Assessment and Management of Patients With Allergic Disorders

LEARNING OBJECTIVES

On completion of this chapter, the learner will be able to:

1. Explain the physiologic events involved with allergic reactions.
2. Describe the types of hypersensitivity.
3. Describe the management of patients with allergic disorders.
4. Describe measures to prevent and manage anaphylaxis.
5. Use the nursing process as a framework for care of the patient with allergic rhinitis.
6. Discuss the different allergic disorders according to type.
The human body is menaced by a host of potential invaders— allergens as well as microbial organisms—that constantly threaten its surface defenses. After penetrating those defenses, these allergens and organisms compete with the body for its nutrients and, if allowed to flourish unimpeded, disrupt its enzyme systems and destroy its vital tissues. To protect against these agents, the body is equipped with an elaborate defense system.

The epithelial cells coating the skin and making up the lining of the respiratory, gastrointestinal, and genitourinary tracts provide the first line of defense. The structure and continuity of these surfaces and the resistance to penetration are initial deterrents to invaders.

One of the most effective defense mechanisms is the body’s capacity to equip itself rapidly with weapons (antibodies) individually designed to meet each new invader, namely specific protein antigens. Antibodies react with antigens in a variety of ways: (1) by coating the antigens’ surfaces if they are particular substances, (2) by neutralizing the antigens if they are toxic, and (3) by precipitating the antigens out of solution if they are dissolved.

The antibodies prepare the antigens so that the phagocytic cells of the blood and the tissues can dispose of them. In some cases, however, the body produces inappropriate or exaggerated responses to specific antigens, and the result is an allergic or hypersensitivity disorder.

**Allergic Reaction: Physiologic Overview**

An allergic reaction is a manifestation of tissue injury resulting from interaction between an antigen and an antibody. Allergy is an inappropriate and often harmful response of the immune system to normally harmless substances. In this case, the substance is termed an allergen. Atopy refers to allergic reactions characterized by the action of IgE antibodies and a genetic predisposition to allergic reactions.

When the body is invaded by an antigen, usually a protein that the body’s defenses recognize as foreign, a series of events occurs in an attempt to render the invader harmless, destroy it, and remove it from the body. When lymphocytes respond to the antigen, antibodies (protein substances that protect against antigens) are produced. Common allergic reactions occur when the immune system of a susceptible person responds aggressively to a substance that is normally harmless (eg, dust, weeds, pollen, danger). Chemical mediators released in allergic reactions may produce symptoms ranging from mild to life-threatening.

The many cells and organs of the immune system secrete various substances important in the immune response. These parts of the immune system must work together to ensure adequate defense against invaders (ie, virus, bacteria, other foreign substances) without destroying the body’s own tissues by an overly aggressive reaction.

**FUNCTION AND PRODUCTION OF IMMUNOGLOBULINS**

Antibodies formed by lymphocytes and plasma cells in response to an immunogenic stimulus constitute a group of serum proteins called immunoglobulins. Grouped into five classes (IgE, IgD, IgG, IgM, and IgA), antibodies can be found in the lymph nodes, tonsils, appendix, and Peyer’s patches of the intestinal tract or circulating in the blood and lymph. Each antibody molecule is composed of two identical heavy (H) chains and two identical light (L) chains. Each chain contains one variable region and one or more constant regions. The constant regions determine the class (IgE, IgD, etc.) of each antibody and allow each class of antibody to interact with specific effector cells and molecules. The variable regions contain antigen-binding sites (Porth, 2002). Antibodies are capable of binding with a wide variety of antigens, which include macromolecules and small chemicals (Abbas & Lichtman, 2001). Antibodies of the IgM, IgG, and IgA classes have definite and well-established protective functions. These include neutralization of toxins and viruses and precipitation, agglutination, and lysis of bacteria and other foreign cellular material. (See Chap. 50 for further discussion of these functions.)

Immunoglobulins of the IgE class are involved in allergic disorders and some parasitic infections, evidenced by elevation of IgE levels. IgE-producing cells are located in the respiratory and intestinal mucosa. Two or more IgE molecules bind together to an allergen and trigger mast cells or basophils to release chemical mediators, such as histamine, serotonin, kinins, slow-reacting substance of anaphylaxis (SRS-A), and the neutrophil factor, which produces allergic skin reactions, asthma, and hay fever.
Antibodies combine with antigens in a special way, likened to keys fitting into a lock. Antigens (the keys) only fit certain antibodies (the locks). Hence, the term “specificity” refers to the specific reaction of an antibody to an antigen. There are many variations and complexities in these patterns. The strength with which one antigen-binding surface of an antibody binds to one epitope, an immunologically active site on an antigen, is known as the affinity of the interaction (Abbas & Lichtman, 2001).

Antibody molecules are bivalent; that is, they have two combining sites. Therefore, the antibody easily becomes a cross-link between two antigen groups, causing them to clump together (agglutination). By this action, foreign invaders are cleared from the bloodstream. Agglutination is the means for determining blood group in laboratory tests.

Role of B Cells

The B cell, or B lymphocyte, is programmed to produce one specific antibody. On encountering a specific antigen, a B cell stimulates production of plasma cells, the site of antibody production. The result is the outpouring of antibodies for the purpose of destroying and removing the antigen.

Role of T Cells

The T cell, or T lymphocyte, assists the B cells in producing antibodies. T cells secrete substances known as lymphokines that encourage cell growth, promote cell activation, direct the flow of cell activity, destroy target cells, and stimulate the macrophages. Macrophages present the antigen to the T cells and initiate the immune response. They also digest antigens and assist in removing cells and other debris. The antigen-binding site of a T cell has a structure much like that of an immunoglobulin. It recognizes epitopes through complementary interactions. Unlike a specific antibody, a T cell does not bind free antigens (Parslow, Stites, Terr & Imboden, 2001).

FUNCTION OF ANTIGENS

Antigens are divided into two groups: complete protein antigens and low-molecular-weight substances. Complete protein antigens, such as animal dander, pollen, and horse serum, stimulate a complete humoral response. (See Chap. 50 for a discussion of humoral immunity.) Low-molecular-weight substances, such as medications, function as haptens (incomplete antigens), binding to tissue or serum proteins to produce a carrier complex that initiates an antibody response. The term “hapten” is derived from the Greek word haptien (to fasten). The proteins or other immunogens that haptens are fastened to are known as carriers (Parslow et al., 2001).

In an allergic reaction, the production of antigen-specific IgE antibodies requires active communication between macrophages, T cells, and B cells. When the allergen is absorbed through the respiratory tract, gastrointestinal tract, or skin, allergen sensitization occurs. The macrophage processes the antigen and presents it to the appropriate T cell. B cells that are influenced by the T cell mature into an allergen-specific IgE immunoglobulin-secreting plasma cell that synthesizes and secretes antigen-specific IgE antibody.

FUNCTION OF CHEMICAL MEDIATORS

Mast cells, which have a major role in IgE-mediated immediate hypersensitivity, are located in the skin and mucous membranes. When mast cells are stimulated by antigens, powerful chemical mediators are released that cause a sequence of physiologic events resulting in symptoms of immediate hypersensitivity (Fig. 53-1). There are two types of chemical mediators: primary, which are preformed and found in mast cells or basophils, and secondary, which are inactive precursors formed or released in response to primary mediators. The most prevalent known primary and secondary mediators are described next. Table 53-1 summarizes the actions of primary and secondary chemical mediators.

Primary Mediators

IgE-mediated inflammation occurs when an antigen binds to the IgE antibodies that occupy certain receptors on mast cells. Within minutes, this binding causes the mast cell to degranulate, releasing certain preformed mediators. A two-phase response results. There is an initial immediate effect on blood vessels, smooth muscle, and glandular secretion. This is followed a few hours later by cellular infiltration of the involved site. This type of inflammatory response is commonly known as an immediate hypersensitivity response (Parslow et al., 2001).
**Table 53-1 • Chemical Mediators of Hypersensitivity**

<table>
<thead>
<tr>
<th>MEDIATORS</th>
<th>ACTION</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary Mediators</strong></td>
<td></td>
</tr>
<tr>
<td>(Preformed and found in mast cells or basophils) Histamine (preformed in mast cells)</td>
<td>Vasodilation Smooth muscle contraction, increased vascular permeability, increased mucus secretion</td>
</tr>
<tr>
<td>Eosinophil chemotactic factor of anaphylaxis (ECF-A) (preformed in mast cells) Platelet-activating factor (PAF) (requires synthesis by mast cells, neutrophils, and macrophages) Prostaglandins (chemically derived from arachidonic acid; require synthesis by cells) Basophil kallikrein (preformed in mast cells)</td>
<td></td>
</tr>
<tr>
<td><strong>Secondary Mediators</strong></td>
<td>Smooth muscle contraction</td>
</tr>
<tr>
<td>(Inactive precursors formed or released in response to primary mediators) Bradykinin (derived from precursor kininogen) Serotonin (preformed in platelets) Heparin (preformed in mast cells) Leukotrienes (derived from arachidonic acid and activated by mast cell degranulation) C, D, and E or slow-reacting substance of anaphylaxis (SRS-A)</td>
<td>Smooth muscle contraction, increased vascular permeability, stimulates pain receptors, increased mucus production Anticoagulant Smooth muscle contraction, increased vascular permeability</td>
</tr>
</tbody>
</table>

**HISTAMINE**

Histamine plays an important role in the immune response. Histamine is released from mast cell granules where it is stored. Maximal intensity is reached within about 15 minutes after antigen contact (Parslow et al., 2001). The effects of histamine release include erythema; localized edema in the form of wheals; pruritus; contraction of bronchial smooth muscle, resulting in wheezing and bronchospasm; dilation of small venules and constriction of larger vessels; and increased secretion of gastric and mucosal cells, resulting in diarrhea. Histamine action results from stimulation of histamine-1 (H1) and histamine-2 (H2) receptors found on different types of lymphocytes, particularly T-lymphocyte suppressor cells and basophils. H1 receptors are found predominantly on bronchiolar and vascular smooth muscle cells. H2 receptors are found on gastric parietal cells.

Certain medications are categorized by their action at these receptors. Diphenhydramine (Benadryl) is an example of an antihistamine, which is a medication displaying an affinity for H1 receptors; cimetidine (Tagamet) and ranitidine (Zantac) are examples of other pharmacologic agents that target H2 receptors to inhibit gastric secretions in peptic ulcer disease.

**EOSINOPHIL CHEMOTACTIC FACTOR OF ANAPHYLAXIS**

Preformed in the mast cells, this chemotactic factor, which affects movement of eosinophils (granular leukocytes) to the site of allergens, is released upon degranulation to inhibit the action of leukotrienes and histamine.

**PLATELET-ACTIVATING FACTOR**

Platelet-activating factor (PAF) is responsible for initiating platelet aggregation at sites of immediate hypersensitivity reactions. It also causes bronchoconstriction and increased vascular permeability. PAF also activates factor XII, or Hageman factor, which induces the formation of bradykinin.

**PROSTAGLANDINS**

Prostaglandins, composed of unsaturated fatty acids, produce smooth muscle contraction as well as vasodilation and increased capillary permeability. The fever and pain that occur with inflammation are due in part to the prostaglandins.

**SECONDARY MEDIATORS**

**LEUKOTRIENES**

Leukotrienes are chemical mediators that initiate the inflammatory response. They are metabolites released by mucosal mast cells. They collectively make up what was once termed “slow-reacting substance of anaphylaxis” (SRS-A). Leukotrienes cause smooth muscle contraction, bronchial constriction, mucus secretion in the airways, and the typical wheal and flare reaction of the skin (Parslow et al., 2001). Compared with histamine, leukotrienes are 100 to 1,000 times more potent in causing bronchospasm. Many manifestations of inflammation can be attributed in part to leukotrienes. Medications categorized as leukotriene antagonists or modifiers (zileuton [Zyflo], zafirlukast [Accolate], montelukast [Singular]) block the synthesis or action of leukotrienes and prevent the signs and symptoms associated with asthma.

**BRADYKININ**

Bradykinin is a polypeptide with the ability to cause increased vascular permeability, vasodilation, hypotension, and contraction of many types of smooth muscle, such as the bronchi (Parslow et al., 2001). Increased permeability of the capillaries results in edema. Bradykinin stimulates nerve cell fibers and produces pain.

**SEROTONIN**

Serotonin is released during platelet aggregation, acting as a potent vasoconstrictor and causing contraction of bronchial smooth muscle.
HYPERSENSITIVITY

Although the immune system defends the host against infections and foreign antigens, immune responses can themselves cause tissue injury and disease. An immune response to an antigen may result in sensitivity to challenge with that antigen; hypersensitivity is a reflection of excessive or aberrant immune responses (Abbas & Lichtman, 2001).

A hypersensitivity reaction is an abnormal, heightened reaction to any type of stimuli. It usually does not occur with the first exposure to an allergen. Rather, the reaction follows a re-exposure after sensitization in a predisposed individual. Sensitization initiates the humoral response or buildup of antibodies. To promote understanding of the immunopathogenesis of disease, hypersensitivity reactions have been classified into four specific types of reactions (Fig. 53-2). Most allergies are identified as either type I or type IV hypersensitivity reactions.

Anaphylactic (Type I) Hypersensitivity

The most severe form of a hypersensitivity reaction is anaphylaxis. This systemic reaction is characterized by edema in many tissues, including the larynx, and is often accompanied by hypotension (Abbas & Lichtman, 2001). Type I or anaphylactic hypersensitivity is an immediate reaction beginning within minutes of exposure to an antigen. This reaction is mediated by IgE antibodies rather than IgG or IgM antibodies. Type I hypersensitivity requires previous exposure to the specific antigen. In turn, the plasma cells produce IgE antibodies in the lymph nodes, where helper T cells aid in promoting this reaction. The IgE antibodies

![Type I Anaphylactic Reaction Diagram]

Type I. An anaphylactic reaction is characterized by vasodilation, increased capillary permeability, smooth muscle contraction, and eosinophilia. Systemic reactions may involve laryngeal stridor, angioedema, hypotension, and bronchial, GI, or uterine spasm; local reactions are characterized by hives. Examples of type I reactions include extrinsic asthma, allergic rhinitis, systemic anaphylaxis, and reactions to insect stings.

![Type II Cytotoxic Reaction Diagram]

Type II. A cytotoxic reaction, which involves the binding of either the IgG or IgM antibody to a cell-bound antigen, may lead to eventual cell and tissue damage. The reaction is the result of mistaken identity when the system identifies a normal constituent of the body as foreign and activates the complement cascade. Examples of type II reactions are myasthenia gravis, Goodpasture’s syndrome, pernicious anemia, hemolytic disease of the newborn, transfusion reaction, and thrombocytopenia.
bind to membrane receptors on mast cells found in connective tissue and basophils. During re-exposure, the antigen binds to adjacent IgE antibodies, activating a cellular reaction that triggers degranulation and the release of chemical mediators (histamine, leukotrienes, and eosinophil chemotactic factor of anaphylaxis [ECF-A]).

Primary chemical mediators are responsible for the symptoms of type I hypersensitivity because of their effects on the skin, lungs, and gastrointestinal tract. When chemical mediators continue to be released, a delayed reaction may occur lasting for up to 24 hours. Clinical symptoms are determined by the amount of the allergen, the amount of mediator released, the sensitivity of the target organ, and the route of allergen entry. Type I hypersensitivity reactions may include both local and systemic anaphylaxis.

**Cytotoxic (Type II) Hypersensitivity**

Type II, or cytotoxic, hypersensitivity occurs when the system mistakenly identifies a normal constituent of the body as foreign. This reaction may be a result of a cross-reacting antibody, possibly leading to cell and tissue damage. Type II hypersensitivity involves the binding of either IgG or IgM antibody to the cell-bound antigen. The result of antigen–antibody binding is activation of the complement cascade (see Chap. 50) and destruction of the cell to which the antigen is bound.

A type II hypersensitivity reaction is associated with several disorders. For example, in myasthenia gravis, the body mistakenly generates antibodies against normal nerve ending receptors. In Goodpasture syndrome, antibodies against lung and renal tissue are generated, producing lung damage and renal failure.

**Type III**

An immune complex reaction is marked by acute inflammation resulting from formation and deposition of immune complexes. The joints and kidneys are particularly susceptible to this kind of reaction, which is associated with systemic lupus erythematosus, serum sickness, nephritis and rheumatoid arthritis. Some signs and symptoms include urticaria, joint pain, fever, rash, and adenopathy (swollen glands).

**Type IV**

A delayed, or cellular, reaction occurs 1 to 3 days after exposure to an antigen. The reaction, which results in tissue damage, involves activity by lymphokines, macrophages, and lysozymes. Erythema and itching are common; a few examples include contact dermatitis, graft-versus-host disease, Hashimoto’s thyroiditis, and sarcoidosis.

**Figure 53-2** (Continued)
A type II hypersensitivity reaction resulting in red blood cell destruction is associated with drug-induced immune hemolytic anemia, Rh-hemolytic disease of the newborn, and incompatibility reactions in blood transfusions (see Chap. 33).

Immune Complex (Type III) Hypersensitivity
Type III, or immune complex, hypersensitivity involves immune complexes formed when antigens bind to antibodies. These complexes are then cleared from the circulation by phagocytic action. When these type III complexes are deposited in tissues or vascular endothelium, two factors contribute to injury: the increased amount of circulating complexes and the presence of vasoactive amines. As a result, there is an increase in vascular permeability and tissue injury. The joints and kidneys are particularly susceptible to this type of injury. Type III hypersensitivity is associated with systemic lupus erythematosus, rheumatoid arthritis, certain types of nephritis, and some types of bacterial endocarditis. These are discussed elsewhere in this text.

Delayed-Type (Type IV) Hypersensitivity
Type IV, or delayed-type hypersensitivity, also known as cellular hypersensitivity, occurs 24 to 72 hours after exposure to an allergen. It is mediated by sensitized T cells and macrophages. An example of this reaction is the effect of an intradermal injection of tuberculin antigen or purified protein derivative (PPD). Sensitized T cells react with the antigen at or near the injection site. Lymphokines are released and attract, activate, and retain macrophages at the site. These macrophages then release lysozymes, causing tissue damage. Edema and fibrin are responsible for the positive tuberculin reaction.

An example of a type IV hypersensitivity reaction is contact dermatitis resulting from exposure to allergens such as cosmetics, adhesive tape, topical medications, medication additives, and plant toxins. The primary exposure results in sensitization. Re-exposure causes a hypersensitivity reaction composed of low-molecular-weight molecules (haptens) that bind with proteins or carriers and are then processed by Langerhans cells in the skin. The symptoms that occur include itching, erythema, and raised lesions.

Assessment

HEALTH HISTORY AND CLINICAL MANIFESTATIONS
A comprehensive allergy history and a thorough physical examination provide useful data for the diagnosis and management of patients with allergic disorders. An assessment form is useful for obtaining and organizing this information (Chart 53-1).

The degree of difficulty and discomfort experienced by the patient because of allergic symptoms and the degree of improvement in those symptoms with and without treatment are assessed and documented. The relationship of symptoms to exposure to possible allergens is noted.

Diagnostic Evaluation

Diagnostic evaluation of the patient with allergic disorders commonly includes blood tests, smears of body secretions, skin tests, and the radioallergosorbent test (RAST). Results of laboratory blood studies provide supportive data for various diagnostic possibilities; however, they are not the major criteria for the diagnosis of allergic disease.

COMPLETE BLOOD COUNT WITH DIFFERENTIAL
The white blood cell (WBC) count is usually normal except with infection. Eosinophils, granular leukocytes, normally make up 1% to 3% of the total number of WBCs. A level between 5% and 15% is nonspecific but does suggest allergic reaction. Higher levels are considered moderate and severe. In moderate eosinophilia, 15% to 40% of blood leukocytes as eosinophils are found in patients with allergic disorders as well as in patients with malignancy, immunodeficiencies, parasitic infections, and congenital heart disease, and those receiving peritoneal dialysis. In severe eosinophilia, 50% to 90% of blood leukocytes as eosinophils are found in the idiopathic hypereosinophilic syndrome.

EOSINOPHIL COUNT
An actual count of eosinophils may be obtained from blood samples or smears of secretions. A total eosinophil count can be obtained from a blood sample by using special diluting fluids that hemolyze erythrocytes and stain the eosinophils. During symptomatic episodes, smears obtained from nasal secretions, conjunctival secretions, and sputum of atopic patients usually reveal eosinophils, indicative of an active allergic response.

TOTAL SERUM IMMUNOGLOBULIN E LEVELS
High total serum IgE levels support the diagnosis of atopic disease. A normal IgE level, however, does not exclude the diagnosis of an allergic disorder. IgE levels are not as sensitive as the paper radioimmunosorbent test (PRIST) and the enzyme-linked immunosorbent assay (ELISA). Indications for determining IgE levels include the following:

- Evaluation of immunodeficiency
- Evaluation of drug reactions
- Initial laboratory screening for allergic bronchopulmonary aspergillosis
- Evaluation of allergy among children with bronchiolitis
- Differentiation of atopic and nonatopic eczema
- Differentiation of atopic and nonatopic asthma and rhinitis

SKIN TESTS
Skin testing entails the intradermal injection or superficial application (epicutaneous) of solutions at several sites. Depending on the suspected cause of allergic signs and symptoms, several different solutions may be applied at several separate sites. These solutions contain individual antigens representing an assortment of allergens, including pollen, most likely to be implicated in the patient’s disease. Positive reactions (wheal and flare) are clinically significant when correlated with the history, physical findings, and results of other laboratory tests.

The results of skin tests complement the data obtained from the history. They indicate which of several antigens are most likely to provoke symptoms and provide some clue to the intensity of the patient’s sensitization. The dosage of the antigen (allergen) injected is also important. Most patients are hypersensitive
**Chart 53-1 • ASSESSMENT**

**Allergy Assessment Form**

<table>
<thead>
<tr>
<th>Name __________________________</th>
<th>Age __________</th>
<th>Sex __________</th>
<th>Date __________</th>
</tr>
</thead>
</table>

**I. Chief complaint:**

**II. Present illness:**

**III. Collateral allergic symptoms:**

- **Eyes:**
  - Pruritus ______
  - Burning ______
  - Lacrimation ______
  - Swelling ______
  - Injection ______
  - Discharge ______
- **Ears:**
  - Pruritus ______
  - Fullness ______
  - Popping ______
  - Frequent infections ______
- **Nose:**
  - Sneezing _____
  - Rhinorrhea _____
  - Obstruction _____
  - Pruritus _____
  - Mouth-breathing _____
  - Purulent discharge _____
- **Throat:**
  - Soreness____
  - Postnasal discharge____
  - Palatal pruritus____
  - Mucus in the morning____
- **Chest:**
  - Cough __________
  - Pain __________
  - Wheezing ______
  - Sputum __________
  - Dyspnea __________
  - Color __________
  - Rest __________
  - Amount __________
  - Exertion __________
  - Color __________
  - Rest __________
- **Skin:**
  - Dermatitis __________
  - Eczema __________
  - Urticaria________

**IV. Family allergies:**

**V. Previous allergic treatment or testing:**

- **Prior skin testing:**
- **Medications:**
  - Antihistamines Improved__________ Unimproved__________
  - Bronchodilators Improved__________ Unimproved__________
  - Nose drops Improved__________ Unimproved__________
  - Hyposensitization Improved__________ Unimproved__________
    - Duration ________
    - Antigens ________
    - Reactions ________
  - Antibiotics Improved__________ Unimproved__________
  - Corticosteroids Improved__________ Unimproved__________

**VI. Physical agents and habits:**

- **Bothered by:**
  - Tobacco for _____ years
  - Alcohol ________
  - Air cond. __________
  - Cigarettes _____ packs/day
  - Heat __________
  - Muggy weather ______
  - Cigars _____ per day
  - Cold __________
  - Weather changes ______
  - Pipes _____ per day
  - Perfumes __________
  - Chemicals __________
  - Never smoked __________
  - Paints __________
  - Hair spray __________
  - Bothered by smoke____
  - Insecticides ______
  - Newspapers ______
  - Cosmetics __________
  - Latex __________

**VII. When symptoms occur:**

- **Time and circumstances of 1st episode:**
- **Prior health:**
  - Course of illness over decades: progressing ________ regressing ________
  - Time of year: __________
    - Perennial ______
    - Seasonal ______
    - Seasonally exacerbated ______
  - Monthly variations (menses, occupation): __________
  - Time of week (weekends vs. weekdays): __________
  - Time of day or night: __________
  - After insect stings: __________

**VIII. Where symptoms occur:**

- **Living where at onset:**
- **Living where since onset:**
- **Effect of vacation or major geographic change:**
- **Symptoms better indoors or outdoors:**
- **Effect of school or work:**
- **Effect of staying elsewhere nearby:**
- **Effect of hospitalization:**
- **Effect of specific environments:**
  - Do symptoms occur around:
    - old leaves _____
    - hay _____
    - lakeside _____
    - barns ______
    - summer homes ______
    - damp basement ______
    - dry attic ______
    - lawnmowing ______
    - animals ______
    - other ______

*(continued)*
to more than one pollen. Under testing conditions, they may not react (although they usually do) to the specific pollens that induce their attacks.

In cases of doubt about the validity of the skin tests, a RAST or a provocative challenge test may be performed. If a skin test is indicated, there is a reasonable suspicion that a specific allergen is producing symptoms in an allergic patient. Several precautionary steps, however, must be observed before skin testing with allergens:

- Testing is not performed during periods of bronchospasm.
- Epicutaneous tests (scratch or prick tests) are performed before other testing methods in an effort to minimize the risk of systemic reaction.
- Emergency equipment must be readily available to treat anaphylaxis.

**Types of Skin Tests**

The methods of skin testing include prick skin tests, scratch tests, and intradermal skin testing (Fig. 53-3). After prick or scratch tests, intradermal skin testing is performed with allergens that did not elicit positive reactions. Because a larger antigen challenge is being used, local or systemic reactions could occur if the same antigens that produced positive skin or scratch reactions are used. The back is the most suitable area of the body for skin testing because it permits the performance of many tests. The multitest applicator is a commercially available device with multiple test heads that allows simultaneous administration of antigens by multiple punctures at different sites.

**Interpretation of Skin Test Results**

Familiarity with and consistent use of a grading system are essential. The grading system used should be identified on a skin test sheet for later interpretation. A positive reaction, evidenced by the appearance of an urticarial wheal (round, reddened skin elevation) (Fig. 53-4), localized erythema (diffuse redness) in the area of inoculation or contact, or pseudopodia (irregular projection at the end of a wheal) with associated erythema is considered indicative of sensitivity to the corresponding antigen.

There may be false-negative results due to improper technique, outdated allergen solutions, and prior use of medications that suppress skin reactivity. Corticosteroids and antihistamines, including allergy medications, suppress skin test reactivity and are usually withheld 48 to 96 hours before testing, depending on the duration of their activity. False-positive skin tests may result from improper preparation or administration of allergen solutions.

Interpretation of positive or negative skin tests must be based on the history, physical examination, and other laboratory test

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**Chart 53-1 • ASSESSMENT (Continued)**

**Allergy Assessment Form**

<table>
<thead>
<tr>
<th>Do symptoms occur after eating:</th>
<th>cheese</th>
<th>mushrooms</th>
<th>beer</th>
<th>melons</th>
<th>bananas</th>
<th>fish</th>
<th>nuts</th>
<th>citrus fruits</th>
<th>other foods (list)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Home:</td>
<td>city</td>
<td>rural</td>
<td>house</td>
<td>age</td>
<td>apartment</td>
<td>basement</td>
<td>damp</td>
<td>dry</td>
<td>heating system</td>
</tr>
<tr>
<td>Bedroom:</td>
<td>Type</td>
<td>Age</td>
<td>Living room:</td>
<td>Type</td>
<td>Age</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pillow</td>
<td></td>
<td></td>
<td>Rug</td>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Mattress</td>
<td></td>
<td></td>
<td>Matting</td>
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<tr>
<td>Blankets</td>
<td></td>
<td></td>
<td>Furniture</td>
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<tr>
<td>Quilts</td>
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<tr>
<td>Furniture</td>
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<td></td>
<td></td>
</tr>
</tbody>
</table>

IX. What does patient think makes symptoms worse? _________________________________________________________________

X. Under what circumstances is patient free of symptoms? _____________________________________________________________

XI. Summary and additional comments: ___________________________________________________________________________

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**Figure 53-3** Intradermal testing. A 0.5-mL or 1-mL sterile syringe with a 26/27 gauge intradermal needle is used to inject 0.02 to 0.03 mL of intradermal allergen. The needle is inserted with the bevel facing upward and the syringe parallel to the skin. The skin is penetrated superficially, and a small amount of the allergen solution is injected to create a bleb (raised area) approximately 5 mm in diameter. A separate sterile syringe and needle are used for each injection. From Taylor, C., Lillis C., & LeMone, P. (2001). Fundamentals of nursing: The art and science of nursing care (4th ed.). Philadelphia: Lippincott Williams & Wilkins.
The following guidelines are used for the interpretation of skin test results:

- Skin tests are more reliable for diagnosing atopic sensitivity in patients with allergic rhinoconjunctivitis than in patients with asthma.
- Positive skin tests correlate highly with food allergy.
- The use of skin tests to diagnose immediate hypersensitivity to medications is limited because metabolites of medications, not the medications themselves, are usually responsible for causing hypersensitivity.

**PROVOCATIVE TESTING**

Provocative testing involves the direct administration of the suspected allergen to the sensitive tissue, such as the conjunctiva, nasal or bronchial mucosa, or gastrointestinal tract (by ingestion of the allergen) with observation of target organ response. This type of testing is helpful in identifying clinically significant allergens in patients with a large number of positive tests. Major disadvantages of this type of testing are the limitation of one antigen per session and the risk of producing severe symptoms, particularly bronchospasm, in patients with asthma.

**RADIOALLERGOSORBENT TEST**

RAST is a radioimmunoassay that measures allergen-specific IgE. A sample of the patient’s serum is exposed to a variety of suspected allergen particle complexes. If antibodies are present, they will combine with radiolabeled allergens. After the serum is centrifuged, radioimmunoassay detects the allergen-specific IgE antibody. Test results are then compared with control values. In addition to detecting an allergen, RAST indicates the quantity of allergen necessary to evoke an allergic reaction. Values are reported on a scale from 0 to 5. Values of 2+ or greater are considered significant. The major advantages of RAST over other tests include decreased risk of systemic reaction, stability of antigens, and lack of dependence on skin reactivity modified by medications. The major disadvantages include the limited allergen selection, reduced sensitivity compared with intradermal skin tests, lack of immediate results, and cost.

**Allergic Disorders**

There are two types of IgE-mediated allergic reactions: atopic and nonatopic disorders. While the underlying immunologic reactions of the two types of disorders are the same, predisposing factors and manifestations are different. The atopic disorders are characterized by a hereditary predisposition and production of a local reaction to IgE antibodies produced in response to common environmental allergens (Kay, 2001a). The nonatopic disorders lack the genetic component and organ specificity of the atopic disorders (Porth, 2002). Examples of atopic disorders are allergic rhinitis, allergic asthma, and atopic dermatitis (Kay, 2001a).

A type I hypersensitivity response results in atopic (allergic) diseases, which affect 10% to 20% of the U.S. population. Genetic factors play a role in susceptibility to these diseases. Disorders characterized as atopic include anaphylaxis, allergic rhinoconjunctivitis, atopic dermatitis, urticaria and angioedema, gastrointestinal allergy, and asthma. Latex allergy may be a type I or type IV hypersensitivity reaction, although true latex allergy is considered to be a type I hypersensitivity reaction (Brehler & Kütting, 2001). Latex allergy is discussed later in this chapter. Contact dermatitis is considered a type IV hypersensitivity reaction.

**ANAPHYLAXIS**

Anaphylaxis is a clinical response to an immediate (type I hypersensitivity) immunologic reaction between a specific antigen and an antibody. The reaction results from IgE antibody. An anaphylactic reaction can be triggered by exposure to an antigen through inhalation, injection, ingestion, or skin contact. It is a severe, life-threatening allergic reaction. It is estimated that 3.3 to 43 million persons in the United States (1.24% to 16.8% of the population) are affected.
Pathophysiology

Anaphylaxis is caused by the interaction of a foreign antigen with specific IgE antibodies found on the surface membrane of mast cells and peripheral blood basophils. The subsequent release of histamine and other bioactive mediators causes activation of platelets, eosinophils, and neutrophils and the coagulation cascade. Smooth muscle spasm, bronchospasm, mucosal edema and inflammation, and increased capillary permeability result. These systemic changes characteristically produce clinical manifestations within seconds or minutes of antigen exposure (Neugut et al., 2001). Closely related to anaphylaxis is an anaphylactoid (anaphylaxis-like) reaction, which is described in Chart 53-2.

Substances that most commonly cause anaphylaxis include foods, medications, insect stings, and latex (Chart 53-3). Foods that are common causes of anaphylaxis include peanuts, tree nuts, shellfish, fish, milk, eggs, soy, and wheat. Many medications have been implicated in anaphylaxis. Those that are most frequently reported include antibiotics (including penicillin), radiocontrast agents, intravenous anesthetics, aspirin and other nonsteroidal anti-inflammatory drugs (NSAIDs), and opioids. Antibiotics and radiocontrast agents cause the most serious anaphylactic reactions, producing reactions in about 1 of every 5,000 exposures. Penicillin is the most common cause of anaphylaxis and accounts for about 75% of fatal anaphylactic reactions in the U.S. each year (Neugut et al., 2001).

Clinical Manifestations

Anaphylactic reactions may be categorized as mild, moderate, and severe systemic reactions. The time from exposure to the antigen to onset of symptoms is a good indicator of the severity of the reaction: the faster the onset, the more severe the reaction (Neugut et al., 2001).

Mild systemic reactions consist of peripheral tingling and a sensation of warmth, possibly accompanied by fullness in the mouth and throat. Nasal congestion, periorbital swelling, pruritus, sneezing, and tearing of the eyes can also be expected. Onset of symptoms begins within the first 2 hours of exposure. Moderate systemic reactions may include flushing, warmth, anxiety, and itching in addition to any of the above symptoms.

Chart 53-2

Anaphylactoid (Anaphylaxis-Like) Reaction

Closely related to anaphylaxis is an anaphylactoid (anaphylaxis-like) reaction, caused by the release of mast cell and basophil mediators triggered by non–IgE-mediated events. An anaphylactoid reaction may occur with medications, food, exercise, and cytotoxic antibody transfusions. The reaction may be local or systemic. Local reactions usually involve urticaria and angioedema at the site of the antigen exposure. Although possibly severe, anaphylactoid reactions are rarely fatal. Systemic reactions occur within about 30 minutes of exposure involving cardiovascular, respiratory, gastrointestinal, and integumentary organ systems. For the most part, an anaphylactoid reaction and its treatment are identical to that of anaphylaxis (Neugut et al., 2001).

Chart 53-3

Common Causes of Anaphylaxis

<table>
<thead>
<tr>
<th>Foods</th>
<th>Medications</th>
<th>Other Pharmaceutical/Biologic Agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peanuts, tree nuts</td>
<td>Antibiotics, especially penicillin</td>
<td>Animal serums (tetanus antitoxin, snake venom antitoxin, rabies antitoxin), antigens used in skin testing</td>
</tr>
<tr>
<td>(walnuts, pecans, cashews, almonds, etc.), shellfish (shrimp, lobster, crab, etc.), fish, milk, eggs, soy, wheat</td>
<td>Radiocontrast agents, anesthetic agents (lidocaine, procaine), vaccines, hormones (insulin, vasopressin, ACTH), aspirin, nonsteroidal anti-inflammatory drugs (NSAIDs)</td>
<td></td>
</tr>
<tr>
<td>Latex</td>
<td>Insect Stings</td>
<td>Medical and nonmedical products containing latex</td>
</tr>
<tr>
<td></td>
<td>Bees, wasps, hornets, yellow jackets, ants, including fire ants</td>
<td></td>
</tr>
</tbody>
</table>

More serious reactions include bronchospasm and edema of the airways or larynx with dyspnea, cough, and wheezing. The onset of symptoms is the same as for a mild reaction.

Severe systemic reactions have an abrupt onset with the same signs and symptoms described above. These progress rapidly to bronchospasm, laryngeal edema, severe dyspnea, cyanosis, and hypotension. Dysphagia (difficulty swallowing), abdominal cramping, vomiting, diarrhea, and seizures can also occur. Cardiac arrest and coma may follow.

Prevention

Strict avoidance of potential allergens is an important preventive measure for the patient at risk for anaphylaxis (Neugut et al., 2001). Patients at risk for anaphylaxis from insect stings should avoid areas populated by insects and should use appropriate clothing, insect repellent, and caution to avoid further stings.

If avoidance of exposure to allergens is impossible, administration of epinephrine is a critical measure to prevent an anaphylactic reaction. People sensitive to insect bites and stings, those who have experienced food or medication reactions, and those who have experienced idiopathic or exercise-induced anaphylactic reactions should always carry an emergency kit that contains epinephrine. The EpiPen from Dey Pharmaceuticals is a commercially available first-aid device that delivers premeasured doses of 0.3 mg (EpiPen) and 0.15 mg (EpiPen Jr.) of epinephrine (Fig. 53-5). The autoinjection system requires no preparation, and the self-administration technique is uncomplicated. The patient must be given an opportunity to demonstrate the correct technique for use; an EpiPen training device is available. Verbal and written information about the emergency kit, as well as strategies to avoid exposure to threatening allergens, must also be provided.

Screening for allergies before a medication is prescribed or first administered is an important preventive measure. A careful history of any sensitivity to suspected antigens must be obtained before administering any medication, particularly in parenteral form, because this route is associated with the most severe anaphylaxis. Nurses caring for patients in any setting (hospital, home, outpatient diagnostic testing sites, long-term care facilities) must assess patients’ risk for anaphylactic reactions. The patient is asked about previous exposure to contrast agents used for diag-
Medical Management

Management depends on the severity of the reaction. Initially, respiratory and cardiovascular functions are evaluated. If the patient is in cardiac arrest, cardiopulmonary resuscitation is instituted. Oxygen is provided in high concentrations during cardiopulmonary resuscitation or when the patient is cyanotic, dyspneic, or wheezing. Epinephrine, in a 1:1,000 dilution, is administered subcutaneously in the upper extremity or thigh and may be followed by a continuous intravenous infusion. Antihistamines and corticosteroids may also be given to prevent recurrences of the reaction and to treat urticaria and angioedema. To maintain blood pressure and normal hemodynamic status, IV fluids (ie, normal saline solution), volume expanders, and vasopressor agents are given. In patients with episodes of bronchospasm or a history of bronchial asthma or chronic obstructive pulmonary disease, aminophylline and corticosteroids may also be administered to improve airway patency and function. If hypotension is unresponsive to vasopressors, intravenous glucagon may be given for its acute inotropic and chronotropic effects. Patients with severe reactions are observed closely for 12 to 14 hours. Because of the potential for recurrence, patients with even mild reactions must be educated concerning this risk.

Nursing Management

If a patient is experiencing an allergic response, the nurse’s initial action is to assess the patient for signs and symptoms of anaphylaxis. The nurse assesses the airway, breathing pattern, and other vital signs. The patient is observed for signs of increasing edema and respiratory distress. Prompt notification of the physician and preparation for initiation of emergency measures (intubation, administration of emergency medications, insertion of intravenous lines, fluid administration, oxygen administration) are important to reduce the severity of the reaction and to restore cardiovascular function. The nurse documents the interventions used and the patient’s response to treatment, vital signs, and laboratory values.

The patient who has recovered from anaphylaxis needs an explanation of what occurred and instruction about avoiding future exposure to antigens and administering emergency medications to treat anaphylaxis. The patient must be instructed about antigens that should be avoided and about other strategies to prevent recurrence of anaphylaxis. All patients who have experienced an anaphylactic reaction should receive a prescription for preloaded syringes of epinephrine. The nurse instructs the patient and family in their use and has the patient and family demonstrate correct administration (Chart 53-4).

ALLERGIC RHINITIS

Allergic rhinitis (inflammation of nasal mucosa; hay fever, chronic allergic rhinitis, pollinosis) is the most common form of respiratory allergy presumed to be mediated by an immediate (type I hypersensitivity) immunologic reaction. It affects about 8% to 10% of the U.S. population (20% to 30% of adolescents). The symptoms are similar to viral rhinitis but are usually more persistent and demonstrate seasonal variation (Tierney, McPhee & Papadakis, 2001). It often occurs with other conditions, such as allergic conjunctivitis, sinusitis, and asthma. Allergic rhinitis is associated with impaired work and school performance and decreased quality of life (Ratner, Ehrlich, Fineman et al., 2002). When untreated, many complications may result, such as allergic asthma, chronic nasal obstruction, chronic otitis media with hearing loss, anosmia (absence of the sense of smell), and, in children, orofacial dental deformities. Early diagnosis and adequate treatment are essential to reduce complications and relieve symptoms.

Because allergic rhinitis is induced by airborne pollens or molds, it is characterized by the following seasonal occurrences:

- Early spring—tree pollen (ox, elm, poplar)
- Early summer—rose pollen (rose fever), grass pollen (Timothy, red-top)
- Early fall—weed pollen (ragweed)

Each year, attacks begin and end at about the same time. Airborne mold spores require warm, damp weather. Although there is no rigid seasonal pattern, these spores appear in early spring, are rampant during the summer, and taper off and disappear by late fall. When untreated, many complications may result, such as allergic asthma, chronic nasal obstruction, chronic otitis media with hearing loss, anosmia (absence of the sense of smell), and, in children, orofacial dental deformities. Early diagnosis and adequate treatment are essential to reduce complications and relieve symptoms.

Pathophysiology

Sensitization begins by ingestion or inhalation of an antigen. On re-exposure, the nasal mucosa reacts by the slowing of ciliary action, edema formation, and leukocyte (primarily eosinophil) infiltration. Histamine is the major mediator of allergic reactions in the nasal mucosa. Tissue edema results from vasodilation and increased capillary permeability.
Clinical Manifestations

Typical signs and symptoms of allergic rhinitis include nasal congestion; clear, watery nasal discharge; intermittent sneezing; and nasal itching. Itching of the throat and soft palate is common. Drainage of nasal mucus into the pharynx initiates multiple attempts to clear the throat and results in a dry cough or hoarseness. Headache, pain over the paranasal sinuses, and epistaxis can accompany allergic rhinitis. The symptoms of this chronic condition depend on environmental exposure and intrinsic host responsiveness. Allergic rhinitis may affect quality of life by also producing fatigue, loss of sleep, and poor concentration (Ratner et al., 2002).

Assessment and Diagnostic Findings

Diagnosis of seasonal allergic rhinitis is based on history, physical examination, and diagnostic test results. Diagnostic tests include nasal smears, peripheral blood counts, total serum IgE, epicutaneous and intradermal testing, RAST, food elimination and challenge, and nasal provocation tests. Results indicative of allergy as the cause of rhinitis include increased IgE and eosinophil levels and positive reactions on allergen testing. False-positive and false-negative responses to these tests, particularly skin testing and provocation tests, may occur.

Medical Management

The goal of therapy is to provide relief from symptoms. Therapy may include one or all of the following interventions: avoidance therapy, pharmacotherapy, and immunotherapy (Kay, 2001b). Verbal instructions must be reinforced by written information. Knowledge of general concepts regarding assessment and therapy in allergic diseases is important so that patients can learn to manage certain conditions as well as prevent severe reactions and illnesses.

AVOIDANCE THERAPY

In avoidance therapy, every attempt is made to remove the allergens that act as precipitating factors. Simple measures and environmental controls are often effective in decreasing symptoms. Examples include use of air conditioners, air cleaners, humidifiers and dehumidifiers, and smoke-free environments. In many cases, it is impossible to avoid exposure to all environmental allergens, so pharmacologic therapy or immunotherapy is needed.

PHARMACOLOGIC THERAPY

Antihistamines. Antihistamines, now classified as H1-receptor antagonists (or H1-blockers), are used in managing mild allergic disorders. (H1-receptor antagonists are used to treat gastric and duodenal ulcers.) H1-blockers bind selectively to H1 receptors, preventing the actions of histamines at these sites. They do not prevent the release of histamine from mast cells or basophils. The H1-antagonists have no effect on H2-receptors, but they do have the ability to bind to nonhistaminic receptors. The ability of certain antihistamines to bind to and block muscarinic receptors underlies several of the prominent anticholinergic side effects of these medications.

Oral antihistamines, which are readily absorbed, are most effective when given at the first occurrence of symptoms because they prevent the development of new symptoms by blocking the actions of histamine at the H1-receptors. The effectiveness of these medications is limited to certain patients with hay fever, vasomotor rhinitis, urticaria (hives), and mild asthma. They are rarely effective in other conditions or in any severe conditions.
Adrenergic agents, vasoconstrictors of mucosal vessels, are used topically (nasal and ophthalmic) in addition to the oral route. The topical route (drops and sprays) causes fewer side effects than oral administration; however, the use of drops and sprays should be limited to a few days to avoid rebound congestion. Adrenergic nasal decongestants are used for the relief of nasal congestion when applied topically to the nasal mucosa. They activate the alpha-adrenergic receptor sites on the smooth muscle of the nasal mucosal blood vessels, reducing local blood flow, fluid exudation, and mucosal edema. Topical ophthalmic drops are used for symptomatic relief of eye irritations due to allergies. Potential side effects include hypertension, dysrhythmias, palpitations, central nervous system stimulation, irritability, tremor, and tachyphylaxis (acceleration of hemodynamic status). Examples of adrenergic decongestants and their routes of administration are found in Table 53-3.

**Mast Cell Stabilizers.** Intranasal cromolyn sodium (Nasalcrom) is a spray that acts by stabilizing the mast cell membrane, thus reducing the release of histamine and other mediators of the allergic response. In addition, it inhibits macrophages, eosinophils, monocytes, and platelets involved in the immune response (Ratner et al., 2002). Cromolyn interrupts the physiologic response to nasal antigens and is used prophylactically before exposure to

| Table 53-2 • Chemical Classes of H1 Antihistamines |
|---------------------------------|---------------------------------|---------------------------------|
| **Classification and Example**  | **Major Side Effects**          | **Nursing Implications**        |
| **Sedating**                    |                                 |                                 |
| Ethanolamines                   | Drowsiness, confusion           | Teach patient to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to drug treatment is stabilized. |
| Ex: diphenhydramine (Benadryl) | Dry mouth, nausea, vomiting     | Suggest sucking on hard candy or ice chips for relief of dry mouth. |
| Piperazines                     | Photosensitivity                 | Encourage use of sunscreen and hat while outdoors. |
| Ex: hydroxyzine (Atarax)        | Urinary retention               | Assess for urinary retention; monitor urinary output.; |
|                                 | Dulls mental alertness; drowsiness | Teach patient to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to drug treatment is stabilized. |
|                                 | Dry mouth                       | Suggest sucking on hard candy or ice chips for relief of dry mouth. |
| Alkylamines                     | Less CNS depression than other groups; best class for daytime use | Teach patient to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to drug treatment is stabilized. |
| Ex: chlorpheniramine            | Gastrointestinal upset          | Administer medication with food or milk to decrease GI distress. Increase fluid intake. |
| (Chlor-Trimeton)                | Drowsiness                      | Teach patient to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to drug treatment is stabilized. |
| Ethylenediamines Ex: triplellamine (PBZ) | Palpitations         | Instruct patient to sit and relax a few minutes before activity. |
| Phenothiazines Ex: promethazine (Phenergan) | Heavy sedation and drowsiness | Teach patient to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to drug treatment is stabilized. |
| **Nonsedating**                 |                                 | Encourage use of humidification at home. |
| loratadine (Claritin)           | Gastrointestinal upset          | Instruct patient to rise from a sitting position slowly. |
| fexofenadine (Allegra)          | Occasional drowsiness and fatigue | Counsel patient to take the medication on an empty stomach. |
| cetirizine (Zyrtec)             | Drowsiness, dry mouth           | Teach patient to avoid alcohol, driving, or engaging in any hazardous activities until CNS response to drug treatment is stabilized. |

Antihistamines are the major class of medications prescribed for the symptomatic relief of allergic rhinitis. The major side effect is sedation, although histamine H1 antagonists are less sedating than earlier antihistamines (Kay, 2001b). Additional side effects include nervousness, tremors, dizziness, dry mouth, palpitations, anorexia, nausea, and vomiting. Antihistamines are contraindicated during the third trimester of pregnancy; for nursing mothers and newborns; in children and elderly people; and in patients whose conditions can be aggravated by muscarinic blockade (ie, asthma, urinary retention, open-angle glaucoma, hypertension, and prostatic hyperplasia).

Newer antihistamines are called second-generation or nonsedating H1-receptor antagonists. Unlike first-generation H1-receptor antagonists, they do not cross the blood–brain barrier and do not bind to cholinergic, serotonin, or alpha-adrenergic receptors. They bind to peripheral rather than central nervous system H1-receptors, causing less sedation. Examples of these medications are loratadine (Claritin), cetirizine (Zyrtec), and fexofenadine (Allegra). These are summarized in Table 53-2.

Adrenergic Agents. Adrenergic agents, vasoconstrictors of mucosal vessels, are used topically (nasal and ophthalmic) in addition to the oral route. The topical route (drops and sprays) causes fewer side effects than oral administration; however, the use of drops and sprays should be limited to a few days to avoid rebound congestion. Adrenergic nasal decongestants are used for the relief of nasal congestion when applied topically to the nasal mucosa. They activate the alpha-adrenergic receptor sites on the smooth muscle of the nasal mucosal blood vessels, reducing local blood flow, fluid exudation, and mucosal edema. Topical ophthalmic drops are used for symptomatic relief of eye irritations due to allergies. Potential side effects include hypertension, dysrhythmias, palpitations, central nervous system stimulation, irritability, tremor, and tachyphylaxis (acceleration of hemodynamic status). Examples of adrenergic decongestants and their routes of administration are found in Table 53-3.
hay fever, medication-induced allergies, and allergic reactions to infections of the lungs should avoid inhaled corticosteroids. Whenever possible, patients with tuberculosis and other bacterial infections of the lungs may become apparent and progress. Into the upper respiratory tract, tuberculosis or untreated bacterial infections (tuberculosis or untreated bacterial infections) are usually mild. Adverse effects (ie, sneezing, local stinging, and burning sensations) are usually mild.

**Corticosteroids.** Intranasal corticosteroids are indicated in more severe cases of allergic and perennial rhinitis that cannot be controlled by more conventional medications such as decongestants, antihistamines, and intranasal cromolyn. These medications include beclomethasone (Beconase, Vancenase), budesonide (Rhinocort), dexamethasone (Decadron Phosphate Turbinaire), flunisolide (Nasalide), fluticasone (Cutivate, Flonase), and triamcinolone (Nasacort).

Because of their anti-inflammatory actions, these medications are equally effective in preventing or suppressing the major symptoms of allergic rhinitis. Corticosteroids are administered by metered-spray devices. If the nasal passages are blocked, a topical decongestant can be used to clear the passages before the administration of the intranasal corticosteroid. Patients must be aware that full benefit may not be achieved for several days to 2 weeks. Adverse effects of intranasal corticosteroids are mild and include drying of the nasal mucosa and burning and itching sensations caused by the vehicle used to administer the medication. Systemic effects are more likely with dexamethasone. Recommended use of this medication is limited to 30 days. Beclomethasone, budesonide, flunisolide, fluticasone, and triamcinolone are deactivated rapidly after absorption, so they do not achieve significant blood levels. Corticosteroids suppress host defenses, so they must be used with caution in patients with tuberculosis or untreated bacterial infections of the lungs. Patients on corticosteroids are at risk for infection and for suppression of typical manifestations of inflammation because host defenses are compromised. Inhaled corticosteroids do not affect the immune system to the same degree as systemic corticosteroids (ie, oral corticosteroids). As corticosteroids are inhaled into the upper respiratory tract, tuberculosis or untreated bacterial infections of the lungs may become apparent and progress. Whenever possible, patients with tuberculosis and other bacterial infections of the lungs should avoid inhaled corticosteroids.

Oral and parenteral corticosteroids are used when conventional therapy has failed and symptoms are severe and of short duration. They can control symptoms of allergic reactions such as hay fever, medication-induced allergies, and allergic reactions to insect stings. Because the response to corticosteroids is delayed, they have little or no value in acute therapy for severe reactions such as anaphylaxis. Patients who receive corticosteroids must be cautioned not to stop taking the medication suddenly or without specific instructions from the physician. The patient is also instructed about side effects, which include fluid retention, weight gain, hypertension, gastric irritation, glucose intolerance, and adrenal suppression. Further discussion of corticosteroids is provided in Chapter 42.

**IMMUNOTHERAPY**

Allergen desensitization (allergen immunotherapy, hyposensitization) is primarily used to treat IgE-mediated diseases by injections of allergen extracts. This type of therapy provides an adjunct to symptomatic pharmacologic therapy and can be used when allergen avoidance is not possible (Parslow et al., 2001). Specific immunotherapy has been used in the treatment of allergic disorders for about 100 years. It consists of administering increasing concentrations of extracts of specific allergens over a long period (Kay, 2001b). Goals of immunotherapy include reducing the level of circulating IgE, increasing the level of blocking antibody IgG, and reducing mediator cell sensitivity. Immunotherapy has been most effective for ragweed pollen; however, treatment for grass, tree pollen, cat, and house dust mite allergens has also been effective.

Correlation of a positive skin test with a positive allergy history is an indication for immunotherapy if the allergen cannot be avoided. The value has been fairly well established in instances of allergic rhinitis and bronchial asthma that are clearly due to sensitivity to one of the common pollens, molds, and household dust. Although helpful in most patients, immunotherapy does not cure the condition. Before immunotherapy is initiated, the patient must understand what to expect and the importance of continuing therapy for several years. When skin tests are performed, the results are correlated with clinical manifestations; treatment is based on the patient’s needs rather than on skin tests.

The most common method of treatment is the serial injection of one or more antigens that are selected in each particular case on the basis of skin tests. This method provides a simple and efficient technique for identifying IgE antibodies to specific antigens. Specific treatment consists of injecting extracts of the pollens or mold spores that cause symptoms in a particular patient. Injections begin with very small amounts and are gradually increased, usually at weekly intervals, until a maximum tolerated dose is attained. Maintenance booster injections are given at 2- to 4-week intervals, frequently for a period of several years, before maximum benefit is achieved.

### Table 53-3 • Adrenergic Decongestants and Their Routes of Administration

<table>
<thead>
<tr>
<th>ADRENERGIC DECONGESTANT</th>
<th>TRADE NAME</th>
<th>ROUTE OF ADMINISTRATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>naphazoline hydrochloride</td>
<td>Privine</td>
<td>Topical</td>
</tr>
<tr>
<td>oxymetazoline hydrochloride</td>
<td>Afrin, Dristan long-lasting, Neo-Synephrine 12 hour, Sinex long-lasting</td>
<td>Topical</td>
</tr>
<tr>
<td>phenylephrine hydrochloride</td>
<td>Neo-Synephrine</td>
<td>Topical</td>
</tr>
<tr>
<td>pseudoephedrine hydrochloride</td>
<td>Sudafed</td>
<td>Oral</td>
</tr>
<tr>
<td>tetrahydrozoline hydrochloride</td>
<td>Collyrium, Murine Plus, Visine</td>
<td>Topical ophthalmic and nasal preparations</td>
</tr>
<tr>
<td>xylometazoline hydrochloride</td>
<td>Neo-Synephrine II, Otrivin</td>
<td>Topical</td>
</tr>
</tbody>
</table>
There are three methods of injection therapy: coseasonal, pre-
seasonal, and perennial. When treatment is given on a coseasonal
basis, therapy is initiated during the season in which the patient
experiences symptoms. This method has been proved ineffective
and is used infrequently. Also, there is an increased risk of sys-
temic reactions. Preseasonal therapy injections are given 2 to
3 months before symptoms are expected, allowing time for hypo-
sensitization to occur. This treatment is discontinued after the
season begins. Perennial therapy is administered all year round,
usually on a monthly basis, and is the preferred method because
it has more effective, longer-lasting results.

Any patient who receives specific immunotherapy is at risk for
general, and potentially fatal, anaphylaxis. This occurs most fre-
quently at the induction or “up-dosing” phase. Attempts have
been made to minimize the risk of systemic reactions by pre-
treating allergen extracts with agents such as formaldehyde. This
approach decreases the binding of the allergen by IgE, but it also
results in decreased immunogenicity (Kay, 2001b).

Because of the risk for anaphylaxis, injections should not be
given by a lay person or by the patient. The patient remains in the
office or clinic for at least 30 minutes after the injection and is ob-
served for possible systemic symptoms. If a large, local swelling de-
velops at the injection site, the next dose should not be increased,
because this may be a warning sign of a possible systemic reaction.

Therapeutic failure is evident when a patient does not experi-
ence a decrease of symptoms within 12 to 24 months, fails to de-
velop increased tolerance to known allergens, and cannot decrease
the use of medications to reduce symptoms. Potential causes of
treatment failure include misdiagnosis of allergies, inadequate
doses of allergen, newly developed allergies, and inadequate en-
vironmental controls.

**NURSING ALERT** Because the injection of an allergen may in-
duce systemic reactions, such injections are given only in a setting
(i.e., physician’s office, clinic) where epinephrine is immediately
available.

**NURSING PROCESS:**
**THE PATIENT WITH ALLERGIC RHINITIS**

**Assessment**

The examination and history of the patient reveal sneezing, often
in paroxysms, thin and watery nasal discharge, itching eyes and
nose, lacrimation, and occasionally headache. The health history
includes a personal or family history of allergy. The allergy as-

ssessment identifies the nature of antigens, seasonal changes in
symptoms, and medication history. The nurse also obtains sub-
jective data about how the patient feels just before symptoms be-
come obvious, such as the occurrence of pruritus, breathing
problems, and tingling sensations. In addition to these symp-
toms, hoarseness, wheezing, hives, rash, erythema, and edema are
noted. Any relationship between emotional problems or stress and
the triggering of allergy symptoms is assessed.

**Diagnosis**

**NURSING DIAGNOSES**

Based on the assessment data, the patient’s major nursing diag-
noses may include the following:

- Ineffective breathing pattern related to allergic reaction
- Deficient knowledge about allergy and the recommended
  modifications in lifestyle and self-care practices
- Ineffective individual coping with chronicity of condition
  and need for environmental modifications

**COLLABORATIVE PROBLEMS/
POTENTIAL COMPLICATIONS**

Based on assessment data, potential complications may include the following:

- Anaphylaxis
- Impaired breathing
- Nonadherence to the therapeutic regimen

**Planning and Goals**

The goals for the patient may include restoration of normal
breathing pattern, increased knowledge about the causes and con-


tral of allergic symptoms, improved coping with alterations and
modifications, and absence of complications.

**Nursing Interventions**

**IMPROVING BREATHING PATTERN**

The patient is instructed and assisted to modify the environment
to reduce the severity of allergic symptoms or to prevent their oc-
currence. The patient is instructed to reduce exposure to people
with upper respiratory infections (URIs). If a URI occurs, the pa-
tient is encouraged to take deep breaths and cough frequently
to ensure adequate gas exchange and prevent atelectasis. The patient
is instructed to seek medical attention because allergy symptoms
along with a URI may compromise adequate lung function.

Compliance with medications and other treatment regimens is
encouraged and reinforced.

**PROMOTING UNDERSTANDING OF ALLERGY
AND ALLERGY CONTROL**

Instruction includes strategies to minimize exposure to allergens,
desensitization procedures, and correct use of medications. The
nurse informs and reminds the patient of the importance of keep-
ing appointments for desensitization procedures because usually
dosages are adjusted on a weekly basis, and missed appointments
may interfere with the dosage adjustment.

Patients also need to understand that medications for allergy
control should be used only when the allergy is apparent. This is
usually on a seasonal basis. Continued use of medications
when not required may cause an increased tolerance to the med-
ication, with the result that the medication is not effective when
needed.

**COPING WITH A CHRONIC DISORDER**

Although allergic reactions are infrequently life-threatening, they
require constant vigilance to avoid allergens and modification of
the lifestyle or environment to prevent recurrence of symptoms.
Allergic symptoms are often present year-round and create dis-
comfort and inconvenience for the patient. Although patients
may not feel ill during allergy seasons, they often do not feel well
either. The need to be alert for possible allergens in the environ-
ment may be tiresome, placing a burden on the patient’s ability
to lead a normal life. Stress related to these difficulties may in turn
increase the frequency or severity of symptoms.

To assist the patient in adjusting to these modifications, the
nurse must have an appreciation of the difficulties encountered
by the patient. The patient is encouraged to verbalize feelings and concerns in a supportive environment and to identify strategies to deal with them effectively.

**MONITORING AND MANAGING POTENTIAL COMPLICATIONS**

**Anaphylaxis and Impaired Breathing**
Respiratory and cardiovascular functioning can be significantly altered during allergic reactions by the reaction itself or by the medications used to treat reactions. The respiratory status is evaluated by monitoring the respiratory rate and pattern and by assessing for breathing difficulties or abnormal lung sounds. The pulse rate and rhythm and blood pressure are monitored to assess cardiovascular status regularly or any time the patient reports symptoms such as itching or difficulty breathing. In the event of signs and symptoms suggestive of anaphylaxis, emergency medications and equipment must be available for immediate use.

**Nonadherence to Therapeutic Regimen**
Knowing about the treatment regimen does not ensure adherence. Having the patient identify potential barriers and explore acceptable solutions for effective management of the condition (eg, installing tile floors rather than carpet, not gardening in the spring) can increase adherence to the treatment regimen.

**PROMOTING HOME AND COMMUNITY-BASED CARE**

**Teaching Patients Self-Care**
The patient is instructed about strategies to minimize exposure to allergens, the actions and adverse effects of medications, and the correct use of medications. The patient should know the names, dose, frequency, actions, and side effects of all medications taken.

Instruction about strategies to control allergic symptoms is based on the needs of the patient as determined by the results of tests, the severity of symptoms, and the motivation of the patient and family to deal with the condition. Suggestions for patients sensitive to dust and mold in the home are given in Chart 53-5.

If the patient is to undergo immunotherapy, the nurse reinforces the physician’s explanation regarding the purpose and procedure. Instructions are given regarding the series of injections, usually given initially every week and then at 2- to 4-week intervals. These instructions include remaining in the physician’s office or the clinic at least 30 minutes after the injection so that emergency treatment may be given if the patient has a reaction; avoiding rubbing or scratching the injection site; and continuing with the series for the period of time required. In addition, the patient and family are instructed about emergency treatment of severe allergic symptoms.

Because antihistamines often produce drowsiness, the patient is cautioned about this and other side effects of the particular medication. Operating machinery, driving a car, and performing activities requiring intense concentration should be postponed. The patient is also informed about the dangers of drinking alcohol when taking these medications because they tend to exaggerate the effects of alcohol.

The patient must be aware of the effects caused by overuse of the sympathomimetic agents in nose drops or sprays. A condition referred to as rhinitis medicamentosa may result (Fig. 53-6). After topical application of the medication, a rebound period may occur in which the nasal mucous membranes become more edematous and congested than they were before the medication was used. Such a reaction encourages the use of more medication, and a cyclical pattern results. The topical agent must be discontinued immediately and completely to correct this problem.

---

**Chart 53-5**

**Home Care Checklist — Allergy Management**

<table>
<thead>
<tr>
<th>At the completion of home care instruction, the patient or caregiver will be able to:</th>
<th>Patient</th>
<th>Caregiver</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Verbalize how to maintain a dust-free environment by removing drapes, curtains, and venetian blinds and replacing them with pull shades; covering the mattress with a hypoallergenic cover that can be zipped; and removing rugs and replacing them with wood flooring or linoleum.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Identify rationale for washing the floor and dusting and vacuuming daily.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Identify rationale for replacing stuffed furniture with wood pieces that can easily be dusted.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• State rationale for wearing a mask whenever cleaning is being done.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Identify rationale for avoiding use of tufted bedspreads, stuffed toys, and feather pillows and replacing them with washable cotton material.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• State rationale for avoiding the use of any clothing that causes itching.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Verbalize ways to reduce dust in the house as a whole by using steam or hot water for heating rather than air and using air filters or air conditioning.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Verbalize ways to reduce exposure to pollens or molds by identifying seasons of the year when pollen counts are high; wearing a mask at times of increased exposure (windy days and when grass is being cut); and avoiding contact with weeds, dry leaves, and freshly cut grass.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• State rationale for seeking air-conditioned areas at the height of the allergy season.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• State rationale for avoiding sprays and perfumes.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• State rationale for use of hypoallergenic cosmetics.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• State rationale for taking prescribed medications as ordered.</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Identify specific foods that may cause allergic symptoms. (Examples of foods that can cause allergic reactions are fish, nuts, eggs, and chocolate.)</td>
<td>✓</td>
<td>✓</td>
</tr>
<tr>
<td>• Develop a list of foods to avoid.</td>
<td>✓</td>
<td>✓</td>
</tr>
</tbody>
</table>
Continuing Care

Follow-up telephone calls to the patient are often reassuring to the patient and family and provide an opportunity for the nurse to answer any questions. The patient is reminded to keep follow-up appointments and is informed about the importance of continuing with treatment. The importance of participating in health promotion activities and health screening is emphasized to the patient.

Evaluation

EXPECTED PATIENT OUTCOMES

Expected patient outcomes may include:

1. Exhibits normal breathing patterns
   a. Demonstrates lungs clear on auscultation
   b. Exhibits absence of adventitious breath sounds (crackles, rhonchi, wheezing)
   c. Has a normal respiratory rate and pattern
   d. Reports no complaints of respiratory distress (shortness of breath, difficulty on inspiration or expiration)
2. Demonstrates knowledge about allergy and strategies to control symptoms
   a. Identifies causative allergens, if known
   b. States methods of avoiding allergens and controlling indoor and outdoor precipitating factors
   c. Removes from the environment items that retain dust
   d. Wears a dampened mask if dust or mold may be a problem
   e. Avoids smoke-filled rooms and dust-filled or freshly sprayed areas
   f. Uses air conditioning for a major part of the day
   g. Takes antihistamines as prescribed; participates in hypo-sensitization program, if applicable
   h. Describes name, purpose, side effects, and method of administration of prescribed medications
   i. Identifies when to seek immediate medical attention for severe allergic responses
   j. Describes activities that are possible, including ways to participate in activities without activating the allergies
3. Experiences relief of discomfort while adapting to the inconveniences of an allergy
   a. Relates the emotional aspects of the allergic response
   b. Demonstrates use of measures to cope positively with allergy
4. Absence of complications
   a. Exhibits vital signs within normal limits
   b. Reports no symptoms or episodes of anaphylaxis (urticaria, itching, peripheral tingling, fullness in the mouth and throat, flushing, or difficulty swallowing) or coughing, wheezing, or difficulty breathing
   c. Demonstrates correct procedure to self-administer emergency medications to treat severe allergic reaction
   d. Correctly states medication names, dose and frequency of administration, and medication actions
   e. Correctly identifies side effects and untoward signs and symptoms to report to physician
   f. Discusses acceptable lifestyle changes and solutions for identified potential barriers for compliance with treatment and medication regimen

CONTACT DERMATITIS

Contact dermatitis (dermatitis venenata), a type IV delayed hypersensitivity reaction, is an acute or chronic skin inflammation that results from direct skin contact with chemicals or allergens. There are four basic types: allergic, irritant, phototoxic, and photoallergic (Table 53-4). Eighty percent of cases are due to excessive exposure to or additive effects of irritants (eg, soaps, detergents, organic solvents) (Tierney et al., 2001). Skin sensitivity may develop after brief or prolonged periods of exposure, and the clinical picture may appear hours or weeks after the sensitized skin has been exposed.

Clinical Manifestations

Symptoms include itching, burning, erythema, skin lesions (vesicles), and edema, followed by weeping, crusting, and finally drying and peeling of the skin. In severe responses, hemorrhagic bullae may develop. Repeated reactions may be accompanied by thickening of the skin and pigmented changes. Secondary invasion by bacteria may develop in skin abraded by rubbing or scratching. Usually, there are no systemic symptoms unless the eruption is widespread.

Assessment and Diagnostic Findings

The location of the skin eruption and the history of exposure aid in determining the condition. In cases of obscure irritants or an unobservant patient, however, diagnosis may be extremely difficult, often involving many trial-and-error procedures before the cause is determined. Patch tests on the skin with suspected offending agents may clarify the diagnosis.

ATOPIC DERMATITIS

Atopic dermatitis is a type I immediate hypersensitivity disorder. A family history is common. The incidence of atopic dermatitis is highest in infants and children. Atopic dermatitis (eczema) affects 10% to 20% of children in Western populations (Kay, 2001b). Most patients have significant elevations of serum IgE and peripheral eosinophilia. Pruritus and hyperirritability of the skin are the most consistent features of atopic dermatitis and are related to large amounts of histamine in the skin. Excessive dryness of the skin with resultant itching is related to changes in lipid content, sebaceous gland activity, and sweating. In response to stroking of the skin, immediate redness appears on the skin and is followed in 15 to 30 seconds by pallor, which persists for
Lesions develop secondary to the trauma of scratching and appear in areas of increased sweating and hypervascularity.

Atopic dermatitis is chronic, with remissions and exacerbations. This condition has a tendency to recur, with remission from adolescence to age 20 (Tierney et al., 2001). Treatment must be individualized.

### Medical Management

Guidelines for treatment include decreasing itching and scratching by wearing cotton fabrics, washing with a mild detergent, humidifying dry heat in winter, maintaining room temperature at 20°C to 22.2°C (68°F to 72°F), using antihistamines such as diphenhydramine (Benadryl), and avoiding animals, dust, sprays, and perfumes. Keeping the skin moisturized with daily baths to hydrate the skin and topical skin moisturizers is encouraged. Topical corticosteroids are used to prevent inflammation, and any infection is treated with antibiotics to eliminate *Staphylococcus aureus* when indicated. Use of low doses of cyclosporine (Neoral, Sandimmune), an immunosuppressive agent, may be effective (Kay, 2001b).

### Nursing Management

Patients who experience atopic dermatitis and their families require assistance and support from the nurse to cope with the disorder. The symptoms are often disturbing to the patient and disruptive to the family. The appearance of the skin may affect the patient’s self-esteem and may affect the patient’s willingness to interact with others. Instructions and counseling about strategies to incorporate preventive measures and treatments into the lifestyle of the family may be helpful.

Patients and family members need to be aware of signs of secondary infection and of the need to seek treatment if infection occurs. The nurse also teaches the patient and family about the side effects of medications used in treatment.

### Dermatitis Medicamentosa (Drug Reactions)

Dermatitis medicamentosa, a type I hypersensitivity disorder, is the term applied to skin rashes induced by the internal administration of certain medications. Although individuals react differently to each medication, certain medications tend to induce eruptions of similar types. Rashes are among the most common adverse reactions to medications and occur in approximately 2% to 3% of hospitalized patients (Tierney et al., 2001).

In general, drug reactions appear suddenly, have a particularly vivid color, present with characteristics that are more intense than the somewhat similar eruptions of infectious origin, and, with the exception of bromide and the iodide rashes, disappear rapidly after the medication is withdrawn. Rashes may be accompanied by systemic or generalized symptoms. Upon discovery of a medication allergy, patients are warned that they have a hypersensi-

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### Table 53-4 • Types, Testing, and Treatment of Contact Dermatitis

<table>
<thead>
<tr>
<th>Type</th>
<th>Etiology</th>
<th>Clinical Presentation</th>
<th>Diagnostic Testing</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergic</td>
<td>Results from contact of skin and allergenic substance. Has a sensitization period of 10–14 days.</td>
<td>Vasodilation and perivascular infiltrates on the dermis Intracellular edema Usually seen on dorsal aspects of hand</td>
<td>Patch testing (contraindicated in acute, widespread dermatitis)</td>
<td>Avoidance of offending material Burow’s solution or cool water compress Systemic corticosteroids (prednisone) for 7–10 days Topical corticosteroids for mild cases Oral antihistamines to relieve pruritus Identification and removal of source of irritation Application of hydrophilic cream or petrolatum to soothe and protect Topical corticosteroids and compresses for weeping lesions Antibiotics for infection and oral antihistamines for pruritus</td>
</tr>
<tr>
<td>Irritant</td>
<td>Results from contact with a substance that chemically or physically damages the skin on a nonimmunologic basis. Occurs after first exposure to irritant or repeated exposures to milder irritants over an extended time.</td>
<td>Dryness lasting days to months Vesiculation, fissures, cracks Hands and lower arms most common areas</td>
<td>Clinical picture Appropriate negative patch tests</td>
<td>Same as for allergic and irritant dermatitis</td>
</tr>
<tr>
<td>Phototoxic</td>
<td>Resembles the irritant type but requires sun and a chemical in combination to damage the epidermis.</td>
<td>Similar to irritant dermatitis</td>
<td>Photopatch test</td>
<td>Same as for allergic and irritant dermatitis</td>
</tr>
<tr>
<td>Photoallergic</td>
<td>Resembles allergic dermatitis but requires light exposure in addition to allergen contact to produce immunologic reactivity.</td>
<td>Similar to allergic dermatitis</td>
<td>Photopatch test</td>
<td>Same as for allergic and irritant dermatitis</td>
</tr>
</tbody>
</table>
tivity to a particular medication and are advised not to take it again. Information identifying the hypersensitivity should be carried with them at all times.

Skin eruptions related to medication therapy suggest more serious hypersensitivities. Frequent assessment and prompt reporting of the appearance of any eruptions are important so that early treatment can be initiated. Some cutaneous drug reactions may be associated with a clinical complex that involves other organs. These are known as complex drug reactions (Tierney et al., 2001).

URTICARIA AND ANGIONEUROTIC EDEMA

Urticaria (hives) is a type I hypersensitive allergic reaction of the skin characterized by the sudden appearance of pinkish, edematous elevations that vary in size and shape and itch and cause local discomfort. They may involve any part of the body, including the mucous membranes (especially those of the mouth), the larynx (occasionally with serious respiratory complications), and the gastrointestinal tract.

Each hive remains for a few minutes to several hours before disappearing. For hours or days, clusters of these lesions may come, go, and return episodically. If this sequence continues for longer than 6 weeks, the condition is called chronic urticaria (Tierney et al., 2001).

Angioneurotic edema involves the deeper layers of the skin, resulting in more diffuse swelling rather than the discrete lesions characteristic of hives. On occasion, this reaction covers the entire back. The skin over the reaction may appear normal but often has a reddish hue. The skin does not pit on pressure, as ordinary edema does. The regions most often involved are the lips, eyelids, cheeks, hands, feet, genitalia, and tongue; the mucous membranes of the larynx, the bronchi, and the gastrointestinal canal may also be affected, particularly in the hereditary type (see section that follows). Swellings may appear suddenly, in a few seconds or minutes, or slowly, in 1 or 2 hours. In the latter case, their appearance is often preceded by itching or burning sensations. Seldom does more than a single swelling appear at one time, although one may develop while another is disappearing. Infrequently, swelling recurs in the same region. Individual lesions usually last 24 to 36 hours. On rare occasions, swelling may recur with remarkable regularity at intervals of 3 to 4 weeks.

HEREDITARY ANGIOEDEMA

Hereditary angioedema, although not an immunologic disorder in the usual sense, is included because of its resemblance to allergic angioedema and because of the seriousness of the condition. Symptoms are due to edema of the skin, the respiratory tract, or the digestive tract. Attacks may be precipitated by trauma or may seem to occur spontaneously.

Clinical Manifestations

When skin is involved, the swelling is usually diffuse, does not itch, and is usually not accompanied by urticaria. Gastrointestinal edema may cause abdominal pain severe enough to suggest the need for surgery. Typically, attacks last 1 to 4 days and are generally harmless. Occasionally, attacks affect the subcutaneous and submucosal tissues in the region of the upper airway and can be associated with respiratory obstruction and asphyxiation. This disorder is inherited as an autosomal dominant trait. Approximately 85% of patients with this disorder have one nonproductive gene; the other 15% have a gene mutation (Parslow et al., 2001).

Medical Management

Attacks usually subside within 3 to 4 days, but during this time the patient should be observed carefully for signs of laryngeal obstruction, which may necessitate tracheostomy as a life-saving measure. Epinephrine, antihistamines, and corticosteroids are usually used in treatment, but their success is limited.

FOOD ALLERGY

IgE-mediated food allergy, a type I hypersensitivity reaction, occurs in 0.1% to 7.0% of the population. Almost any food can cause allergic symptoms. Any food can contain an allergen that results in anaphylaxis. The most common offenders are seafood (lobster, shrimp, crab, clams, fish), legumes (peanuts, tree nuts, peas, beans, licorice), seeds (sesame, cottonseed, caraway, mustard, flaxseed, and sunflower seeds), nuts, berries, egg white, buckwheat, milk, and chocolate (Parslow et al., 2001). Peanut and tree nut (ie, cashew, walnut) allergies are responsible for most severe food allergy reactions (Sicherer, Munoz-Furlong, Burks et al., 1999).

One of the dangers of food allergies is that they may be hidden in other foods and not apparent to those susceptible to the allergen. For example, peanuts and peanut butter are often used in salad dressings and Asian, African, and Mexican cooking and may result in severe allergic reactions, including anaphylaxis. Previous contamination of equipment with allergens (ie, peanuts) during preparation of another food product (ie, chocolate cake) is enough to produce anaphylaxis in those with severe allergy.

Clinical Manifestations

Clinical symptoms are classic allergic symptoms (urticaria, atopic dermatitis, wheezing, cough, laryngeal edema, angioedema) and gastrointestinal symptoms (itching; swelling of lips, tongue, and palate; abdominal pain; nausea; cramps; vomiting; and diarrhea).

Assessment and Diagnostic Findings

A careful diagnostic workup is required in any patient with a suspected food hypersensitivity. Included are a detailed allergy history, a physical examination, and pertinent diagnostic tests. When testing for allergy, skin testing is used to identify the source of symptoms and is useful in identifying specific foods as causative agents.

Medical Management

Therapy for food hypersensitivity includes elimination of the food responsible for the hypersensitivity (Chart 53-6). Pharmacologic therapy is necessary in patients who cannot avoid exposure to offending foods or patients with multiple food sensitivities not responsive to elimination measures. Medication therapy involves the use of H1- and H2-blockers, antihistamines, adrenergic agents, corticosteroids, and cromolyn sodium.
Many food allergies disappear with time, particularly in children. About one third of proven allergies disappear in 1 to 2 years if the patient carefully avoids the offending food.

**Nursing Management**

In addition to participating in management of the allergic reaction, the nurse focuses on preventing future exposure of the patient to the food allergen. If a severe allergic or anaphylactic reaction to food allergens has occurred, the nurse must instruct the patient and family about strategies to prevent its recurrence. The patient is instructed about the importance of carefully assessing food prepared by others as well as hidden sources of food allergens and of avoiding locations and facilities where those allergens are likely to be present. The patient and family must be knowledgeable about early signs and symptoms of allergic reactions and must be proficient in emergency administration of epinephrine if a reaction occurs. The nurse also advises the patient to wear a medical alert bracelet or to carry identification and emergency equipment at all times.

**SERUM SICKNESS**

The illness known as serum sickness is an example of an immune complex type III hypersensitivity. It has traditionally resulted from the administration of therapeutic antiserum of animal sources for the treatment or prevention of infectious diseases, such as tetanus, pneumonia, rabies, diphtheria, botulism, and venomous snake and black widow spider bites. With the advent of human antitetanus serum and antibiotics, classic serum sickness is much less common now. However, various medications (primarily penicillin) may cause a serum sickness–like reaction similar to that caused by foreign sera.

**Clinical Manifestations**

Symptoms are due to a reaction and immunologic attack on the serum or medication. Antibodies appear to be of the IgE and IgM classes. Early manifestations, beginning 6 to 10 days after the administration of the medication, include an inflammatory reaction at the site of injection of the medication, followed by regional and generalized lymphadenopathy. There is usually a skin rash, which may be urticarial or purpuric. Joints are frequently tender and swollen. Vasculitis may occur in any organ but is most commonly observed in the kidney, resulting in proteinuria and, occasionally, casts in the urine. There may be mild to severe cardiac involvement. Peripheral neuritis may cause temporary paralysis of the upper extremities or may be widespread, causing Guillain-Barré syndrome.

**Medical Management**

The usual course lasts for several days to a few weeks if untreated, but the patient responds promptly and completely if treated with antihistamines and corticosteroids. Aggressive therapy, including ventilator support, may be necessary if peripheral neuritis and Guillain-Barré syndrome occur.

**Nursing Management**

See Chapter 64 for nursing management of Guillain-Barré syndrome.

**LATEX ALLERGY**

Latex allergy, the allergic reaction to natural rubber proteins, has been implicated in rhinitis, conjunctivitis, contact dermatitis, urticaria, asthma, and anaphylaxis. Latex allergy and hypersensitivity were first reported in 1927 (Parslow et al., 2001). Although the prevalence of latex allergy is unknown, since 1989 the number of cases of latex allergy has steadily increased (Parslow et al., 2001). This increase may be due to the widespread use of latex gloves with implementation of universal and now standard precautions in response to the AIDS epidemic, changes in the manufacturing of gloves to speed the process to meet the increased demand for gloves, and greater awareness about latex allergy and its signs and symptoms.

Natural rubber latex is derived from the sap of the rubber tree (*Hevea brasiliensis*). The conversion of the liquid rubber latex into a finished product entails the addition of more than 200 chemicals. The proteins in the natural rubber latex (*Hevea proteins*) or the various chemicals that are used in the manufacturing process are thought to be the source of the allergic reactions. Not all objects composed of latex have the same ability to stimulate an allergic response. For example, the antigenicity of latex gloves can differ widely depending on their manufacturing method.

Populations at risk include health care workers, patients with atopic allergies or multiple surgeries, people working in factories manufacturing latex products, females, and patients with spina bifida. Because more food handlers, hairdressers, auto mechanics, and police are now wearing latex gloves, they may also be at risk for latex allergy. It is estimated that 1% to 3% of the general population has an allergy to latex and that 10% to 17% of health care workers are sensitized. Patients are at risk for anaphylactic reactions due to contact with latex during medical treatments, especially surgical procedures. About 19% of anaphylactic reactions associated with anesthesia are caused by allergy to latex (Brehler & Küttig, 2001).
Food that has been handled by individuals wearing latex gloves may stimulate an allergic response. Cross-reactions have been reported in people who are allergic to certain food products, such as kiwis, bananas, pineapples, passion fruits, avocados, and chestnuts.

Routes of exposure to latex products can be cutaneous, percutaneous, mucosal, parenteral, and aerosol. The most frequent source of exposure is cutaneous, which usually involves the wearing of natural latex gloves. The powder used to facilitate putting on latex gloves can become a carrier of latex proteins from the gloves; when the gloves are put on or removed, the particles become airborne and can be inhaled or can settle on skin, mucous membranes, or clothing. Mucosal exposure can occur from the use of latex condoms, catheters, airways, and nipples. Parenteral exposure can occur from intravenous lines or hemodialysis equipment. In addition to latex-derived medical devices, many household items also contain latex. Examples of medical and household items containing latex and a list of alternative products are found in Table 53-5. It is estimated that over 40,000 medical devices and nonmedical products contain latex (Brehler & Kütting, 2001).

### Clinical Manifestations

Several different types of reactions to latex are possible. Irritant contact dermatitis, a nonimmunologic response, may be due to mechanical skin irritation or an alkaline pH associated with latex gloves. Common symptoms of irritant dermatitis include erythema and pruritus. These symptoms can be eliminated by changing glove brands or using powder-free gloves. Use of hand lotion before donning latex gloves may worsen the symptoms because lotions may leach latex proteins from the gloves, increasing skin exposure and the risk of developing true allergic reactions (Burt, 1998).

Delayed hypersensitivity to latex, a type IV allergic reaction mediated by T cells in the immune system, is localized to the area of exposure and is characterized by symptoms of contact dermatitis, including vesicular skin lesions, papules, pruritis, edema, erythema, and crusting and thickening of the skin. These symptoms usually appear on the back of the hands. This reaction is thought to be due to chemicals that are used for manufacturing latex products. It is the most common allergic reaction to latex. Although not usually life-threatening, delayed hypersensitivity

<table>
<thead>
<tr>
<th>Table 53-5 • Selected Products Containing Natural Rubber Latex and Latex-Free Alternatives</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRODUCTS CONTAINING LATEX</td>
</tr>
<tr>
<td><strong>Hospital Environment</strong></td>
</tr>
<tr>
<td>Ace bandage (brown)</td>
</tr>
<tr>
<td>Adhesive bandages, Band-Aid dressing, Telfa</td>
</tr>
<tr>
<td>Anesthesia equipment</td>
</tr>
<tr>
<td>Blood pressure cuff, tubing, and bladder</td>
</tr>
<tr>
<td>Catheters</td>
</tr>
<tr>
<td>Catheter leg bag straps</td>
</tr>
<tr>
<td>Crutch axillary pads and hand grips, tips</td>
</tr>
<tr>
<td>ECG pads</td>
</tr>
<tr>
<td>Elastic compression stockings</td>
</tr>
<tr>
<td>Gloves</td>
</tr>
<tr>
<td>IV catheters</td>
</tr>
<tr>
<td>IV rubber injection ports</td>
</tr>
<tr>
<td>Levin tube</td>
</tr>
<tr>
<td>Medication vials</td>
</tr>
<tr>
<td>Penrose drains</td>
</tr>
<tr>
<td>Prepackaged enema kits</td>
</tr>
<tr>
<td>Pulse oximeters</td>
</tr>
<tr>
<td>Resuscitation bags</td>
</tr>
<tr>
<td>Stethoscope tubing</td>
</tr>
<tr>
<td>Syringes—single use (Monoject, B &amp; D)</td>
</tr>
<tr>
<td>Suction tubing</td>
</tr>
<tr>
<td>Tapes</td>
</tr>
<tr>
<td>Thermometer probes</td>
</tr>
<tr>
<td>Tourniquets</td>
</tr>
<tr>
<td>Theraband</td>
</tr>
<tr>
<td><strong>Home Environment</strong></td>
</tr>
<tr>
<td>Balloons</td>
</tr>
<tr>
<td>Diapers, incontinence pads</td>
</tr>
<tr>
<td>Condoms, diaphragms</td>
</tr>
<tr>
<td>Feminine hygiene pad</td>
</tr>
<tr>
<td>Wheelchair cushions</td>
</tr>
</tbody>
</table>

*Confirmation is essential to verify that all items are latex-free before using, especially if risk of latex allergy.
Diagnostic Testing

The diagnosis of latex allergy is based on the history and diagnostic test results (Parslow et al., 2001). Sensitization is detected by skin testing, RAST, or ELISA. Skin tests have been unreliable because of variability in the techniques used; however, a new standardized skin testing reagent is expected to be available in the near future. Skin tests should be done only by clinicians who have expertise in their administration and interpretation and who have the necessary equipment available to treat local or systemic allergic reactions to the reagent (Hamilton & Adkinson, 1998). Nasal challenge and dipstick tests may be useful in the future as screening tests for latex allergy.

Medical Management

The best treatment available for latex allergy is the avoidance of latex products, but this is often difficult because of the widespread use of latex-based products. Patients who have experienced an anaphylactic reaction to latex should be instructed to wear medical identification. Antihistamines and an emergency kit containing epinephrine should be provided to these patients, along with instructions about emergency management of latex allergy symptoms. Patients should be counseled to notify all health care workers as well as local paramedic and ambulance companies about their allergy. Warning labels can be attached to car windows to alert police and paramedics about the driver’s or passenger’s latex allergy in case of a motor vehicle crash. Individuals with latex allergy should be provided with a list of alternative products and referred to local support groups; they are also urged to carry their own supply of nonlatex gloves.

People with type I latex sensitivity may be unable to continue to work if a latex-free environment is not possible. This may occur with surgeons, dentists, operating room personnel, or intensive care nurses. Occupational implications for employees with type IV latex sensitivity are usually easier to manage by changing to nonlatex gloves and avoiding direct contact with latex-based medical equipment. Although latex-specific immunotherapy has been reported, this method of treatment remains experimental (Brehler & Küttig, 2001).

Nursing Implications

The research findings suggest that nitrile medical examination gloves are an acceptable alternative to latex gloves and that vinyl and copolymer gloves provide less protection to the wearer. These findings suggest the need for health care providers and facilities to use care in selecting the type and manufacturer of gloves to protect the wearer during patient care. The researchers indicated that studies using larger samples are warranted and should address other variables that may affect the barrier effectiveness of gloves during use, including duration of use, presence of powder, glove size, hand dominance, complexity of tasks performed while wearing gloves, and the use of instruments.
those at particularly high risk (eg, patients with spina bifida, patients who have undergone multiple surgical procedures). Every time an invasive procedure must be performed, the nurse should consider the possibility of latex allergies. Nurses working in operating rooms, intensive care units, short procedure units, and emergency departments need to pay particular attention to latex allergy. See Chapter 19 for a latex allergy screening form.

Although the type I reaction is the most significant of the reactions to latex, care must be taken in the presence of irritant contact dermatitis and delayed hypersensitivity reaction to avoid further exposure of the individual to latex. Patients with latex allergy are advised to notify their health care providers and to wear a medical information bracelet. Patients must become knowledgeable about what products contain latex and what products are safe, nonlatex alternatives. They must also become knowledgeable about signs and symptoms of latex allergy and emergency treatment and self-injection of epinephrine in case of allergic reaction.

Nurses can be instrumental in establishing and participating in multidisciplinary committees to address latex allergy and to promote a latex-free environment. Latex allergy protocols and education of staff about latex allergy and precautions are important strategies to reduce this growing problem and to ensure assessment and prompt treatment of affected individuals.

New Approaches to Treatment of Allergic Diseases

Although allergen-specific immunotherapy reduces symptoms for several years after it is discontinued, this approach to management is limited in terms of usefulness because of its potential adverse effects, particularly anaphylaxis, and the relatively crude allergen extracts involved. Newer approaches to the treatment of allergic diseases to overcome these limitations are being evaluated and include the use of substances such as naturally occurring isoforms of allergens from plants and trees. These isoforms are less likely to stimulate anaphylactic reactions. Use of recombinant allergens is expected to eliminate variation between batches of allergen. Other experimental approaches include the use of DNA vaccines and monoclonal antibodies and other strategies to block IgE or its synthesis (Kay, 2001b).

Critical Thinking Exercises

1. A 45-year-old man arrives at the emergency department where he states that he is severely allergic to bees and was stung by a wasp approximately 10 minutes ago. He tells you that he has been in anaphylactic shock in the past as a result of bee stings. He tells you that he received allergy injections for bee allergy approximately 10 years ago. He tells you that he has been in anaphylactic shock in the past as a result of bee stings. Upon assessing the patient, you note that he has generalized hives and urticaria on his body, and he is complaining of his throat swelling. What would be your immediate course of action?

2. A patient with severe allergies is to receive instructions about self-administration of epinephrine if she experiences anaphylaxis. Develop a teaching plan for this patient and identify outcomes to measure the effectiveness of your teaching. How would you modify your teaching if the patient reports a severe fear of injection? If the patient has a profound hearing loss? If the patient is visually impaired?

3. A 28-year-old man has a surgical procedure for a varicocoele. A Penrose drain is inserted into his scrotum during the surgical procedure in the operating room. The patient develops erythema and significant edema of the scrotum inconsistent with the procedure that was performed. The circulating nurse had noted on the operative checklist that the patient was allergic to latex. What would be your immediate course of action? How could you ensure that a similar situation would not occur in the future? What course of action could you take to ensure a latex-free environment for patients who require such an environment?

REFERENCES AND SELECTED READINGS

Books

Journals
Asterisks indicate nursing research articles.


RESOURCES AND WEBSITES

American Academy of Allergy, Asthma and Immunology, 611 East Wells Street, Milwaukee, WI 53202; (800) 822-ASMA; http://www.aaai.org.

Centers for Disease Control and Prevention, 1600 Clifton Road, Atlanta, GA 30333; (404) 639-3311 or (800) 311-3435; http://www.cdc.gov.

Food Allergy & Anaphylaxis Network, 10400 Eaton Place, Suite 107, Fairfax, VA 22030; (800) 929-4040; http://www.foodallergy.org; e-mail: faan@foodallergy.org.

National Institute of Allergy and Infectious Disease, NIAID Office of Communications and Public Liaison, NIH, Bldg. 31, Room 7A50, 31 Center Drive, MSC 2520, Bethesda, MD 20893; (301) 496-5717; http://www.niaid.nih.gov.

National Institutes of Health, 9000 Rockville Pike, Bethesda, MD 20892; (301) 496-4000; http://www.nih.gov.