CHAPTER

44

Alterations in Skin Function and Integrity

The skin is one of the most versatile organs of the body. It forms a protective surface that prevents many harmful substances, including microorganisms, from entering the body; it retards the loss of body fluids; it helps regulate body temperature; and it houses sensory receptors, contains immune system cells, synthesizes chemicals, and excretes small quantities of waste products. The skin is also unique in that the manifestations of disease or injury are immediately observable. It also provides a sensitive reflection of many internal disorders. A number of systemic diseases are manifested by skin disorders (*e.g.*, rash associated with systemic lupus erythematosus and jaundice caused by liver disease).

STRUCTURE OF THE SKIN

The skin is one of the largest organs of the body and accounts for about 7% of total body weight in the average adult. The skin, which is also called the *integument*, which simply means "covering," serves as an interface between the internal and external environments.[1,2] The skin is tough, yet pliable, allowing it to endure the effects of any number of external agents. Although the skin may become bruised, lacerated, burned, or infected, it has remarkable properties that allow for a continuous cycle of healing, shedding, and cell regeneration.

Layers of the Skin

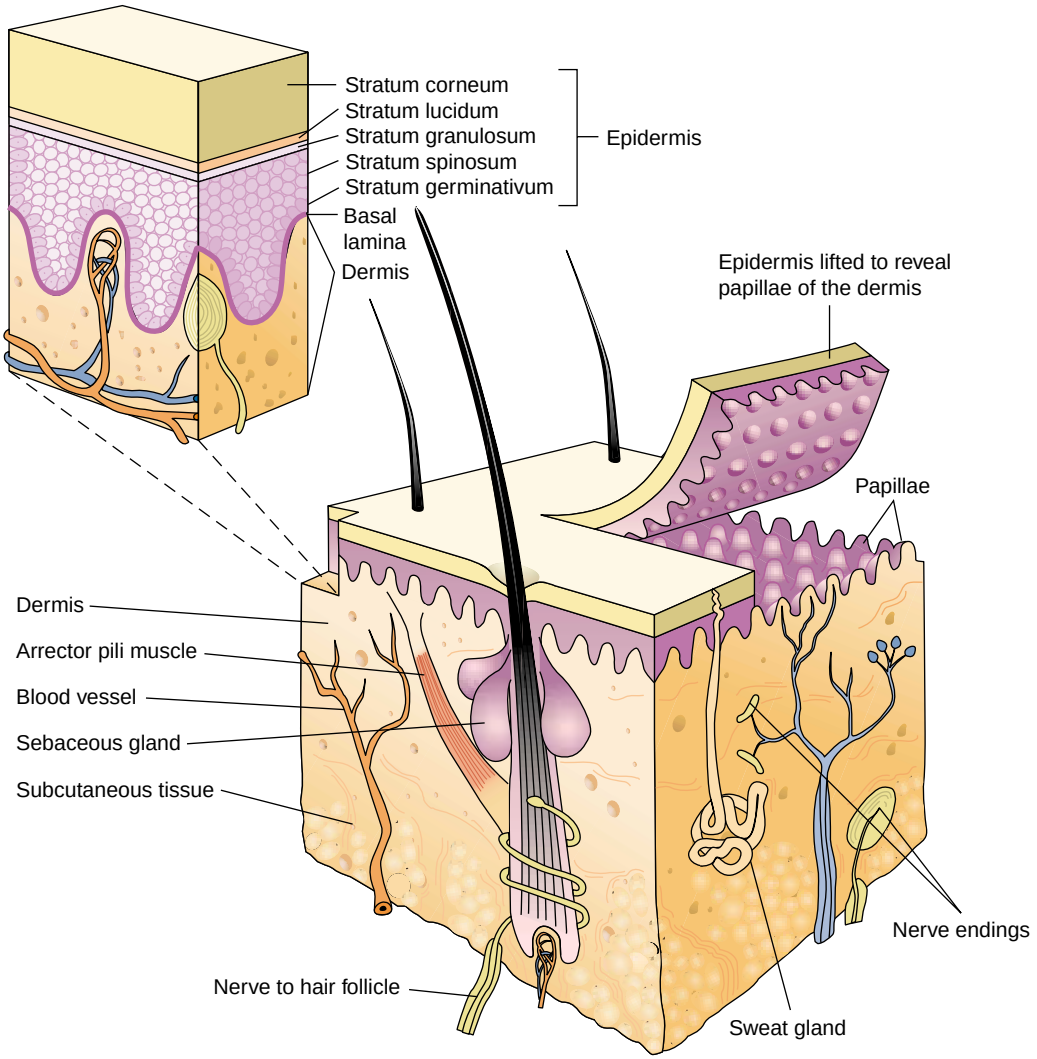
The skin is composed of three rather distinct layers: the epidermis, dermis, and subcutaneous tissues. The layer of subcutaneous tissue binds the dermis to the underlying tissues of the body (Fig. 44-1). The basal lamina (basement membrane) is a layer of intercellular and extracellular matrices that serves as an interface between the dermis and the epidermis. It provides for adhesion of the dermis to the epidermis and serves as a selective filter for molecules moving between the two layers. It is also a major site of immunoglobulin and complement deposition in skin disease. The basal lamina is involved in skin disorders that cause bullae or blister formation.

Epidermis

The epidermis contains the keratinocytes, which produce a fibrous protein called *keratin*, which is essential to the protective function of skin. Because of its high keratin content, the outer layer of the epidermis has a rough, horny texture. In addition to the keratinocytes, the epidermis has three other types of cells that arise from its basal layer: melanocytes that produce a pigment called *melanin*, which is responsible for skin color; Merkel's cells that provide sensory information; and Langerhans' cells that link the epidermis to the immune system. The epidermis contains openings for two types of glands: sweat glands, which produce watery secretions, and sebaceous glands, which produce an oily secretion called *sebum*.

Keratinocytes. The keratinocytes, or keratin-forming cells, are the major cells of the epidermis. They develop into five distinct layers, or strata, as they divide and mature: the stratum germinativum, the stratum spinosum, the stratum granulosum, the stratum lucidum, and the stratum corneum.

The deepest layer, the *stratum germinativum* or *stratum basale*, consists of a single layer of basal cells that are attached to the basal lamina. The basal cells are the only epidermal cells that are mitotically active. All cells of the epidermis arise from this layer. As new cells form in the basal layer, the older cells change shape and are pushed outward (Fig. 44-2). As these cells near the surface, they die and their cytoplasm is converted to keratin. It normally takes 3 to 4 weeks for the epidermis to replicate itself. This cell turnover is greatly accelerated in diseases such as psoriasis.



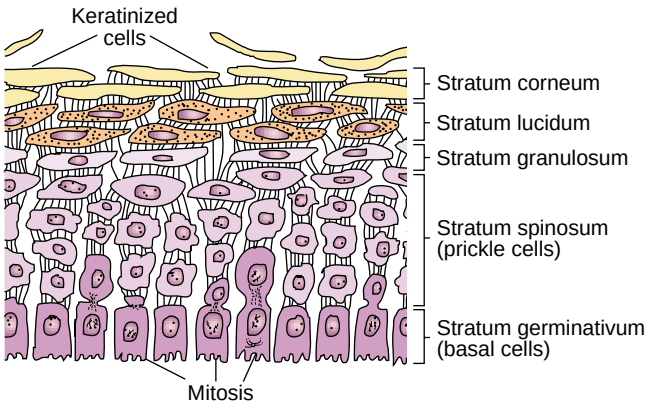
■ **FIGURE 44-1** ■ Three-dimensional view of the skin.

KEY CONCEPTS

SKIN STRUCTURES AND THE MANIFESTATIONS OF SKIN DISORDERS

- The skin has two layers, an outer epidermis and an inner dermis, separated by a basement membrane, all of which can contribute to the development and symptomatology of skin disorders.
- The epidermis, which is avascular, is composed of four to five layers of stratified squamous keratinized epithelial cells that are formed in the deepest layer of the epidermis and migrate to the skin surface to replace cells that are lost during normal skin shedding. The papulosquamous dermatoses, such as psoriasis, involve increased epidermal cell turnover with marked thickening of the epidermis. The avascular layers of the epidermis also serve as a site for superficial fungal infections.
- The basement membrane is a thin adhesive layer that cements the epidermis to the dermis. This is the layer involved in blister formation.
- The dermis is a connective tissue layer that separates the epidermis from the underlying subcutaneous fat layer. It contains the blood vessels that produce hyperemic skin responses and nerve fibers that are the source of pain, discomfort, and itch associated with skin disorders.
- The Langerhans' cells of the epidermis, which bind antigen; the dermal dendrocytes of the dermis, which have phagocytic properties; and immune cells (T cells and mast cells), which are found in the dermis, contribute to the antigen-antibody responses affecting the skin.
- Sebaceous glands in the skin produce an oily secretion called sebum, which is secreted into the hair follicles on the skin. The hair follicle and sebaceous gland form the pilosebaceous unit, which is the site of acne lesions.

The second layer, the *stratum spinosum*, is formed as cells from the basal cell layer move outward toward the skin surface. The stratum spinosum is two to four layers thick. The cells of this layer are commonly referred to as *prickle cells* because they develop a spiny appearance as their cell borders interact. The third layer, the *stratum granulosum* is only a few cells thick; it is composed of flatter cells containing protein granules called *keratohyalin granules*. The *stratum lucidum*, which lies just superficial to the stratum granulosum, is a thin, transparent layer mostly confined to the palms of the hands and soles of the feet. It consists of transitional cells that retain some of the functions of living skin cells from the layers below but otherwise resemble the cells of the stratum corneum.



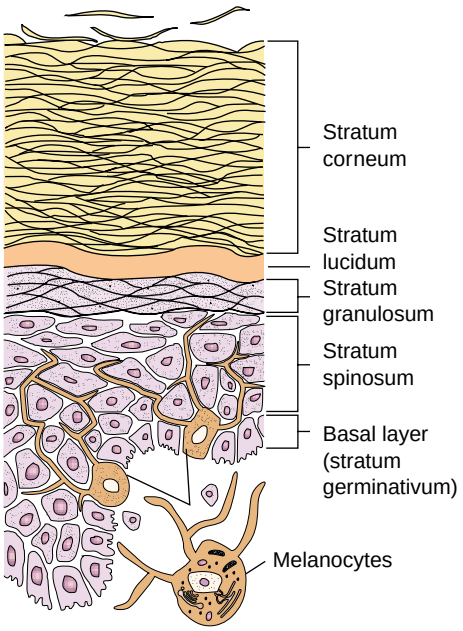
■ **FIGURE 44-2** ■ Epidermal cells. The basal cells undergo mitosis, producing keratinocytes that change their size and shape as they move upward, replacing cells that are lost during normal cell shedding.

The top or surface layer of the epidermis is the *stratum corneum*. It is made up of stratified layers of dead keratinized cells that are constantly shedding. The stratum corneum contains the most cell layers and the largest cells of any zone of the epidermis. It ranges from 15 layers thick in areas such as the face to 25 layers or more on the arm. Specialized areas, such as the palms of the hands or soles of the feet, have 100 or more layers.

Melanocytes. The melanocytes are pigment-synthesizing cells that are located at or in the basal layer. They function to produce pigment granules called *melanin*, the black or brown substance that gives skin its color. The melanocytes have long, cytoplasm-filled extensions that extend between the keratinocytes. Although the melanocytes remain in the basal layer, melanin is transferred to the keratinocytes through these extensions (Fig. 44-3). Each melanocyte is capable of supplying several keratinocytes with melanin. The primary function of melanin is to protect the skin from harmful ultraviolet sun rays. Exposure to the sun's ultraviolet rays increases the production of melanin, causing tanning to occur. The amount of melanin in the keratinocytes determines a person's skin color. All people have relatively few or no melanocytes in the epidermis of the palms of the hands or soles of the feet.

The ability to synthesize melanin depends on the ability of the melanocytes to produce an enzyme called *tyrosinase*, which converts the amino acid tyrosine to a precursor of melanin. A genetic lack of this enzyme results in a clinical condition called *albinism*. Persons with this disorder lack pigmentation in the skin, hair, and iris of the eye.

Langerhans' Cells. Langerhans' cells are star-shaped macrophages of the immune system (see Chapter 8). They arise from the bone marrow and migrate to the epidermis, where they help activate the immune system. Their slender dendritic, or threadlike processes, extend through the keratinocytes in the epidermis, forming a more or less continuous network recognizing foreign antigens (Fig. 44-4). The Langerhans' cells bind antigen to their surface, process it, and then, bearing the processed antigen, migrate from the epidermis into lymphatic vessels and then into regional lymph nodes. They are the only



■ **FIGURE 44-3** ■ Melanocytes. The melanocytes, which are located in the basal layer of the skin, produce melanin pigment granules that give skin its color. The melanocytes have threadlike cytoplasmic-filled extensions that are used in passing the pigment granules to the keratinocytes.

immune cells in the skin known to be capable of antigen-presentation and therefore may be responsible for allergic reactions affecting the skin.

Dermis

The dermis is the connective tissue layer that separates the epidermis from the subcutaneous fat layer. It supports the epidermis and serves as its primary source of nutrition. The two layers of the dermis, the papillary dermis and the reticular dermis, are composed of cells, fibers, ground substances, nerves, and blood vessels. The hair and glandular structures are embedded in this layer and continue through the epidermis.

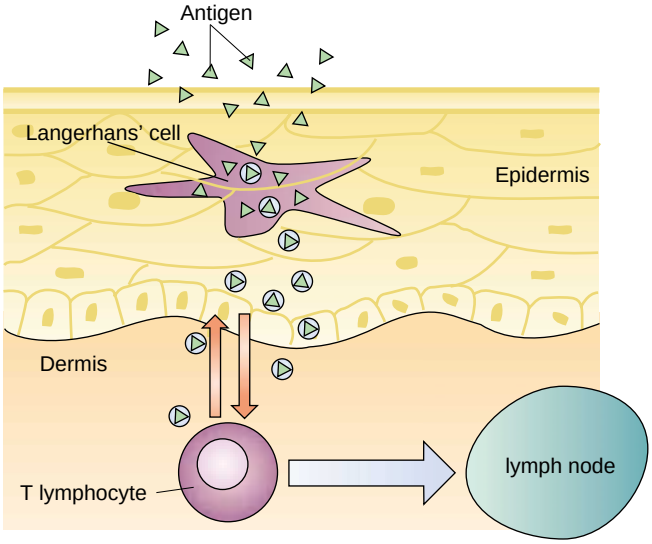
Papillary Dermis. The papillary dermis is a thin, superficial layer that lies adjacent to the epidermis. It consists of collagen fibers and ground substance. This layer is densely covered with conical projections called *dermal papillae* (see Fig. 44-1). The basal cells of the epidermis project into the papillary dermis, forming *rete ridges*. It is believed that the dense structure of the dermal papillae serves to minimize the separation of the dermis and the epidermis. Dermal papillae contain capillary venules that nourish the epidermal layers of the skin. Lymph vessels and nerve tissue also are found in this layer.

Reticular Dermis. The reticular dermis is the thicker area of the dermis and forms the bulk of the dermal layer. The reticular dermis is characterized by a complex meshwork of three-dimensional collagen bundles interconnected with large elastic fibers and ground substance, a viscid gel that is rich in mucopolysaccharides. The collagen fibers are oriented parallel to the body's surface in any given area. Collagen bundles may be organized lengthwise, as on the abdomen, or in round clusters, as in the heel. The direction of surgical incisions is often determined by this organizational pattern.

The reticular dermis also contains dendritic cells with threadlike projections, called *dermal dendrocytes*. Dermal dendrocytes, which have phagocytic properties, are believed to possess antigen-presenting functions and play an important part in the immune function of the skin. Immune cells found in the dermis include macrophages, T cells, mast cells, and fibroblasts. The major type of T-cell–mediated immune response in the skin is delayed-type hypersensitivity (see Chapter 10). The mast cells play a prominent role in IgE-mediated hypersensitivity responses.

Subcutaneous Tissue

The subcutaneous tissue layer consists primarily of loose connective and fatty tissues that lend support to the vascular and neural structures supplying the outer layers of the skin. There is controversy about whether the subcutaneous tissue should be considered an actual layer of the skin. Because the eccrine glands and deep hair follicles extend to this layer and several skin diseases involve the subcutaneous tissue, the subcutaneous tissue may be considered part of the skin.



■ **FIGURE 44-4** ■ Langerhans' cells.

Innervation and Blood Supply

The innervation of the skin is complex. The skin, with its accessory structures, serves as an organ for receiving sensory information from the environment. The dermis is well supplied with sensory neurons as well as nerves that supply the blood vessels, sweat glands, and arrector pili muscles. The receptors for touch, pressure, heat, cold, and pain are widely distributed in the dermis. The papillary layer of the dermis is supplied with free nerve endings that serve as nociceptors (*i.e.*, pain receptors) and thermoreceptors. The dermis also contains encapsulated pressure-sensitive receptors that detect pressure and touch.

The arterial vessels that nourish the skin form two plexuses (*i.e.*, collection of blood vessels), one located between the papillary and reticular layers of the dermis and the other be-

tween the dermis and the subcutaneous tissue layer. Capillary flow that arises from vessels in this plexus extends up and nourishes the overlaying epidermis by diffusion. Blood leaves the skin by way of small veins that accompany the subcutaneous vessels. The lymphatic system of the skin, which aids in combating certain skin infections, also is limited to the dermis.

Most of the skin's blood vessels are under sympathetic nervous system control. The sweat glands are innervated by cholinergic fibers but controlled by the sympathetic nervous system. Likewise, the sympathetic nervous system controls the arrector pili (pilomotor) muscles that cause elevation of hairs on the skin. Contraction of these muscles tends to cause the skin to dimple, producing "goose bumps."

In summary, the skin, which forms the major barrier between the internal organs and the external environment, is primarily an organ of protection. The skin is composed of three layers, the epidermis, dermis, and subcutaneous tissues. The skin also houses a variety of appendages, including hair, nails, and sebaceous and sweat glands. The epidermis, the outermost layer of the skin, contains five layers, or strata. The major cells of the epidermis are the keratinocytes, melanocytes, Langerhans' cells, and Merkel's cells. The stratum germinativum, or basal layer, is the source of the cells in all five layers of the epidermis. The keratinocytes, which are the major cells of the epidermis, are transformed from viable keratinocytes to dead keratin as they move from the innermost layer of the epidermis (*i.e.*, stratum germinativum) to the outermost layer (*i.e.*, stratum corneum). The melanocytes are pigment-synthesizing cells that give skin its color.

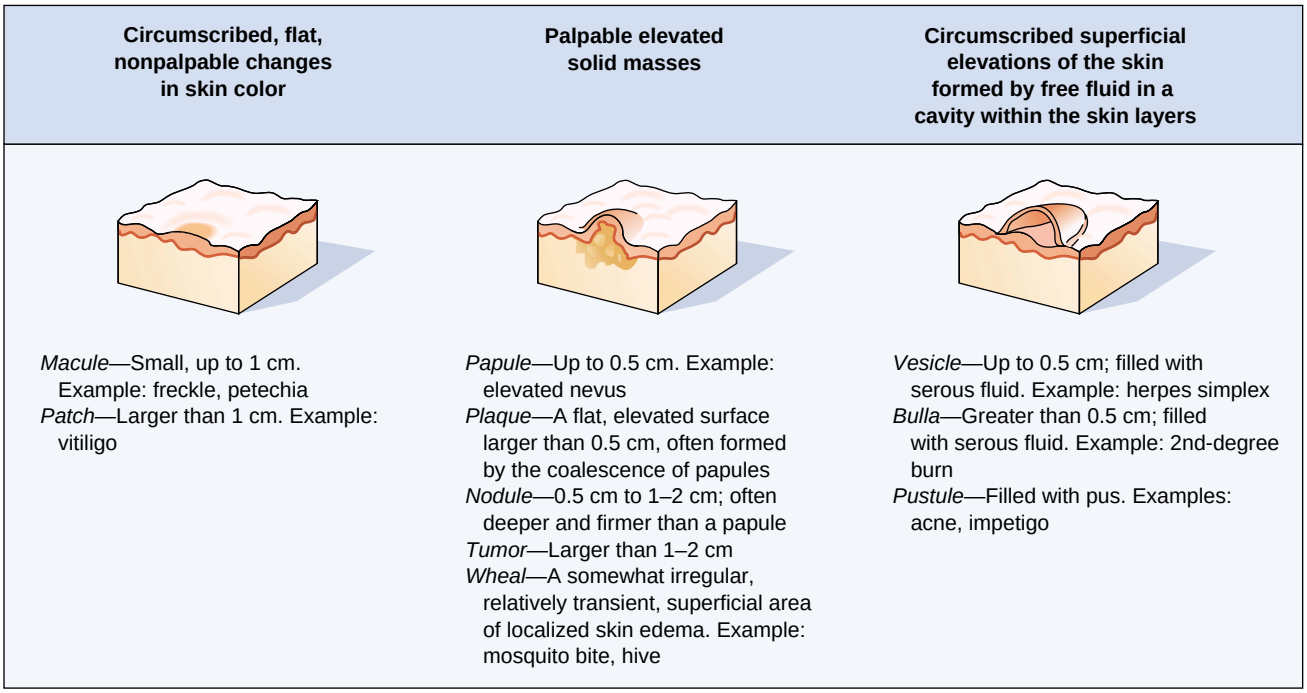
The dermis provides the epidermis with support and nutrition and is the source of blood vessels, nerves, and skin appendages. Sensory receptors for touch, pressure, heat, cold, and pain are widely distributed in the dermis. Both the epidermis and dermis participate in the immune functions of the skin; the Langerhans' cells of the epidermis process foreign antigens for presentation to immune cells (T cells, macrophages, mast cells, and fibroblasts) in the dermis. The subcutaneous tissue layer consists primarily of fat and connective tissues that lend support to the vascular and neural structures supplying the outer layers of the skin.

MANIFESTATIONS OF SKIN DISORDERS

Skin disorders are manifested by a variety of primary lesions and rashes (Fig. 44-5). Commonly, secondary lesions result from overtreatment, scratching, and infection that accompanies the primary skin disorders.

Lesions, Rashes, and Vascular Disorders

Rashes are temporary eruptions of the skin, such as those associated with childhood diseases, heat, diaper irritation, or drug-induced reactions. The term *lesion* refers to a traumatic or pathologic loss of normal tissue continuity, structure, or function. The components of a rash sometimes are referred to as *lesions*. Rashes and lesions may range in size from a fraction of a millimeter (*e.g.*, the pinpoint spots of petechiae) to many



■ **FIGURE 44-5** ■ Primary lesions may arise from previously normal skin. Authorities vary somewhat in their definitions of skin lesions by size. Dimensions given should be considered approximate. (Bates B.B. [1995]. *A guide to physical examination and history taking* [6th ed.]. Philadelphia: J.B. Lippincott)

centimeters (*e.g.*, decubitus ulcer, or pressure sore). They may be blanched (white), erythematous (reddened), hemorrhagic or purpuric (containing blood), or pigmented. Repeated rubbing and scratching can lead to lichenification (thickened and roughened skin characterized by prominent skin markings caused by repeated scratching or rubbing) or excoriation (lesion caused by breakage of the epidermis, producing a raw linear area). Skin lesions may occur as primary lesions arising in previously normal skin, or they may develop as secondary lesions resulting from other disease conditions.

A *blister* is a vesicle or fluid-filled papule. Blisters of mechanical origin form from the friction caused by repeated rubbing on a single area of the skin. Friction blisters most commonly occur on the palmar and plantar surfaces of the hands and feet where the skin is thick enough to form a bleb. Blisters also develop from first-degree and second-degree partial-thickness burns. Histologically, there is degeneration of epidermal cells and a disruption of intercellular junctions that causes the layers of the skin to separate. As a result, fluid accumulates, and a noticeable bleb forms on the skin surface.

A *callus* is a hyperkeratotic plaque of skin caused by chronic pressure or friction. It represents a hyperplasia of the dead keratinized cells that make up the cornified or horny layer of the skin. Increased cohesion between cells results in hyperkeratosis and decreased skin shedding. *Corns* are small, well-circumscribed, conical keratinous thickenings of the skin. They usually appear on the toes from rubbing or ill-fitting shoes. The actual corn may be either hard with a central horny core or soft, as commonly seen between the toes. They may appear on the hands as an occupational hazard. Corns on the feet often are painful, whereas corns on the hands may be asymptomatic.

Telangiectasis are dilated superficial blood vessels, capillaries, or terminal arteries that appear either red or bluish. They can appear by themselves or as a part of other skin disorders such as rosacea or basal cell carcinoma.

Pruritus

Pruritus, or the sensation of itch, is a symptom common to many skin disorders. Generalized itching in the absence of a primary skin disease may be symptomatic of other organ disorders, such as chronic renal disease, diabetes, or biliary disease. Warmth, touch, and vibration also can act locally to trigger the itch phenomenon.

Itch is mediated by cutaneous receptors. Substances such as histamine, bradykinin, substance P, and bile salts act locally to stimulate the itch receptors. Prostaglandins are modulators of the itch response, lowering the threshold for other mediators. One type of itch, sometimes referred to as *central itch*, is perceived as occurring on the skin, but originates in the central nervous system (CNS).[3] For example, the pain reliever morphine promotes itch by acting on central opioid receptors in the CNS.

Scratching, the well-known response to itch, is a neurologic reflex that to varying degrees can be controlled by the individual. Although scratching may temporarily relieve itch, many types of itch are not easily localized and are not relieved by scratching. In many people, excoriations and thickened papular areas develop at the site of repeated scratching or rubbing.

Variations in Black Skin

Some skin disorders common to African Americans are not commonly found in European Americans. Similarly, some skin disorders, such as skin cancers, affect light-skinned persons more commonly than dark-skinned persons. Because of these differences, serious skin disorders may be overlooked, and normal variations in darker skin may be mistaken for anomalies. Skin color is determined by the melanin produced by the melanocytes. Although the number of melanosomes in dark and white skin are the same, black skin produces more melanin and produces it faster than does white skin. Because of their skin color, blacks are better protected against skin cancer and the premature wrinkling and aging of the skin that occurs with sun exposure.

Some conditions common in people with black skin are too much or too little color. Areas of the skin may darken after injury, such as a cut or scrape, or after disease conditions, such as acne.[4] These darkened areas may take many months or years to fade. Dry or "ashy" skin also can be a problem for people with black skin. It often is uncomfortable, and it also is easily noticed because it gives the skin an ashen, or grayish, appearance. Although using a moisturizer may help relieve the discomfort, it may cause a worsening of acne in predisposed persons.

Normal variations in skin structure and skin tones often make evaluation of dark skin difficult (Table 44-1). The darker pigmentation can make skin pallor, cyanosis, and erythema more difficult to observe. Therefore, verbal histories must be relied on to assess skin changes. The verbal history should include clients' descriptions of their normal skin tone. Changes in skin color, in particular hypopigmentation and hyperpigmentation, often accompany ethnic skin disorders and are very important signs to observe for when diagnosing skin conditions.

TABLE 44-1 Common Normal Variations in Dark Skin

Variation	Appearance
Futcher (Voigt's) line	Demarcation between darkly pigmented and lightly pigmented skin in upper arm; follows spinal nerve distribution; common in black and Japanese populations
Midline hypopigmentation	Line or band of hypopigmentation over the sternum, dark or faint, lessens with age; common in Latin American and black populations
Nail pigmentation	Linear dark bands down nails or diffuse nail pigmentation, brown, blue or blue-black
Oral pigmentation	Blue to blue-gray pigmentation of oral mucosa; gingivae also affected
Palmar changes	Hyperpigmented creases, small hyperkeratotic papules, and tiny pits in creases
Plantar changes	Hyperpigmented macules, can be multiple with patchy distribution, irregular borders, and variance in color

(Developed from information in Rosen T., Martin S. [1981]. *Atlas of black dermatology*. Boston: Little, Brown)

In summary, skin lesions and rashes are the most common manifestations of skin disorders. Rashes are temporary skin eruptions. Lesions result from traumatic or pathologic loss of the normal continuity, structure, or function of the skin. Lesions may be vascular in origin; they may occur as primary lesions in previously normal skin; or they may develop as secondary lesions resulting from primary lesions. Blisters, calluses, and corns result from rubbing, pressure, and frictional forces applied to the skin.

Pruritus and dry skin are symptoms common to many skin disorders. Scratching because of pruritus can lead to excoriation, infection, and other complications.

Normal variations in black skin often make evaluation difficult and result in some disorders being overlooked. Changes in color, especially hypopigmentation or hyperpigmentation, often accompany the skin disorders of dark-skinned people.

SKIN DAMAGE CAUSED BY ULTRAVIOLET RADIATION

The skin is the protective shield against harmful ultraviolet rays from the sun. Skin cancers and other skin disorders such as early wrinkling and aging of the skin have been attributed to the damaging effects of sunlight.

Ultraviolet Rays

The earth's sunlight is measured in wavelengths ranging from approximately 290 nm in the ultraviolet region up to approximately 2500 nm in the infrared region. Ultraviolet (UV) radiation is divided into three types: UVC, UVB, and UVA. UVC rays are short (100 to 289 nm) and do not pass through the earth's atmosphere. However, they can be produced artificially and are damaging to the eyes. UVB rays are 290 to 320 nm. These are the rays that are primarily responsible for nearly all the skin effects of sunlight. They are more commonly referred to as *sunburn rays*. UVA rays are 321 to 400 nm. These rays, which can pass through window glass, are more commonly referred to as *suntanning rays*. In general, it takes approximately 1000 times more UVA to match the untoward effects of UVB. Nonetheless, UVA contributes to many skin alterations. Artificial sources of UVA, such as tanning salons, may produce the same effects as UVB.[5]

With UV exposure, skin cells release vasoactive and injurious chemicals, resulting in vasodilation and sunburn. Melanin in the stratum corneum absorbs UV radiation as a means of preventing destruction of the lower skin layers. The skin responds to UV exposure with an increase in melanin content. Components of the immune system in the skin, especially Langerhans' cells, also respond to UV radiation. The number of immune cells is decreased, and their activity is lessened by UV exposure.[6] It is thought that the immune cells are important in removing sun-damaged cells with malignant potential.

Sunburn

Sunburn is caused by excessive exposure of the epidermal and dermal layers of the skin to UV radiation, resulting in an erythematous inflammatory reaction. Sunburn ranges from mild to severe. A mild sunburn consists of various degrees of skin redness. Inflammation, vesicle eruption, weakness, chills, fever, malaise, and pain accompany more severe forms of sunburn. Scaling and peeling follow any overexposure to sunlight. Black skin also burns and may appear grayish or gray-black.

Severe sunburns are treated with wet Burow's solution soaks and topical creams and lotions to limit inflammation and pain.[7] Extensive second- and third-degree burns may require hospitalization and specialized burn care techniques.

Drug-Induced Photosensitivity

Some drugs are classified as photosensitive drugs because they produce an exaggerated response to ultraviolet light. Examples include some of the anti-infective agents (sulfonamides, tetracyclines, nalidixic acid), antihistamines (cyproheptadine, diphenhydramine), antipsychotic agents (phenothiazines, haloperidol), diuretics (thiazides, acetazolamide, amiloride), and nonsteroidal anti-inflammatory drugs (phenylbutazone, ketoprofen, naproxen).[8] Severe sunburn can result when persons taking these drugs are exposed to sunlight.

Drug-induced photosensitivity, such as UVA photosensitivity induced by the psoralens, may be used in treating skin conditions, such as psoriasis, that respond well to ultraviolet radiation exposure. Because an increased incidence of cancerous lesions has been reported in people who have been treated with these agents, their use requires caution and careful surveillance.

Sunscreens and Other Protective Measures

The ultraviolet rays of sunlight or other sources can be either completely or partially blocked from the skin surface by sunscreens. The U.S. Food and Drug Administration (FDA) requires a *sun protection factor* (SPF) rating on all commercial preparations based on their ability to obstruct ultraviolet radiation absorption. The ratings usually are on a scale of 1 to 30+; higher ratings block more sunlight.[9] Products with a higher SPF screen out more UVB rays, which are responsible for acute sun damage.

There are two primary types of sunscreens available on the market—chemical (soluble) agents and physical (insoluble) agents.[9] Chemical agents (*e.g.*, para-aminobenzoic acid [PABA]) protect the skin from absorbing sunlight, and physical agents (*e.g.*, micronized titanium dioxide and microfine zinc) work by reflecting sunlight.

Other protective measures include knowledge about sunlight and how to protect the skin. Shielding the skin with protective clothing and hats or head coverings helps decrease ultraviolet radiation exposure.

In summary, there has been an alarming increase in skin cancers since the early 1980s, and repeated exposure to the ultraviolet rays of the sun has been implicated as its principal cause. Solar and artificial sources of radiation contribute to the amount of radiation to which human beings are exposed. Sunburn, which is caused by excessive exposure to ultraviolet radiation, is an erythematous inflammatory reaction, ranging from mild to severe. Photosensitive drugs can also produce an exaggerated response to ultraviolet light when they are taken in combination with sun exposure. Sunscreens are protective agents that work by either reflecting sunlight or preventing its absorption.

PRIMARY DISORDERS OF THE SKIN

Primary skin disorders are those originating in the skin. They include infectious processes, acne and rosacea, papulosquamous dermatoses, allergic disorders and drug reactions, and arthropod infestations. Although most of these disorders are not life threatening, they can affect the quality of life.

Infectious Processes

The skin is subject to invasion by a number of microorganisms, including fungi, bacteria, and viruses. Normally, the skin flora, sebum, immune responses, and other protective mechanisms guard the skin against infection. Depending on the virulence of the infecting agent and the competence of the host's resistance, infections may result.

Fungal Infections

Fungi are free-living, saprophytic, plantlike organisms, certain strains of which are considered part of the normal skin flora.[10] Fungal or mycotic infections of the skin are traditionally classified as superficial or deep. The superficial mycoses, more commonly known as *tinea* or *ringworm*, invade only the superficial keratinized tissue (skin, hair, and nails). Deep fungal infections involve the epidermis, dermis, and subcutis. Infections that typically are superficial may exhibit deep involvement in immunosuppressed individuals.

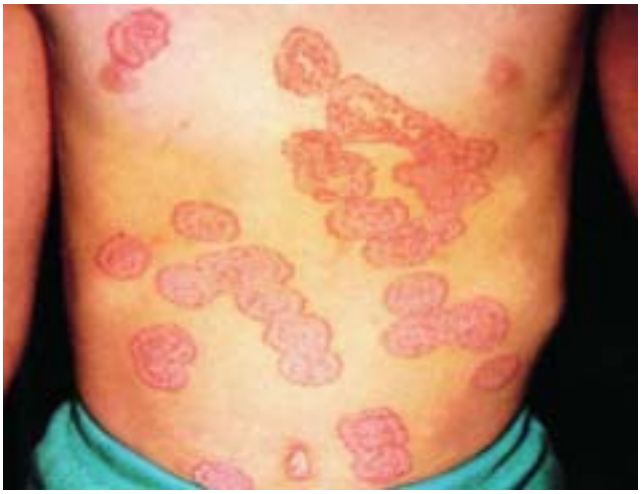
The fungi that cause superficial mycoses live on the dead keratinized cells of the epidermis. They emit an enzyme that enables them to digest keratin, which results in superficial skin scaling, nail disintegration, or hair breakage, depending on the location of the infection. Deeper reactions involving vesicles, erythema, and infiltration are caused by the inflammation that results from exotoxins liberated by the fungus. Fungi also are capable of producing an allergic or immune response.

Superficial Fungal Infections. Superficial fungal infections affect various parts of the body, with the lesions varying according to site and fungal species. Tinea can affect the body (tinea corporis), face and neck (tinea faciei), scalp (tinea capitis), hands (tinea manus), feet (tinea pedis), or nails (tinea unguium).

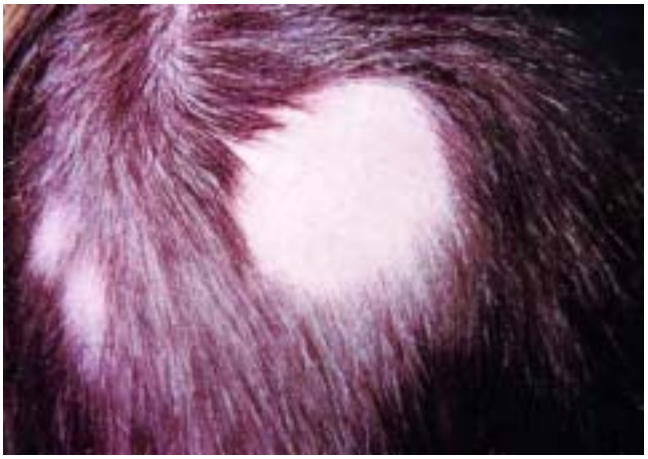
Tinea corporis (ringworm of the body) can be caused by any of the fungi. Although tinea corporis affects all ages, children seem most prone to infection. Transmission is most commonly from kittens, puppies, and other children who have infections. The lesions vary, depending on the fungal agent. The most common types of lesions are oval or circular patches on exposed skin surfaces and the trunk, back, or buttocks (Fig. 44-6). Less common are foot and groin infections. The lesion begins as a red papule and enlarges, often with a central clearing. Patches have raised red borders consisting of vesicles, papules, or pustules. The borders are sharply defined, but lesions may coalesce. Pruritus, a mild burning sensation, and erythema frequently accompany the skin lesion.

Tinea capitis (ringworm of the scalp) occurs in two forms: primary (noninflammatory) and secondary (inflammatory). Depending on the invading fungus, the lesions of the *noninflammatory type* can vary from grayish, round, hairless patches to balding spots or black dots on the head. The lesions vary in size and are most commonly seen on the back of the head (Fig. 44-7). Mild erythema, crust, or scale may be present. The individual usually is asymptomatic, although pruritus may exist. Children between 3 and 14 years of age are primarily affected, although an increasing number of adults are receiving diagnoses of the infection. The *inflammatory type* of tinea capitis has a rapid onset, and lesions usually are localized to one area. The initial lesion consists of a pustular, scaly, round patch with broken hairs. A secondary bacterial infection is common and may lead to a painful, circumscribed, boggy, and indurated lesion called a *kerion*. The highest incidence is among children and farmers who work with infected animals.

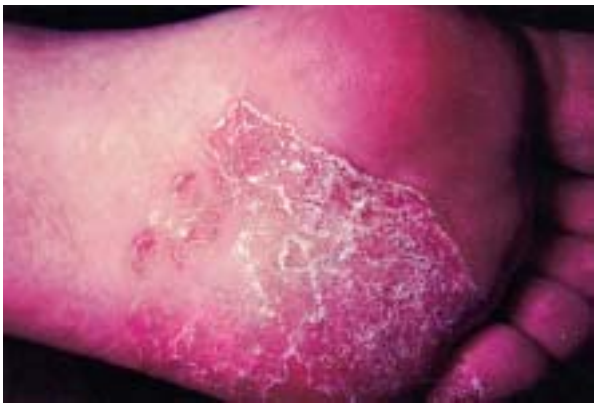
Tinea pedis (athlete's foot, or ringworm of the feet) is a common dermatosis primarily affecting the spaces between the toes, the soles of the feet, or the sides of the feet (Fig. 44-8). The lesions vary from a mildly scaling lesion to a painful, exudative, erosive, inflamed lesion with fissuring. Lesions often are accompanied by pruritus, pain, and foul odor. Some persons are prone to chronic tinea pedis, whereas others have a



■ **FIGURE 44-6** ■ Tinea of the body caused by *Microsporum canis.* (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



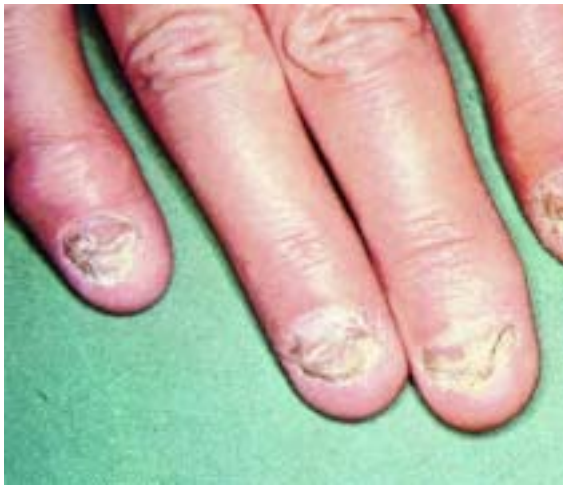
■ **FIGURE 44-7** ■ Tinea of the scalp caused by *Microsporum audouinii.* (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



■ **FIGURE 44-8** ■ Chronic tinea of sole of the foot caused by *Trichophyton rubrum.* (Schering Corp.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

milder form that is exacerbated during hot weather or when the feet are exposed to moisture or occlusive shoes. *Tinea manus* (ringworm of the hands) usually is a secondary infection with tinea pedis as the primary infection. In contrast to other skin disorders such as contact dermatitis and psoriasis, which affect both hands, tinea manus usually occurs only on one hand. The characteristic lesion is a blister on the palm or finger surrounded by erythema. Chronic lesions are scaly and dry. Cracking and fissuring may occur. The lesions may spread to the plantar surfaces of the hand. If chronic, tinea manus may lead to tinea of the fingernails.

Tinea unguium is a dermatophyte infection (onychomycosis) of the nails. Toenails are involved more commonly than fingernails. Toenail infection is common in persons prone to chronic infections of tinea pedis. Often, the infection in the toenails becomes a ready source for future infections of the foot. It may begin from a crushing injury to a toenail or from the spread of tinea pedis. The infection often begins at the tip of the nail, where the fungus digests the nail keratin. Initially, the nail appears opaque, white, or silvery (Fig. 44-9). The nail



■ **FIGURE 44-9** ■ Tinea of the fingernail caused by *Trichophyton rubrum.* (Duke Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

then turns yellow or brown. The condition often remains unchanged for years. During this time it may involve only one or two nails and may produce little or no discomfort. Gradually, the nail thickens and cracks as the infection spreads. Permanent discoloration and distortion result as the nail separates from the underlying epidermis. Less common forms of tinea unguium are superficial white onychomycosis, in which areas of the nails become powdery white and erode, and proximal subungual onychomycosis (PSO), in which there is rapid invasion of the nail, leaving it white with no additional thickening of the nail. Although it is one of the less common forms of tinea unguium, PSO has increased among people with acquired immunodeficiency syndrome (AIDS).

Diagnosis of superficial fungal infections is primarily done by microscopic examination of skin scrapings for fungal spores, the reproducing bodies of fungi. Potassium hydroxide (KOH) preparations are used to prepare slides of skin scrapings.[11] KOH disintegrates human tissue and leaves behind the threadlike filaments, called *hyphae*, that grow from the fungal spores. Cultures also may be done.

Superficial fungal infections may be treated with topical or systemic antifungal agents. Tinea treatment usually follows diagnosis confirmed by KOH preparation or culture, particularly if a systemic agent is to be used. Topical agents, both prescription and over-the-counter preparations, are commonly used in the treatment of tinea infections; however, outcome success often is limited because of the lengthy duration of treatment, poor compliance, and high rates of relapse at specific body sites.

The oral systemic antifungal agents include griseofulvin, the azoles, and the allylamines.[11] Griseofulvin is a fungicidal agent derived from a species of *Penicillium* that is used only in the treatment of dermatophytoses. It acts by binding to the keratin of newly forming skin, protecting the skin from new infection. Because its action is to prevent new infection, it must be administered for 2 to 6 weeks to allow for skin replacement. The azoles (*e.g.*, ketoconazole, itraconazole, and fluconazole) are a group of synthetic antifungal drugs that act by inhibiting the fungal enzymes needed for the synthesis of ergosterol, which is an essential part of fungal cell membranes. Terbinafine, a synthetic allylamine, acts by interrupting ergosterol synthesis, causing the accumulation of a metabolite that is toxic to the fungus. In contrast to griseofulvin, the synthetic agents are fungicidal (*i.e.*, kill the fungus) and thus are more effective in shorter treatment periods.[10] Some of the oral agents can produce serious side effects, such as hepatic toxicity, or interact adversely with other medications being taken. A number of the synthetic fungicides (*e.g.*, ketoconazole, miconazole, clotrimazole, and terbinafine) are available as topical preparations and produce less severe side effects. Topical corticosteroids may be used in conjunction with antifungal agents to relieve itching and erythema secondary to inflammation.

Candidal Infections. Candidiasis (moniliasis) is a fungal infection caused by *Candida albicans*. This yeastlike fungus is a normal inhabitant of the gastrointestinal tract, mouth, and vagina (see Chapter 35). The skin problems result from the release of irritating toxins on the skin surface. Some persons are predisposed to candidal infections by conditions such as diabetes mellitus, antibiotic therapy, pregnancy, use of birth control pills, poor nutrition, and immunosuppressive diseases.

Oral candidiasis may be the first sign of infection with human immunodeficiency virus (HIV).

C. albicans thrives in warm, moist intertriginous areas of the body. The rash is red with well-defined borders. Patches erode the epidermis, and there is scaling. Mild to severe itching and burning often accompany the infection. Severe forms of infection may involve pustules or vesiculopustules. In addition to microscopic analysis, a candidal infection often can be differentiated from a tinea infection by the presence of satellite lesions. These satellite lesions are maculopapular and are found outside the clearly demarcated borders of the candidal infection. Satellite lesions often are diagnostic of diaper rash complicated by *Candida*. The appearance of candidal infections varies according to the site (Table 44-2).

Diagnosis usually is based on microscopic examination of skin or mucous membrane scrapings placed in KOH solution. Depending on the site of infection and extent of involvement, topical and oral antifungal agents may be used in treatment.

Bacterial Infections

Bacteria are considered normal flora of the skin. Most bacteria are not pathogenic, but when pathogenic bacteria invade the skin, superficial or systemic infections may develop. Bacterial skin infections are commonly classified as primary or secondary infections. Primary infections are superficial skin infections such as impetigo or ecthyma. Secondary infections consist of deeper cutaneous infections, such as infected ulcers. Diagnosis usually is based on cultures taken from the infected site. Treatment measures include antibiotic therapy and measures to promote comfort and prevent the spread of infection.

Impetigo. Impetigo is a common superficial bacterial infection caused by *staphylococci* or *group A β-hemolytic streptococci* (GABHS), or both. It is common among infants and young children, although older children and adults occasionally contract the disease. Impetigo initially appears as a small vesicle or pustule or as a large bulla on the face or elsewhere on the body. As the primary lesion ruptures, it leaves a denuded area that discharges a honey-colored serous liquid that hardens on the skin surface and dries as a honey-colored crust with a "stuck-on" appearance (Fig. 44-10). New vesicles erupt within hours. Pruritus often accompanies the lesions, and the skin excoriations that result from scratching multiply the infection sites. A possible complication of untreated GABHS impetigo is poststreptococcal glomerulonephritis (see Chapter 23). Topical mupirocin, which has few side effects, may be effective for limited disease. If the area is large or if there is concern about complications, systemic antibiotics are used.

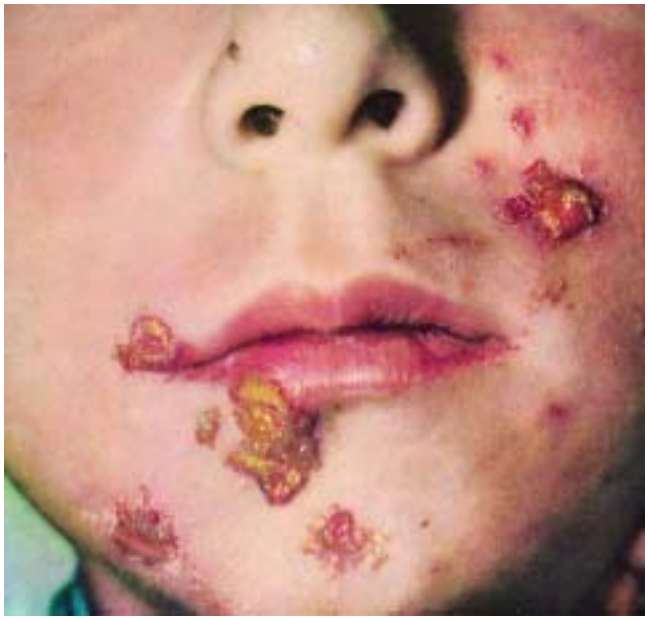
Ecthyma is an ulcerative form of impetigo, usually secondary to minor trauma. It is caused by GABHS, *Staphylococcus aureus*, or *Pseudomonas*. It frequently occurs on the buttocks and thighs of children (Fig. 44-11). The lesions are similar to those of impetigo. A vesicle or pustule ruptures, leaving a skin erosion or ulcer that weeps and dries to a crusted patch, often resulting in scar formation. With extensive ecthyma, there is a low-grade fever and extension of the infection to other organs. Treatment usually involves the use of systemic antibiotics.

Viral Infections

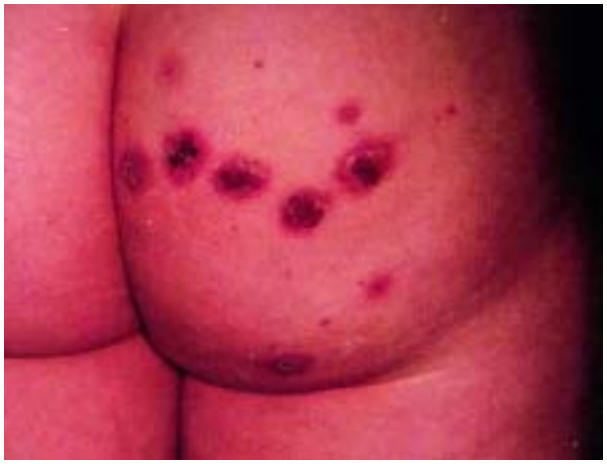
Viruses are intracellular pathogens that rely on live cells of the host for reproduction. They have no organized cell structure but consist of a DNA or RNA core surrounded by a protein coat. The viruses seen in skin lesion disorders tend to be DNA-containing viruses. Viruses invade the keratinocyte, begin to reproduce, and cause cellular proliferation or cellular death. The rapid increase in viral skin diseases has been attributed to the use of corticosteroid drugs, which have immunosuppressive qualities, and the use of antibiotics, which alter the bacterial flora of the skin. As the number of bacterial infections

TABLE 44-2 Candidal Infections: Locations and Appearance of Lesions

Location	Appearance
Breasts, groin, axillae, anus, umbilicus, toe or fingerwebs	Red lesions with well-defined borders and presence of satellite lesions; lesions may be dry or moist
Vagina	Red, oozing lesions with sharply defined borders and inflamed vagina; cervix may be covered with moist, white plaque; cheesy, foul-smelling discharge; presence of pruritus and burning
Glans penis (balanitis)	Red lesions with sharply defined borders; penis may be covered with white plaque; presence of pruritus and burning
Mouth (thrush)	Creamy white flakes on a red, inflamed mucous membrane; papillae on tongue may be enlarged
Nails	Red, painful swelling around nail bed; common in persons who often have their hands in water



■ **FIGURE 44-10** ■ Impetigo of the face. (Abner Kurten, *Folia Dermatologica*. No. 2. Geigy Pharmaceuticals.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



■ **FIGURE 44-11** ■ Ecthyma on the buttocks of a 13-year-old boy. (Glaxo-Wellcome Co.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

has decreased, there has been a proportional rise in viral skin diseases.

Verrucae. Verrucae, or warts, are common, benign papillomas caused by DNA-containing human papillomaviruses (HPV). The lesions are circumscribed, symmetrical epidermal neoplasms that are often elevated above the skin and often appear papillary. Histologically, there is an irregular thickening of the stratum spinosum and greatly increased thickening of the stratum corneum.

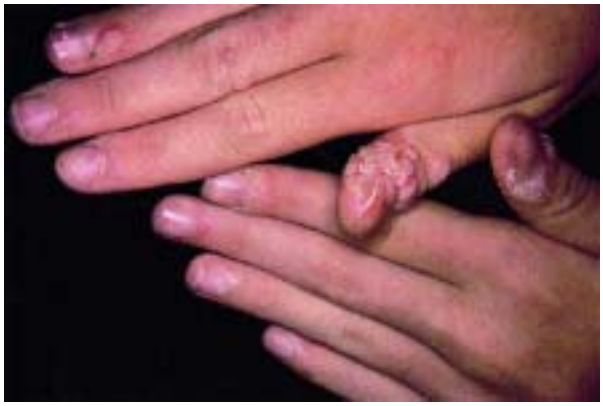
There are more than 50 types of HPVs found on the skin and mucous membranes of humans that cause several different kinds of warts, including skin warts and genital warts.[12] The skin warts, caused by HPV types 1, 2, 3, and 4, usually are not precancerous. HPV transmission usually occurs through breaks in skin integrity. They are known as common warts, flat warts, and plantar warts. *Common warts* are papillary growths, slightly raised above the skin surface, and varying in size from pin-head sized to large clusters of pea-sized tumors (Fig. 44-12). They are seen most commonly on the hands. Common hand warts can be transmitted by biting the cuticles surrounding the nail. *Flat warts* are small flat tumors that are often barely visible but can occur in clusters of 10 or more. They are commonly seen on the forehead and the dorsum of the hand. Plantar warts are flat to slightly raised painful growths that extend deep into the skin. They are frequently transmitted to the abraded, softened heels of children in gym showers or swimming areas. Genital warts are discussed in Chapter 35. Genital warts are sexually transmitted; some types of HPV may increase the risk of genital (cervix, vulva, and penis) cancers.

Treatment usually is directed at inducing a "wart-free" period without producing scarring. Warts resolve spontaneously when immunity to the virus develops. The immune response may be delayed for years. Removal is usually done by applying a keratolytic agent, such as salicylic acid, that breaks down the wart tissue, or by freezing with liquid nitrogen. Various types of laser surgery, electrosurgery, and the use of cytotoxic or antiviral therapy also have been successful in wart eradication.

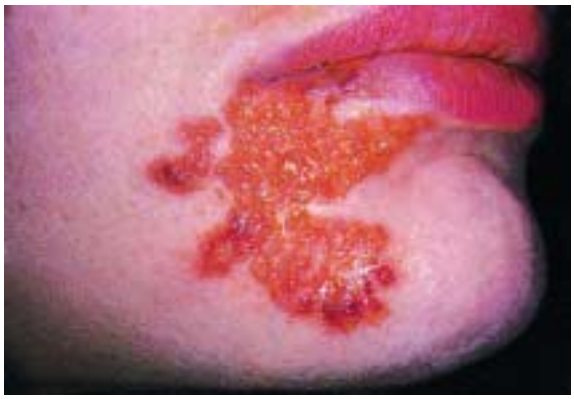
Herpes Simplex. Herpes simplex virus (HSV) infections of the skin and mucous membrane (*i.e.*, cold sore or fever blister) are common. Two types of herpesviruses infect humans: type 1 and type 2. HSV-1 usually is confined to the oropharynx, and the organism is spread by respiratory droplets or by direct contact with infected saliva. Genital herpes usually is caused by HSV-2 (see Chapter 35), although HSV-1 also can cause genital herpes.

Infection with HSV-1 may present as a primary or recurrent infection. Primary HSV-1 infections usually are asymptomatic. Symptomatic disease occurs most frequently in young children (1 to 5 years of age). Symptoms include fever, sore throat, painful vesicles, and ulcers of the tongue, palate, gingiva, buccal mucosa, and lips. Primary infection results in the production of antibodies to the virus so that recurrent infections are more localized and less severe. After an initial infection, the herpesvirus persists in the trigeminal and other dorsal root ganglia in the latent state. It is likely that many adults were exposed to HSV-1 during childhood and therefore have antibodies to the virus.

The recurrent lesions of HSV-1 usually begin with a burning or tingling sensation. Vesicles and erythema follow and progress to pustules, ulcers, and crusts before healing (Fig. 44-13). The lesion is most common on the lips, face, and mouth. Pain



■ **FIGURE 44-12** ■ Common and periungual warts. (Reed & Carnrick Pharmaceuticals.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



■ **FIGURE 44-13** ■ Recurrent herpes simplex of the face. (Dermik Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

is common, and healing takes place within 10 to 14 days. Precipitating factors may be stress, sunlight exposure, menses, or injury. Individuals who are immunocompromised may have severe attacks.

There is no cure for oropharyngeal herpes simplex; most treatment measures are palliative. Penciclovir cream, a topical antiviral agent, applied at the first symptom may be used to reduce the duration of an attack. Application of over-the-counter topical preparations containing antihistamines, antipruritics, and anesthetic agents along with aspirin or acetaminophen may be used to relieve pain. Oral antiviral drugs that inhibit herpesvirus replication may be used prophylactically to prevent recurrences. Sunscreen preparations applied to the lips can prevent sun-induced herpes simplex.

Herpes Zoster. Herpes zoster (shingles) is an acute, localized vesicular eruption distributed over a dermatomal segment of the skin. It is caused by the same herpesvirus, varicella-zoster, that causes chickenpox. It is believed to be the result of reactivation of a latent varicella-zoster virus that was dormant in the sensory dorsal root ganglia since a childhood infection. During an episode of herpes zoster, the reactivated virus travels from the ganglia to the skin of the corresponding dermatome. Although herpes zoster is not as contagious as chickenpox, the reactivated virus can be transmitted to nonimmune contacts.

The incidence of herpes zoster increases with age; it occurs 8 to 10 times more frequently in persons older than 60 years than in younger persons.[13] The normal age-related decrease in cell-mediated immunity is thought to account for the increased viral activation in this age group.[14] Other persons at increased risk because of impaired cell-mediated immunity are those with conditions such as HIV infection and certain malignancies, chronic corticosteroid users, and those undergoing cancer chemotherapy and radiation therapy.

The lesions of herpes zoster typically are preceded by a prodrome consisting of a burning pain, tingling sensation, extreme sensitivity of the skin to touch, and pruritus along the affected dermatome. This may be present for 1 to 3 days or longer before the appearance of the rash. During this time, the pain may be mistaken for a number of other conditions, such as heart disease, pleurisy, various musculoskeletal disorders, or gastrointestinal disorders.

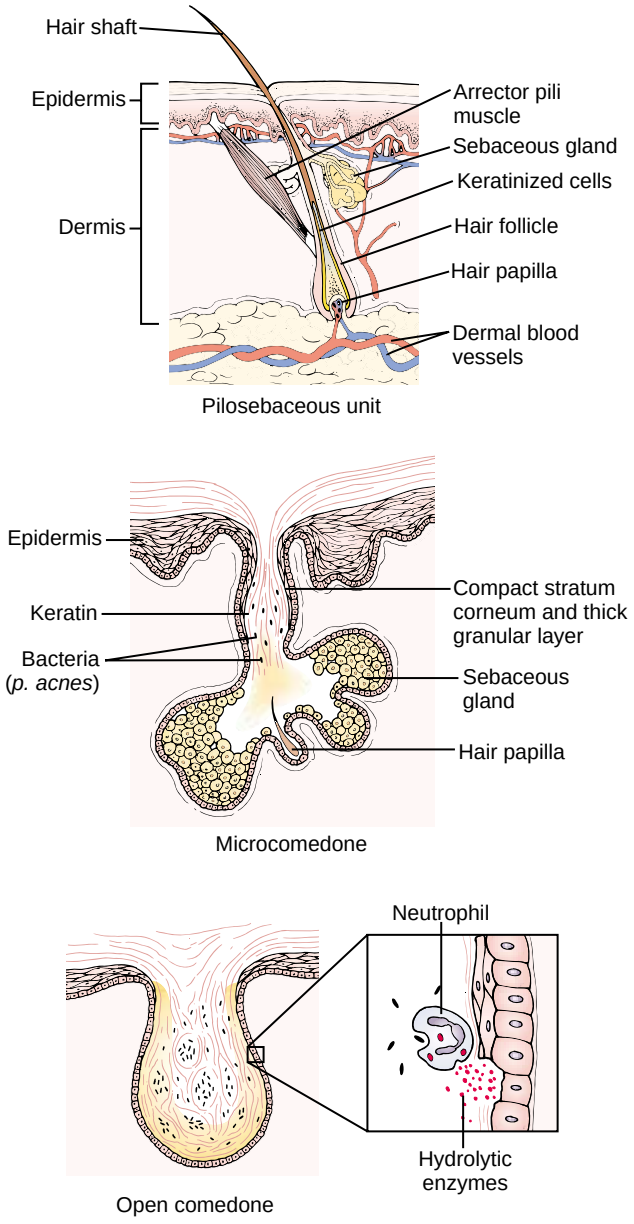
The rash appears as an eruption of vesicles with erythematous bases that are restricted to skin areas supplied by sensory neurons of a single or associated group of dorsal root ganglia (Fig 44-14). In immunosuppressed persons, the lesions may extend beyond the dermatome. Eruptions usually are unilateral in the thoracic region, trunk, or face. New crops of vesicles erupt for 3 to 5 days along the nerve pathway. The vesicles dry, form crusts, and eventually fall off. The lesions usually clear in 2 to 3 weeks.

Serious complications can accompany eruptions. Eye involvement can result in permanent blindness and occurs in a large percentage of cases involving the ophthalmic division of the trigeminal nerve (see Chapter 38). Pain can persist for several months after the rash disappears. Postherpetic neuralgia, which is pain that persists longer than 1 to 3 months after the resolution of the rash, is an important complication of herpes zoster (see Chapter 38).[14]

The treatment of choice for herpes zoster is the administration of an antiviral agent. The treatment is most effective when started within 72 hours of rash development.[15,16] Narcotic analgesics, tricyclic antidepressants or anticonvulsant drugs, and nerve blocks may be used for the management of herpetic pain. Oral corticosteroids sometimes are used to reduce the inflammation that may contribute to the pain.

Acne and Rosacea

Acne is a disorder of the pilosebaceous unit (hair follicle and sebaceous gland). The hair follicle is a tubular invagination of the epidermis in which hair is produced. The sebaceous glands empty into the hair follicle, and the pilosebaceous unit opens to the skin surface by means of a widely dilated opening called a pore (Fig. 44-15). The sebaceous glands produce a complex lipid mixture called sebum, from the Latin word meaning *tallow* or *grease*. Sebum consists of a mixture of free fatty acids, triglycerides, diglycerides, monoglycerides, sterol esters, wax es-



■ **FIGURE 44-15** ■ The pathogenesis of acne vulgaris. The pilosebaceous unit (**A**) and the development of a microcomedone (whitehead) (**B**), and an open comedone with an accumulation of melanin (**C**).

ters, and squalene. Sebum production occurs through what is called a *holocrine process*, in which the sebaceous gland cells that produce the sebum are completely broken down and their lipid contents are emptied through the sebaceous duct into the hair follicle. The amount of sebum produced depends on two factors: the size of the sebaceous gland and the rate of sebaceous cell proliferation. The sebaceous glands are largest on the face, scalp, and scrotum, but are present in all areas of the skin except for the soles of the feet and palms of the hands. Sebaceous cell proliferation and sebum production are uniquely responsive to direct hormonal stimulation by androgens. In men, testicular androgens are the main stimulus for sebaceous activity; in women, adrenal and ovarian androgens maintain sebaceous activity.

Acne lesions consist of *comedones* (whiteheads and blackheads), papules, pustules, nodules, and, in severe cases, cysts. *Whiteheads* are pale, slightly elevated papules with no visible orifice. *Blackheads* are plugs of material that accumulate in sebaceous glands that open to the skin surface. The color of blackheads results from melanin that has moved into the sebaceous glands from adjoining epidermal cells. *Papules* are raised areas less than 5 mm in diameter. *Pustules* have a central core of purulent material. *Nodules* are larger than 5 mm in diameter and may become suppurative or hemorrhagic. Suppurative nodules often are referred to as *cysts* because of their resemblance to inflamed epidermal cysts. Acne lesions are divided into noninflammatory and inflammatory lesions. Noninflammatory acne consists primarily of comedones. Inflammatory acne consists of papules, pustules, nodules, and cysts. The inflammatory lesions are believed to develop from the escape of sebum into the dermis and the irritating effects of the fatty acids contained in the sebum.

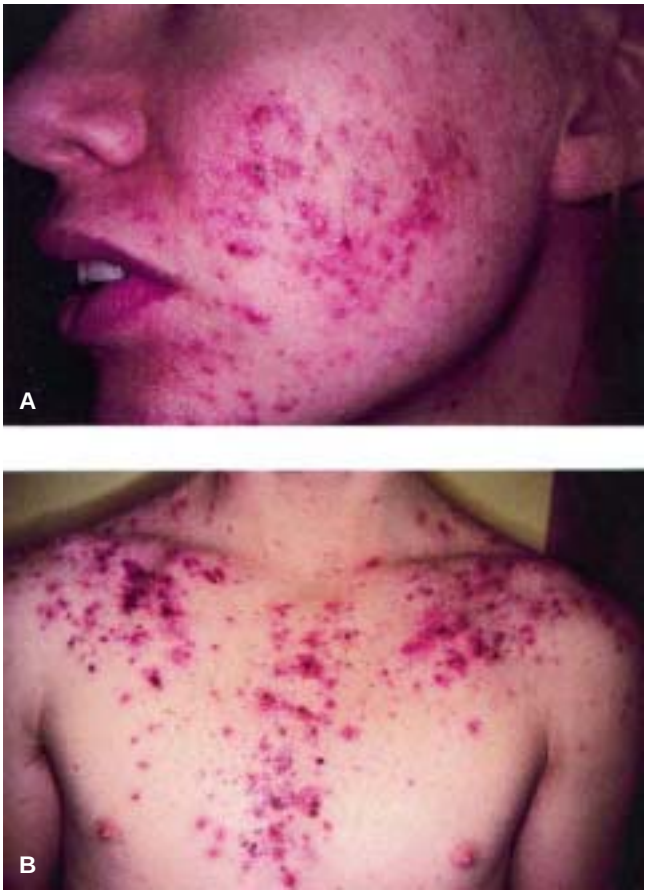
Two types of acne occur during different stages of the life cycle: acne vulgaris, which is the most common form among adolescents and young adults, and acne conglobata, which develops later in life.[17] Other types of acne occur in association with various etiologic agents and influences.

Acne Vulgaris

Acne vulgaris is a common skin condition of adolescence and young adults. The condition is so common during adolescence that is often regarded as a normal part of the maturing process. The acne vulgaris lesions, which consist of comedones (whiteheads and blackheads) or inflammatory lesions (pustules, nodules, or cysts), are found primarily on the face and neck and to a lesser extent, on the back, chest, and shoulders (Fig. 44-16).

The cause of acne vulgaris remains unknown. Several factors are believed to contribute to acne, including (1) the influence of androgens on sebaceous cell activity, (2) increased proliferation of the keratinizing epidermal cells that form the sebaceous cells, (3) increased sebum production in relation to the severity of the disease, (4) decreased amounts of linoleic acid in the sebum, and (5) the presence of the *Propionibacterium acnes*, the microorganism responsible for inflammatory stage of the disorder.[17,18] These factors probably are interrelated. Increased androgen production results in increased sebaceous cell activity, with a resultant plugging of the pilosebaceous ducts. The excessive sebum provides a medium for the growth of *P. acnes*. The *P. acnes* organism contains lipases that break down the free fatty acids that produce the acne inflammation.

Treatment of acne focuses on clearing up existing lesions, preventing new lesions from forming, and limiting scar formation.[19,20] Milder forms of acne usually respond to proper cleansing and application of topical keratolytic agents that act chemically to break down keratin, loosen comedones, and exert a peeling effect on the skin. The treatment measures for moderate to severe acne are directed at correcting the defect in epidermal cell proliferation, decreasing sebaceous gland activity, reducing the *P. acnes* population, and limiting the inflammatory process. Often, a combination of keratolytic and antibacterial agents is used. Topically applied antibiotics do not affect existing lesions but prevent future lesions by decreasing the amount of *P. acnes*



■ **FIGURE 44-16** ■ (**A**) Acne of the face and (**B**) acne of the chest. (Hall J.C. [1999]. *Sauer's manual of skin diseases* [8th ed., p. 118]. Philadelphia: Lippincott Williams & Wilkins)

on the skin, thereby reducing subsequent inflammation formed from the presence of sebaceous fatty acid metabolites.

Severe cases of acne are managed with topical vitamin A, systemic antibiotics, and oral retinoids or acid form of vitamin A. The action of topical vitamin A (tretinoin) has been attributed to decreased cohesiveness of epidermal cells and increased epidermal cell turnover. This is thought to result in increased extrusion of open comedones and transformation of closed comedones into open ones. The oral retinoids (isotretinoin) have revolutionized the treatment of recalcitrant cases of acne and cystic acnes. Although the exact mode of action is unknown, isotretinoin decreases sebaceous gland activity, prevents new comedones from forming, reduces the *P. acnes* count through sebum reduction, and has an anti-inflammatory effect. Because of its many side effects, it is used only in persons with severe acne. Isotretinoin is a known teratogen and should not be used in women who are pregnant or may become pregnant.

Acne Conglobata

Acne conglobata occurs later in life and is a chronic form of acne. Comedones, papules, pustules, nodules, abscesses, cysts, and scars occur on the back, buttocks, and chest. Lesions occur to a lesser extent on the abdomen, shoulders, neck, face, upper arms, and thighs.[17] The comedones have multiple openings. Their discharge is odoriferous, serous, and purulent or mucoid. Healing leaves deep keloidal lesions. Affected persons have anemia with increased white blood cell counts, sedimentation rates, and neutrophil counts. The treatment is difficult and stringent. It often includes debridement, systemic corticosteroid therapy, oral retinoids, and systemic antibiotics.

Rosacea

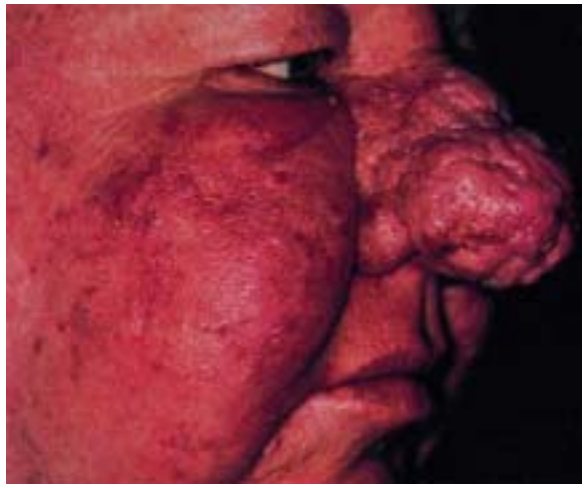
Rosacea, formerly called *acne rosacea*, is a chronic acneform type eruption of the butterfly area of the face that occurs in middle-age and older adults. It is easily confused with acne and may coexist with it. In the early stage of development, there are repeated episodes of blushing, eventually progressing to a persistent erythema on the nose and cheeks that sometimes extends to the forehead and chin (Fig. 44-17). This stage often occurs before 20 years of age. As the person ages, the erythema persists, and telangiectasia with or without acneiform components (*e.g.*, comedones, pustules, nodules) develops. After years of affliction, acne rosacea may develop into an irregular bullous hyperplasia (thickening) of the nose, known as *rhinophyma*. The sebaceous follicles and openings enlarge, and the skin color changes to a purple-red. Rhinophyma occurs mainly in men.[17]

The cause of rosacea remains unknown. It is seen more commonly in fair-skinned persons and has been called "the curse of the Celts." The capillaries in the involved facial areas display an increased reactivity to heat and other vascular stimulating agents such as highly spiced food and alcohol.

Treatment measures are similar to those used for acne vulgaris. Rhinophyma can be treated surgically. Because of increased capillary reactivity, people with rosacea are instructed to avoid heat, sunlight, hot liquids, highly seasoned foods, and alcohol.

Atopic Eczema and Nummular Eczema

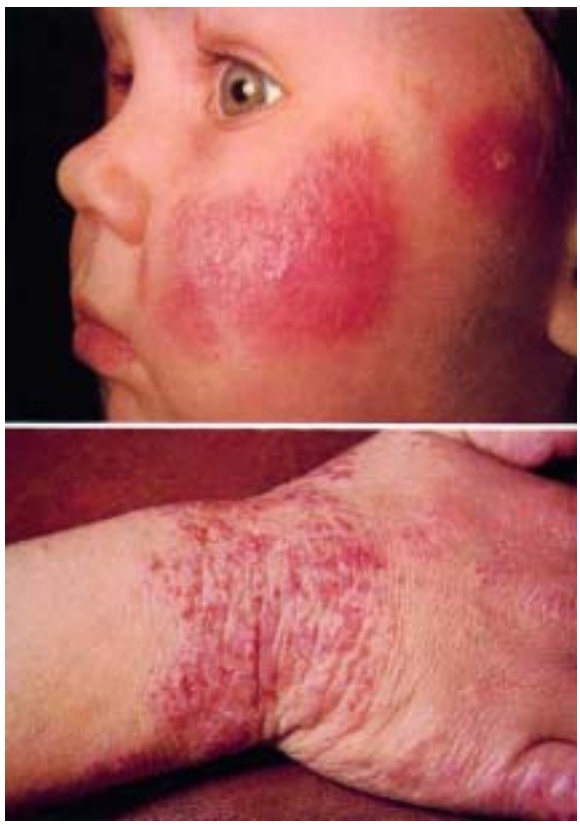
Atopic eczema (atopic dermatitis) is a common skin disorder that occurs in two clinical forms: infantile and adult.[21,22] It is associated with a type I hypersensitivity reaction (see Chap-



■ **FIGURE 44-17** ■ Chronic rosacea with rhinophyma. (Hoechst Marion Roussel Pharmaceuticals, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

ter 10). There usually is a family history of asthma, hay fever, or atopic dermatitis. The infantile form is characterized by vesicle formation, oozing, and crusting with excoriations. It usually begins in the cheeks and may progress to involve the scalp, arms, trunk, and legs (Fig. 44-18). The skin of the cheeks may be paler, with extra creases under the eyes, called *Dennie-Morgan folds*. There is marked follicle involvement in persons with black skin. Lesions may be hypopigmented or hyperpigmented or both on a black-skinned person. The infantile form usually becomes milder as the child ages, often disappearing by the age of 15 years. Adolescents and adults usually have dry, leathery, and hyperpigmented or hypopigmented lesions located in the antecubital and popliteal areas. These may spread to the neck, hands, feet, eyelids, and behind the ears. Itching may be severe with both forms. Secondary infections are common.

Treatment of atopic eczema is designed to target the underlying abnormalities such as dryness, pruritus, superinfection, and inflammation. It involves allergen control, basic skin care, and medications. Because dry skin and pruritus often exacerbate the condition, hydration of the skin is essential to treating atopic dermitis. Mild or healing lesions may be treated with lotions containing a mild antipruritic agent. Acute weeping lesions are treated with soothing lotions, soaks, or wet dressings. Topical corticosteroids provide an effective form of treatment but can cause local and systemic side effects. Because of their side effects, systemic corticosteroids usually are reserved for severe cases.



■ **FIGURE 44-18** ■ Atopic eczema on an infant's face and on a wrist. (Dome Chemicals.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

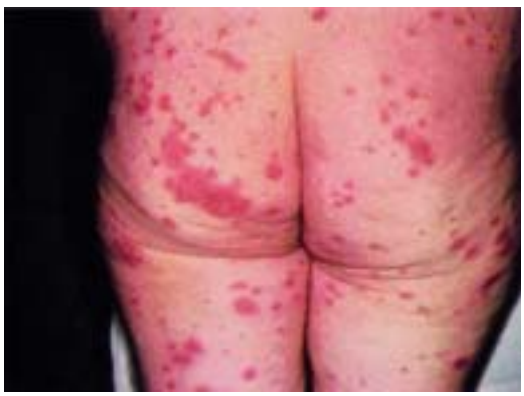
The lesions of nummular eczema (discoid eczema) are coin-shaped (nummular) papulovesicular patches mainly involving the arms and legs (Fig. 44-19). Lichenification and secondary bacterial infections are common. It is not unusual for the initial lesions seemingly to heal, followed by a secondary outbreak of mirror-image lesions on the opposite side of the body. Most nummular eczema is chronic, with weeks to years between exacerbations. Exacerbations occur more frequently in the cold winter months. The exact cause of nummular eczema is unknown. There usually is a history of asthma, hay fever, or atopic dermatitis. Ingestion of iodides and bromides usually aggravates the condition. Treatment is palliative. Frequent bathing, foods rich in iodides and bromides, and stress should be reduced, whereas the environmental humidity should be increased. Topical corticosteroids, coal tar preparations, and ultraviolet light treatments are prescribed as necessary.

Urticaria

Urticaria, or hives, is characterized by edematous plaques, called *wheals*, that are accompanied by intense itching. Wheals typically appear as raised pink or red areas surrounded by a paler halo. They blanch with pressure and vary in size from a few millimeters to centimeters. Thicker lesions that result from massive transudation of fluid into the dermis or subcutaneous tissue are referred to as *angioedema*. Although angioedema lesions can occur on any skin surface, they typically involve the larynx, causing hoarseness or sore throat, or mucosal surface of the gastrointestinal tract, causing abdominal pain.

Histamine, released from mast cells, is the most common mediator of urticaria. It causes hyperpermeability of the microvessels of the skin and surrounding tissue, allowing fluid to leak into the tissues, causing edema and wheal formation.[23,24] A variety of immunologic, nonimmunologic, physical, and chemical stimuli can cause urticaria.

Urticaria can be acute or chronic. Daily or almost daily episodes of urticaria persisting for longer than 6 weeks are considered to be chronic. The most common causes of acute urticaria are foods or drinks, medications, or exposure to pollens or chemicals. Food is the most common cause of acute urticaria in children. Although nonsteroidal anti-inflammatory drugs, including aspirin, do not normally cause urticaria, they may exacerbate pre-existing disease.



■ **FIGURE 44-19** ■ Nummular eczema of the buttocks. (Johnson & Johnson.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

Chronic urticaria affects primarily adults and is twice as common in women as in men. Usually its cause cannot be determined despite extensive laboratory tests. Some forms of chronic urticaria are associated with histamine-releasing autoantibodies. In rare cases, urticaria is a manifestation of underlying disease, such as certain cancers, collagen diseases, and hepatitis. There is an association between chronic urticaria and autoimmune thyroid disease (*e.g.*, Hashimoto's thyroiditis, Graves' disease, toxic multinodular thyroiditis). A hereditary deficiency of a C1 (complement 1) inhibitor also can cause urticaria and angioedema.

Physical urticarias constitute another form of chronic urticaria.[35] Physical urticarias are intermittent, usually last less than 2 hours, are produced by appropriate stimuli, have distinctive appearances and locations, and are seen most frequently in young adults. Dermographism, or skin writing, is one form of physical urticaria in which wheals appear in response to simple rubbing of the skin (Fig. 44-20). The wheals follow the pattern of the scratch or rubbing, appearing within 10 minutes, and dissolving completely within 20 minutes. Other types of physical urticaria are cholinergic (*i.e.*, exercise-induced), cold, delayed pressure, solar (*i.e.*, sunlight), aquagenic (*i.e.*, water), vibratory, and external (localized heat-induced). Appropriate challenge tests (*e.g.*, application of an ice cube to the skin to initiate development of cold urticaria) are used to differentiate physical urticaria from chronic urticaria due to other causes.

Most types of urticaria are treated with antihistamines: drugs that block histamine type 1 (H_1) and less frequently, H_1 in combination with histamine type 2 (H_2). They control urticaria by inhibiting vasodilation and escape of fluid into the surrounding tissues. Severe urticaria and angioedema are treated with epinephrine. Oral corticosteroids may be used in the treatment of refractory urticaria.

Drug-Induced Skin Eruptions

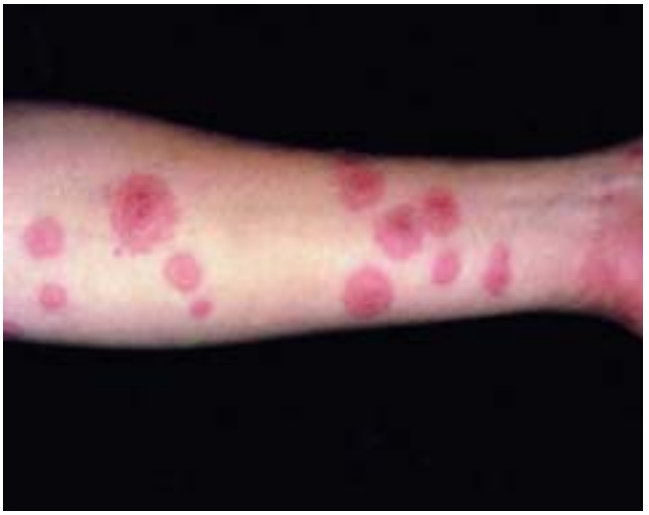
Almost any drug can cause a localized or generalized skin eruption. Topical drugs usually are responsible for a localized contact dermatitis type of rash, whereas systemic drugs cause generalized skin lesions. Most drug eruptions are morbilliform (*i.e.*, measles-like) or exanthematous. Drug eruptions usually occur within 1 to 2 weeks after starting therapy. Some drug reactions progress to more severe skin lesions, necessitating prompt medical attention. Drug-induced skin reactions mimic almost all other skin lesions described in this chapter.

The diagnosis of a drug sensitivity depends almost entirely on accurate reporting by the person because the lesions from drug sensitization vary greatly. Treatment of mild cases is aimed at eliminating the offending drug while treating the symptoms. In mild cases, the lesions usually clear within 7 to 14 days after withdrawal of the offending drug. Severe drug eruptions often require systemic corticosteroid therapy and antihistamines.

Three types of bullous skin manifestations that result from drug reactions end in epidermal skin detachment: erythema multiforme minor, Stevens-Johnson syndrome (erythema multiforme major), and toxic epidermal necrolysis. The latter two are rare occurrences, but they can be life threatening. Both usually are caused by sensitivity to such drugs as sulfonamides and anticonvulsants. Although erythema multiforme minor may be drug induced, it more frequently occurs after infections, especially with herpes simplex. It is self-limiting, with a small amount of skin detachment at the lesion sites.

The lesions of erythema multiforme minor and Stevens-Johnson syndrome are similar. The primary lesion of both is a round, erythematous papule, resembling an insect bite. Within hours to days, these lesions change into several different patterns. The individual lesions may enlarge and coalesce, producing small plaques, or they may change to concentric zones of color appearing as "target" or "iris" lesions (Fig. 44-21). The outermost rings of the target lesions usually are erythematous; the central portion usually is opaque white, yellow, or gray (dusky). In the center, small blisters on the dusky purpuric macules may form, giving them their characteristic target-like appearance. Although there is wide distribution of lesions over the body surface area, there is a propensity for them to occur on the face and trunk. With Stevens-Johnson syndrome, there is more skin detachment.

■ **FIGURE 44-20** ■ Dermographism on a patient's back. (Dermik Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



■ **FIGURE 44-21** ■ Erythema-multiforme–like eruption on the patient's arm. Notice the dusky, target-like appearance. (Dermik Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

Toxic epidermal necrolysis is the most serious and life-threatening drug reaction. The person experiences a prodromal period of malaise, low-grade fever, and sore throat. Within a few days, widespread erythema and large, flaccid bullae appear, followed by the loss of the epidermis. This leaves a denuded and painful dermis. The skin surrounding large denuded areas may have the typical target-like lesions seen with Stevens-Johnson syndrome. The skin separates easily from the dermis with lateral pressure. The epithelium of mucosal surfaces, especially the mouth and eyes, may be involved.

These three types of bullous skin eruptions are seemingly quite similar. The diagnostic boundary for erythema multiforme minor is that it usually occurs after herpes simplex infection and is self-limiting. Precise diagnostic boundaries between Stevens-Johnson syndrome and toxic epidermal necrolysis have not been established. However, it is generally agreed that cases involving less than 10% of the body surface area are called *Stevens-Johnson syndrome*, and detachment of more than 30% of the epidermis is labeled *toxic epidermal necrolysis*.[25,26] The mortality rate associated with Stevens-Johnson syndrome is less than 5%, and that for toxic epidermal necrolysis is 30% or greater.[26]

The skin detachment associated with these drug reactions is different from the desquamation (*i.e.*, peeling) discussed with other skin disorders. For example, with scarlet fever there is peeling of the stratum corneum, the dead keratinized layer. In the bullous disorders discussed here, there is full-thickness detachment (*i.e.*, peeling of the entire epidermis down to the dermis). This leaves the person vulnerable to multiple problems, such as loss of body fluid and thermal control, nutritional deficits, and electrolyte imbalance.

Treatment of erythema multiforme minor and less severe cases of Stevens-Johnson syndrome includes relief of symptoms using compresses, antipruritic drugs, and topical anesthetics. Corticosteroid therapy may be indicated in moderate cases, although its use is a matter of controversy. For severe cases of Stevens-Johnson syndrome or toxic epidermal necrolysis, hospitalization is required for fluid replacement, antibiotics, respiratory care, analgesics, and moist dressings. When large areas of skin are detached, the care is similar to that provided to patients with thermal burns.

Papulosquamous Dermatoses

Papulosquamous dermatoses are a group of skin disorders characterized by scaling papules and plaques. Among the major papulosquamous diseases are psoriasis, pityriasis rosea, and lichen planus.

Psoriasis

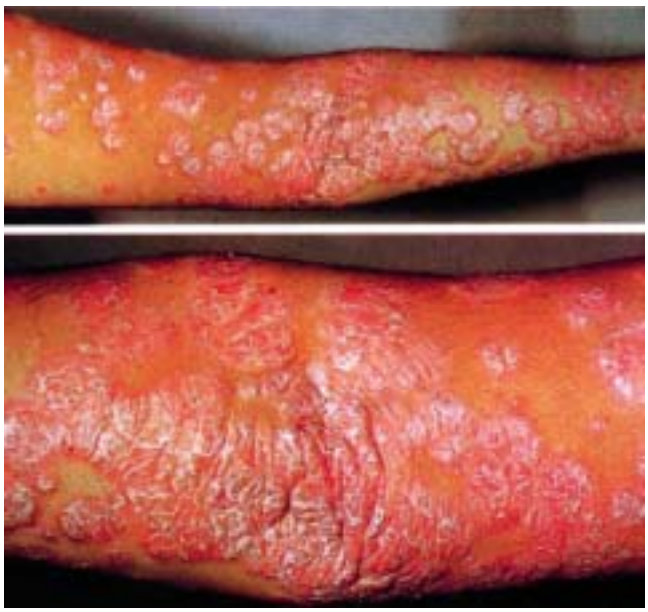
Psoriasis is a common skin disease characterized by circumscribed red, thickened plaques with an overlying silvery-white scale. Psoriasis occurs worldwide, although the incidence is lower in warmer, sunnier climates. In the United States, it affects 2.6% or 6 million Americans.[27] The average age of onset is in the third decade; its prevalence increases with age. Approximately one third of patients have a genetic history, indicating a hereditary factor. Childhood onset of the disease is more strongly associated with a family history than psoriasis occurring in adults older than 30 years.[28] There appears to be an association between psoriasis and arthritis. Psoriatic arthritis occurs in 5% to 7% of persons with psoriasis (see Chapter 43).

The cause of psoriasis remains poorly understood and is probably multifactorial. There is evidence of a genetic component. The more severe the disease, the greater is the likelihood of a familial background. Environmental factors may also play a role. A variety of stimuli, such as physical injury, infections, use of certain drugs, and photosensitivity, may precipitate the development or exacerbation of lesions in people who are predisposed to the disease. The reaction of the skin to an original trauma of any type is called the *Köbner reaction*.

Histologically, psoriasis is characterized by increased epidermal cell turnover with marked epidermal thickening, a process called *hyperkeratosis*. The migration time of the keratinocyte from the basal cell layer to the stratum corneum decreases from the normal 14 days to approximately 4 to 7 days. The granular layer of the epidermis is thinned or absent. There is also an accompanying thinning of the epidermal cell layer that overlies the tips of the dermal papillae (suprapapillary plate), and the blood vessels within dermal papillae become tortuous and dilated. These capillary beds show permanent damage even when the disease is in remission or has resolved. The close proximity of the vessels in the dermal papillae to the hyperkeratotic scale accounts for multiple, minute bleeding points that are seen when the scale is lifted.[12]

The lesions of plaque-type psoriasis may occur anywhere on the skin but most often involve the elbows, knees, and scalp (Fig. 44-22). The primary lesions are papules that vary in shape. The papules form thick red plaques with a silvery scale. In darker-skinned persons, the plaques may appear purple. There may be excoriation, thickening, or oozing from the lesions. A differential diagnostic finding is that the plaques bleed from minute points when removed.

There is no cure for psoriasis. The goal of treatment is to suppress the signs and symptoms of the disease: hyperkeratosis, epidermal inflammation, and abnormal keratinocyte



■ **FIGURE 44-22** ■ Psoriasis on the elbows of a 17-year-old girl. (Roche Laboratories.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

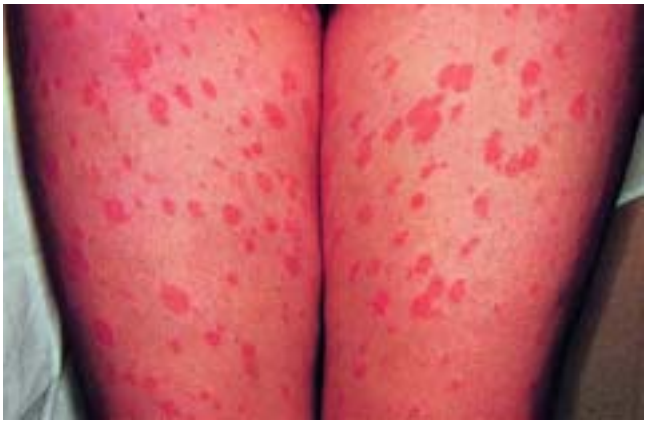
differentiation.[27,28] Treatment measures are divided into topical and systemic approaches. Psoriasis has long been treated topically with keratolytic agents, coal tar products, and anthralin.[17] Topical and systemic corticosteroids may be used, depending on the severity of the disease. Severe, generalized psoriasis may be treated with agents such as methotrexate. Methotrexate, which is used for cancer treatment, is an antimetabolite that inhibits DNA synthesis and prevents cell mitosis. Phototherapy after the administration of a psoralen has proven to be effective in many severe cases. Methoxsalen, a psoralen, exerts its actions when exposed to UVA radiation in 320- to 400-nm wavelengths. Methoxsalen is given orally before UVA exposure. Activated by the UVA energy, methoxsalen inhibits DNA synthesis, thereby preventing cell mitosis and decreasing the hyperkeratosis that occurs with psoriasis. The combination treatment regimen of psoralen and UVA is known by the acronym PUVA.

Pityriasis Rosea

Pityriasis rosea is a rash that primarily affects young adults. The origin of the rash is unknown, but it is believed to be caused by an infective agent. Numerous viruses have been investigated, but no conclusive evidence has been found. The incidence is highest in winter. Cases occur in clusters and among persons who are in close contact with each other, indicating an infectious spread. However, there are no data to support communicability. It may be an immune response to any number of agents.

The characteristic lesion is an oval macule or papule with surrounding erythema (Fig. 44-23). The lesion spreads with central clearing, much like tinea corporis. This initial lesion is a solitary lesion called the *herald patch* and is usually on the trunk or neck. As the lesion enlarges and begins to fade away (2 to 10 days), successive crops of lesions appear on the trunk and neck. The lesions on the back have a characteristic "Christmas tree" pattern. The extremities, face, and scalp may be involved. Mild to severe pruritus may occur.

The disease is self-limited and usually disappears within 6 to 8 weeks. Treatment measures are palliative and include topical steroids, antihistamines, and colloid baths. Systemic corticosteroids may be indicated in severe cases.



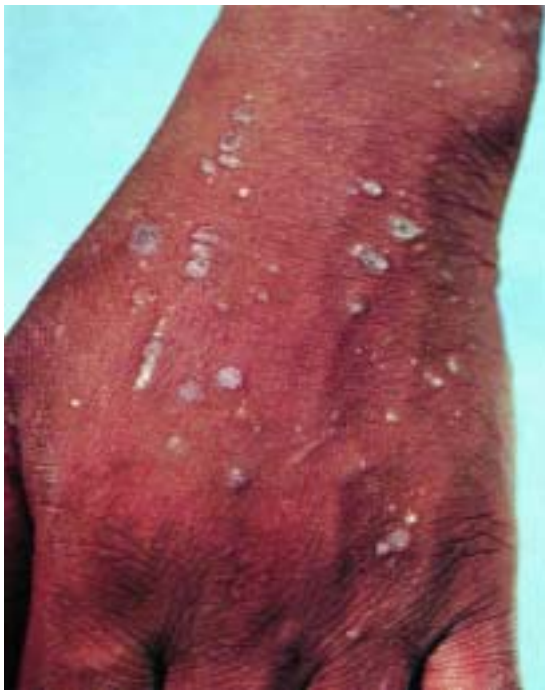
■ **FIGURE 44-23** ■ Pityriasis rosea of the thighs. (Syntex Laboratories.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

Lichen Planus

The term *lichen* is of Greek origin and means "tree moss." The term is applied to skin disorders characterized by small (2 to 10 mm), flat-topped papules with irregular, angulated borders (Fig. 44-24).[17] Lichen planus is a relatively common chronic, pruritic disease. It involves inflammation and papular eruption of the skin and mucous membranes. There are variations in the pattern of lesions (*e.g.*, annular, linear) and differences in the sites (*e.g.*, mucous membranes, genitalia, nails, scalp). The characteristic lesion is a purple, polygonal papule covered with a shiny, white, lacelike pattern. The lesions appear on the wrist, ankles, and trunk of the body. Most persons who have skin lesions also have oral lesions, appearing as milky white lacework on the buccal mucosa or tongue.

The etiology of lichen planus is unknown. There is increasing evidence of a cell-mediated response involving the epidermal-dermal junction with damage to the basal cell layer. Although the cause of most cases of lichen planus is unknown, some are linked to medication use or hepatitis C virus infection. The most common offending agents include gold, antimalarial agents, thiazide diuretics, beta blockers, nonsteroidal anti-inflammatory agents, quinidine, and angiotensin-converting enzyme inhibitors.[29]

Diagnosis is based on the clinical appearance of the lesions and the histopathologic findings from a punch biopsy. For most persons, lichen planus is a self-limited disease. Treatment measures include discontinuation of all medications, followed by treatment with topical corticosteroids and occlusive dressings. Antipruritic agents are helpful in reducing itch. Systemic corticosteroids may be indicated in severe cases. Intralesional corticosteroid injections also may be used.



■ **FIGURE 44-24** ■ Lichen planus of the dorsum of the hand and wrist. Notice the violacenous color of the papules and the linear Köbner's phenomenon. (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

Lichen Simplex Chronicus

Lichen simplex chronicus is a localized lichenoid dermatitis. The term *lichen simplex* denotes that there was no known predisposing skin disorder in the affected person. It is characterized by the occurrence of itchy, reddened, thickened, and scaly patches of dry skin. Persons with the condition may have a single or, less frequently, multiple lesions. The lesions are seen most commonly at the nape of the neck, wrist, ankles, or anal area. The condition usually begins as a small pruritic patch, which culminates in a repetitive cycle of itching and scratching that develops into a chronic dermatosis. Because of the chronic itching and scratching, excoriations and lichenification with thickening of the skin develop, often giving the appearance of tree bark. Treatment consists of measures to decrease scratching of the area. A moderate-potency corticosteroid is often prescribed to decrease the itching and subsequent inflammatory process.

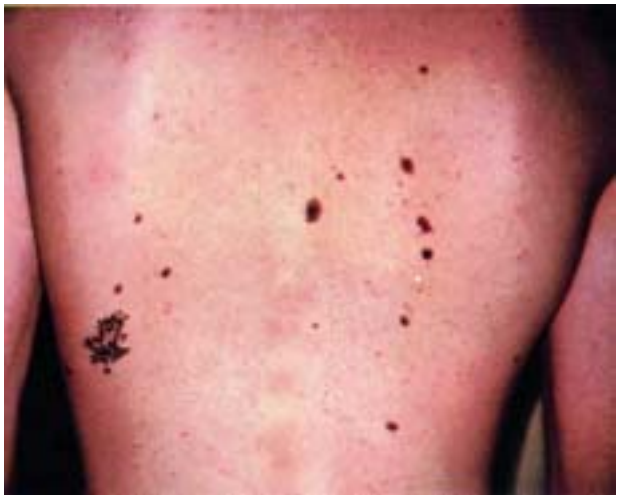
In summary, primary disorders of the skin include pigmentary skin disorders, infectious processes, inflammatory conditions, immune disorders, and allergic reactions. Pigmentary skin disorders include vitiligo, albinism, and melasma. Although the causes of the disorders vary, all involve changes in the amount of melanin produced by the melanocytes. These disorders appear in people of every skin type; however, the manifestations of the disorders vary among light-skinned and dark-skinned persons. Superficial fungal infections are called *dermatophytoses* and are commonly known as *tinea* or *ringworm*. Impetigo, which is caused by staphylococci or β-hemolytic streptococci, is the most common superficial bacterial infection. Viruses are responsible for verrucae (warts), herpes simplex type 1 lesions (cold sores or fever blisters), and herpes zoster (shingles). Noninfectious inflammatory skin conditions such as acne, lichen planus, psoriasis, and pityriasis rosea are of unknown origin. They usually are localized to the skin and are rarely associated with specific internal disease. Allergic skin responses involve the body's immune system and are caused by hypersensitivity reactions to allergens, environmental agents, drugs, and other substances.

NEVI AND SKIN CANCERS

Nevi

Nevi, or moles, are common congenital or acquired tumors of the skin that are benign. Almost all adults have nevi, some in greater numbers than others. Nevi can be pigmented or nonpigmented, flat or elevated, and hairy or nonhairy.

Nevocellular nevi are pigmented skin lesions resulting from proliferation of melanocytes in the epidermis or dermis. Nevocellular nevi are tan to deep brown, uniformly pigmented, small papules with well-defined and rounded borders. They are formed initially by melanocytes with their long dendritic extensions that are normally interspersed among the basal keratinocytes.[12] These melanocytes are transformed into round or oval melanin-containing cells that grow in nests or clusters along the dermal-epidermal junction. Because of their location, these lesions are called *junctional nevi* (Fig. 44-25). Eventually, most junctional nevi grow into the surrounding dermis as nests



■ **FIGURE 44-25** ■ Junctional nevi of the back of a 16-year-old patient. (Owen Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

or cords of cells. *Compound nevi* contain epidermal and dermal components. In older lesions, the epidermal nests may disappear entirely, leaving a *dermal nevi*. Compound and dermal nevi usually are more elevated than junctional nevi.

Another form of nevi, the *dysplastic nevi*, are important because of their capacity to transform to malignant melanomas. Although the association between dysplastic nevi and malignant melanoma was made more than 175 years ago, it was not until 1978 that the role of dysplastic nevi as a precursor of malignant melanoma was described in detail. Dysplastic nevi are larger than other nevi (often >5 mm in diameter). Their appearance is one of a flat, slightly raised plaque with a pebbly surface, or a target-like lesion with a darker, raised center and irregular border. They vary in shade from brown and red to flesh tones and have irregular borders. A person may have hundreds of these lesions. Unlike other moles or nevi, they occur on both sun-exposed and covered areas of the body. Dysplastic nevi have been documented in multiple members of families prone to the development of malignant melanoma.[12]

Because of the possibility of malignant transformation, any mole that undergoes a change warrants immediate medical attention. The changes to observe and report are changes in size, thickness, or color, and itching or bleeding.

Skin Cancer

There has been an alarming increase in skin cancers during the past several decades. Since the 1970s, the incidence rate of malignant melanoma, the most serious form of skin cancer, has increased significantly.[30] These increases are, on average, 4% per year, from 5.7 per 100,000 in 1973 to 13.8 per 100,000 in 1996, with approximately 47,700 new cases and 9600 deaths each year from melanoma. There also are approximately 1.3 million cases each year of highly curable basal cell and squamous cell cancers.

The rising incidence of skin cancer may be attributed primarily to increased sun exposure associated with societal and lifestyle shifts in the United States. The thinning of the ozone layer in the earth's stratosphere is thought to be an important

KEY CONCEPTS

SKIN CANCERS

- Increased and unprotected exposure to the ultraviolet rays of sunlight produces sunburn and increases the risk for development of skin cancer.
- The melanocytes, which protect against sunburn through increased production of melanin and suntanning, are particularly vulnerable to the adverse effects of unprotected exposure to ultraviolet light. Malignant melanoma, which is a malignant tumor of melanocytes, is a rapidly progressive and metastatic form of skin cancer.
- Basal cell carcinoma and squamous cell carcinoma, which also reflect the effects of increased sun exposure, are less aggressive forms of skin cancer and are more easily cured.

factor in this incidence rate. Society's emphasis on suntanning also is implicated. Persons have more leisure time and spend increasing amounts of time in the sun with uncovered skin.

Although the factors linking sun exposure to skin cancer are incompletely understood, both total cumulative exposure and altered patterns of exposure (in the case of melanoma) are strongly implicated. Basal cell and squamous cell carcinomas are associated with total cumulative exposure to ultraviolet radiation, whereas melanomas are associated with intense intermittent exposure. Thus, basal cell and squamous cell carcinomas occur more commonly on maximally sun-exposed parts of the body, such as the face and back of the hands and forearms. In contrast, melanomas occur most commonly in areas of the body that are exposed to the sun intermittently, such as the back in men and the lower legs in women. It is more common in persons with indoor occupations whose exposure to sun is limited to weekends and vacations.

Malignant Melanoma

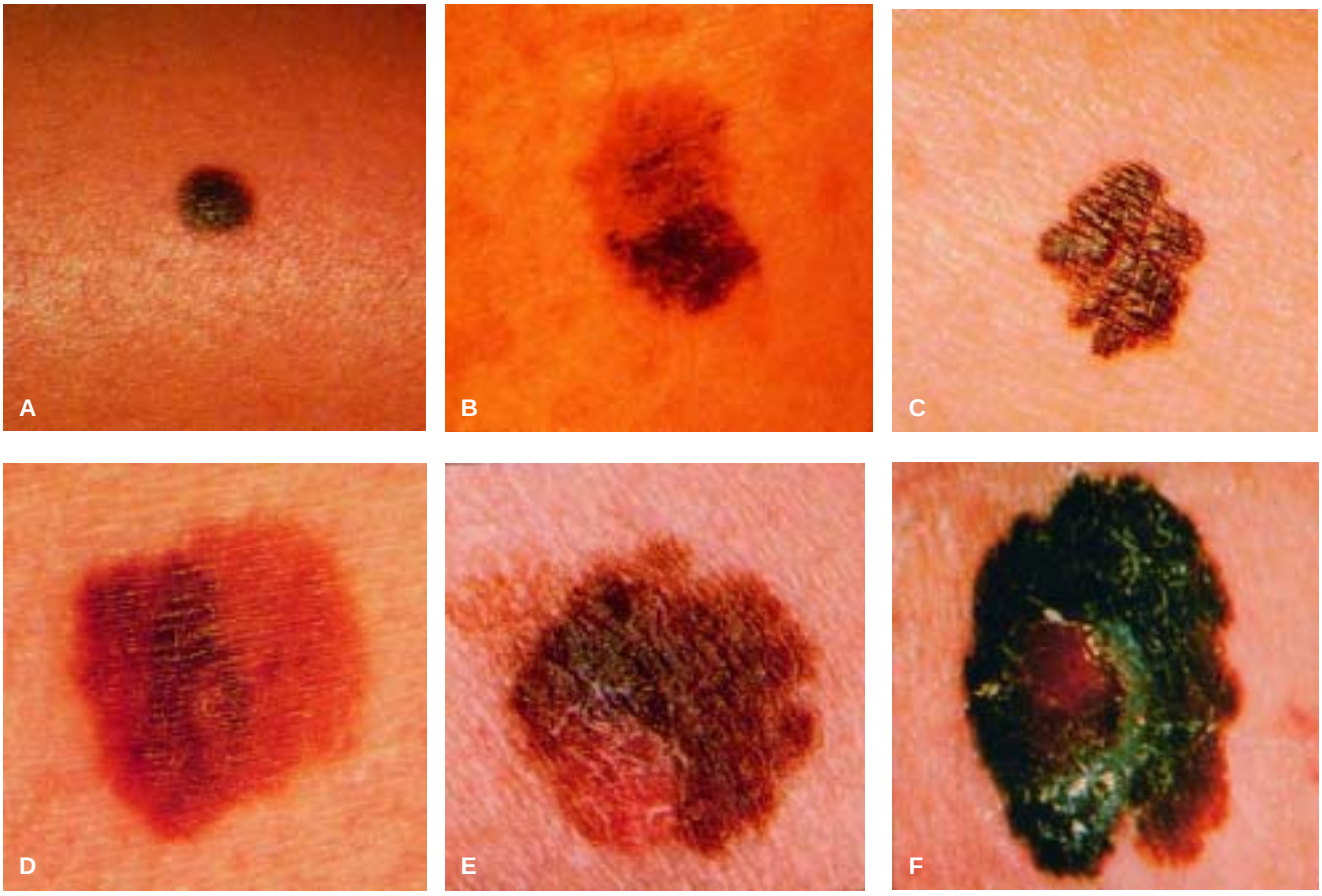
Malignant melanoma is a malignant tumor of the melanocytes. It is a rapidly progressing, metastatic form of cancer. The increased incidence of melanoma that has occurred during the past several decades has been attributed to an increase in sun exposure. The risk is greatest in fair-skinned people, particularly those with blond or red hair who sunburn and freckle easily. Fortunately, the increased risk of melanoma has been associated with a concomitant increase in the 5-year survival rate, from approximately 40% in the 1940s to 90% at present.[31] Public health screening measures, early diagnosis, increased knowledge of precursor lesions, and greater public knowledge of the disease may account for earlier intervention.

Severe, blistering sunburns in early childhood and intermittent intense sun exposures (trips to sunny climates) contribute to increased susceptibility to melanoma in young and middle-age adults. Roughly 90% of malignant melanomas in whites occur on sun-exposed skin. However, in African Americans and Asians, roughly 67% occur on non–sun-exposed areas, such as mucous membranes and subungual, palmar, and plantar surfaces.[32] Although sun exposure remains a significant risk factor for melanoma, other potential risk factors have been identified, including atypical mole/dysplastic nevus syndrome, immunosuppression, prior PUVA therapy, and exposure to ultraviolet light at tanning salons. Using statistical analysis, it has been determined that six factors independently influence the risk for development of malignant melanoma: family history of malignant melanoma, presence of blond or red hair, presence of marked freckling on the upper back, history of three or more blistering sunburns before 20 years of age, history of 3 or more years of an outdoor job as a teenager, and presence of actinic keratosis. Persons with two of these risk factors had a 3.5-fold increased risk of malignant melanoma, and those with three or more risk factors had a 20-fold increased risk.[31]

Malignant melanomas differ in size and shape. Usually, they are slightly raised and black or brown. Borders are irregular, and surfaces are uneven. Most seem to arise from pre-existing nevi or new molelike growths (Fig. 44-26). There may be surrounding erythema, inflammation, and tenderness. Periodically, melanomas ulcerate and bleed. Dark melanomas are often mottled with shades of red, blue, and white. These three colors represent three concurrent processes: melanoma growth (blue), inflammation and the body's attempt to localize and destroy the tumor (red), and scar tissue formation (white). Malignant melanomas can appear anywhere on the body. Although they frequently are found on sun-exposed areas, sun exposure alone does not account for their development. In men, they are found frequently on the trunk, head, neck, and arms; in women, they commonly are found on the legs.

Four types of melanomas have been identified: superficial spreading, nodular, lentigo maligna, and acral lentiginous.[30] *Superficial spreading melanoma* is characterized by a raised-edged nevus with lateral growth. It has a disorderly appearance in color and outline. This lesion tends to have biphasic growth, horizontally and vertically. It typically ulcerates and bleeds with growth. This type of lesion accounts for 70% of all melanomas and is most prevalent in persons who sunburn easily and have intermittent sun exposure. *Nodular melanomas*, which account for 15% to 30% of melanomas, are raised dome-shaped lesions that can occur anywhere on the body. They are commonly a uniform blue-black color and tend to look like blood blisters. Nodular melanomas tend to rapidly invade the dermis from the start with no apparent horizontal growth phase. *Lentigo maligna* melanomas, which account for 4% to 10% of all melanomas, are slow growing, flat nevi that occur primarily on sun-exposed areas of elderly persons. Untreated lentigo maligna tends to exhibit horizontal and radial growth for many years before it invades the dermis to become lentigo maligna melanoma. *Acral lentiginous melanoma*, which accounts for 2% to 8% of melanomas, occurs primarily on the palms of the hands, soles of the feet, nail beds, and mucous membranes. It has the appearance of lentigo maligna. Unlike other types of melanomas, it has a similar incidence in all ethnic groups.

Because virtually all the known risks of melanoma are related to susceptibility and magnitude of ultraviolet light exposure, protection from the sun's rays plays a critical role in the prevention of malignant melanoma. Early detection is critical with malignant melanoma. Regular self-examination of the total skin surface in front of a mirror under good lighting provides a method for early detection. It requires that a person undress completely and examine all areas of the body using a full



■ **FIGURE 44-26** ■ (**A**) Normal mole with even, round contour and sharply defined borders. (**B**) Changes in appearance of a mole: *asymmetry.* (**C**) Changes in appearance of a mole: *border irregularity.* (**D**) Changes in appearance of a mole: *color and uneven pigmentation.* (**E**) Changes in the appearance of a mole: *diameter greater than 6 mm.* (**F**) Changes in the surface of a mole: *scaliness, oozing, and bleeding.* (American Cancer Society. [1995]. *What you should know about melanoma.* Dallas: American Cancer Society)

mirror, handheld mirror, and handheld hair dryer (to examine the scalp). An *ABCD* rule has been developed to aid in early diagnosis and timely treatment of malignant melanoma.[31] The acronym stands for *a*symmetry, *b*order irregularity, *c*olor variegation, and *d*iameter greater than 0.6 cm (pencil eraser size). People should be taught to watch for these changes in existing nevi or the development of new nevi, as well as other alterations such as bleeding or itching.

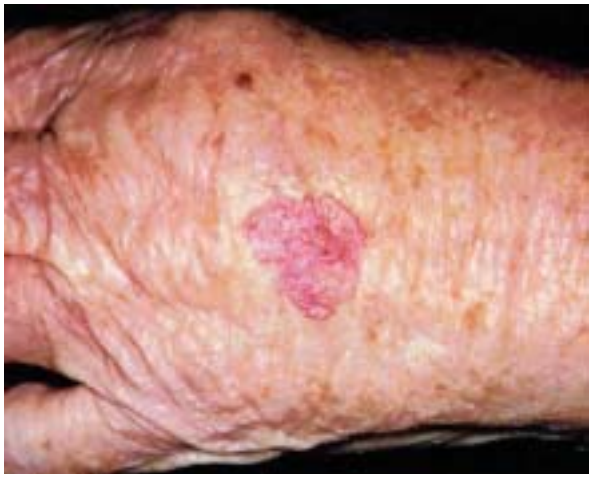
Diagnosis of melanoma is based on biopsy findings from a suspect lesion. Treatment is usually surgical excision, the extent of which is determined by the thickness of the lesion, invasion of the deeper skin layers, and spread to regional lymph nodes. Deep and wide excisions, with possible use of skin grafts, are used. Current cancer treatment, such as immunotherapy and chemotherapy, is indicated when the disease becomes systemic. Interferon alfa-2b is approved by the FDA for treatment of melanoma.[30,31]

The prognosis for malignant melanoma varies. It depends on factors such as tumor thickness (measured in millimeters), anatomic site, type of lesion, and levels of invasion (degree of penetration in the anatomic layers of the skin). Tumor thickness is an important factor in determining prognosis in persons with malignant melanoma.

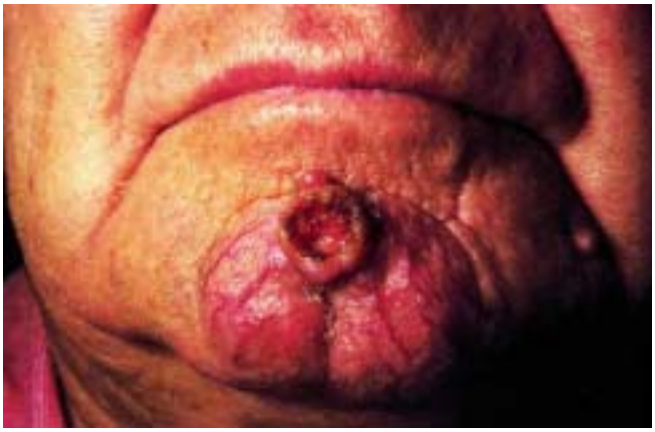
Basal Cell Carcinoma

Basal cell carcinoma is the most common skin cancer in humans, accounting for 75% of all nonmelanoma skin cancers.[34] Like other skin cancers, basal cell carcinoma has increased in incidence during the past several decades. Basal cell carcinoma usually occurs in persons who were exposed to great amounts of sunlight. The incidence is twice as high among men as women and greatest in the 55- to 75-year-old age group.

Basal cell carcinoma usually is a nonmetastasizing tumor that extends wide and deep if left untreated. These tumors are seen most frequently on sun-exposed areas of the body, such as the head and neck, but do occur on other skin surfaces that were not exposed to the sun (Fig. 44-27). Although there are several histologic types of basal cell carcinoma, nodular ulcerative and superficial basal cell carcinomas are the most frequently occurring types. *Nodular ulcerative basal cell carcinoma* is the most common type.[34] It is a nodulocystic structure that begins as a small, flesh-colored or pink, smooth, translucent nodule that enlarges with time. Telangiectatic vessels frequently are seen beneath the surface. Over the years, a central depression forms that progresses to an ulcer surrounded by the original shiny, waxy border. Basal cell carcinoma in darker-skinned persons usually is darkly pigmented



■ **FIGURE 44-27** ■ Basal cell carcinoma and wrinkling of the hand. (Syntex Laboratories.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



■ **FIGURE 44-28** ■ Squamous cell carcinoma of the chin. (Syntex Laboratories, Westwood Pharmaceuticals.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

and frequently misdiagnosed as other skin diseases, including melanoma.

The second most common form is *superficial basal cell carcinoma*, which is seen most often on the chest or back. It begins as a flat, nonpalpable, erythematous plaque. The red, scaly areas slowly enlarge, with nodular borders and telangiectatic bases. This type of skin cancer is difficult to diagnose because it mimics other dermatologic problems.

All suspected basal cell carcinomas should undergo biopsy for diagnosis. The treatment depends on the site and extent of the lesion. The most important treatment goal is complete elimination of the lesion. Also important is the maintenance of function and optimal cosmetic effect. Curettage with electrodesiccation, surgical excision, irradiation, and chemosurgery are effective in removing all cancerous cells. Patients should be checked at regular intervals for recurrences.

Squamous Cell Carcinoma

Squamous cell carcinomas are malignant tumors of the outer epidermis. They are commonly found on the sun-exposed area of the skin of people with fair complexions.[34] Metastasis is more common with squamous cell carcinoma than with basal cell carcinoma.

The mechanisms of squamous cell carcinoma development are unclear. Most squamous cell cancers occur in sun-exposed areas of the skin, and persons who spend much time outdoors, have lighter skin, and live in lower latitudes are more affected. The increase in the incidence of squamous cell carcinomas is consistent with increased ultraviolet radiation exposure. Other suspected causes include exposure to arsenic (*i.e.*, Bowen's disease), gamma radiation, tars, and oils.

There are two types of squamous cell carcinoma: intraepidermal and invasive. *Intraepidermal squamous cell carcinoma* remains confined to the epidermis for a long time. However, at some unpredictable time, it may penetrate the basement membrane to the dermis and metastasize to the regional lymph nodes. It then converts to *invasive squamous cell carcinoma*. The invasive type can develop from intraepidermal carcinoma or from a premalignant lesion (*e.g.*, actinic keratoses). It may be slow growing or fast growing with metastasis.

Squamous cell carcinoma is a red-scaling, keratotic, slightly elevated lesion with an irregular border, usually with a shallow chronic ulcer (Fig. 44-28). Later lesions grow outward, show large ulcerations, and have persistent crusts and raised, erythematous borders. The lesions characteristically occur on the nose, forehead, helixes of the ears, lower lip, and back of the hands. In blacks, the lesions may appear as hyperpigmented nodules and occur more frequently on non–sun-exposed areas.

Treatment measures are aimed at the removal of all cancerous tissue using methods such as electrosurgery, excision surgery, chemosurgery, or radiation therapy. After treatment, the person is observed for the remainder of his or her life for signs of recurrence. The recurrence rate is roughly 50%, with a 70% metastatic rate.[35]

In summary, nevi are moles that usually are benign. Because they may undergo cancerous transformation, any mole that undergoes a change warrants immediate medical attention. There has been an alarming increase in skin cancers during the past few decades. Repeated exposure to the ultraviolet rays of the sun has been implicated as the principal cause of skin cancer. Neoplasms of the skin include malignant melanoma, basal cell carcinoma, and squamous cell carcinoma. Malignant melanoma is a malignant tumor of the melanocytes. It is a rapidly progressing, metastatic form of cancer. The most important clinical sign is the change in size, shape, and color of a pigmented skin lesion, such as a mole. Early detection through skin self-examination is critical. Squamous cell carcinoma and basal cell carcinoma are of epidermal origin. Basal cell carcinomas are the most common form of skin cancer among whites. They are slow-growing tumors that rarely metastasize. Squamous cell carcinoma is common in pale-skinned elderly persons. The two types of squamous cell carcinoma are intraepidermal and invasive. Intraepidermal squamous cell carcinoma remains confined to the epidermis for a long time. Invasive squamous cell carcinoma can develop from intraepidermal carcinoma or from premalignant lesions such as actinic keratoses.

AGE-RELATED SKIN MANIFESTATIONS

Many skin problems occur more commonly in certain age groups. Because of aging changes, infants, children, and elderly persons tend to have different skin problems.

Skin Disorders in Infants and Children

Skin disorders in infants and children may be different from those in older children and adults. Certain skin disorders such as diaper rash and cradle cap are seen only in infants. Other conditions such as measles and chickenpox are more common in school age children.

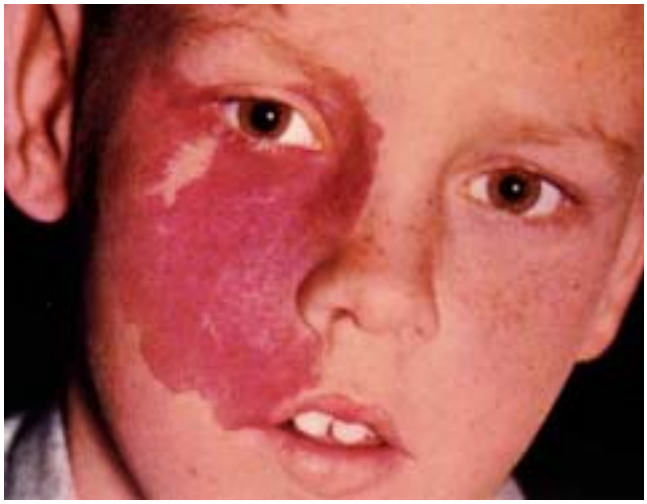
Skin Disorders in Infants

Infancy connotes the image of perfect, unblemished skin. For the most part, this is true. However, several congenital skin lesions, such as mongolian spots, hemangiomas, and nevi, are associated with the early neonatal period.

Vascular and Pigmented Birthmarks. Pigmented and vascular lesions comprise most birthmarks.[36] Pigmented birthmarks represent abnormal migration or proliferation of melanocytes. Mongolian spots are caused by selective pigmentation. They usually occur on the buttocks or sacral area and are seen commonly in Asians and blacks. Nevi or moles are small, tan to brown, uniformly pigmented solid macules. *Nevocellular nevi* are formed initially from aggregates of melanocytes and keratinocytes along the dermal-epidermal border. *Congenital melanocytic nevi* are collections of melanocytes that are present at birth or develop within the first year of life. They present as macular, papular, or plaquelike pigmented lesions of various shades of brown, with a black or blue focus. The texture of the lesions varies, and they may be with or without hair. They usually are found on the hands, shoulders, buttocks, entire arm, or trunk of the body. Some involve large areas of the body in garment-like fashion. They usually grow proportionately with the child. Congenital melanocytic nevi are clinically significant because of their association with malignant melanoma.

Vascular birthmarks are cutaneous anomalies of angiogenesis and vascular development.[36] Two types of vascular birthmarks commonly are seen in infants and small children: bright red raised strawberry hemangiomas and flat, reddish-purple port-wine stains.

The strawberry hemangiomas begin as small, red lesions that are noticed shortly after birth. Hemangiomas are benign vascular tumors produced by proliferation of the endothelial cells. They are seen in approximately 5% to 10% of 1-year-old children.[37] Female infants are three times as likely as male infants to have hemangiomas, and there is an increased incidence in premature infants. Approximately 35% of these lesions are present at birth, and the remainder develop within a few weeks after birth. Hemangiomas typically undergo an early period of proliferation during which they enlarge, followed by a period of slow involution in which the growth is reversed, and finally complete resolution. Most strawberry hemangiomas disappear before 5 to 7 years of age without leaving an appreciable scar. Hemangiomas can occur anywhere in the body. Hemangiomas of the airway can be life threatening. Ulceration, the most frequent complication, can be painful and carries the risk of infection, hemorrhage, and scarring.[37]



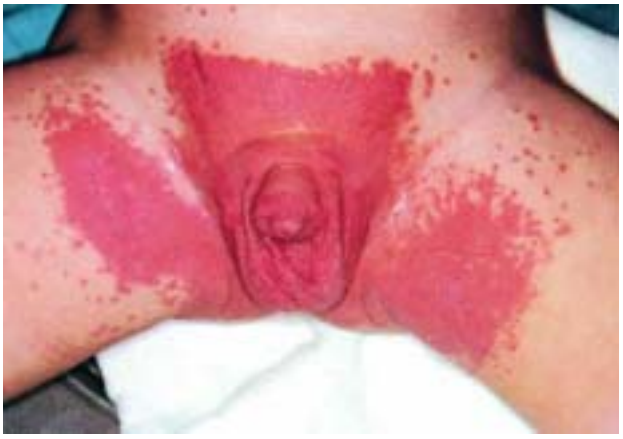
■ **FIGURE 44-29** ■ Port-wine stain on the face of a boy. (Ortho Dermatology Corp.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

Port-wine stains are pink or red patches that can occur anywhere on the body and are very noticeable (Fig. 44-29). They represent slow-growing capillary malformations that grow proportionately with the child and persist throughout life. Port-wine stains usually are confined to the skin but may be associated with vascular malformations of the eye or leptomeninges over the cortex, leading to cognitive disorders, seizures, and other neurologic deficits. Cover-up cosmetics are used in an attempt to conceal their disfiguring effects. Laser surgery has been used effectively in the treatment of port-wine stains.

Diaper Rash. The appearance of diaper rash ranges from simple (*i.e.*, widely distributed macules on the buttocks and anogenital areas) to severe (*i.e.*, beefy, red, excoriated skin surfaces in the diaper area).[38] It results from a combination of ammonia and other breakdown products of urine. The treatment includes measures to minimize or prevent skin wetness. It includes frequent diaper changes with careful cleaning of the irritated area to remove the waste products. This is particularly important in hot weather. Exposing the irritated area to air is helpful. Secondary candidal (*i.e.*, yeast) infections may occur and require additional treatment (Fig. 44-30).

Prickly Heat. Prickly heat (heat rash) results from constant maceration of the skin because of prolonged exposure to a warm, humid environment. Maceration leads to midepidermal obstruction and rupture of the sweat glands. Although commonly seen during infancy, prickly heat may occur at any age. The treatment includes removing excessive clothing, cooling the skin with warm water baths, drying the skin with powders, and avoiding hot, humid environments.

Cradle Cap. Cradle cap is a greasy crust or scale formation on the scalp. It usually is attributed to infrequent and inadequate washing of the scalp. Cradle cap is treated by mild shampooing and gentle combing to remove the scales. Sometimes oil can be left on the head for minutes to several hours, softening



■ **FIGURE 44-30** ■ *Candida* intertrigo after a course of oral antibiotics in a 1-year-old child. (Owen Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

the scales before scrubbing. Other emulsifying ointments or creams may be helpful in difficult cases. The scalp may need to be rubbed firmly to remove the buildup of keratinized cells.

Skin Manifestations of Common Infectious Diseases

Infectious childhood diseases that produce rashes include roseola infantum, rubella, rubeola, and varicella. Although these diseases are seen less frequently because of successful immunization programs and the use of antibiotics, they still occur. Immunization has greatly reduced the incidence of rubella and rubeola. Varicella vaccine is also available.

Roseola Infantum. Roseola infantum (*i.e.*, exanthema subitum) is a contagious viral disease of infants and young children, most frequently between 6 and 18 months of age. It is caused by human herpesvirus-6 and produces a characteristic maculopapular rash covering the trunk and spreading to the appendages. The rash is preceded by an abrupt onset of high fever (≤105°F), inflamed tympanic membranes, and coldlike symptoms usually lasting 3 to 4 days. These symptoms improve at approximately the same time the rash appears. Unlike rubella, no cervical or postauricular lymph node adenopathy occurs. Roseola infantum frequently is mistaken for rubella. Rubella usually can be excluded by the age of the child and the absence of lymph node adenopathy. In general, rubella does not develop in children younger than 6 to 9 months of age because they retain some maternal antibodies. Blood antibody titers may be taken to determine the actual diagnosis. In most cases, there are no long-term effects associated with this disease.

Rubella. Rubella (*i.e.*, 3-day measles or German measles) is a childhood disease caused by the rubella virus. It is characterized by a diffuse, punctate, macular rash that begins on the trunk and spreads to the arms and legs. Mild febrile states occur; usually the fever is less than 100°F. Postauricular, suboccipital, and cervical lymph node adenopathy is common. Coldlike symptoms (*e.g.*, nasal congestion and cough) usually accompany the disease.

Rubella usually has no long-lasting sequelae; however, the transmission of the disease to pregnant women early in their gestation periods may result in congenital rubella syndrome. Among the clinical signs of congenital rubella syndrome are cataracts, microcephaly, mental retardation, deafness, patent ductus arteriosus, glaucoma, purpura, and bone defects.

Rubeola. Rubeola (measles, hard measles, 7-day measles) is an acute, highly communicable viral disease caused by *Morbillivirus*. The characteristic rash is macular and blotchy; sometimes the macules become confluent. The rubeola rash usually begins on the face and spreads to the appendages. There are several accompanying symptoms: a fever of 100°F or greater, Koplik's spots (*i.e.*, small, irregular red spots with a bluish-white speck in the center) on the buccal mucosa, and mild to severe photosensitivity. The patient commonly has coldlike symptoms, general malaise, and myalgia. In severe cases, the macules may hemorrhage into the skin tissue or onto the outer body surface. This form is called *hemorrhagic measles*. The course of measles is more severe in infants, adults, and malnourished children. There may be severe complications, including otitis media, pneumonia, and encephalitis. Antibody titers are determined for a conclusive diagnosis of rubeola.

Varicella. Varicella (chickenpox) is a common communicable childhood disease. It is caused by the varicella-zoster virus, which also is the agent in herpes zoster (shingles). The characteristic skin lesion occurs in three stages: macule, vesicle, and granular scab. The macular stage is characterized by development within hours of macules over the trunk of the body, spreading to the limbs, buccal mucosa, scalp, axillae, upper respiratory tract, and conjunctiva. During the second stage, the macules form vesicles with depressed centers. The vesicles break open, and a scab forms during the third stage. Crops of lesions occur successively, so that all three forms of the lesion usually are visible by the third day of the illness.

Mild to extreme pruritus accompanies the lesions, which can lead to scratching and subsequent development of secondary bacterial infections. Chickenpox also is accompanied by coldlike symptoms and sometimes photosensitivity. Mild febrile states usually occur, typically beginning 24 hours before lesion outbreak. Side effects, such as pneumonia, septic complications, and encephalitis, are rare.

Varicella in adults may be more severe, with a prolonged recovery rate and greater chances for development of varicella pneumonitis or encephalitis. Immunocompromised persons may experience a chronic, painful type.

Skin Disorders in the Elderly

Elderly persons experience a variety of age-related skin disorders and exacerbations of earlier skin problems. Aging skin is believed to involve a complex process of actinic (solar) damage, normal aging, and hormonal influences.[39] Actinic changes primarily involve increased occurrence of lesions on sun-exposed surfaces of the body.

Normal Age-Related Changes

Normal aging consists of changes that occur on areas of the body that have not been exposed to the sun. They include thinning of the dermis and the epidermis, diminution in subcutaneous tissue, a decrease in and thickening of blood vessels, and a decrease in the number of melanocytes, Langerhans' cells,

and Merkel's cells. The keratinocytes shrink, but the number of dead keratinized cells at the surface increases. This results in less padding and thinner skin, with color and elasticity changes. The skin also loses its resistance to environmental and mechanical trauma. Tissue repair takes longer.

With aging, there is also less hair and nail growth, and there is permanent hair pigment loss. There is normally less hormonally influenced sebaceous gland activity, although the glands in the facial skin may increase in size. The skin in most persons older than 70 years becomes dry, rough, scaly, and itchy. When there is no underlying pathology, it is called *senile pruritus*. Itching and dryness often become worse during the winter, when the need for home heating lowers the humidity.

Common Skin Lesions

The most common skin lesions in the elderly are skin tags, keratoses, lentigines, and vascular lesions. Most are actinic manifestations; they occur as a result of exposure to sun and weather throughout the years.

Skin Tags. Skin tags are soft, brown or flesh-colored papules. They occur on any skin surface but most frequently the neck, axilla, and intertriginous areas. They range in size from a pinhead to the size of a pea. Skin tags have the normal texture of the skin. They are benign and can be removed with scissors or electrodesiccation for cosmetic purposes.

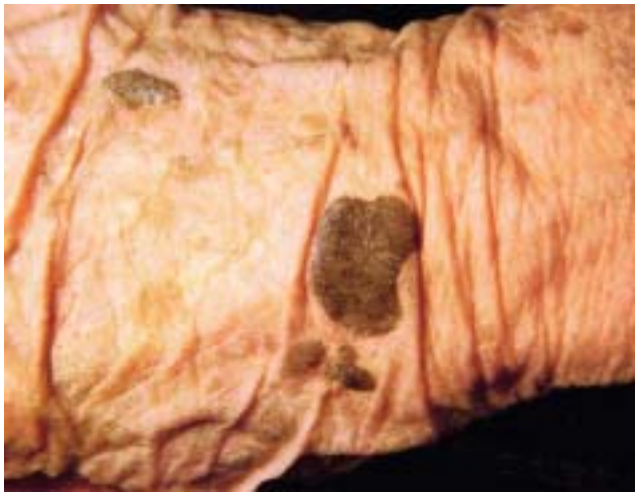
Keratoses. A *keratosis* is a horny growth or an abnormal growth of the keratinocytes. A *seborrheic keratosis* (*i.e.*, seborrheic wart) is a benign, sharply circumscribed, wartlike lesion that has a stuck-on appearance (Fig. 44-31). They vary in size up to several centimeters. They are usually round or oval, tan, brown, or black lesions. Less pigmented ones may appear yellow or pink. Keratoses can be found on the face or trunk as a solitary lesion or sometimes by the hundreds. Seborrheic keratoses are benign, but they must be watched for changes in color, texture, or size, which may indicate malignant transformation to squamous cell carcinoma.

Actinic keratoses are the most common premalignant skin lesions that develop on sun-exposed areas. The lesions usually are less than 1 cm in diameter and appear as dry, brown scaly areas, often with a reddish tinge. Actinic keratoses often are multiple and more easily felt than seen (Fig. 44-32). They often are indistinguishable from squamous cell carcinoma without biopsy. A hyperkeratotic form also exists that is more prominent and palpable. Often, there is a weathered appearance of the surrounding skin. Slight changes, such as enlargement or ulceration, may indicate malignant transformation. Most actinic keratoses are treated with 5-fluorouracil cream, which erodes the lesions. Roughly 20% of actinic keratoses convert to squamous cell carcinomas.

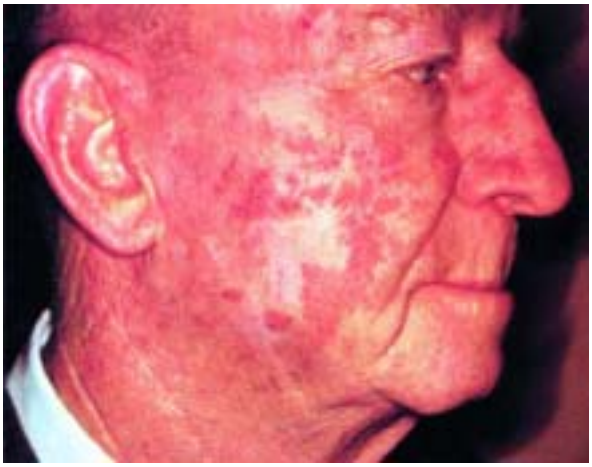
Lentigines. A *lentigo* is a well-bordered brown to black macule, usually less than 1 cm in diameter. *Solar lentigines* are tan to brown, benign spots on sun-exposed areas. They are commonly referred to as *liver spots*. Creams and lotions containing hydroquinone (*e.g.*, Eldoquin, Solaquin) may be used temporarily to bleach the spots. These agents inhibit the synthesis of new pigment without destroying existing pigment. Higher concentrations are available by prescription. Successful treatment depends on avoiding sun exposure and consistent use of sunscreens. Liquid nitrogen applications have been successful in eradicating senile lentigines.

Lentigo maligna (*i.e.*, Hutchinson's freckle) is a slowly progressive (≤20 years) preneoplastic disorder of melanocytes. It occurs on sun-exposed areas, particularly the face. The lesion is a pigmented macule with a well-defined border and grows to 5 cm or sometimes larger. As it grows throughout the years, it may become slightly raised and wartlike. If untreated, a true malignant melanoma often develops. Surgery, curettage, and cryotherapy have been effective at removing the lentigines. Careful monitoring for conversion to melanoma is important.

Vascular Lesions. Vascular lesions are vascular tumors with chronically dilated blood vessels. The small blood vessels lie in



■ **FIGURE 44-31** ■ Large seborrheic keratoses on the hand of an 84-year-old woman. (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)



■ **FIGURE 44-32** ■ Multiple actinic keratoses of the face of an 80-year-old man. (Dermik Laboratories, Inc.) (Sauer G.C., Hall J.C. [1996]. *Manual of skin diseases* [7th ed.]. Philadelphia: Lippincott-Raven)

the middle to upper dermis. *Senile angiomas* (cherry angiomas) are smooth, cherry-red or purple, dome-shaped papules. They usually are found on the trunk. *Telangiectases* are single dilated blood vessels, capillaries, or terminal arteries that appear on areas exposed to sun or harsh weather, such as the cheeks and the nose. The lesions can become large and disfiguring. Pulsed dye lasers have been effective in removing them. *Venous lakes* are small, dark blue, slightly raised papules that have a lakelike appearance. They occur on exposed body parts, particularly the backs of the hands, ears, and lips. They are smooth and compressible. Venous lakes can be removed by electrosurgery, laser therapy, or surgical excision if a person desires.

In summary, some skin problems occur in specific age groups. Common in infants are diaper rash, prickly heat, and cradle cap. Infectious childhood diseases that are characterized by rashes include roseola infantum, rubella, rubeola, and varicella.

Changes in skin that occur with aging involve a complex process of actinic damage, normal aging, and hormonal influences. With aging, there is thinning of the dermis and the epidermis, diminution in subcutaneous tissue, lessening and thickening of blood vessels, and a slowing of hair and nail growth. Dry skin is common among the elderly, becoming worse during the winter months. Among the skin lesions seen in the elderly are skin tags, keratoses, lentigines, and vascular skin lesions.

REVIEW QUESTIONS

- Contrast and compare the following lesions: macule, patch, papule, plaque, nodule, pustule, blister, callus, and corn.
- Describe the three types of ultraviolet radiation and relate them to sunburn, aging skin changes, and the development of skin cancer.
- Relate the behavior of fungi to the production of superficial skin lesions associated with tinea or ringworm.
- State the cause and describe the appearance of impetigo and ecthyma.
- Compare the viral causes, manifestations, and treatments of verrucae, herpes simplex, and herpes zoster lesions.
- Describe the pathogenesis of acne vulgaris and relate it to measures used in treating the disorder.
- Describe the differences and similarities between erythema multiforme minor, Stevens-Johnson syndrome, and toxic epidermal necrolysis.
- Define the term *papulosquamous* and use the term to describe the lesions associated with psoriasis, pityriasis rosea, and lichen planus.
- Describe the origin of nevi and state their relationship to skin cancers.
- Compare the appearance and outcome of basal cell carcinoma, squamous cell carcinoma, and malignant melanoma.
- Describe the distinguishing features of rashes associated with the common infectious childhood diseases: roseola infantum, rubeola, rubella, chickenpox, and scarlet fever.
- Describe the appearance of skin tags, keratoses, lentigines, and vascular lesions that are commonly seen in the elderly.

connection

Visit the Connection site at connection.lww.com/go/porth for links to chapter-related resources on the Internet.

REFERENCES

1. Harrist T.J., Schapiro B., Quinn T.R., et al. (1999). The skin. In Rubin E., Farber J.L. (Eds.), *Pathology* (3rd ed., pp. 1237–1298). Philadelphia: Lippincott Williams & Wilkins.
2. Watson K.R. (2000). Structure of the skin. In Hall J.C. (Ed.), *Sauer's manual of skin diseases* (8th ed., pp. 1–5). Philadelphia: Lippincott Williams & Wilkins.
3. Greaves M.H. (1998). In Champion R.H., Burton J.L., Burns D.A., et al. (Eds.), *Textbook of dermatology* (6th ed., pp. 617–618). Oxford: Blackwell Science.
4. American Academy of Dermatology. (1987). Black skin. [On-line]. Available: http://www.aad.org/pamphlets/ black.html.
5. Diffey B.L. (1992). Human exposure to ultraviolet violet radiation. In Marks R., Plewig G. (Eds.), *The environmental threat to the skin* (pp. 3–9). London: Martin Dunitz.
6. Gilchrest B.A., Eller M.S., Geller A.C., et al. (1999). The pathogenesis of melanoma induced by ultraviolet radiation. *New England Journal of Medicine* 340, 1341–1348.
7. Hall J.C. (Ed.). (2000). *Sauer's manual of skin diseases* (8th ed., p. 295). Philadelphia: Lippincott Williams & Wilkins.
8. Drug Facts and Comparisons Staff. (2000). *Drug facts and comparisons 2000* (54th ed., p. 1714). St. Louis: Facts and Comparisons.
9. Epstein J., Kaplan L., Levine N. (2000). The value of sunscreens. *Patient Care* 34 (11), 103–107.
10. Brooks G.F., Butel J.S., Ornston L.N. (1995). *Medical microbiology* (20th ed., pp. 531–536). Norwalk, CT: Appleton & Lange.
11. Noble S.L., Forbes R.C. (1998). Diagnosis and management of common tinea infections. *American Family Physician* 58, 177.
12. Murphy G.F. (2003). The skin. In Kumar V., Cotran R.S., Robbins S.L. (Eds.), *Basic pathology* (7th ed., pp. 789–807). Philadelphia: W.B. Saunders.
13. Gilden D.H., Kleinschmidt-DeMasters B.K., LaGuardia J.J., et al. (2000). Neurologic complications of reactivation of varicella-zoster virus. *New England Journal of Medicine* 342, 635–645.
14. Stankus S.J., Dlugopolski M., Packer D. (2000). Management of herpes zoster (shingles) and postherpetic neuralgia. *American Family Physician* 61, 2437–2444, 2447–2448.
15. Tyring S.K. (1996). Early treatment of herpes zoster. *Hospital Practice* 31, 137–144.
16. Landow K. (2000). Acute and chronic herpes zoster. An ancient scourge yields to timely therapy. *Postgraduate Medicine* 107 (7), 107–118.
17. Hall J.C. (Ed.). (2000). *Sauer's manual of skin diseases* (8th ed., pp. 114–126, 127–144). Philadelphia: Lippincott Williams & Wilkins.
18. Usantine R.P., Quan M.A., Strick R. (1998). Acne vulgaris: A treatment update. *Hospital Practice* 33 (2), 111–127.
19. Krowchuk D.P. (2000). Managing acne in adolescents. *Pediatric Clinics of North America* 47, 841–857.
20. Leyden J.J. (1997). Therapy for acne vulgaris. *New England Journal of Medicine* 336, 1156–1163.
21. Correale C.E., Walker C., Craig T.J. (1999). Atopic dermatitis: A review of diagnosis and treatment. *American Family Physician* 60, 1191–1210.

22. Kristal L., Klein P.A. (2000). Atopic dermatitis in infants and children. *Pediatric Clinics of North America* 47, 877–894.
23. Scott C.B., Moloney M.F. (1996). Physical urticaria. *Nurse Practitioner* 21 (11), 42–59.
24. Greaves M.W. (1995). Chronic urticaria. *New England Journal of Medicine* 332, 1767–1772.
25. Bastuji-Garin S., Rzany B., Stern R.S., et al. (1993). Clinical classification of cases of toxic epidermal necrolysis, Stevens-Johnson syndrome, and erythema multiforme. *Archives of Dermatology* 129, 92–96.
26. Roujeau J.C., Stern R.S. (1994). Severe adverse cutaneous reactions to drugs. *New England Journal of Medicine* 331, 1272–1284.
27. Koo J.Y. (1999). Current consensus and update on psoriasis therapy: A perspective from the U.S. *Journal of Dermatology* 26, 723–733.
28. Camisa C. (1994). *Psoriasis* (pp. 3, 30, 31, 55). Boston: Blackwell.
29. Katta R. (2000). Lichen planus. *American Family Physician* 61, 3319–3324, 3327–3328.
30. Urist M.M., Heslin M.J., Miller D.M. (2001). Malignant melanoma. In Lenhard R.E., Osteen R.T., Gansler T. (Eds.), *Clinical oncology* (pp. 553–563). Atlanta: American Cancer Society.
31. Rigel D.S., Carucci J.A. (2000). Malignant melanoma: Prevention, early detection, and treatment in the 21st century. *CA: A Cancer Journal for Clinicians* 50, 215–236.
32. Halder R.M., Bridgeman-Shah S. (1995). Skin cancer in African Americans. *Cancer* 75, 667–673.
33. Jerant A.F., Johnson J.T., Sheridan C.M., et al. (2000). Early detection and treatment of cancer. *American Family Physician* 62, 357–368, 375–376, 381–382.
34. Preston D.S., Stern R.S. (1992). Nonmelanoma cancers of the skin. *New England Journal of Medicine* 327, 1649–1662.
35. Frankel D.H. (1992). Squamous cell carcinoma of the skin. *Hospital Practice* 27, 99–106.
36. Dohil M.A., Baugh W.P., Eichenfield L.F. (2000). Vascular and pigmented birthmarks. *Pediatric Clinics of North America* 47, 783–810.
37. Drolet B.A., Esterly N.B., Frieden I.J. (1999). Hemangiomas in children. *New England Journal of Medicine* 341, 173–181.
38. Kazaks E.L., Lane A.T. (2000). Diaper dermatitis. *Pediatric Clinics of North America* 47, 909–918.
39. Bolognia J.L. (1995). Aging skin. *American Journal of Medicine* 98, 99S–103S.