assays are useful to identify autoantibodies. Other diagnostic tests are generally specific to the suspected disorder and to identifying the degree of tissue damage and destruction. Although the manifestations of these disorders can often be managed, a cure typically is not possible unless the affected target tissue is removed (e.g., colectomy for the client with ulcerative colitis).

Diagnosis

Serologic assays are used to identify and measure antibodies directed toward host tissue antigens or normal cellular components. Many detectable autoantibodies are not specific to a single autoimmune disorder and are used to establish the autoimmune

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process rather than the specific disorder. Although healthy people often have low levels of autoantibodies, levels are much higher in clients affected by an autoimmune disorder. The following serologic assays may be ordered:

- *Antinuclear antibody (ANA)* detects antibodies produced to DNA and other nuclear material. These antibodies can cause tissue damage characteristic of autoimmune disorders, such as SLE. The client's serum is combined with nuclear material and tagged antihuman antibody to detect ANA-antihuman antibody complexes. A negative, or normal, result is a titer <1:20. When complexes are detected at higher titer levels (>1:20), the test is positive for ANA. This test is not specific for SLE, because high levels of ANA may be present in rheumatoid arthritis (RA) and cirrhosis of the liver; nevertheless, 95% of clients with SLE have a positive ANA titer.
- *Lupus erythematosus (LE) cell test* is also used to detect SLE and monitor its treatment. Neutrophils that contain large masses of phagocytized DNA from the nuclei of PMNs are called LE cells. Like the ANA, the LE cell prep is nonspecific for SLE. A positive result may also be seen in RA or with medications such as isoniazid, penicillin, phenytoin, procainamide, streptomycin, or sulfonamide drugs.
- *Rheumatoid factor (RF)* is an immunoglobulin present in the serum of approximately 80% of clients with rheumatoid arthritis. Low titer levels (<1:20) may be present in the elderly. An RF titer 1:80 or higher indicates RA. A titer between 1:20 and 1:80 could indicate SLE, scleroderma, or liver cirrhosis (Pagana & Pagana, 2002).
- *Complement assay* may also be useful in identifying autoimmune disorders. In these disorders, complement may be consumed in the development of antigen–antibody complexes. Decreased levels are seen on examination. Both total complement level and amounts of individual components of the complement cascade can be determined.

Medications

Various approaches are used in the treatment of autoimmune disorders. Anti-inflammatory medications such as aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs), and corticosteroids may be prescribed to reduce the inflammatory response and minimize tissue damage. (Refer to Chapter 9 ∞ for additional detail on these drugs.) When these agents are not effective or well tolerated by the client, slow-acting anti-inflammatory medications may be prescribed. Slow-acting or antirheumatic drugs include such medications as gold salts, hydroxychloroquine (Plaquenil), and penicillamine. Their use is further detailed in Chapter 42 ∞. Cytotoxic drugs may be used in combination with plasmapheresis in treating many autoimmune disorders. Cytotoxic drugs are discussed in further detail in the next section of this chapter. Disease modifying antirheumatic drugs (DMARDs) reduce signs and symptoms, reduce or prevent joint damage, and preserve the structure and function of the joints in patients with RA. These drugs may reduce health costs for clients with RA and allow them to remain active and productive. The most common DMARDs in current use are methotrexate (Rheumatrex), sulfasalazine (Azulfidine), hydroxychloroquine (Plaquenil), leflunomide (Arava), and cyclosporine (Sandimmune, Neoral).

Another class of antirheumatic drugs, referred to as *biologicals* or *biological response modifiers,* consists of laboratory-produced proteins that decrease the inflammatory process. These antibodies bind tumor necrosis factor alpha (TNF-α) and interleukin-1, both inflammatory elements. These medications include infliximab (Remicade) or adalimumab (Humira), etanercept (Enbrel), and anakinra (Kineret).

NURSING CARE

Nursing care measures for the client with an autoimmune disorder are individualized and tailored to needs dictated by manifestations of the disorder. Nurses often will be involved with the client in an outpatient setting such as an office or home, evaluating the client's response to therapy and self-care management.

Consider the following nursing diagnoses in planning care for the client with an autoimmune disorder:

- *Activity Intolerance* related to inflammatory effects of autoimmune disorder
- *Ineffective Coping* related to chronic disease process
- *Interrupted Family Processes* related to lack of understanding about autoimmune disorder and its effects
- *Ineffective Protection* related to disordered immune function
- *Risk for Ineffective Therapeutic Regimen Management* related to lack of understanding

Community-Based Care

Because many autoimmune disorders are chronic, teaching the client and family about the disorder and its management is a key nursing care component. The client may be taking drugs with multiple side effects or long-term effects, necessitating effective teaching. Clients with autoimmune disorders often do not appear to be ill, making it difficult for friends and families to understand their care needs. The chronicity of these disorders also puts the client at high risk for unproven remedies and quackery. Provide psychologic support, listening, and teaching. In addition, suggest resources such as local support groups and the American Autoimmune Related Diseases Association.

THE CLIENT WITH A TISSUE TRANSPLANT

Since the first kidney transplant was performed from one identical twin to the other in 1954, organ and tissue transplantation have become increasingly popular and viable treatment options. The transplantation of avascular tissues, such as skin, cornea, bone, and heart valves, is considered routine, with little need for tissue matching and **immunosuppression**. Transplants of organs (e.g., the kidney, heart, heart and lung, liver, and bone marrow) are increasingly common and are no longer considered experimental or extraordinary procedures. Common organ transplants are outlined in Table 13–2.

Transplant success is closely tied to obtaining an organ with tissue antigens as close to those of the recipient as possible. As noted earlier in this chapter, every body cell has cell surface antigens known as human leukocyte antigens that are unique to the individual. Although identical twins may have the same

TABLE 13–2 **Organ Transplants**

ORGAN	GRAFT TYPE	INDICATIONS FOR TRANSPLANT	SUCCESS RATE
Kidney	Allograft; may be isograft	End-stage renal disease	81–89% at 5 years
Heart	Allograft	End-stage cardiac disease refractory to medical management	72.5% at 5 years
Lung	Allograft	Pulmonary hypertension, cystic fibrosis, pulmonary fibrosis, chronic obstructive pulmonary disease	46% at 5 years
Liver	Allograft	Severe liver dysfunction due to chronic active hepatitis, primary biliary cirrhosis, sclerosing cholangitis	72–78% 5-year survival
Bone marrow	Autograft or allograft	Leukemia, aplastic anemia, congenital immunologic defects	30–70% cure
Skin	Autograft, allograft, or xenograft	Severe burns, plastic surgery	>95% at 5 years
Cornea	Allograft	Corneal ulceration and opacification	>95% at 5 years
Pancreas	Allograft	Pancreatic insufficiency, diabetes	80.3% pancreas
Islet cells	Allograft (multiple donor)	Type 1 diabetes mellitus	100% >2 years

Source: Data from *Current Medical Diagnosis and Treatment* (44th ed.) by L. M. Tierney, S. J. McPhee, and M. A. Papadakis, 2005, New York: Lange Medical Books/McGraw-Hill; US Transplant.org; Scientific registry of transplant recipients, accessed 10/23/05. http://www.ustransplant.org/

HLA type, the chance is reduced to 1 in 4 for siblings, and less than 1 in several thousand for unrelated individuals (Tierney et al., 2005). Matching the HLA type of the donor and recipient as closely as possible decreases the potential for rejection of the transplanted organ or tissue but does not eliminate it.

Pathophysiology

An **autograft**, a transplant of the client's own tissue, is the most successful type of tissue transplant. Skin grafts are the most common examples of autografts. Increasingly, autologous bone marrow transplants and blood transfusions are being used to reduce immunologic responses. When the donor and recipient are identical twins, the term **isograft** is used. Because of the high likelihood of an HLA match, the success of these grafts is good and rejection episodes are mild. Few people, however, have an identical twin to provide tissue for donation; and when the need is for an organ such as the heart, liver, or lungs, a living-donor transplantation is not possible. Most often, organ and tissue transplants are **allografts**, which are grafts between members of the same species but who have different genotypes and HLA. Allografts may come from living donors; examples are bone marrow, blood, and a kidney. Most often, however, organs for transplantation are obtained from a cadaver. Donors are typically people who meet the criteria for brain death; are less than 65 years old; and are free of systemic disease, malignancy, or infection, including HIV, hepatitis B, or hepatitis C. The organ is removed immediately before or after cardiac arrest and preserved until it is transplanted into the waiting recipient. Finally, **xenograft** is a transplant from an animal species to a human. These transplants are the least successful but may be used in selected instances, such as the use of pig skin as a temporary covering for a massive burn.

Tissue typing is used to determine **histocompatibility**, the ability of cells and tissues to survive transplantation without immunologic interference by the recipient. Tissue typing is performed in an attempt to match the donor and recipient as closely as possible for HLA type and blood type (ABO, Rh) and to identify preformed antibodies to the donor's HLA.

Both antibody-mediated and cell-mediated immune responses are involved in the complex process of transplant rejection. Host macrophages process donor antigen, presenting it to T and B lymphocytes. Activated lymphocytes (B and T cells) produce both antibody- and cell-mediated effects. Killer T cells bind with cells of the transplanted organ, resulting in cell lysis. Helper T cells stimulate the multiplication and differentiation of B cells, and antibodies are produced to graft endothelium. Complement activation or antibody-dependent cell-mediated cytotoxicity leads to transplant cell destruction. Rejection typically begins after the first 24 hours of the transplant, although it may present immediately. Rejection episodes are characterized as hyperacute, acute, or chronic, as summarized in Table 13–3.

Hyperacute tissue rejection occurs immediately to 2 to 3 days after the transplant of new tissue. Hyperacute rejection is due to preformed antibodies and sensitized T cells to antigens in the donor organ. Hyperacute rejection is most likely to occur in clients who have had a previous organ or tissue transplant, such as a blood transfusion. Hyperacute rejection may be evident even before the transplant procedure is completed. The grafted organ initially appears pink and healthy, but soon becomes soft and cyanotic as blood flow is impaired. Organ function deteriorates rapidly, and symptoms of organ failure develop.

Acute tissue rejection is the most common and treatable type of rejection episode. It occurs between 4 days and 3 months after the transplant. Acute rejection is mediated primarily by the cellular immune response, resulting in transplant cell destruction. The client experiencing acute rejection demonstrates manifestations of the inflammatory process, with fever, redness, swelling, and tenderness over the graft site. Signs of impaired function of the transplanted organ may be noted (e.g., elevated blood urea nitrogen [BUN] and creatinine, liver enzyme and bilirubin elevations, or elevated cardiac enzymes and signs of cardiac failure).

Chronic tissue rejection occurs from 4 months to years after transplant of new tissue. Chronic rejection is most likely the re-

TABLE 13–3 **Transplant Rejection Episodes**

TYPE	CAUSE	PRESENTATION	TREATMENT
Hyperacute	Preexisting antibodies to donor ABO or HLA antigens	Occurs within minutes to hours or days of the transplant Rapid deterioration of organ function	The transplant usually cannot be saved; prevent with crossmatch, and use antimetabolites or anti-inflammatory drugs before surgery.
Acute	Primarily a cell-mediated immune response to HLA antigens; antibody-mediated response may also contribute	Occurs within days to months after the transplant Signs of inflammation and impaired organ function	Increase immunosuppression using steroids, cyclosporine, monoclonal antibodies, or antilymphocyte globulins.
Chronic	Probably antibody-mediated response; may also involve inflammatory damage to vessel endothelium	Occurs 4 months to years after the transplant Gradual deterioration of organ function	None; loss of graft will occur, requiring retransplant.

sult of antibody-mediated immune responses. Antibodies and complement are deposited in transplant vessel walls, causing narrowing and decreased function of the organ due to ischemia. The gradual deterioration of transplanted organ function is seen with chronic tissue rejection.

Graft-versus-host disease (GVHD) is a frequent and potentially fatal complication of bone marrow transplant. When there is no close match between donor and recipient HLA, immunocompetent cells in the grafted tissue recognize host tissue as foreign and mount a cell-mediated immune response. If the host is immunocompromised, as is often the case when a bone marrow transplant is performed, host cells are unable to destroy the graft and instead become the targets of destruction. Of clients with very closely matched bone marrow, 30% to 60% nevertheless develop GVHD. Acute GVHD occurs within the first 100 days following a transplant and primarily affects the skin, liver, and gastrointestinal tract. The client develops a maculopapular pruritic rash beginning on the palms of the hands and soles of the feet. The rash may spread to involve the entire body and lead to desquamation. Gastrointestinal manifestations include abdominal pain, nausea, and bloody diarrhea. GVHD that lasts longer than 100 days is said to be chronic. If it is limited to the skin and liver, the prognosis is good. If multiple organs are involved, the prognosis is poor (Porth, 2005).

INTERDISCIPLINARY CARE

Pretransplant care and post-transplant care are directed toward reducing the risk that transplanted tissue will be rejected or result in GVHD. Diagnostic studies are directed first at identifying a suitable donor, then at monitoring the immune response to the transplant. Immunosuppressive therapy with medications is a vital part of post-transplant care. Indeed, the development of effective immunosuppressive drugs is responsible for the success of organ transplants using allografts.

Diagnosis

The following diagnostic tests may be ordered prior to organ or tissue transplantation:

- *Blood type and Rh factor* of both the donor and recipient are determined. Although there is some question about the benefit of histocompatibility testing prior to transplant of a cadaver organ, there is no question about the need for ABO blood group compatibility.
- *Crossmatching* of the client's serum against the donor's lymphocytes is performed to identify any preformed antibodies against antigens on donor tissues. If present, these antibodies would likely result in an immediate or hyperacute graft rejection with probable loss of the transplant.
- *HLA histocompatibility testing* identifies donors with an HLA type close to that of the recipient. It is used primarily to identify living donors for bone marrow and kidney transplant. Because of GVHD, histocompatibility tests to identify an identical or very close HLA match are particularly important in bone marrow transplant. HLA tests are performed using lymphocytes from a blood sample. The sample should not be obtained within 72 hours of a blood transfusion, because this will interfere with results.
- *Mixed lymphocyte culture (MLC) assay tests* also are used to determine histocompatibility between the donor and the recipient. This test identifies whether mononuclear cells of the recipient will react against the potential donor's leukocyte antigens. The disadvantage of this test is that results cannot be obtained until 7 to 10 days later (Chernecky & Berger, 2004). If the intended recipient is severely immunocompromised, the results may be falsely negative. Clients treated with chemotherapy within 2 weeks of specimen collection are potentially immunocompromised.
- *Ultrasonography* or *magnetic resonance imaging (MRI)* of the transplanted organ may be performed to evaluate its size, perfusion, and function.
- *Tissue biopsies* of the transplanted organ are performed routinely to assess for evidence of tissue rejection.

Medications

Prior to transplantation, several antibiotic and antiviral drugs may be prescribed, including the following:

- Trimethoprim-sulfamethoxazole (Septra, Bactrim), which decreases the incidence of gram-negative bacterial infections

- Acyclovir (Zovirax), which prevents the development of herpes simplex virus pneumonia in bone marrow transplant recipients
- Ganciclovir (Cytovene), which prevents the development of cytomegalovirus (CMV) pneumonia with bone marrow transplant recipients.

The mainstays of drug therapy for clients following a tissue or organ transplant are immunosuppressive agents. Varying regimens of these drugs are used, depending on the transplanted tissue and the medical center; however, a combination of corticosteroids and cyclosporine is common for maintenance therapy. Antilymphocyte therapy and the use of monoclonal antibodies are increasingly common in the immediate post-transplant period and for treating steroid-resistant rejection episodes.

Corticosteroids, primarily prednisone (Deltasone, others) and methylprednisolone (Solu-Medrol, others), were among the first medications used to prevent transplant rejection, and they remain important agents today. Although the exact anti-inflammatory and immunosuppressive activity of corticosteroids is unknown, they are known to suppress production of interleukin-1 and -2, decrease monocyte migration, and suppress proliferative and cytotoxic T-cell activity. Although they are very effective, large doses of corticosteroids used post-transplant are associated with significant adverse effects. Wound healing is impaired, and the metabolism of fats, proteins, and carbohydrates is altered. Blood glucose increases with steroid use, impairing glucose control. Fat distribution changes, producing a cushingoid appearance with moon facies, increased truncal fat, and "buffalo hump." Fluid retention and hypertension are potential problems, as are osteoporosis, gastrointestinal bleeding, and emotional disturbances.

Azathioprine (Imuran) has been in use as an immunosuppressant for more than 25 years and continues to be a component of many regimens. Azathioprine inhibits both cell-mediated and antibody-mediated immunity, although its activity is more specific for T cells than B cells. Because it is rapidly metabolized by the liver, azathioprine can be given to clients with impaired renal function but may not be effective in clients with impaired hepatic function. Bone marrow suppression is the most common adverse effect of this drug, necessitating frequent evaluation of the complete blood count (CBC). Hepatotoxicity, pancreatitis, and increased risk of neoplasm are also associated with azathioprine administration. Nursing responsibilities related to azathioprine are listed in the Medication Administration box on the following pages. Clients who cannot tolerate azathioprine may receive a newer immunosuppressant, mycophenolate mofetil (CellCept). Primarily, it is prescribed following renal and cardiac transplants. See Figure 13–6 ■.

Cyclosporine has contributed significantly to the success of organ transplantation since its introduction in the 1970s. Cyclosporine inhibits T-cell function and the normal cell-mediated immune response. The incidence of cyclosporine toxicity and side effects is related to blood levels, so blood levels are monitored closely. Cyclosporine is both nephrotoxic and hepatotoxic, especially at high doses. Observable toxic effects include hypertension and CNS symptoms such as flushing or tingling of the extremities, confusion, visual disturbances, and seizures or coma.

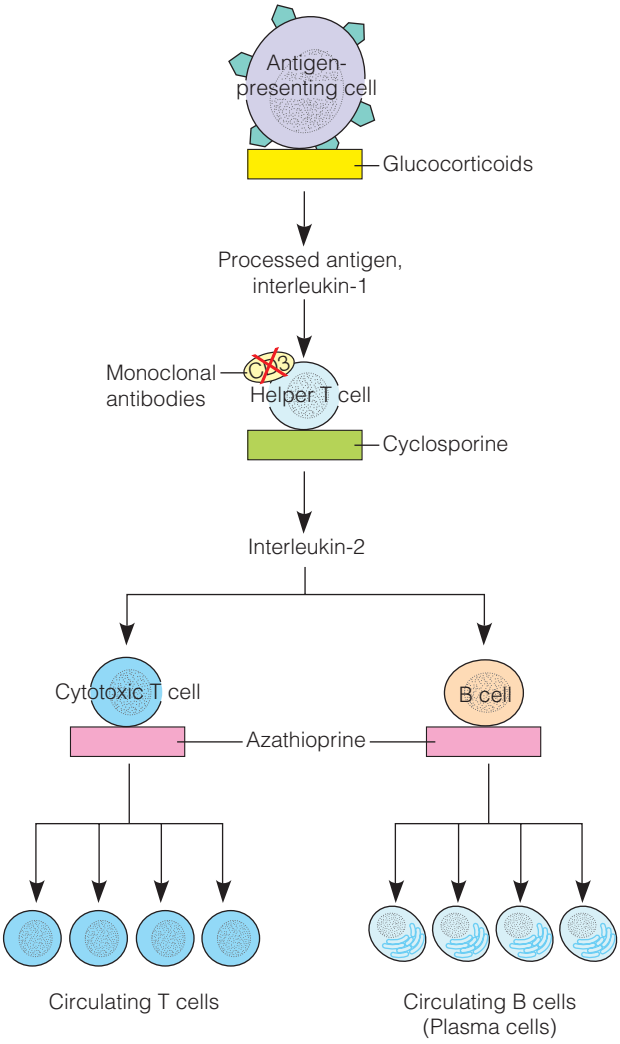
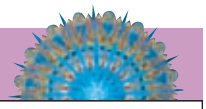


Figure 13–6 ■ Sites of action of immunosuppressive agents.

Muromonab-CD3, also known as OKT3 or Orthoclone, is the first monoclonal antibody produced for therapeutic use in humans. As a monoclonal antibody, OKT3 is specific to T cells, blocking their generation and function. It binds with a surface antigen on T cells, inactivating and removing them from circulation. It also blocks killer T cells attached to the graft. Because of significant side effects, the use of OKT3 is limited primarily to treatment of steroid-resistant rejection. Two newer monoclonal antibodies, basiliximab (Simulect) and daclizumab (Zenapax), are a combination of mouse and human antibodies and cause fewer side effects.

Polyclonal antilymphocyte antibodies are also used as adjunctive immunosuppressant therapy. These are administered as antilymphocyte globulin (ALG) or antithymocyte globulin (ATG). These globulins contain antibodies against both T and B cells, as well as other mononuclear leukocytes. When administered, they deplete circulating lymphocytes, platelets, and granulocytes.

MEDICATION ADMINISTRATION **Immunosuppressive Agents**



T-CELL SUPPRESSORS

Cyclosporine (Sandimmune)
Tacrolimus (Prograf)
Sirolimus (Rapamune)

These drugs inhibit T-cell development and activation. They are given concurrently with a glucocorticoid and in combination with other immunosuppressants and inhibit immune system activity and organ rejection.

Nursing Responsibilities

- Monitor BUN and creatinine for evidence of nephrotoxicity.
- Teach the signs and symptoms of infection unique to immunesuppressed individuals. A temperature of 100.6°F is significant evidence of infection. A sore throat may be a manifestation. Other signs and symptoms of inflammation and infection may be absent.
- Teach clients good hygiene to avoid infection with special emphasis on hand washing and avoiding infected individuals.
- Monitor blood pressure and availability and use of antihypertensive medications.
- Teach to avoid grapefruit juice, which can raise cyclosporine levels by 50% to 200% and increase the risk of toxicity. Sirolimus should not be taken with grapefruit juice. Sirolimus increases cholesterol and triglycerides. Lipid-lowering drugs may be necessary to prevent hyperlipidemias.

CYTOTOXIC AGENTS

Azathioprine (Imuran)
Cyclophosphamide (Cytoxan)
Methotrexate (Rheumatrex, Trexall)
Mycophenolate (CellCept)

Certain drugs that are identified as cytotoxic or antineoplastic agents are effective as immunosuppressive agents. They act by decreasing the proliferation of cells within the immune system and are widely used to prevent rejection following a tissue or organ transplant. They are usually administered concurrently with corticosteroid therapy, allowing lower doses of both preparations and resulting in fewer side effects.

Nursing Responsibilities

- Monitor blood count, with particular attention to the WBC and platelet counts. Notify the physician if WBCs fall below 4000 or platelets below 75,000.
- Monitor renal and liver function studies, including creatinine, BUN, creatinine clearance, and liver enzymes. Report abnormal levels to the physician.
- Administer the drug as ordered. Administer oral preparations with food to minimize gastrointestinal effects. Antacids may be ordered.
- Have client increase fluids to maintain good hydration and urinary output, void frequently, and avoid taking in the evening, which promotes dwelling of the drug in the bladder overnight.
- Monitor intake and output.
- Monitor for signs of abnormal bleeding, bleeding gums, bruising, petechiae, joint pain, hematuria, and black or tarry stools.
- Use meticulous hand washing and other appropriate measures to protect the client from infection. Assess for signs of infection.
- Pulmonary fibrosis is a rare (<1%) potential adverse effect of cyclophosphamides. Therefore, monitor respiratory function using pulmonary function studies and monitor for clinical signs of dyspnea or cough.

Health Education for the Client and Family

- Avoid large crowds and situations where exposure to infection is probable.
- Report signs of infection, such as chills, fever, sore throat, fatigue, or malaise, to the physician.
- Use contraceptive measures to prevent pregnancy while on immunosuppressive therapy; these drugs are teratogenic.
- Avoid the use of aspirin or ibuprofen while taking these drugs. Report any signs of bleeding to the physician. Many over-the-counter products contain aspirin; check labels for aspirin.
- With cyclophosphamide, amenorrhea may occur. The menses will resume after the drug is discontinued.
- If taking cyclophosphamide, report difficulty breathing or cough to the physician.

MONOCLONAL ANTIBODY

- Muromonab-CD3 or OKT3 (Orthoclone); Basiliximab and Daclizumab

This monoclonal antibody against T cells is formed by immunizing a mouse with an antigen to produce a specific antibody. Lymphocytes producing the antibody, OKT3, are cloned, and the antibody is harvested. When injected into humans, OKT3 binds with a surface antigen on T cells, removing them from circulation and inactivating those bound to allograft cells. Due to the high incidence of adverse effects, the first two doses of OKT3 are administered by a physician and the client closely observed for 2 hours following each dose.

Nursing Responsibilities

- Be sure a chest x-ray has been performed within 24 hours preceding initiation of OKT3 therapy and that no congestion is present. The risk of anaphylaxis is greater in the client with fluid overload.
- Premedicate as ordered with hydrocortisone, acetaminophen, and diphenhydramine to reduce potential adverse effects.
- Position a crash cart or code cart with emergency medications in the client's room or in proximity to it.
- After each of the first two doses, monitor vital signs every 15 minutes for 2 hours, then every 30 minutes for 2 hours.
- After the first two doses, administer a 2.5- to 10-mg dose by intravenous push over 1 to 2 minutes.
- Observe closely for potential adverse effects, including chills and fever; tachycardia; headache and tremor; hypertension or hypotension; nausea, vomiting, and diarrhea; chest pain, dyspnea, and wheezing.
- OKT3 can also cause anaphylaxis; observe for evidence of urticaria, angioedema, laryngeal edema, wheezing, or other signs of anaphylactic reaction.
- Monitor CBC for evidence of leukopenia or pancytopenia.
- Assess for infection. Remember that typical signs of infection, including symptoms such as fever and inflammation, may be masked or reduced by immunosuppressive therapy.

(continued)

MEDICATION ADMINISTRATION **Immunosuppressive Agents (continued)**

Health Education for the Client and Family

- Teach about the drug and its purpose.
- Discuss potential adverse and side effects, and emphasize the need to report symptoms promptly.
- Inform the client that adverse effects are most likely to occur following the first two doses, necessitating close observation at that time. Reassure the client that this is standard protocol for this medication.

ANTILYMPHOCYTE GLOBULINS

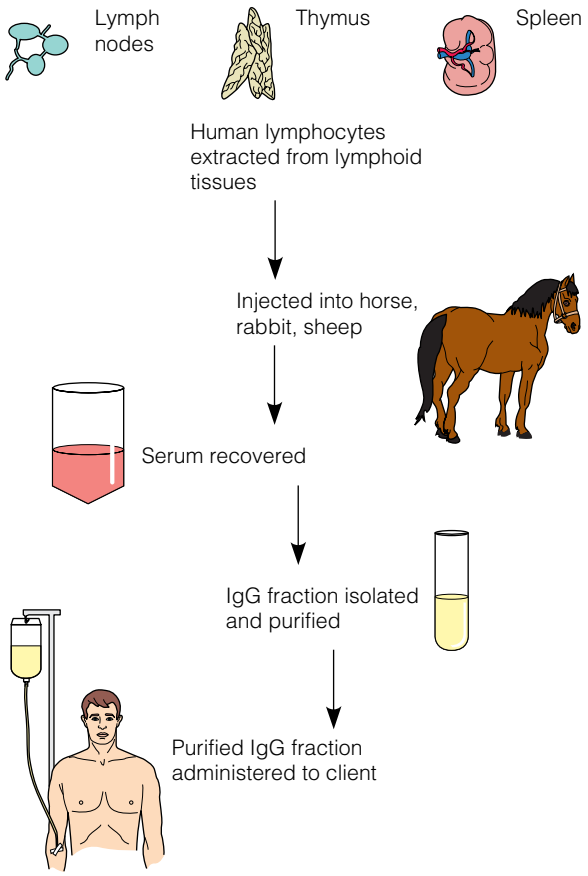
Antithymocyte globulin or ATG (ATGAM)
Antilymphocyte globulin or ALG

These globulins containing antilymphocyte antibodies are produced by immunizing horses (the main source), rabbits, or sheep with human lymphocytes to stimulate production of antibodies (see figure at right). Serum from the animal is then recovered, and the active IgG fraction is isolated, purified, and administered parenterally to the client. It binds with peripheral lymphocytes and mononuclear cells, removing them from circulation.

ATG or ALG is used both to induce immunosuppression immediately following a transplant and to treat steroid-resistant rejection episodes. As with monoclonal antibody, multiple side effects are associated with ATG or ALG.

Nursing Responsibilities

- Perform a skin test for sensitivity to horse serum prior to initial dose. Report any positive reaction to the physician and hold administration until desensitization therapy has been completed.
- Premedicate as ordered with acetaminophen and diphenhydramine prior to each dose. Steroids may also be administered before the initial dose. Have epinephrine and hydrocortisone injections available at the bedside in case of anaphylactic reaction.
- Administer by intravenous infusion into a central line over 4 to 6 hours.
- Monitor vital signs hourly while medication is infusing.
- Assess for adverse effects, including chills and fever, erythema, and pruritus. Notify the physician; these may be treated symptomatically.
- Monitor CBC daily, notify the physician if WBC falls to less than 3000/mm^3 or platelet count to less than 100,000/mm^3. The medication may be stopped or reduced.
- Assess renal function studies to monitor for serum sickness. Report complaints of joint pain.
- Monitor for signs of infection, and report any signs promptly.



A horse is inoculated with washed human lymphocytes, stimulating the production of immunoglobulin with polyclonal antilymphocyte antibodies. These are then extracted from horse serum, purified, and administered intravenously to the client.

Health Education for the Client and Family

- Explain the need for special precautions and close monitoring while this drug is being administered.
- Instruct the client to report any adverse effects, including malaise or joint pain, promptly.
- Ask the client to report any evidence of easy bruising, bleeding gums, or black stools.
- Teach family members about the importance of not exposing the client to persons with infectious diseases.

NURSING CARE

The client who has an organ or tissue transplant has both immediate and long-term nursing care needs. Both the client and the family must be considered in providing nursing care.

Health Promotion

Part of the health promotion activities focus on preventing the need for a tissue transplant. It is important to increase public awareness regarding unhealthy lifestyle behaviors, such as excessive alcohol consumption and illegal drug use, and their relationship to organ failure. Clients with chronic diseases such as diabetes mellitus and hypertension must understand that inadequate management of these disorders could lead to end-stage renal disease. Other risk factors may simply relate to a person's heredity; understanding how heredity could affect future health might influence the client's lifestyle choices.

Assessment

Assessment data collected following a tissue transplant focus on identifying potential rejection episodes. Further focused

assessments are described with nursing interventions in the next section.

Nursing Diagnoses and Interventions

Because of the continuing risk of transplant rejection and the need for immunosuppression, *Ineffective Protection* and *Risk for Impaired Tissue Integrity* are priority nursing care foci. The client's underlying disease process, the transplant, and the continuing need for immunosuppressive drug therapy also have emotional and psychologic consequences. Many nursing diagnoses, such as *Powerlessness* or *Ineffective Coping,* may be appropriate. The diagnosis *Anxiety* related to potential transplant rejection is considered in this section.

Ineffective Protection

Ineffective protection is a problem for the transplant client at all stages. Before the transplant occurs, failure of the affected organ may put the client at risk for infection and other multisystemic problems. Incisions and invasive perioperative procedures impair skin and mucous membrane protection from infectious organisms and other antigens. Immunosuppressive drugs given postoperatively to prevent graft rejection disarm the immune response to a certain extent, increasing the risk of infections and neoplastic growths.

PRACTICE ALERT

Use strict aseptic technique in changing dressings and caring for invasive catheters such as intravenous lines and indwelling urinary catheters to protect against external and resident host microorganisms.

- Wash hands on entering room and before providing direct care. *Hand washing removes transient organisms from the skin, reducing the risk of transmission to the client.*
- Assess frequently for signs and symptoms of infection. Monitor the temperature and vital signs every 4 hours. Assess for evidence of inflammation, abnormal wound drainage, changes in urine or other body secretions, complaints of pain, or behavior changes that may indicate infection. Culture abnormal wound drainage. *The client on immunosuppressive therapy is more susceptible to infection, and usual signs and symptoms may not be evident. Both the temperature and inflammatory response can be suppressed by therapy. Prompt identification and intervention for infection is important in the immunosuppressed client.*
- Monitor laboratory values, including CBC and tests of organ function; report changes to the physician. *An elevation in the WBC count with increased numbers of immature cells (bands) or a decline in function of the transplanted organ (e.g., a rising BUN and creatinine in the renal transplant client) may be early indications of infection or transplant failure.*
- Initiate reverse or protective isolation procedures as indicated by the client's immune status. *These procedures further protect the severely immunocompromised client from infection.*
- Instruct ill family members and visitors to avoid contact with the client. *A "minor" upper respiratory infection can be a significant illness in the immunocompromised host.*
- Help ensure adequate nutrient intake, offering supplementary feedings as indicated or maintaining parenteral nutrition if necessary. *Adequate nutrition is important for healing and immune system function.*
- Change intravenous bags and tubing at least every 24 hours, and change peripheral intravenous sites every 48 to 72 hours, unless contraindicated. Remove invasive catheters and lines as soon as they are no longer necessary. *Changing lines and sites is important to reduce bacterial contamination. Fewer invasive lines provide fewer sites for bacterial invasion of the body.*
- Emphasize the importance of washing hands thoroughly after using the bathroom and before eating. *This reduces the risk of infection with endogenous organisms.*
- Provide good mouth care. *Good mouth care reduces the population of oral microorganisms and helps maintain an intact mucous membrane lining.*
- Monitor for potential adverse effects of medications:
  - Thrombocytopenia and possible bleeding
  - Fluid retention with edema and possible hypertension
  - Loss of bone density, osteoporosis, and possible pathologic fractures
  - Renal or hepatic toxicity
  - Cardiac effects, particularly in the presence of fluid retention and hypervolemia.

  Medications used to maintain immunosuppression and preserve the allograft have many potential adverse effects that can alter normal protective and homeostatic mechanisms.

Risk for Impaired Tissue Integrity: Allograft

As noted, the risk for transplant rejection is highest in the initial postoperative period, but it is never completely eliminated for the client who has had an allograft. The client who has had a bone marrow transplant has the additional risk of developing GVHD, which can affect the integrity of skin, mucous membranes, and other organs.

- Administer immunosuppressive therapy as prescribed. *Suppression of the immune response is necessary to reduce the risk of graft destruction by normal immune responses and to preserve the graft's function.*
- Assess for evidence of graft rejection, including tenderness, erythema, and swelling over the site; sudden weight gain, edema, and hypertension; chills and fever; malaise; and an increased WBC count and sedimentation rate. Report any changes immediately. *Early identification of rejection allows adjustment of medication regimens and, possibly, preservation of the graft.*
- Monitor results of laboratory studies for function of the transplanted organ. *With a functional graft, results (e.g., renal or liver function studies) will improve; a functional decline may be an early indicator of rejection.*
- Assess for and report signs of GVHD immediately, including maculopapular rash, erythema of the skin and possible desquamation, hair loss, abdominal cramping and diarrhea, or jaundice with elevated bilirubin and liver enzymes (AST, ALT). *GVHD is a potentially lethal complication in the immunosuppressed client and necessitates immediate intervention.*

- Stress the importance of maintaining immunosuppressive therapy and reporting signs of graft rejection promptly to the physician. *Continued immunosuppression and prompt treatment of rejection are vital to preserving graft function.*

Anxiety

The client who undergoes an organ or tissue transplantation often faces the unwelcome choices of death from organ failure or receiving an organ that his or her body will likely attempt to reject. In most cases, the client understands that to receive this transplant, someone else must die and be willing to give up an organ. When the transplant comes from a living donor (bone marrow or kidney), the client may worry not only about himself or herself, but also about the condition of the donor. Fear of rejection and guilt may be even greater in this instance.

- Assess level of anxiety by noting such cues as expressions of apprehension, fear, or inadequacy; facial expression, tension, or shakiness; difficulty focusing; helplessness; poor eye contact; and restlessness. *Clients may have difficulty identifying or verbalizing feelings of fear and anxiety. Nonverbal cues are often useful in recognizing states of anxiety.*
- Provide opportunities to express feelings. Use opening statements such as "Facing an organ transplant must be very stressful." Listen attentively. *Encouragement and active listening allow the client to express feelings of anxiety or fear.*
- Arrange tasks to allow as much time with the client as possible. When leaving, tell the client when you will return. *Time spent with the client facilitates the development of trust.*
- Provide clear, concise directions. *Highly anxious clients have difficulty focusing and retaining information.*
- Encourage involvement in care but do not request unnecessary decisions. *The client needs to feel a sense of control but may become irritated if asked to make decisions unrelated to the situation.*
- Encourage family members to remain with the client as much as possible. *This can help reduce the client's anxiety.*
- Encourage the use of coping behaviors that have been effective for the client in the past. *Coping mechanisms and behaviors help lower anxiety to a more acceptable level.*
- Reduce or eliminate environmental stressors to the extent possible. *This gives the client a better sense of control.*
- Assist with stress reduction and relaxation techniques, such as guided imagery, meditation, and muscle relaxation. *These techniques help the client gain control over physical responses to anxiety.*
- Arrange for a counselor or mental health specialist to work with the client. *Counseling can help the client identify and deal with his or her feelings.*
- Assess the client's preference and desire for spiritual counseling prior to transplant. *Because there is a risk of death if the transplant fails, the client may want to discuss his or her concerns with a spiritual counselor.*

Community-Based Care

Teaching of the client and family regarding an organ or tissue transplant begins well before the transplant and continues throughout hospitalization and follow-up treatment.

Initial teaching focuses on the options, risks, and potential benefits of the transplant itself. Include the procedure by which the organ is selected and obtained, as well as the procedure by which it is transplanted into the client. If a living related donor is an option, discuss the risks and benefits for both the client and the donor. Outline the post-transplant treatment regimen, including any lifestyle changes that may be necessary.

Following the transplant, provide verbal and written instructions, including the following:

- Manifestations of transplant rejection and the importance of notifying the physician
- Immunosuppressive drug regimen and side effects
- Wound care
- Avoiding exposure to infectious diseases, particularly respiratory infections, and wearing a mask when going outside
- Meticulous personal hygiene, hand washing technique, and frequent mouth care
- Wearing a Medic-Alert bracelet or tag
- Follow-up visits to the physician or clinic
- Helpful resources:
  - American Council on Transplantation
  - Local and state support groups related to specific organ transplant, such as the National Kidney Foundation.

IMPAIRED IMMUNE RESPONSES

Disorders of impaired immune system responses may be either congenital or acquired. Often the function of either T or B cells is impaired, reducing the body's ability to defend against foreign antigens or abnormal host tissue.

No matter what the cause, clients with immunodeficiency disorders demonstrate an unusual susceptibility to infection. When the antibody-mediated response is primarily affected, the client is at particular risk for severe and chronic bacterial infections. These clients also do not develop long-lasting immunity to such diseases as chickenpox and are prone to recurrent cases. Clients with a defect of cell-mediated immunity tend to develop disseminated viral infections such as herpes simplex and CMV. Candidiasis and other fungal infections are also common. Because T cells are involved with activating antibody-mediated immune responses as well, overwhelming bacterial infections may occur. Immunodeficiency in its most severe form occurs when both antibody-mediated and cell-mediated responses are impaired. Clients with combined immunodeficiency are susceptible to all varieties of infectious organisms, including those not normally considered to be pathogens.

Most immunodeficiency diseases are genetically determined and rare. They affect children more than adults. The noted exception is AIDS, an infectious disease caused by a virus.

THE CLIENT WITH HIV INFECTION

In 1981, five cases of *Pneumocystis carinii* pneumonia (PCP) and 26 cases of a rare cancer, Kaposi's sarcoma, were diagnosed in young, previously healthy homosexual males in Los Angeles and New York City. The term **acquired immunodeficiency syndrome (AIDS)** was ascribed to this new phenomenon to describe the immune system deficits associated with these opportunistic disorders. Prior to this time, both PCP and Kaposi's had been seen only in elderly, debilitated, or severely immunodeficient people. Other groups at risk for AIDS were soon identified: injection drug users, persons with hemophilia, recipients of blood transfusions, and immigrants from Haiti.

Research to identify the cause of this apparently new disease progressed feverishly, and in 1983, a common antibody was identified in clients with AIDS. The **human immunodeficiency virus (HIV)** was isolated in 1984. It then became apparent that AIDS was the final, fatal stage of HIV infection.

It began, like so many epidemics, with a few isolated cases, and has become a worldwide plague. (See the accompanying Focus on Cultural Diversity box.) AIDS invades our lives in ways we never imagined—testing our scientific knowledge, probing our private values, and eluding a vaccine or a cure. Progression of HIV-positive disease to AIDS has slowed because of the effectiveness of highly active antiretroviral therapy (HAART) (Centers for Disease Control and Prevention [CDC], 2006). This change in progression to AIDS makes monitoring of AIDS less useful as an indicator of infected cases. For that reason, the CDC developed new surveillance methods based on infection rates in high-risk populations.

Although the incidence of HIV has leveled and mortality due to AIDS has declined, the epidemic is far from over. In the United States it continues to disproportionately affect African Americans, males who have sex with males (MSM), and high-risk heterosexuals. High-risk heterosexuals are those who have sex with individuals known to be infected with HIV or with high risk for having HIV. Two separate elements are reported in the surveillance data: HIV infection rate and AIDS occurrence. HIV infection is dependent on exposure to the virus; conversion to AIDS is related to access and use of HAART. HIV new cases are highest among MSM and high-risk heterosexuals; AIDS occurrence continues among MSM, high-risk heterosexuals, and African Americans. (CDC, 2006).

Incidence and Prevalence

Through 2004, the CDC (2004) estimated that 1,039,000 to 1,185,000 persons in the United States were living with HIV/AIDS, with 24% to 27% undiagnosed and unaware of their HIV infection. By the end of December 2004, a cumulative total of 944,305 cases of AIDS had been reported. Deaths among people with AIDS had decreased from 50,610 in 1995 to 15,798 in 2004, more than likely the result of improved treatments rather than a decline in spread of the disease (CDC, 2006). A continued decline in deaths is dependent on access to quality care and treatment and continued development of treatments for those already heavily treated (Tierney et al., 2005).

The risk factors for HIV infection are behavioral. Among adults in the United States, 60% of reported cases are in men who have sex with other men, including homosexuals, bisexuals, and such groups as prison populations. Unprotected anal intercourse is the major route of transmission in this group. Injection drug use is the second leading risk factor, accounting for approximately 25% of cases. Sharing of needles and other drug paraphernalia is the primary route of transmission in this group. Heterosexual intercourse with an infected drug user and exchanging sex for drugs are major risk factors for women. Hemophiliacs who require large amounts of intravenous clotting factors and people infected through blood transfusion account for a small number of cases, approximately 2% (CDC, 2004).

The majority of women were infected through heterosexual contact (75%) with the remaining 25% through injection drug use. Among risk groups, the most rapid increases are noted in young gay and bisexual men, women, and inner-city injection drug users, especially African Americans and Hispanics (CDC, 2004). In the United States, the rate of male adult/adolescent HIV/AIDS cases (per 100,000 population) reported was 131.6 among African Americans, 60.2 among Hispanics, 20.8 among American Indians/Alaska Natives, 18.7 among Caucasians, and 13.9 among Asians/Pacific Islanders (CDC, 2004). The rapid increase of AIDS cases among women is of special concern; those numbers increased from 7% of cases in 1985 to 27% of newly reported cases in 2003 (CDC, 2004).

In addition, AIDS in the adult over age 50 accounts for approximately 10% of all reported cases in the United States (CDC, 2004). Declining immune system function in older adults significantly increases their risk for contracting HIV/AIDS, along with the belief that they cannot be affected by it. Just as younger persons with HIV/AIDS contract the diseases primarily through sexual intercourse, so does the elderly population. Because older adults are beyond childbearing years, they often fail

FOCUS ON CULTURAL DIVERSITY
HIV/AIDS

There are an estimated 36.2 million people infected with AIDS worldwide, with virtually every country in the world reporting cases of AIDS (Joint United Nations Programme on HIV/AIDS, 2000). The highest incidence is found in sub-Saharan Africa, South and Southeast Asia, the United States, western Europe, South America, and Canada. Approximately 70% of all people infected with HIV or who have AIDS live in sub-Saharan Africa, and another 16% live in South and Southeast Asia, largely in Thailand and India. The most common mode of transmission is heterosexual intercourse. The cofactors of general health status, the presence of genital ulcers, and the number of sexual partners correlate with incidence (Tierney et al., 2005).

Critical Thinking Questions

1. How accurate is informed consent for a clinical trial in Third World countries if investigators are dependent on translators? What factors could impact the validity of the consent?
2. Do you think it is ethical to do clinical trials in countries outside the origin of a product?

to use condoms when engaging in sexual activity. Manifestations may be overlooked by healthcare professionals, leading to a delayed diagnosis and increased severity of the disease.

HIV is a retrovirus transmitted by direct contact with infected blood and body fluids. Significant concentrations of the virus are present in blood, semen, vaginal and cervical secretions, and cerebrospinal fluid (CSF) of infected individuals. It is also found in breast milk and saliva. Sexual contact is the primary mode of transmission. HIV is also transmitted through contact with infected blood via needle sharing during injection drug use or by transfusion. Approximately 13% to 40% of infants born to HIV-positive mothers are infected perinatally. Breast-feeding is a route of transmission and should be avoided (Tierney et al., 2005).

Less than 0.04% of people voluntarily donating blood (a process that generally excludes people with high-risk behavior) are found to be HIV positive. HIV is not transmitted by casual contact, nor is there any evidence of its transmission by

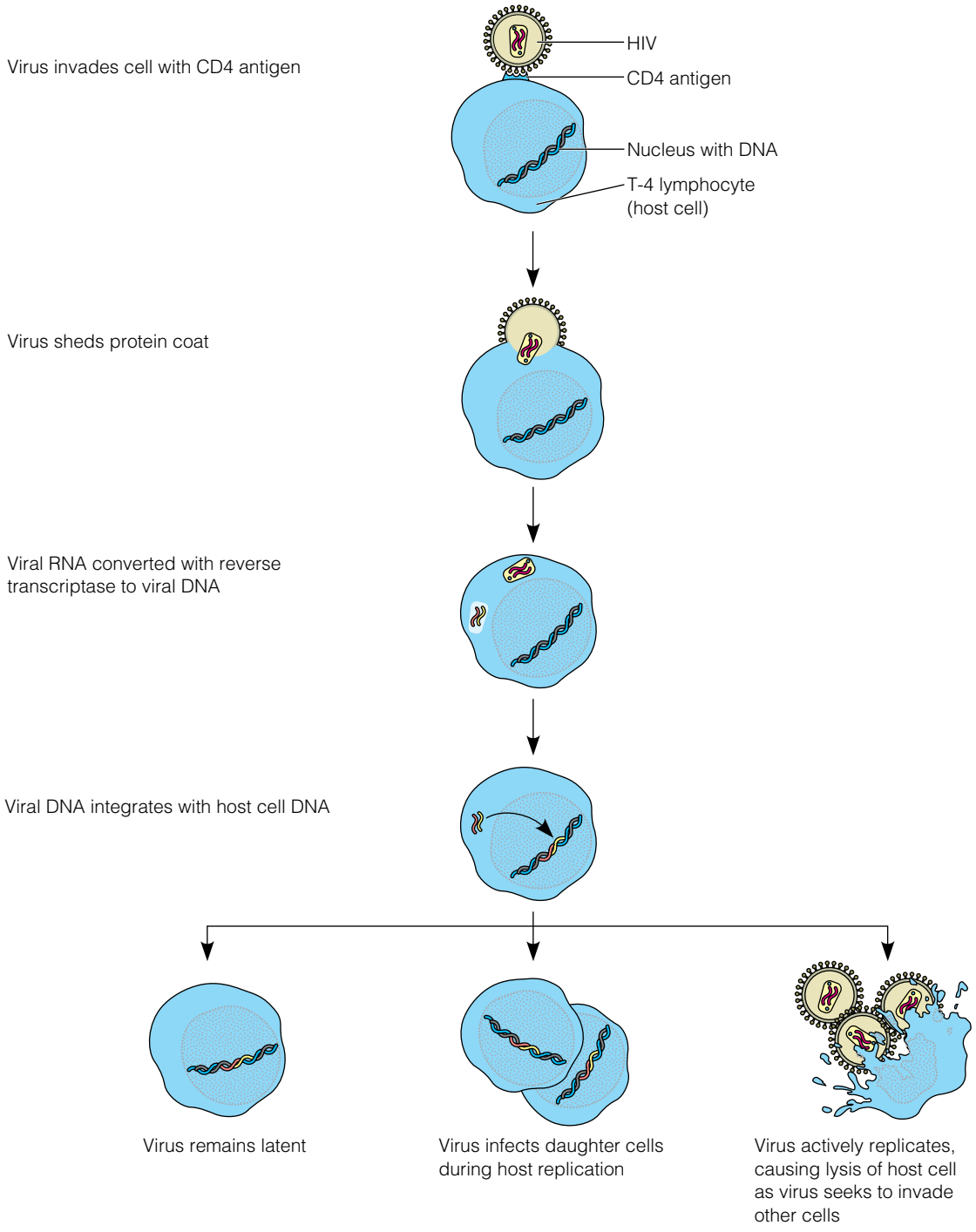


Figure 13–7 ■ How HIV infects and destroys CD4 cells.

vectors such as mosquitoes. Blood donation also poses no risk of contracting HIV to the donor, because only new sterile equipment is used. A small but real occupational risk exists for healthcare workers. Percutaneous exposure to infected blood or body fluids through a needle-stick injury or nonintact skin is the primary route of transmission. Documented evidence indicates that parenteral exposure poses a 0.3% risk of becoming HIV positive (Carrico, 2001; Tierney et al., 2005). Mucosal exposures, such as splashing in the eyes or mouth, pose a much smaller risk.

Pathophysiology and Manifestations

As mentioned, HIV is a retrovirus, meaning it carries its genetic information in RNA. On entry into the body, the virus infects cells that have the CD4 antigen. Once inside the cell, the virus sheds its protein coat and uses an enzyme called *reverse transcriptase* to convert the RNA to DNA (Figure 13–7 ■). This viral DNA is then integrated into host cell DNA and duplicated during normal processes of cell division. Within the cell, the virus may remain latent or become activated to produce new RNA and to form *virions*. The virus then buds from the cell surface, disrupting its cell membrane and leading to destruction of the host cell.

Although the virus may remain inactive in infected cells for years, antibodies are produced to its proteins, a process known as **seroconversion**. These antibodies are usually detectable 6 weeks to 6 months after the initial infection. Helper T or CD4 cells are the primary cells infected by HIV. It also infects macrophages and certain cells of the CNS. Helper T cells play a vital role in normal immune system function, recognizing foreign antigens and infected cells and activating antibody-producing B cells. They also direct cell-mediated immune activity and influence the phagocytic activity of monocytes and macrophages. The loss of these helper T cells leads to the immunodeficiencies seen with HIV infection (Porth, 2005). Figure 13–8 ■ illustrates the typical course of HIV infection.

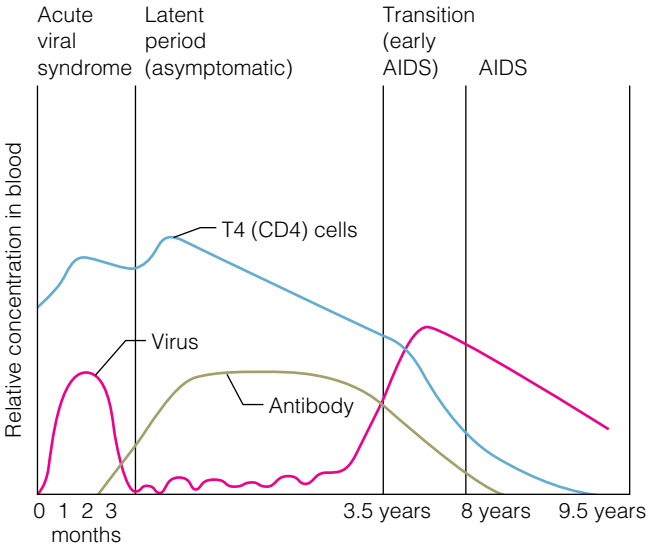


Figure 13–8 ■ The progression of HIV infection. Acute illness develops shortly after the virus is contracted, corresponding with a rapid rise in viral levels. Antibodies are formed and remain present throughout the course of infection. Late in the disease, viral activation results in a marked increase in virus while CD4 (T4) cells diminish as they are destroyed with viral replication. Antibody levels gradually decrease as immune function is impaired.

The clinical manifestations of HIV infection range from no symptoms to severe immunodeficiency with multiple opportunistic infections and cancers (see the Manifestations box below). It appears that the majority of clients develop an acute mononucleosis-type illness within days to weeks after contracting the virus. Typical manifestations include fever, sore throat,

MANIFESTATIONS of HIV Infection and AIDS

I. Acute Retroviral Syndrome (ARS) or Primary HIV Infection
- Fever
- Sore throat
- Arthralgias and myalgias
- Headache
- Rash
- Nausea, vomiting, and abdominal cramping

II. Asymptomatic Infection
- None; converts to seropositive status

III. Persistent Generalized Lymphadenopathy
- Enlargement of two or more extrainguinal sites for more than 3 months

IV. Other Acute Disease Symptoms
- General malaise, fatigue
- Low grade fever
- Night sweats
- Involuntary weight loss
- Skin dryness, or rashes

V. Other Diseases and AIDS
A. *AIDS Dementia Complex*
B. *Secondary Infectious Diseases*
- *Pneumocystis carinii* pneumonia
- *Mycobacterium* tuberculosis
- *Mycobacterium avium* complex
- Candidiasis
- Cryptosporidiosis
- Cryptococcosis
- Toxoplasmosis
- Herpes simplex or herpes zoster
- Cytomegalovirus

C. *Secondary Cancers*
- Kaposi's sarcoma
- Non-Hodgkin's lymphoma
- Cervical dysplasia and cervical cancer

D. *Other Conditions*
- Pelvic inflammatory disease
- Human papillomavirus

BOX 13–2 Classification System for HIV Infection and Expanded AIDS Surveillance Case Definition for Adolescents and Adults

	Diagnostic Categories	Clinical Categories	
	A		
CD4 + T-cell Categories	**Asymptomatic, Acute (Primary) HIV or Persistent Generalized Lymphadenopathy (PGL)**	**B Symptomatic, Not (A) or (C) Conditions**	**C AIDS-Indicator Conditions**
(1) ≥500/mm^3	A1	B1	C1
(2) 200–499/mm^3	A2	B2	C2
(3) <200/mm^3	A3	B3	C3

As of January 1, 1993, people with AIDS-indicator conditions (clinical category C) and those in categories A3 or B3 were considered to have AIDS.

Clinical Category A

One or more of the following conditions in an adolescent or adult with documented HIV infection and without conditions in categories B and C:

- Asymptomatic HIV infection
- Persistent generalized lymphadenopathy
- Acute HIV infection with accompanying illness or history of acute HIV infection

Clinical Category B

Examples of conditions but are not limited to:

- Candidiasis, oral (thrush), or vulvovaginal (persistent, frequent, or poorly responsive to therapy)
- Cervical dysplasia/cervical carcinoma *in situ*
- Constitutional symptoms, such as fever (38.5°C) or diarrhea exceeding 1 month duration
- Hairy leukoplakia
- Herpes zoster involving at least two distinct episodes
- Pelvic inflammatory disease
- Peripheral neuropathy

Clinical Category C

- Candidiasis of bronchi, trachea, or lungs; esophagus
- Coccidioidomycosis
- Cryptococcosis
- Cryptosporidiosis with persistent diarrhea
- Cytomegalovirus infection (other than of liver, spleen, or lymph nodes)
- CMV retinitis
- HIV encephalopathy
- Herpes simplex: chronic ulcers or bronchitis, pneumonitis, or esophagitis
- *Mycobacterium avium* complex or disseminated
- *Mycobacterium tuberculosis*
- *Pneumocystis carinii* pneumonia
- Progressive multifocal leukoencephalopathy
- *Salmonella* septicemia
- Toxoplasmosis of the brain
- Kaposi's sarcoma
- Cervical cancer, invasive
- Lymphoma
- HIV wasting syndrome

Source: Adapted from "Revised Classification System for HIV Infection and Expanded Case Definition for AIDS Among Adolescents and Adults," 1993, *MMWR, CDC Recommendations and Reports, 41*(RR 17), pp. 1–19.

arthralgias and myalgias, headache, rash, and lymphadenopathy. Pathologic changes are also noted in the CNS of many infected individuals although the mechanism of neurologic dysfunction is unclear. The client may also experience nausea, vomiting, and abdominal cramping. The client often attributes this initial manifestation of HIV infection to a common viral illness such as influenza, upper respiratory infection, or stomach virus.

Following this acute illness, clients enter a long-lasting asymptomatic period. Although the virus is present and can be transmitted to others, the infected host has few or no symptoms. Clearly, the majority of HIV-infected persons are in this stage of the disease. The length of the asymptomatic period varies widely, but its mean length is estimated to be 8 to 10 years.

Some clients with few other symptoms develop persistent generalized lymphadenopathy. This is defined as enlargement of two or more lymph nodes outside the inguinal chain with no other illness or condition to account for the lymphadenopathy.

The move from asymptomatic disease or persistent lymphadenopathy to AIDS is often not clearly defined. The client may complain of general malaise, fever, fatigue, night sweats, and involuntary weight loss. Persistent skin dryness and rash may be a problem. Diarrhea is common, as are oral lesions such as hairy leukoplakia, candidiasis, and gingival inflammation and ulceration. The development of advanced HIV typically occurs 10 to 11 years after initial infection; this varies according to the viral load, rate of disease progression, and the development of resistance to antiretroviral therapy (Kenny, 2004).

With the development of significant constitutional disease, neurologic manifestations, or opportunistic infections or cancers, the client has manifestations that are characteristic of AIDS and a very poor prognosis. HIV infection and AIDS may be classified by using the CDC's matrix classification system. Under this system, HIV disease is determined by the presence of clinical symptoms (clinical categories A, B, and C) and by T4 cell counts (categories 1, 2, and 3) (Box 13–2). When a client's T4 cell count falls to <200, he or she has late-stage AIDS, <50 is end stage AIDS (Coyne, Lyne, & Watson, 2002).

When clinical manifestations develop, the outcome varies. With antiretroviral therapy (ART), many clients are living longer after being diagnosed with AIDS. Today PCP is most commonly diagnosed in those who are undiagnosed or have a late diagnosis of HIV infection or who fail to take prophylactic

antibiotics when their CD4 count is <200. ART is credited with decreasing the incidence of opportunistic infections and improving survival (CDC, 2004). The time of survival has increased from about 13 months at the start of the AIDS epidemic; however, survival after diagnosis of HIV-related lymphomas still averages less than 8 months.

AIDS Dementia Complex and Neurologic Effects

Neurologic manifestations of HIV are common, affecting 40% to 60% of clients with AIDS. Included among the manifestations are dementia, delirium, and seizures. They result from both the direct effects of the virus on the nervous system and opportunistic infections.

AIDS dementia complex is the most common cause of mental status changes for clients with HIV infection. This dementia results from a direct effect of the virus on the brain and affects cognitive, motor, and behavioral functioning. Fluctuating memory loss, confusion, difficulty concentrating, lethargy, and diminished motor speed are typical manifestations of AIDS dementia complex. Clients become apathetic, losing interest in work and social and recreational activities. As the complex progresses, the client develops severe dementia with motor disturbances such as ataxia, tremor, spasticity, incontinence, and paraplegia (Kasper et al., 2005; Porth, 2005).

Infections and lesions common with AIDS may also affect the CNS. Toxoplasmosis and non-Hodgkin's lymphoma are space-occupying lesions that may cause headache, altered mental status, and neurologic deficits. Cryptococcal meningitis and CMV infection also are common in people with AIDS. CNS complications have declined with the use of HAART therapy (Tierney et al., 2005).

Peripheral nervous system manifestations are also common in HIV-infected clients. Sensory neuropathies with manifestations of numbness, tingling, and pain in the lower extremities affect about 30% of clients with AIDS. A Guillain-Barré type of inflammatory demyelinating polyneuropathy can also occur, resulting in progressive weakness and paralysis.

Opportunistic Infections

Opportunistic infections are the most common manifestations of AIDS, often occurring simultaneously. The risk of opportunistic infections is predictable by the T4 or CD4 cell count. The normal CD4 cell count is greater than 1000/mm^3. When the CD4 count falls to less than 500/mm^3, manifestations of immunodeficiency are seen. With a count of less than 200/mm^3, opportunistic infections and cancers are likely.

PNEUMOCYSTIS CARINII PNEUMONIA *Pneumocystis carinii* pneumonia is the most common opportunistic infection affecting clients with AIDS. Approximately 75% to 80% of clients develop PCP at some point in their disease (Tierney et al., 2005). It tends to be recurrent, and is the cause of death in about 20% of clients with AIDS. PCP is caused by a common environmental fungus that is not pathogenic in clients with intact immune systems.

Unlike many pneumonias, the manifestations of PCP are nonspecific and may progress insidiously. Clients often present with fever, cough, dyspnea, tachypnea, and tachycardia. Complaints of mild chest pain and sputum may also be present. Breath sounds may initially be normal. With severe disease, the client may present with cyanosis and significant respiratory distress.

TUBERCULOSIS An estimated 4% of clients with AIDS develop tuberculosis, contributing significantly to the rise in incidence of this disease in the United States. In some clients, active tuberculosis results from reactivation of a prior infection. In other clients, it is a new, primary disease facilitated by impaired immune function. Rapid progression, diffuse pulmonary infiltrates, and disseminated disease occur more commonly in clients with AIDS. Multiple-drug-resistant strains of tuberculosis present a significant problem (Tierney et al., 2005).

Clients with pulmonary tuberculosis present with a cough productive of purulent sputum, fever, fatigue, weight loss, and lymphadenopathy. Disseminated disease affects the bone marrow, bone, joints, liver, spleen, CSF, skin, kidneys, gastrointestinal tract, lymph nodes, brain, and other sites.

CANDIDIASIS *Candida albicans* infection is a common opportunistic infection in clients with AIDS. It is usually manifested as oral thrush or esophagitis. In women, vaginal candidiasis is frequent and often recurrent. Oral thrush presents as white, friable plaques on the buccal mucosa or tongue and, in the HIV-infected client, is often the first indication of progression to AIDS. Clients with esophagitis have difficulty swallowing and substernal pain or burning that increases with swallowing.

MYCOBACTERIUM AVIUM COMPLEX *Mycobacterium avium* complex (MAC) affects up to 25% of clients with AIDS, typically occurring late in the course of the disease when CD4 cell counts are less than 50/mm^3. MAC is more common in women than men. MAC is caused by organisms commonly found in food, water, and soil. It is a major cause of "wasting syndrome" in persons with AIDS (Figure 13–9 ■). Manifestations of MAC include chills and fever, weakness, night sweats, abdominal pain and diarrhea, and weight loss. Nearly every organ can be infected, and most people with MAC develop disseminated disease.

OTHER INFECTIONS Herpes virus infections are common in clients with AIDS and may be severe. CMV can affect the retina, the gastrointestinal tract, or lungs. Disseminated herpes simplex or herpes zoster may occur, although severe mucocutaneous manifestations are more common.

Figure 13–9 ■ Wasting syndrome in a client with AIDS.

Parasitic infections with *Toxoplasma gondii* and *Cryptococcus neoformans* commonly affect the CNS. Toxoplasmosis occurs as encephalitis or an intracerebral mass lesion. Changes in mental status, focal neurologic signs, and seizures may result. *Cryptococcus* infection may present as either meningitis or disseminated disease, primarily affecting the lungs. *Cryptosporidium,* a protozoon affecting the gastrointestinal tract, is an important cause of prolonged diarrhea in AIDS clients. Bacterial salmonella infections are also a relatively common cause of diarrhea.

Women with AIDS have a high incidence of pelvic inflammatory disease (PID). Although the pathogens appear to be the same as those in PID affecting non-HIV-infected women, the disease is more severe. Inpatient treatment with intravenous antibiotics is often necessary.

Secondary Cancers

As cell-mediated immune function declines, the risk of malignancy increases. The CDC classification of AIDS currently includes four cancers: Kaposi's sarcoma, non-Hodgkin's lymphoma, primary lymphoma of the brain, and invasive cervical carcinoma.

KAPOSI'S SARCOMA **Kaposi's sarcoma (KS)** is often the presenting symptom of AIDS. It remains the most common cancer associated with the disease. Kaposi's sarcoma is caused by a virus called the Kaposi sarcoma-associated herpesvirus, also known as human herpesvirus 8. Men who have sex with men not only have a risk of HIV infection, but are also more likely to be infected with the virus responsible for KS. Women who have sex with these men also have a risk of HIV and KS. The virus associated with KS appears to be mainly transmitted through sexual contact, although cases have also been reported in injection drug users. People whose immune system is suppressed because they have received an organ transplant have a 1 in 200 risk of developing KS (American Cancer Society, 2005).

A tumor of the endothelial cells lining small blood vessels, KS presents as vascular macules, papules, or violet lesions affecting the skin and viscera (Figure 13–10 ■). The face is a common site for skin lesions, especially the tip of the nose and pinnae of the ears. Common sites for visceral disease include the gastrointestinal tract, lungs, and lymphatic system.

The lesions of KS are usually painless initially, but may become painful as the disease progresses. Internally, the tumors may obstruct organ function or cause bleeding. When the lungs are involved, gas exchange may be severely impaired, resulting in pulmonary hemorrhage. This disease may progress slowly or rapidly. KS is an indicator of late-stage HIV disease, with an average survival time of 18 months after diagnosis.

LYMPHOMAS Lymphomas are malignancies of the lymphoid tissue, including lymphocytes, lymph nodes, and the lymphoid organs such as the spleen and bone marrow. In AIDS, two lymphomas are common, non-Hodgkin's lymphoma (including Burkitt's lymphoma) and primary lymphoma of the brain. Hodgkin's disease also occurs five times more frequently in clients with HIV infection than in those without. The CNS is the usual site for these lymphomas, although they may be found in the bone marrow, gastrointestinal tract, liver, skin, and mucous membranes. They are aggressive tumors, growing and spreading rapidly. Headache and changes in mental status are common early symptoms of lymphomas affecting the CNS.

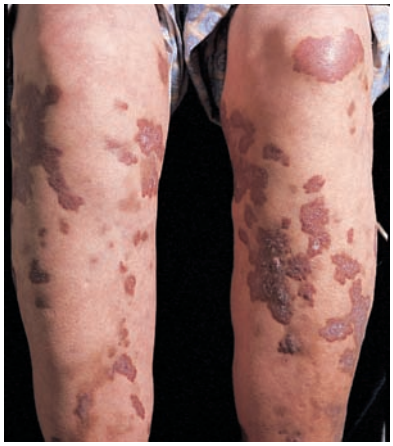


Figure 13–10 ■ Kaposi's sarcoma lesions.
Source: Zeva Oelbaum/Peter Arnold, Inc.

CERVICAL CANCER Of women with HIV infection, 40% have cervical dysplasia. Cervical cancer develops frequently and tends to be aggressive. Women with concurrent HIV infection and cervical cancer usually die of the cervical cancer, not AIDS. Because of this, it is recommended that women with HIV infection have Papanicolaou (Pap) smears every 6 months and aggressive treatment of cervical dysplasia with colposcopic examination and cone biopsy.

INTERDISCIPLINARY CARE

Although multiple research studies to identify a cure for HIV infection and AIDS are under way, no cure is currently available. This fact, plus the apparent universally fatal nature of the disease, make prevention a vital strategy in HIV care. New treatments are under investigation (Box 13–3).

The goals of care for the client with HIV disease are as follows:

- Early identification of the infection
- Promoting health-maintenance activities to prolong the asymptomatic period as long as possible
- Prevention of opportunistic infections
- Treatment of disease complications, such as cancers
- Providing emotional and psychosocial support.

Diagnosis

Diagnostic testing is used to screen and identify the infection, as well as to monitor the client's disease and immune status. The following diagnostic tests may be ordered. The likelihood that a positive screening test truly indicates the presence of HIV infection decreases as HIV prevalence in the tested population becomes lower. Therefore, false-positive HIV test results are more likely in settings where the tested population prevalence is lower than in settings where the tested population prevalence is higher. When a preliminary, positive rapid test is explained to clients, phrases like "a good chance of being infected" or "very likely infected" can be used to indicate the likelihood of

BOX 13–3 Investigational Immune-Based Treatment for HIV

HIV infection progressively alters the function of and destroys CD4 + lymphocytes. CD4 + cells are essential to the function of the immune system, including the body's ability to respond to infections. These cells initiate, direct, and regulate immune responses and may also directly attack infected cells. They also are a source of cytokines, the chemical messengers of the immune system. Destruction of CD4 + cells by HIV devastates the immune system, facilitating the development of fatal infections and neoplasms in the infected person. Immune-based treatments indirectly affect the HIV by improving the function of the immune system through actions that inhibit cytokines, replenish cytokines, or restore immune function. These treatments, used alone or in combination with antiretroviral drugs, are being investigated for use in the treatment of HIV.

Inhibiting Cytokines

Tumor necrosis factor alpha (TNF-α) is a cytokine secreted by activated monocytes and macrophages in response to infection, infestation, or tumor growth. It causes a proliferation of B cells and T cells. However, high levels of this cytokine may actually facilitate the development of disease by blocking the normal inflammatory response. It is believed that blocking the effect of TNF-α can suppress HIV production, although caution must be used.

Replenishing Cytokines

Some cytokines (interleukin-2, interleukin-12, and interferon alpha) may be helpful in treating HIV by stimulating the production of killer cells as well as increasing the function of lymphocytes. The interferons are part of the body's first line of defense against viruses. All of these agents have toxic side effects, requiring careful nursing assessment and care.

Restoring Immune System Function

HIV infection not only destroys CD4 + cells, but also eventually destroys the lymphoid organs, such as bone marrow and the thymus gland. Lymphocytes, including the CD4 + cells, are derived from stem cells in bone marrow and mature in the thymus. Two investigational treatments to restore the immune system are bone marrow transplant and thymus transplant. Bone marrow transplants have been used to correct other types of immune disorders (such as leukemia or lymphoma) but have yet to be effective in persons with HIV. A few thymus transplants have been done in HIV-infected persons, but have provided only temporary benefits.

HIV infection and qualified based on the HIV prevalence in the setting and the client's individual risk

Further testing is always required to confirm a reactive screening test result.

- *Enzyme-linked immunosorbent assay (ELISA)* is the most widely used screening test for HIV infection. The ELISA test was developed in 1985 to screen blood donors. ELISA tests for HIV antibodies; it does not detect the virus. Therefore, a client may have a negative ELISA test early in the course of infection, before detectable antibodies have developed. The test has a 99.5% or higher sensitivity when performed at least 13 weeks after infection. This means that more than 99.5% of tests performed on blood containing HIV antibodies will show a positive result. False positives can occur; therefore, an initial positive result is always tested repeatedly and confirmed using a different method of antibody detection, usually the Western blot.

The FDA has licensed more than one rapid test; these are widely used because results can be given immediately. Immediate notification is critical because many clients tested for HIV do not return to learn the results; many cannot be located to give the test results and educate about safe behaviors whether they are positive or negative for HIV. Although confirmation of results is dependent on testing with a second source, ELISA or Western blot test, learning results immediately gives the client more information to make wise choices about his or her behaviors and self-care.

- *Western blot antibody testing* is more reliable but more time consuming and more expensive than ELISA. When combined with ELISA, however, a specificity of greater than 99.9% is achieved. Specificity is a measure of the probability that a negative test result indicates that no antibodies are present. In this test, the client's serum is mixed with HIV proteins to detect reaction. If antibodies to HIV are present, a detectable antigen–antibody response will occur.
- *HIV viral load tests* measure the amount of actively replicating HIV. Levels correlate with disease progression and response to antiretroviral medications. Levels greater than 5000 to 10,000 copies/mL indicate the need for treatment.
- *CBC* is performed to detect anemia, leukopenia, and thrombocytopenia, which are often present in HIV infection. Lymphopenia (or low levels of lymphocytes) is especially common in this disease.
- *CD4 cell count* is the most widely used test to monitor the progress of the disease and guide therapy. The CD4 cell count correlates very closely with the immunodeficiency disorders seen in AIDS. AIDS is now defined not only by the presence of opportunistic infections and other diseases indicative of immunodeficiency, but also by HIV-seropositive status and a CD4 count of less than 200/mm^3 or a percentage of CD4 lymphocytes of less than 14%. CD4 counts are recommended every 3 to 6 months for all people with HIV disease.

In addition to these widely used tests, several other diagnostic tests may be performed:

- *Blood culture for HIV* provides the most specific diagnosis but is an expensive and cumbersome test that is not widely available in the United States.
- *Immune-complex-dissociated p24 assay* is a test for p24 (HIV) antigen in the blood. This antigen indicates active reproduction of HIV and tends to be positive prior to seroconversion and with advanced disease. It is most useful in monitoring disease progression and the antiviral activity of experimental medications (Pagana & Pagana, 2002; Tierney et al., 2005).

Other diagnostic tests are used primarily to detect secondary cancers and opportunistic infections in the client with HIV. Tests ordered are both general and specific to the client's manifestations and may include the following:

- *Tuberculin skin testing* to detect possible tuberculosis infection
- *MRI* of the brain to identify lymphomas
- *Specific cultures and serology examinations for opportunistic infections* such as PCP, toxoplasmosis, and others
- *Pap smears* every 6 months for early detection of cervical cancer in women with cervical dysplasia (Tierney et al., 2005).

Medications

Pharmacologic management of the client with HIV disease has four primary foci: (1) to suppress the infection itself, decreasing symptoms and prolonging life, (2) to provide prophylaxis of opportunistic infections, (3) to stimulate hematopoietic response, and (4) to treat opportunistic infections and malignancies. Effectiveness of treatment is monitored by viral load and CD4 cell counts; positive results are indicated by a reduction in viral load along with preserving the CD4 count above 350 mm^3. Treatment is recommended when the CD4 count falls below 200 mm^3. Clients with symptoms of severe disease are treated regardless of their CD4 level or viral load, so monitoring these individuals may reveal higher levels of CD4 or lower viral load. Initiating therapy in asymptomatic individuals with higher CD4 levels did not show protective effect and was thought to perhaps increase viral resistance. Today the drugs have been combined and dosing schedules simplified, which helps clients adhere to medication administration schedules. Currently, researchers are using clinical trials of asymptomatic clients receiving HAART to evaluate alternating drug regimens to prevent drug resistance by the viral organisms (Martinez-Picado et al., 2003).

Four classes of drugs used in antiretroviral treatment include nucleoside reverse transcriptase inhibitors (NRTIs), nonnucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), and entry inhibitors. HAART combines three or four antiretroviral drugs to reduce the incidence of drug resistance. Combination therapies increase the likelihood of decreasing viral load and symptoms but also burden clients with complicated and expensive medication schedules. Clients beginning the HAART protocol must understand the benefits, risks, costs, and effects on daily life. HAART does not eradicate HIV infection. HAART medications are expensive, the newer triple combinations such as Trizivir cost about $1030 for 60 doses, a 30-day supply. This costs approximately $13,400 per year, and does not include medications to prevent or treat opportunistic infections or cancer (Tierney et al., 2005). These medications are scheduled for specific times throughout the day; therefore, leading a normal life becomes a challenge. In addition, all HAART medications cause major adverse reactions leading to less than perfect adherence, as with most chronic diseases, but in this case, the outcome could be fatal.

Each client must be able to adhere to the treatment regimen. It may be preferable to delay initiating therapy until the client is able to agree to adhere so irregular dosing does not lead to viral resistance. Some providers gauge client ability to follow the HAART regimen by the client's success with prophylaxis for an opportunistic infection.

Several methods to promote and ensure adherence are being used and studied. Wroe and Thomas (2003) found it helpful to distinguish between intentional and unintentional nonadherence and treat them as separate entities. Clients' beliefs and internal logic were found to impact intentional nonadherence; preparing clients for the effects of HAART therapy by focusing on lessening clients' reasons for not taking medication is believed to reduce intentional nonadherence. Enriquez and McKinsey (2004) emphasize the role of nursing in preventing drug resistance by assessing patients' readiness to adhere to the treatment regimen and intervening to overcome identified barriers to adherence prior to initiating therapy.

Another approach to adherence is the use of electronic monitoring devices (EMD) (Bova et al., 2005). By placing a microprocessor in a medication cap, records are created of the time, date, and frequency of bottle opening. Although this method does not guarantee that the medication will be taken even if the cap is removed, the record created is a source for follow-up and discussion between the provider and the client. Whether the client is asked to keep a diary of taking the medication, using an EMD to keep a record, or relying on pill count, adherence to medication regimen is critically important.

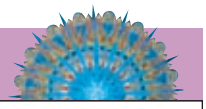
Ingersoll and Heckman (2005) found the most effective provider–client relationship for fostering adherence is a balance of appropriate challenge and support. Providers who were never confrontational seem to be perceived by clients as giving permission to be less adherent. Although depression, substance use, and financial considerations undoubtedly influence adherence to HAART therapy, and need to be addressed, provider–client relationships seem to have the most influence on adherence behavior.

NUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS The NRTIs (also called nucleoside analogs) inhibit the action of viral reverse transcriptase, a retroviral enzyme that catalyzes the substrates for conversion and copying of viral RNA to DNA sequences. This enzyme is necessary for viral integration into cellular DNA and replication. The nucleoside analogs act as a chemical decoy for building blocks of the formation of the DNA copy, preventing the RNA from being copied into DNA. Each drug substitutes for a particular nucleoside base at different points on the chain. See the accompanying Medication Administration guidelines below for this group of drugs.

Zidovudine (Retrovir, AZT) was the first antiretroviral agent approved for use with HIV infection. It remains in widespread use and has been shown to decrease symptoms and prolong the lives of clients with AIDS. Zidovudine is often given to clients with a CD4 cell count of less than 500 because of evidence that it slows the progression to severe disease (Tierney et al., 2005). Zidovudine may also be used prophylactically following a documented parenteral exposure to HIV. AZT is used in combination with ddI, ddC, or 3TC.

- Didanosine (ddI, Videx) also inhibits reverse transcriptase and viral replication. It is used in combination therapy with AZT.
- Stavudine (d4T, Zerit) is a retroviral inhibitor that has been shown to increase CD4 cell counts and decrease serum p24

MEDICATION ADMINISTRATION Antiretroviral Nucleoside Analogs



ZIDOVUDINE (AZT, AZIDOTHYMIDINE)

Zidovudine is the first antiretroviral agent developed to treat HIV infection. It interferes with reverse transcriptase, thus inhibiting replication of the virus. The usual dose is 300 mg twice daily. It is administered orally. Dose-limiting side effects are anemia and neutropenia.

Nursing Responsibilities

- Assess for possible contraindications to therapy including allergic response or a CD4 count of greater than 350/mm^3.
- Administer by mouth, instructing the client to swallow capsules whole.
- Assess for adverse effects. Nausea and headache are common. They may be self-limiting, decreasing with time, or significant and continuing, necessitating a change of therapy. Nausea and neutropenia are treated with erythropoietin (epoetin alpha) and G-CSF (filgrastim).
- Assess CBC with differential and creatine phosphokinase. Notify the physician of significant changes.

Health Education for the Client and Family

- Zidovudine will not cure HIV infection but slows its progress and reduces significant symptoms.
- Take the drug at least 0.5 hour before or 1 hour after meals if tolerated.
- With this and all antiretroviral drugs, it is important to emphasize that the client is still infective and can pass the infection to others. Use safer sex practices and other measures to prevent transmission to partners. Do not donate blood or breast-feed.
- Notify the physician if signs of an infection or adverse response to zidovudine develop: sore throat, swollen lymph glands, fever; unusual fatigue or weakness; easy bruising, bleeding gums, or an injury that will not heal; persistent or intractable nausea; muscle pain or wasting.
- Continue all scheduled follow-up visits and laboratory studies to monitor for drug toxicity.
- Check with the physician before taking any prescription or over-the-counter drug.

DIDANOSINE (DDI, VIDEX)

As with zidovudine, didanosine does not kill HIV but inhibits its replication within the cells. Its activity is similar to that of zidovudine. Didanosine has been shown to increase CD4 cell counts and lower p24 antigen levels (Tierney et al., 2005). Didanosine is used alone for clients who are intolerant or resistant to zidovudine. It is also being used with zidovudine in combination therapy regimens. Didanosine does not cause the anemia associated with zidovudine, but it may cause neutropenia. Didanosine is also associated with an increased risk of pancreatitis, peripheral neuropathy, and dry mouth.

Nursing Responsibilities

- Assess for possible contraindications to didanosine therapy, including previous episodes of pancreatitis and impaired renal or liver function.
- Administer as directed. Tablets are to be chewed thoroughly or dissolved in 1 ounce of water at room temperature. The powder form is dissolved in water prior to administration, and diarrhea is attributable to the buffering agent used in this formula.
- Administer with caution to clients taking vincristine, rifampin, pentamidine, ethambutol, or metronidazole; the action of both drugs may be affected by concurrent administration. Intravenous pentamidine and trimethoprim-sulfamethoxazole taken concurrently may increase the risk of acute and fatal pancreatitis.
- Didanosine interferes with the absorption of ketoconazole and dapsone. Doses of these drugs should be scheduled at least 2 hours apart from didanosine doses.
- Evaluate for therapeutic response and possible adverse effects. Notify the physician if manifestations of peripheral neuropathy, diarrhea, depression, or other adverse effects develop.
- Stop the drug and notify the physician immediately if the client develops manifestations of pancreatitis or hepatic failure, including nausea and vomiting, severe abdominal pain, elevated bilirubin, or elevated serum enzymes (e.g., amylase, AST, ALT).

Health Education for the Client and Family

- Take the drug as directed. The prescribed two-tablet dose must always be taken to get the required amount of antacid to prevent the drug from being destroyed by stomach acid.
- Take on an empty stomach, at least 1 hour before or 2 hours after meals.
- Do not use alcohol while taking didanosine; alcohol may increase the risk of pancreatitis.
- Stop the drug and call the doctor immediately if nausea, vomiting, abdominal pain, or diarrhea develops. These may indicate pancreatitis.
- Call the doctor if extremity pain, weakness, numbness, or tingling occurs. These side effects usually disappear when didanosine is discontinued.
- Other side effects to report to the physician include unusual bleeding or bruising, fatigue, weakness, fever, or persistent sore throat.

ABACAVIR

Abacavir is a nucleoside analog with activity against some HIV strain resistant to other nucleoside drugs. It is prepared in combination with zidovudine and lamivudine (Trizivir), and one tablet is taken twice daily. This combination drug is composed exclusively of nucleoside analogs, lacking NNRTIs or PIs. As such it is less effective at decreasing viral load and allowing immune system enhancement, but the ease of administration makes it a useful drug for clients who cannot adhere to more complex regimens. The main toxicity is a hypersensitivity response in approximately 5% of clients manifested with flulike symptoms. Avoid repeated use in those individuals.

Nursing Responsibilities

- Assess for possible hypersensitivity reactions, anemia, and neutropenia.
- Evaluate for desired effect of increased CD4 counts and lower blood levels of p24 antigen.
- Notify the physician if the client develops evidence of pancreatitis, impaired hepatic function, or painful peripheral neuropathy.

Health Education for the Client and Family

- Take without regard to food or water.
- Check with the physician before taking any other prescription or over-the-counter medication.
- Report all signs and symptoms of hypersensitivity to this drug.
- Report to the physician signs of infection or changes in condition.

antigen levels. Current use is for clients who are intolerant of AZT.

- Lamivudine (3-TC, Epivir) is used for low CD4 cell counts or symptomatic disease as a first-line treatment in combination with AZT.
- Abacavir (Ziagen) is a potent inhibitor of reverse transcriptase; however, it may cause serious hypersensitivity reactions.
- Zidovudine plus lamivudine (Combivir) is a combination drug in use to decrease HIV zidovudine-resistant strains.

PROTEASE INHIBITORS Protease is a viral enzyme necessary for the formation of specific viral protein needs for viral assembly and maturation. PIs bond chemically with protease to block the function of the enzyme and result in the production of immature, noninfectious viral particles. When combined with other antiviral drugs, these chemicals increase the chance of eliminating the virus by interfering with different stages of its life cycle. However, viral resistance occurs rather quickly. PIs inhibit and induce metabolism of other drugs, so their use with other medications and the dose of those medications must be carefully planned. Some drugs will circulate longer because their metabolism is inhibited; others will be speedily metabolized and eliminated.

Protease inhibitors and nucleoside analogs are associated with serious metabolic derangements. These include elevated cholesterol and triglycerides, insulin resistance and diabetes mellitus, and changes in body fat composition, which are particularly distressing to the clients. These body fat changes are primarily abdominal obesity and skeletal wasting. This set of symptoms is referred to as lipodystrophy (Tierney et al., 2005). Elevated cholesterol should be treated with pravastatin or atorvastatin. Lovastatin and simvastatin react to PIs, so they need to be avoided. Reduction of dietary sources of cholesterol should be made.

- Saquinavir (Invirase) is used in combination with nucleoside analogs to treat progression of the disease.
- Ritonavir (Norvir) is used in combination with nucleoside analogs to treat progression of the disease.
- Indinavir (Crixivan) is used in combination with nucleoside analogs to treat progression of the disease.
- Nelfinavir (Viracept) is used in cases of failure of or intolerance to other protease inhibitors.
- Amprenavir (Agenerase) is the newest protease inhibitor.
- Lopinavir/Ritonavir (Kaletra) is the first combination of protease inhibitors active against some HIV strains resistant to other protease inhibitors.

NONNUCLEOSIDE REVERSE TRANSCRIPTASE INHIBITORS Nevirapine (Viramune), delavirdine (Rescriptor), and efavirenz (Sustiva) are NNRTIs that may be used in combination with nucleoside analogs and protease inhibitors. However, one limitation to NNRTIs is the high incidence of cross-resistance to NRTIs. Some studies have shown that nevirapine and efavirenz may significantly reduce serum levels of the protease inhibitors. Only one NNRTI should be used at the same time. Nevirapine has a reported risk for liver toxicity and Stevens-Johnson syndrome (Bartlett & Weber, 2005).

ENTRY INHIBITORS: ENFUVIRTIDE (FUZEON) This new class of drugs became available in 2003. These entry or fusion inhibitors prevent HIV from entering target cells by binding to the protein envelope that surrounds the virus. When bound to the drug, the virus cannot morph in order to fit and adhere to cell membranes (Covington, 2005). Adding this new class of drug to the regimen of heavily pretreated individuals improves CD4 counts and lowers viral loads (Tierney et al., 2005).

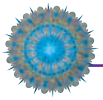
Other agents may also be administered in combination with antiretroviral therapy. Interferons, which are naturally occurring lymphokines, have been used alone and in combination. Alpha-interferon may be used to treat KS and in combination with zidovudine to slow disease progression. Gamma-interferon is also used. As more drugs become available, the burden to choose the best regimen increases for the healthcare provider. The most important limiting factor when choosing a regimen is client adherence. Second to that is selecting an effective combination of drugs without overlapping toxicities or toxicities so debilitating that adherence will be further impaired.

Some clients undergoing HAART are developing body composition changes and metabolic abnormalities associated with the therapy, especially the PIs. Increased fat deposition in the midsection, breasts, and neck with atrophy in the face, buttocks, and extremities describes the body composition changes; metabolic abnormalities include increased low-density lipoprotein cholesterol and triglycerides and insulin resistance. The combination of changes is consistent with metabolic syndrome, which increases the risk of cardiovascular disease and diabetes. These conditions are commonly treated with medications. Robinson (2005) has observed these serious changes in HIV-positive clients and hopes to prevent and treat the changes with diet and exercise without adding to the polypharmacy already experienced by those undergoing HAART.

A number of pharmacologic agents are used to prevent and treat opportunistic infections and malignancies in the client with HIV. These agents are outlined in Table 13–4.

Many clients at some point require an implanted venous access device, such as a Groshong catheter, to facilitate blood sampling, intravenous medication administration, transfusions, and parenteral nutrition. See Chapter 14 ∞ for nursing care of the client with an intravenous access device implant.

It is recommended that all HIV-infected clients receive pneumococcal, influenza, hepatitis B, and *Haemophilus influenzae b* vaccines. Persons with a positive PPD and negative chest x-ray are given prophylactic isoniazid. When the client's CD4 cell count falls to less than 200, prophylactic treatment for PCP is begun, usually with trimethoprim-sulfamethoxazole. Clients with a CD4 count of less than 100 are started on prophylactic treatment for MAC.



NURSING CARE

The client with HIV and AIDS has many care needs, including both physical and psychosocial support (see the Nursing Research box on page 360). Because there is as yet no cure or effective treatment for HIV disease, many of these needs fall within the realm of nursing to promote knowledge and understanding, self-care, comfort, and quality of life. As with many diseases that have an ultimately fatal outcome, the course of HIV infection may well be affected by the client's

TABLE 13–4 **Pharmacologic Treatment of Common Opportunistic Infections and Malignancies in HIV Disease**

CONDITION	TREATMENT	POTENTIAL ADVERSE EFFECTS
Infections		
Pneumocystis carinii pneumonia	Trimethoprim/sulfamethoxazole Pentamidine	Rash, neutropenia, anemia, thrombocytopenia, Stevens-Johnson syndrome Hypotension, altered blood glucose levels, hypocalcemia, anemia and leukopenia, liver and renal toxicity, pancreatitis
Tuberculosis	Combination drug therapy using isoniazid, rifampin, ethambutol, pyrazinamide, or streptomycin	Multiple; see Chapter 38
Candidiasis Oral thrush	Clotrimazole troches Nystatin suspension	Few toxic responses noted for either medication
Esophagitis or recurrent vaginitis	Ketoconazole Fluconazole Amphotericin B	Hepatitis, adrenal insufficiency Hepatitis Bone marrow toxicity, acute renal or hepatic failure; nausea, vomiting; chills, fever, headache
Mycobacterium avium complex	Combination therapy using • Clarithromycin, plus • Clofazimine • Ethambutol • Rifampin • Ciprofloxacin • Amikacin	 • Hepatitis, nausea, diarrhea • Diarrhea, nausea, vomiting; skin discoloration, pruritus, rash • Thrombocytopenia, hepatitis, optic neuritis • Bone marrow depression, renal failure, hepatitis • Nausea, rash • Bone marrow depression, renal failure, ototoxicity, hepatitis
Cytomegalovirus	Ganciclovir Foscarnet	Bone marrow depression, fever Renal failure, electrolyte imbalances, seizures
Herpes simplex or herpes zoster	Acyclovir	Nausea, vomiting, diarrhea; CNS effects; renal failure
Toxoplasmosis	Pyrimethamine, plus sulfadiazine or clindamycin and folinic acid	Bone marrow depression, rash; respiratory failure; nausea, vomiting, abdominal pain; hematuria
Malignancies		
Kaposi's sarcoma	Intralesional vinblastine	Inflammation and pain at injection site
Lymphoma	Combination chemotherapy	Nausea, vomiting; bone marrow toxicity; alopecia

social support systems, control, perceived self-efficacy in management, and coping mechanisms.

As the epidemic continues, nurses are providing care for increasing numbers of clients with HIV infection at various stages of disease. These clients are not only in special care settings, but also on general units, maternal–child units, hospice, and home settings. As clients with HIV disease live longer, nurses will increasingly encounter clients in whom HIV disease is a secondary diagnosis, with another primary diagnosis, for example, seizures, heart disease, diabetes mellitus, or an operative procedure.

Prevention

To date, no safe immunization to protect against HIV infection has been developed. Education, counseling, and behavior modification are the primary tools for AIDS prevention. The benefit of education and behavior modification is evident in the homosexual male population. The incidence of new HIV infections in this population has declined dramatically in high-prevalence cities. Nurses play a vital role in providing education about this epidemic and infection prevention for individuals and communities.

All sexually active individuals need to know how HIV is spread. Following are the only *totally* safe sex practices:

- No sex
- Long-term mutually monogamous sexual relations between two uninfected people
- Mutual masturbation without direct contact

Clients who do engage in sexual activity need to know and practice safer sex (Box 13–4). Reducing the number of sexual partners—for example, by entering into and remaining in a long-term mutually monogamous relationship with an uninfected partner—reduces the risk. Clients should not engage in unprotected sex, especially if the HIV status of the partner is unknown. Latex condoms have been shown to reduce the risk of transmitting HIV. Their effectiveness is improved when nonoxynol-9, a spermicide, is used for lubrication; however, it may cause genital ulcers which can facilitate HIV transmission. To be effective, condoms must be used with every sexual encounter involving vaginal, oral, or anal intercourse. They also need to be applied and removed properly. A female condom is also available for use.

Healthcare workers exposed to HIV infection or adults who experience a high-risk exposure to HIV may choose postexposure prophylaxis. Risk of exposure for healthcare workers may

MEDIALINK Case Study: HIV Prevention

NURSING RESEARCH Evidence-Based Practice and Nurses' Willingness to Care for People with AIDS

As reported by the Centers for Disease Control and Prevention, the number of deaths from AIDS has declined. This is believed to be the result of both the slowing of the epidemic and of improved treatment, which has lengthened the life span of people with AIDS. However, as treatment continues to improve survival, a key challenge will be the increasing number of people living with HIV and AIDS—and the additional resources needed for services, treatment, and care.

Several studies have found that some professional nurses and students are resistant to caring for clients with AIDS. One study by Sherman (1996) examined relationships among moral choices and nurses' willingness to care for clients with AIDS. The willingness to provide care for clients with this illness involves moral choices about one's own mortality (death anxiety), spirituality, and social support. In a survey of 220 registered nurses employed in eight hospitals in the New York Metropolitan area, Sherman found that willingness to care for AIDS patients was positively correlated with spirituality and perceived social support and negatively correlated with death anxiety. It is suggested that nurses' willingness to care for people with AIDS may be related not only to nurses' personal values and beliefs (expressed in spirituality) but also to their professional identity and role expectations.

Addressing nursing reluctance to treat clients with HIV infection, nursing educators Valois, Turgeon, Godin, Blondeau, and Cote (2001) researched the impact of persuasive messages on nursing students' beliefs and attitudes about caring for HIV-positive clients. Nursing education certainly increases knowledge and awareness of the science of HIV infection, but it may not modify attitudes or behaviors. According to the underlying theory of the research, individuals who receive evidence-based persuasive messages may develop favorable beliefs that will alter their willingness to perform a given behavior. Three main types of beliefs were considered in this study: behavioral belief (related to the expected consequences of adopting a behavior); normative belief (related to perceived social pressures by significant others resulting from adopting a behavior); and control belief (related to resources or barriers that seem to facilitate or hamper adoption of the behavior).

In three sessions, the student nurses in the experimental group were given positive persuasive messages about caring for HIV-infected clients. The persuasive messages were compelling and specific to caring for clients with HIV; case studies provided opportunities for the students to discuss the elements of the case within the framework of the persuasive messages. Students in the control group studied the science of caring for clients with HIV but did not receive the persuasive messages. When beliefs and attitudes about caring for HIV-positive clients were compared, the researchers found significantly greater willingness to provide care in the experimental group. Nursing students proved to be well prepared and motivated to receive this information.

IMPLICATIONS FOR NURSING

Standards of professional nursing clearly state that nurses will care for people with AIDS. To increase nurses' willingness to do so, students need to be better socialized into their roles and responsibilities. Providing information within an evidence-based framework, analyzing and defining effective nursing care in client cases, and utilizing the standards of professional nursing support the development of positive attitudes. Discussions within the classroom and clinical settings provide a safe means of bringing fears into the open and sharing experiences. Student groups can serve as support groups, improving communications, decreasing isolation and anxiety, and improving self-esteem and morale. Within the work setting, perceived support from colleagues and administrators as well as increased contact with people with AIDS are important factors in making caring a rewarding and positive experience.

CRITICAL THINKING IN CLIENT CARE

1. These studies were of student nurses and registered nurses. What differences do you think might have been found between the two groups today?
2. Carefully consider each of the following clients with AIDS and write a brief paragraph about how you would feel if you were assigned to care for them:
   a. A heterosexual female, age 25
   b. A homosexual male, age 35
   c. A newborn baby girl
   d. A 40-year-old single mother of three teenagers
   e. A 30-year-old homeless drug user
   f. A 17-year-old male with hemophilia, infected by blood transfusions

be through needle sticks or cuts with a sharp object, contact with mucous membrane or nonintact skin, semen, vaginal secretions, fluids contaminated with visible blood, and possibly CSF, synovial fluid, and pleural, peritoneal, pericardial, or amniotic fluids. CDC guidelines recommend treatment with HAART, which includes two NRTIs for lower risk exposures and the addition of a third drug for higher risk exposure. A 4-week course of treatment is recommended and should be started within 72 hours, preferably within 2 to 3 hours of exposure (Bartlett & Weber, 2005).

The most difficult group of high-risk people to reach and educate has been injection drug users. People in this group should never share needles, syringes, or other drug paraphernalia. Many cities have initiated needle-exchange programs, providing a sterile needle and syringe in exchange for a used one. A fresh solution of household bleach and water in a 1:10 ratio is effective to clean paraphernalia when sterile supplies are not available. It is important to also teach people in this population about safer sex practices, because most heterosexual HIV transmission occurs between injection drug users and their partners.

Screening of voluntary blood donors and donated blood supplies has reduced the risk of transmission by transfusion to 1 in 100,000. Because current blood-screening methods use antibody testing, receiving donated blood continues to carry a small risk. Clients in the *window period* between contraction of the virus and the development of detectable antibodies are able to transmit the virus to others, even though they do not yet test positive for HIV. This window period usually lasts from 6 weeks to 6 months; rarely, it lasts up to 1 year. When possible, encourage clients to use autologous transfusion, donating their own blood prior to an anticipated surgery. Seeking donations from family members is not encouraged for several reasons.

BOX 13–4 Guidelines for Safer Sex

- Practice mutual monogamy; if you are not in a mutually monogamous relationship, limit the number of sexual partners.
- Do not engage in unprotected sex, especially if HIV status of your partner is unknown. (Remember that a person may be infected and infective for up to 6 months before converting to seropositive status.)
- When entering into a new monogamous relationship, both partners should undergo HIV testing initially. If both are negative, practice abstinence or safer sex for 6 months, followed by retesting. If results still indicate that both partners are negative, sexual activity can probably be considered safe.
- Use latex condoms for oral, vaginal, or anal intercourse; avoid natural or animal skin condoms, which allow passage of HIV.
- For vaginal or anal sex, lubricate the condom with the spermicidal agent nonoxynol-9 for additional protection.
- Do not use an oil-based lubricant such as petroleum jelly, which can result in condom damage; water-based lubricants are acceptable.
- Women should carry and use a female condom.
- Remember that use of other means of birth control, such as oral contraceptives, provide no protection against HIV; barrier protection with a condom is necessary.
- Engage in safer sexual practices that are less damaging to sensitive tissues (e.g., mutual masturbation, avoiding anal or oral sex).
- Do not use drugs or alcohol.
- Do not share needles, razors, toothbrushes, sexual toys, or other items that may be contaminated with blood or body fluids.
- If HIV positive:
  a. Do not engage in unprotected sexual activity.
  b. Inform all current and former sexual partners of HIV status.
  c. Inform all healthcare personnel—primary care providers, physicians, and dentists in particular—of HIV status.
  d. Do not donate blood, plasma, blood products, sperm, organs, or tissue.
  e. If female, do not become pregnant.

Family members may have engaged in high-risk behaviors but lie about their risk because of embarrassment or fear of discovery. Furthermore, the family member may have a different blood type or have other contraindications to donating.

Encourage HIV-positive clients to abstain from donating blood, organs, or sperm. They should understand tactics to avoid exchange of body fluids by not sharing needles or other drug paraphernalia, not sharing razors, and not obtaining a tattoo. Stress the importance of informing all medical personnel providing direct care (especially anyone performing a dental, surgical, or obstetric procedure) about the diagnosis.

Healthcare workers can prevent most exposures to HIV by using standard precautions (refer to Appendix A and see Figure 13–11 ■). Testing to determine HIV status remains voluntary and relies on the use of antibody-screening methods. It is therefore impossible to identify every client who is HIV positive. With standard precautions, all clients are treated alike, eliminating the need to know the client's HIV status. All high-risk body fluids are treated as if they are infectious, and barrier precautions are used to prevent skin, mucous membrane, or percutaneous exposure to them. Counseling and testing are provided to healthcare workers with a documented needle-stick exposure. Some clinicians and facilities recommend prophylactic AZT therapy after needle-stick or splash exposure; however, it must be initiated immediately, and its effectiveness has yet to be established.

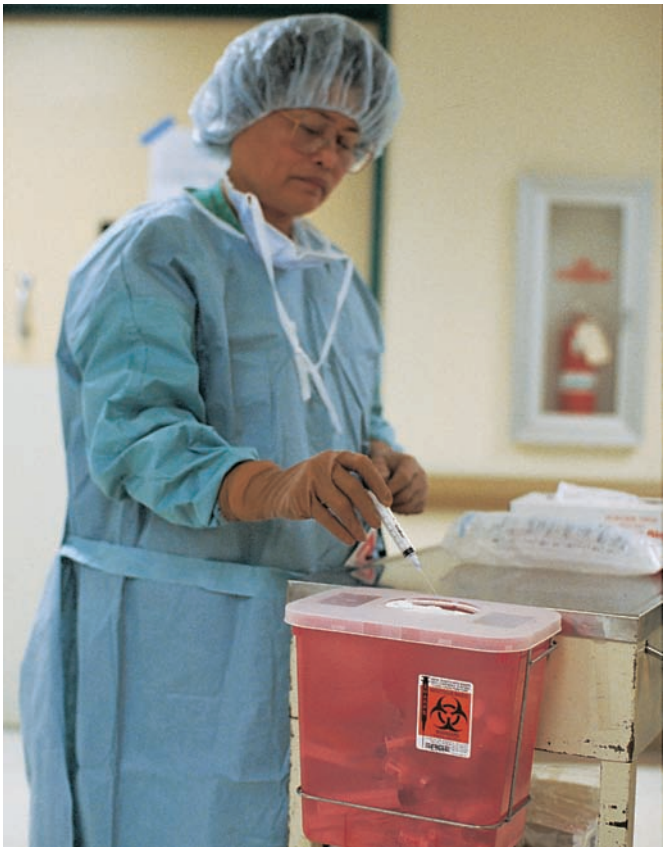


Figure 13–11 ■ This nurse is disposing of a needle and syringe in a special container, a necessary practice to avoid the transmission of HIV through needle sticks with contaminated needles.

Assessment

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

- *Health history:* risk factors (transfusion, unprotected sex, needle exposure), infections (sexually transmitted infections, hepatitis, tuberculosis), medications, recreational drug use, foreign travel, pets
- *Physical assessment:* height, weight, nutrition, skin and mucous membranes, vision, lymph nodes, breath sounds, abdominal tenderness, motor strength, coordination, cranial nerves, gait, deep tendon reflexes, genitourinary examination, mental status. Remember that symptoms must be interpreted and reported by the client. Like pain, the presence and severity of dyspnea are determined and reported

by the client. We must believe what the client tells us. Assessment is the basis for differential diagnosis; fitting appropriate treatment to the correct etiology is critical. For example, delirium is an acute confusional state and, unlike dementia, is reversible. There are effective nursing interventions for these conditions (Coyne et al., 2002).

Nursing Diagnoses and Interventions

Nursing care needs for the client with HIV infection change over the course of the disease. Preventive healthcare measures, health maintenance activities, education, and support of coping mechanisms are important in the early stages of the disease. Counseling the client with a new diagnosis of HIV infection is vital. HIV infection and AIDS continue to carry a social stigma that may interfere with the client's usual support systems and coping mechanisms. As the disease progresses and the client experiences more physical symptoms, direct care needs become more important while the need for psychosocial support continues. Acute exacerbation of opportunistic infections may necessitate hospitalization, but typically the client is managed at home. See the accompanying Nursing Care Plan on page 363.

Ineffective Coping

On receiving the test results indicating HIV seropositive status, the person with HIV infection is faced with multiple issues rarely affecting other clients. First and foremost, HIV is a disease for which there is no known cure and which is, at this time, thought to be almost universally fatal. Social support systems, family relationships, and the ability to obtain and retain useful work and health insurance may be disrupted by the disease. The client may experience guilt about his or her lifestyle and how the disease was contracted. As the disease progresses, social isolation, fatigue, body image changes, medication side effects, and multiple other issues affect the client's abilities to cope.

- Assess social support network and usual methods of coping. *This will help both the nurse and the client identify people and mechanisms that can help the client cope more effectively with the disease.*
- If possible, assign a primary nurse, whether the setting is home health, hospice, or acute care. *This helps promote the development of a therapeutic and trusting relationship and provides for continuity of care.*
- Plan for consistent, uninterrupted time with the client. *Time and a consistent presence encourage the client to express feelings and work through issues related to HIV infection.*
- Interact at every opportunity outside of providing specific nursing care treatments. *This purposeful interaction communicates caring and acceptance without fear of HIV disease.*
- Support the client's social network. *Nontraditional families may offer more support than the traditional family. This in turn may necessitate a liberal interpretation of the term* family *if unit policy is* immediate family only.
- Promote interaction between the client, significant others, and family. *Hospitalization and manifestations of HIV disease may bring about isolation from others and decrease the client's ability to cope.*
- Encourage involvement in making care decisions. *This gives the client a greater sense of self-worth and control over the situation, increasing coping abilities.*
- Set and maintain limits on manipulative and other destructive behaviors. *The client who is unable to limit inappropriate behaviors needs the external control established by setting limits.*
- Assist to accept responsibility for actions without blaming others. *Effective coping cannot occur without accepting responsibility for one's actions.*
- Support positive coping behaviors, decisions, actions, and achievements. *As self-esteem is enhanced, coping improves* (Côté & Pepler, 2005).

Impaired Skin Integrity

Dryness, malnutrition, immobility from fatigue, and skin lesions on pressure sites contribute to impaired integrity of the skin for the client with HIV disease. Maintaining skin integrity is important because of the progressive and debilitating nature of the disease. It is also a consideration both as the first line of defense against infection in an immunosuppressed client and as a site for secondary manifestations such as KS and herpes.

- Monitor the skin frequently for lesions and areas of breakdown. *Early identification of impaired skin integrity allows prompt intervention.*
- Monitor lesions for signs of infection or impaired healing. *Infection or poor tissue perfusion not only impairs healing but may lead to further skin breakdown.*
- Turn at least every 2 hours, more frequently if necessary. *Turning decreases unrelieved pressure on bony prominences and improves circulation to the tissues.*
- Use pressure-relieving devices, such as pressure and egg-crate mattresses, or sheep skin pads for elbows and heels. *These devices provide prophylactic relief of pressure.*
- Keep skin clean and dry using mild, nondrying soaps or oils for cleansing. *Night sweats and diarrhea, if present, can cause breakdown and damage to the skin. Frequent cleansing with nondrying products discourages bacterial growth, thus reducing the risk of infection.*

PRACTICE ALERT

Applying protective creams to reddened areas in the rectal area protects skin from the caustic effects of diarrhea.

- Massage around but not over affected pressure sites to increase circulation to the surrounding tissue. *Massaging over the affected area can cause skin breakdown.*
- If blisters are noted, leave intact, and dress with a hydrocolloid (DuoDERM) dressing. *Blisters provide natural sterile coverings for damaged tissue, improving healing and preventing bacterial invasion.*
- Caution against scratching. If confused, trim fingernails and use mitts or soft restraints to prevent scratching. Check for circulation of hands and fingers frequently if mitts or restraints are used. *Scratching and skin damage allow bacteria to be introduced into lesions, increasing the risk of infection. Tight or restrictive restraints or mitts may compromise circulation.*

MEDIALINK Care Plan Activity: A Client with AIDS

NURSING CARE PLAN A Client with HIV Infection

Sara Lu is a 26-year-old elementary school teacher who lives with her parents and two younger sisters. Ms. Lu is very close to her parents and sisters; they share everything with each other. During the required physical for admission to graduate school, Ms. Lu tells her physician that lately she has felt fatigued. She also states that she has had a persistent sore throat, intermittent bouts of diarrhea, and mild shortness of breath for about a month. She takes no routine medications other than a daily multivitamin and an occasional acetaminophen tablet for a headache. She is active in a drama club in her community, and she jogs 3 miles three to four times a week. She is engaged to be married; her wedding date is 6 months away. Her fiancé is the only person with whom she has had sexual relations. Her sexual activity has been unprotected. Ms. Lu has a history of open heart surgery 7 years ago to correct a congenital valve defect. She has been physically healthy since that time, until about a month or two ago. The physician orders a mononucleosis test, ELISA, Western blot analysis, CD4 T-cell count, a p24 antigen test, and an erythrocyte sedimentation rate (ESR). She has been asked to return in 1 week for follow-up.

ASSESSMENT

On Ms. Lu's follow-up visit, Carole Kee, RN, obtains her nursing history. Ms. Lu continues to have flulike symptoms but has improved somewhat. She states that she just has not been as active as usual and is worried about her health. Her appetite has decreased because of soreness in her mouth, and she has noted some whitish patches on her tongue and cheeks.

A chest x-ray film reveals no abnormality. The results of her laboratory tests are as follows:

- ELISA: positive for antibodies against HIV
- Western blot analysis: positive for antibodies against HIV
- p24 antigen test: positive for circulating HIV antigens
- ESR: increased to 25 mm/h (normal for women is 15 to 20 mm/h; normal for men is 10 to 15 mm/h)
- CD4 T-cell count: 599/mm^3 (normal range is 600 to 1200 mm^3).

Ms. Lu's physical examination reveals that she has enlarged lymph nodes in her neck and white patches on her oral mucosa. Her skin is warm to the touch. Her vital signs are as follows: T 99.9°F (37.7°C), P 84, R 20, and BP 120/78.

Ms. Lu is told of the results of her laboratory tests and the medical diagnosis of HIV infection. Ms. Lu is obviously distressed and wants to know how this happened, its meaning, whether she has infected her loved ones, and whether she will get better.

DIAGNOSES

- *Imbalanced Nutrition: Less than Body Requirements* related to soreness in mouth
- *Risk for Deficient Fluid Volume* related to decreased fluid intake and diarrhea
- *Risk for Infection* related to altered immune protection
- *Anxiety* related to diagnosis and fear
- *Deficient Knowledge* about the HIV disease process

EXPECTED OUTCOMES

- Maintain adequate nutrition for optimal body and cellular function.
- Consume at least 2500 mL of fluid per day.
- Remain free of infections and their complications.
- Verbalize anxiety and use appropriate coping mechanisms.
- Verbalize and demonstrate knowledge of HIV disease.
- Verbalize measures to prevent HIV transmission to others, including safer sex practices.

PLANNING AND IMPLEMENTATION

- Monitor daily weight and intake and output.
- Monitor dietary habits and serum albumin levels.
- Teach Ms. Lu the importance of consuming a nutritionally balanced diet and maintaining adequate fluid intake.
- Suggest strategies for coping with anorexia and nausea.
- Provide dietary consultation referral.
- Encourage oral care before and after meals.
- Assess bowel sounds and monitor elimination pattern.
- Administer antiemetic and antimotility medications as ordered.
- Monitor for signs of dehydration, such as poor skin turgor, oliguria, and orthostatic hypotension.
- Increase fluid to 2500 mL daily.
- Use strict aseptic technique for all invasive procedures.
- Teach Ms. Lu to avoid exposure to infection and people with known illnesses.
- Administer antiretroviral medications and antibiotics as prescribed, and monitor response.
- Encourage maintenance of regular physical exercise.
- Provide opportunities for Ms. Lu to verbalize her feelings.
- Avoid false reassurances.
- Provide appropriate and adequate information about HIV/AIDS.
- Teach safer sex practices and other measures to prevent HIV transmission.
- Teach anxiety-controlling techniques, such as deep breathing and meditation.

EVALUATION

Ms. Lu is eager to learn about her illness and wants her family to come with her for further explanation. She states that she is sure her fiancé will be available as well. Ms. Lu is taking home antifungal medication, diet plans, and a schedule for increased exercise. She will return in 1 week for counseling and in 1 month for a follow-up physical.

CRITICAL THINKING IN THE NURSING PROCESS

1. How does age affect the body's response to fighting HIV? What other factors affect the risk of HIV infection and its progression?
2. Are the laboratory results for Ms. Lu a true indication that she is HIV positive? What additional tests might be ordered?
3. What is the most likely source of Ms. Lu's infection? What measures are used to reduce this risk, and how did she contract HIV? What is another possible source of Ms. Lu's HIV infection?
4. Ms. Lu says that her fiancé would like to have a child. How will you counsel her regarding pregnancy and childbearing?

See Evaluating Your Response in Appendix C.

- Avoid the use of heat or occlusive dressings. *Heat can further dry and damage the skin; occlusive dressings may impair circulation and lead to ulceration.*
- Prevent skin shearing by using a turnsheet and adequate personnel when repositioning. *Shearing causes tissue trauma that can lead to decubitus ulcers.*
- Encourage ambulation if possible; if the client is confined to bed, encourage active or passive range-of-motion exercises. *Activity increases circulation, decreases pressure and skin breakdown, and helps maintain muscle tone.*
- Monitor nutritional intake and albumin levels. *Maintenance of optimal nutrition decreases the risk of tissue breakdown and improves resistance to infection.*

Imbalanced Nutrition: Less than Body Requirements

Many factors associated with HIV disease, including manifestations of the disease itself, put the client at risk for altered nutrition and weight loss. Nausea and anorexia may be manifestations of the disease or the result of antiretroviral therapy. Chronic diarrhea is a common manifestation of constitutional HIV disease. Wasting syndrome is also common. It is manifested by involuntary weight loss of greater than 10% to 15% of baseline weight, severe diarrhea, fever, and chronic fatigue and weakness. The exact cause of wasting syndrome is unclear, but the diarrhea and fatigue contribute, as does the increased metabolic rate associated with fever. Oral and esophageal candidiasis and KS of the gastrointestinal tract may cause painful swallowing, making eating difficult and thereby contributing to anorexia. Poor nutritional status in the client with HIV can ultimately result in altered comfort, a change in body image, muscle wasting, increased risk of infection, and higher mortality and morbidity.

- Assess nutritional status, including weight; body mass; caloric intake; and laboratory studies, such as total protein and albumin levels, hemoglobin, and hematocrit. *These factors provide a baseline to determine the effectiveness of interventions.*
- Identify possible causes of altered nutrition. *Identification of causes provides direction for planned interventions.*
- Administer prescribed medications for candidiasis and other manifestations as ordered. *Eliminating this opportunistic infection improves comfort and facilitates food intake. Topical viscous anesthetic can help reduce pain and improve oral intake.*
- Administer antidiarrheal medications after stools and antiemetics prior to meals. Provide antipyretics as needed to control fever. *Reducing diarrhea will improve nutrient absorption; preprandial medication with an antiemetic reduces nausea and improves food intake. Reduction of fever lowers the body's metabolic demands.*

PRACTICE ALERT
High-fiber foods can increase intestinal motility and the incidence of diarrhea.

- Provide a diet high in protein and kilocalories. *A high-protein, high-kilocalorie diet provides the necessary nutrients to meet metabolic and tissue healing needs.*
- Offer soft foods and serve small portions. *Soft foods are easily digested. Small portions are more appealing to the anorectic or nauseated client.*
- Involve in meal planning and encourage significant others to bring favorite foods from home. *The client is more likely to consume adequate amounts of preferred foods. Allowing food choices enhances the client's sense of control.*
- Assist with eating as needed. *Fatigue and weakness can prevent the client from eating an adequate amount of food.*
- Provide supplementary vitamins and enteral feedings, such as Ensure. *This improves nutritional status and caloric intake.*
- Provide or assist with frequent oral hygiene. *Oral hygiene improves comfort and appetite, and reduces the risk of mucosal lesions.*
- Administer appetite stimulants, such as megestrol (Megace) and dronabinol (Marinol) as ordered. *Both drugs may increase appetite and promote weight gain.*

Ineffective Sexuality Patterns

The diagnosis of HIV infection can significantly alter the client's expressions of sexuality. Guilt over the diagnosis may interfere with libido. The client may be angry with a significant other or partner if that person was the probable source of infection. The client may fear spreading the disease to others via sexual relations. As the disease progresses, its manifestations can affect body image and self-esteem, impairing sexuality. Other symptoms, such as nausea, fatigue, and weakness, may also interfere with libido and sexual satisfaction.

- Examine your feelings about sexuality, your role in dealing with a client's sexuality, the client's lifestyle, and sexual preferences. *To deal effectively with the client's concerns, it is vital that the nurse be comfortable with his or her own feelings of sexuality and be able to accept the client's lifestyle. Referring the client to another nurse or counselor may be necessary.*
- Establish a trusting, therapeutic relationship through the use of time, active listening, caring, and self-disclosure. Maintain a nonthreatening, nonjudgmental attitude toward the client. *Sexuality is a private issue that will be uncomfortable or impossible for the nurse and client to discuss without a mutually trusting relationship.*
- Provide factual information about HIV infection and its effects. *This helps the client separate fears and myths from reality.*
- Discuss safer sex practices, including hugging, cuddling, nonsexual contact, the use of latex condoms and spermicidal lubricant, and mutual masturbation. *Alternative forms of sexual activity and expressing affection can allow the client and significant other to remain close throughout the course of the disease.*
- Encourage discussion of fears and concerns with significant other. *Open communication helps them to deal with issues related to sexuality.*
- For the client without a significant other, stress the need to continue to meet people and develop social relationships while practicing safer sex. *The risk of isolation is high in the client with HIV infection, and relationships with others help the client to cope with the disease.*

- Refer the client and significant other to local support groups for people and partners of people with HIV. *Support groups provide a social and support network of people facing the same issues.*

Using NANDA, NIC, and NOC

Chart 13–1 shows links between NANDA, NIC, and NOC when caring for the client with AIDS.

Community-Based Care

Teaching needs for both the client and significant other are extensive. The primary need is information about the disease, its spread, and its expected course. The client and family need current factual information to plan realistically and to combat myths, misperceptions, and prejudices. At the same time, it is important to include information about current research and progress in treating the disease to maintain a sense of hopefulness.

The following topics should be discussed with the client and family to prepare for home care:

- Guidelines for safer sex practices
- Nutrition, rest and exercise, stress reduction, lifestyle changes, and maintaining a positive outlook
- Infection prevention and transmission including hand washing and wearing gloves when handling client's secretions or excretions
- Importance of regular medical follow-up and monitoring of immune status
- Signs and symptoms of opportunistic infections and malignancies, as well as other symptoms that should be reported
- Medications and adverse effects
- Use and care of implanted venous access devices, total parenteral nutrition, intravenous pumps and continuous medication delivery systems, and intravenous or aerosolized medications
- Cessation of smoking, alcohol, and recreational or illicit drug use
- Home health services

NANDA, NIC, AND NOC LINKAGES

CHART 13–1 The Client with an HIV Infection

NANDA

Fatigue

↓

NIC

Energy Management

Nutrition Management

Support System Enhancement

↓

NOC

Endurance

Nutritional Status: Energy

Stress Level

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.

- Hospice and respite care services
- Community resources, such as support groups, social agencies, and counselors
- Helpful resources:
  - CDC National AIDS Hotline
  - Gay Men's Health Crisis Network
  - National Association of People with AIDS
  - National Organization for HIV over Fifty.

MEDIALINK AIDS/HIV Support and Resources

EXPLORE MEDIALINK

Prentice Hall Nursing MediaLink DVD-ROM

Audio Glossary
NCLEX-RN® Review
Crossword Puzzle: The Immune System

Animation/Video

Histamine
Immune System
Immune System in the Older Adult

COMPANION WEBSITE www.prenhall.com/lemone

Audio Glossary
NCLEX-RN® Review
Care Plan Activity: A Client with AIDS
Case Study: HIV Prevention
MediaLink Application: At Risk for HIV/AIDS
Links to Resources

CHAPTER HIGHLIGHTS

- The immune system is a complex combination of cellular and humoral components that protect against disease. Immunity develops when the body recognizes foreign proteins as "non-self" and develops nonspecific inflammatory responses and specific cellular responses to each foreign antigen.
- Clients suffer when the immune system is excessively or inadequately responsive, or when recognition of self fails and reactions escalate against self. The latter occurs in autoimmune diseases.
- With aging, there is a general decline in the sensitivity and regulation of the immune system, often resulting in autoimmune disease.
- Hypersensitivities are excessive responses to antigens that result in harm to the client. These range from benign to severely life threatening. Damage to host tissue is caused by chemicals of the immune response, destruction of cells, or creation of large antigen–antibody complexes that accumulate in the kidney glomerular capillaries.
- Allergic reactions are treated pharmacologically to prevent or moderate allergic responses. Another method of dampening allergic responses is by desensitization, a weekly process of introducing increasing amounts of known allergens subdermally.
- Clients must be taught that the safest practice is to avoid contact with all known allergens.
- Latex allergy is a problem for healthcare professionals. Repeated exposure to latex-containing equipment and gloves results in delayed hypersensitivity.
- Any type of allergic reaction has the potential to escalate to anaphylaxis. Respiratory arrest and cardiac failure are risks with full blown allergic reactions. Nurses must recognize early signs and symptoms and immediately signal for emergency care.
- Intentional immunosuppression is an essential step in preventing transplant rejection. The client receiving a transplanted organ will be treated with immune-suppressing drugs to prevent initial rejection, to maintain the transplant, and to halt any rejection process that may develop. Clients will take the immune-suppressing, antirejection drugs for their lifetime. The drugs prevent cytokine production that upregulates an immune reaction and targets the transplanted organ. Most immunosuppressing drugs are nephrotoxic; immunosuppression places clients at greater risk for infection and cancers.
- HIV/AIDS continues to spread and many clients are unaware they have the virus. AIDs is a profoundly immune-suppressed condition that results from viral destruction of cellular components of host immunity.
- A major change in the AIDS epidemic is the disease profile, which has benefited from HAART. HAART is a combination of drugs that limits viral replication and host susceptibility to opportunistic infections and cancer. Clients are living much longer with the disease without progression to AIDS. "Pill burden" refers to the number of pills the client must take daily to maintain immune function; side effects of the combination of drugs which make up HAART are appearing as clients live longer. In addition to increased susceptibility to infections and cancers, clients suffer from a dementia peculiar to AIDs.

TEST YOURSELF NCLEX-RN® REVIEW

1 Which one of the following conditions is caused by a type I IgE-mediated hypersensitivity reaction?
   1. autoimmune hemolytic anemia
   2. systemic lupus erythematosus
   3. graft rejection
   4. anaphylaxis

2 A client received a liver transplant 1 day ago. If the client were to develop an acute transplant rejection episode, when should the nurse expect to see the manifestations?
   1. approximately 4 days to 3 months later
   2. approximately 2 days later
   3. within the first 24 hours
   4. within the first 8 hours

3 The nurse notes a cough, shortness of breath, and tachypnea in a client with AIDS. Which opportunistic infection is probably causing these manifestations?
   1. *Toxoplasma gondii*
   2. cytomegalovirus
   3. *Pneumocystis carinii*
   4. *Cryptococcus neoformans*

4 Which of the following explanations should the nurse give to a client who has tested positive for HIV?
   1. "You have been diagnosed with AIDS."
   2. "At this point, AIDS is not active in your blood."
   3. "This means that you will not develop AIDS in the future."
   4. "Antibodies to the AIDS virus are present in the blood."

5 Clients taking zidovudine (Retrovir) should be monitored for which of the following adverse reactions?
   1. cardiotoxicity
   2. leukopenia
   3. nephrotoxicity
   4. polycythemia

6 The order of administering antigens in allergy testing is based on prevention of anaphylaxis. Which method should be used first?
   1. inhalation
   2. prick test
   3. intradermal injection
   4. subcutaneous injection

7 If a hypersensitivity response is suspected when blood products are infusing, the nurse should:
   1. discard the product immediately.
   2. replace all tubing and attach a new line with NS.
   3. backflush the line and run the NS attached at the Y tubing.
   4. remove the intravenous catheter and establish access distal to the site.

8 Protease inhibitors and nucleoside analogs share correlations to metabolic abnormalities including:
   1. lactose intolerance.
   2. diabetes mellitus.
   3. Hashimoto's thyroiditis.
   4. systemic lupus erythematosus.

9 The priority when initiating or changing HIV drug therapy regimens is:
1. cost of therapy.
2. access to dental care.
3. toxicities associated with each drug.
4. client willingness to adhere to the drug regimen.

10 Clients receiving kidney transplants will receive immunosuppressant therapy. The agent used to induce immunosuppression immediately following a transplant is often:
1. azathioprine.
2. corticosteroids.
3. muromonab-CD3.
4. antithymocyte globulin.

See Test Yourself answers in Appendix C.

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