CHAPTER 13

Integumentary Function

LEARNING OBJECTIVES

- Discuss normal integumentary anatomy and physiology.
- Differentiate congenital integumentary disorders.
- Differentiate integumentary changes and conditions associated with aging.
- Differentiate inflammatory integumentary disorders.
- Differentiate infectious integumentary disorders.
- Differentiate traumatic integumentary disorders.
- Differentiate pressure injuries.
- Differentiate integumentary cancers.
- Apply understanding of various common integumentary disorders such as age-related lesions, infectious and inflammatory disorders, traumatic and pressure injuries, acne, and skin cancer.
- Develop diagnostic and treatment considerations for various integumentary disorders.

The integumentary system protects the body from pathogen invasions, regulates temperature, senses environmental changes, and maintains water balance. This system comprises the skin, nails, hair, mucous membranes, and glands. Disorders of the integumentary structures can result in numerous issues because of the extensive functions of this system. Such disorders can stem from a wide range of causes, including congenital defects, advancing age, inflammation, infections, and cancers.

The skin, just like a book cover, can provide an abundance of information pertaining to internal bodily functions. Many skin conditions, such as birthmarks, are mild and may not require treatment, whereas others, such as skin cancer, can be life threatening.

Anatomy and Physiology

The skin, along with the nails, hair, mucous membranes, and glands, constitutes the integumentary system. In addition to participating in sensory functions, the integumentary system plays a key role in immunity, temperature regulation, and water balance. Moreover, this system excretes small amounts of waste products. The integumentary system is the body's largest organ system, covering all external surfaces and accounting for approximately 15% of the body's weight.

The skin consists of three layers—the epidermis, the dermis, and the hypodermis (**Figure 13-1**). The epidermis, or outermost layer of the skin, comprises squamous epithelia, or flat sheets of cells. The dermis, the middle layer, is composed of dense, irregular connective tissue and very little fatty tissue. The dermis includes nerves, hair follicles, smooth muscle, glands, blood vessels, and lymphatic vessels. The hypodermis, or subcutaneous tissue, is the innermost layer of the skin, consisting of soft, fatty tissue as well as blood vessels, nerves, and immune cells (e.g., macrophages).

Epidermis

The epidermis consists of five distinct layers (**Figure 13-2**).

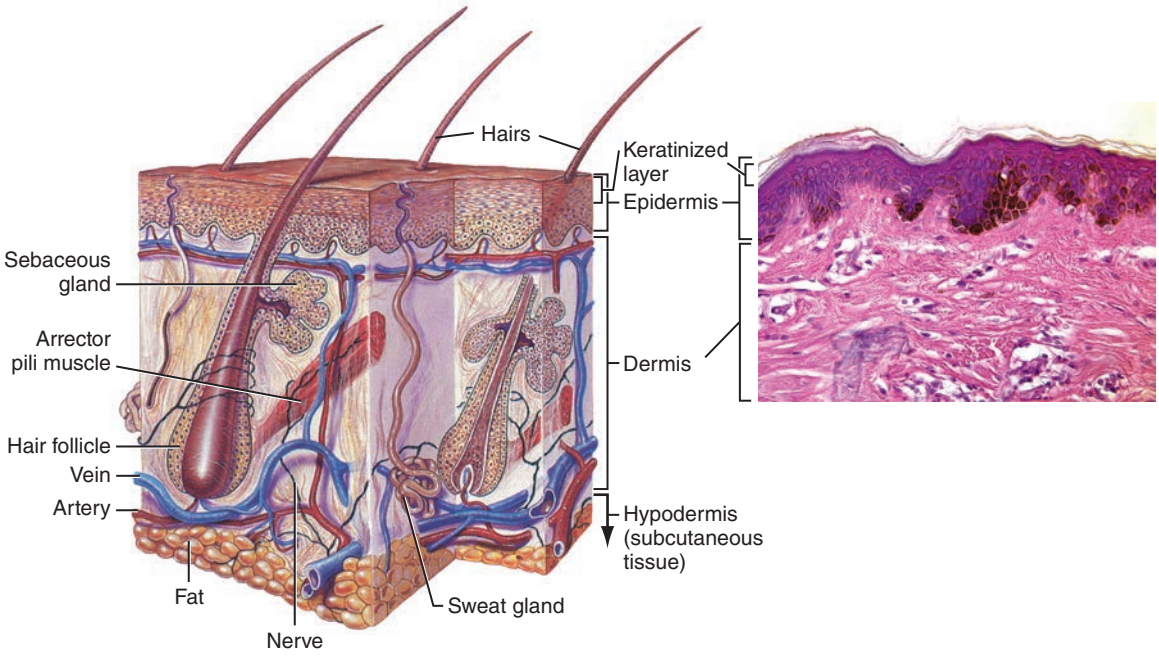


Figure 13-1 The layers of the skin.

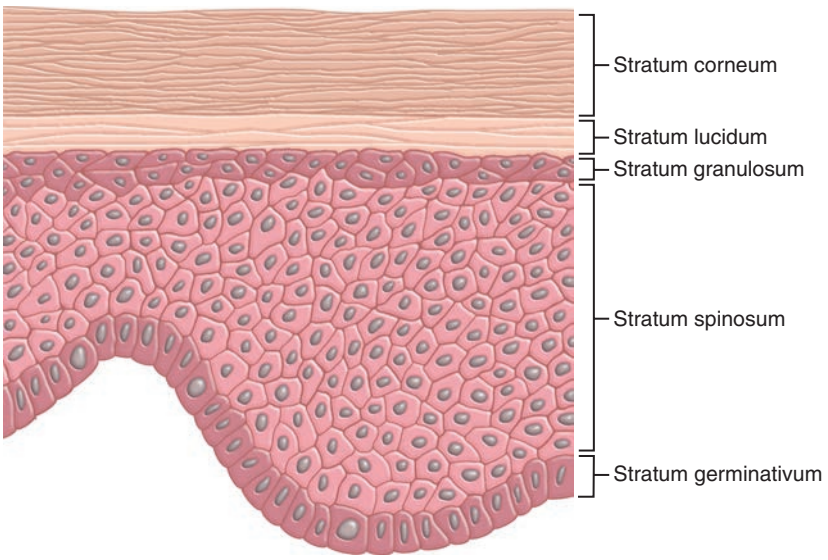


Figure 13-2 The layers of the epidermis.

1. **Stratum corneum:** The outermost layer of body protection against environment. The stratum corneum is composed of waterproof keratin.
2. **Stratum lucidum:** The location of transitional cells that maintain function. Cells appear as stratum corneum cells.
3. **Stratum granulosum:** The location of cell loss or continued cell keratin production.
4. **Stratum spinosum:** The location of basal cell differentiation into cells such as keratinocytes. Keratin production commences in this layer. The cells continue migrating toward the stratum corneum.
5. **Stratum germinativum:** The innermost layer attached to basal cells (the basement membrane) that separate the epidermis from dermis. Basal cells remain in the stratum germinativum and undergo

mitosis to produce keratinocytes. Specialized cells, known as Merkel cells, are connected to afferent nerves with receptors responsible for the sensation of touch. Although located throughout the body, the Merkel cells are abundant in areas used often in detecting touch (fingers, toes, lips, mouth). The basement membrane is a selectively permeable membrane.

New cells proliferate from the innermost basal layer and push upward toward the stratum corneum. The outer layers often contain 25 sheets of dead cells that are continuously shed; however, the number of layers can vary around the body, with the palms and soles having about 100 layers.

Keratinocytes in these layers produce keratin, a protein that strengthens skin, and melanocytes produce melanin, a pigment that gives the skin, hair, and eyes color. The types of melanin are eumelanin and pheomelanin. Eumelanin (brown and black pigments) protects the skin from ultraviolet (UV) rays. Eumelanin causes tanning and freckles. Pheomelanin (yellow and red pigment) reacts to ultraviolet light, and this reaction possibly explains why fair-skinned people are more susceptible to skin cancer.

Everyone has the same number of melanocytes. The difference in skin tones (light to dark) in individuals is explained by the amount of melanin produced, which is genetically determined. Melanocyte stimulating hormone (MSH) is an adrenocorticotropic hormone produced by the anterior pituitary gland, and it is produced from the cleavage of proopiomelanocortin from which adrenocorticotropic hormone (ACTH) is also derived. Stimulation of MSH 1 receptors results in the production of melanin by the melanocytes. The stimulation occurs in response to exposure to ultraviolet light.

In addition, MSH influences various other processes in the body. Some examples include when fat cells release leptin, MSH acts on receptors in the hypothalamus to suppress appetite. MSH release can influence aldosterone, antiinflammatory processes, and sexual behavior. Addison's disease is a disorder (primary adrenal insufficiency) that causes diffuse hyperpigmentation as a result of the MSH effects from elevated ACTH.

Other important cells in the epidermis are Langerhans cells, which are part of the skin's immunologic function. Langerhans cells are dendritic cells (antigen-presenting cells), formed in the bone marrow, that recognize foreign antigens and transport them to the lymphatic system. These cells also produce cytokines involved in the immune system (see the immunity chapter).

Dermis and Appendages

The dermis is the layer between the epidermis and subcutaneous layers. This layer has many structures and contains collagen, a protein. The collagen is in a substance known as hyaluronic acid, which helps the skin to retain moisture. Many lotions and topical skin products contain hyaluronic acid to help with dry skin. There are various glands in the dermis, including the following:

- **Sebaceous glands:** These glands produce sebum, which moisturizes and protects the skin. The sebaceous glands are located throughout the body except for the palms, soles, and sides of the feet. The glands' secretory activity is controlled by genetics and hormones such as testosterone, and the glands are inactive until adolescence. During adolescence, the glands increase in size and produce more sebum, leading to inflammation—one component of acne vulgaris.
- **Sweat glands:** Two types—eccrine and apocrine glands—are located throughout the skin:
  - **Eccrine glands:** Also known as merocrine glands, these glands secrete sweat (sodium chloride and water) through skin pores in response to the sympathetic nervous system. The eccrine gland function includes temperature regulation by secreting water. The eccrine glands are located throughout the body except on the lips and genitalia.
  - **Apocrine glands:** These glands open into hair follicles in the axillae, scalp, face, and external genitalia. Skin bacteria convert apocrine secretions (oily) into chemicals that cause human body odor.

The dermis also consists of hair, which is keratinized and emanates from hair follicles. Hair phases—growth (anagen), atrophy (catagen), and rest (telogen)—are influenced by hormones. Hair follicles are often part of sebaceous glands, and together they form the pilosebaceous unit. Several disorders stem from the pilosebaceous unit, such as acne vulgaris. Other structures of the dermis include nerves, smooth muscle, blood vessels, and lymphatic vessels.

Subcutaneous Tissue

The subcutaneous layer—the hypodermis—connects the dermis to the muscle. The subcutaneous layer is made of fat cells (adipose tissue) and connective tissue that function as a cushion for the body and provides insulation from cold temperatures. Blood vessels, nerves, and macrophages are also located in this layer.

Nails

Nails consist of hard, keratinized plates located on the fingers and toes (**Figure 13-3**). Nail growth is continuous and occurs in a structure known as the nail matrix. The end of growth is a crescent-shaped white area known as the lunula. The cuticle is at the lower part of the nail, and the paronychium is the tissue that surrounds the border. The nail bed is vascular, so it can be a window into oxygenation status (e.g., cyanosis, pallor, or clubbing). The nail can develop disorders such as onychomycosis, and nail bed abnormalities can be a sign of an underlying systemic disorder (e.g., leukonychia in liver or heart disease).

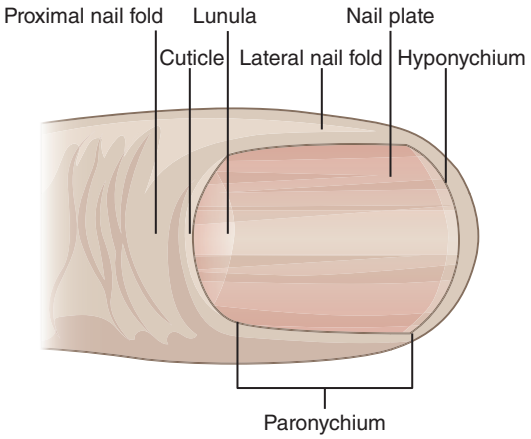


Figure 13-3 Anatomy of the nail.

UNDERSTANDING CONDITIONS THAT AFFECT THE INTEGUMENTARY SYSTEM

There are approximately 2,200 disorders of the skin, so when considering alterations of the integumentary system, organizing them based on their basic underlying pathophysiology can increase understanding. Skin disorders can be organized by how they occurred (e.g., infections, allergens, and trauma) and by the anatomic structure predominantly affected (e.g., sebaceous gland or the dermal layer). Disorders can also be organized by those that are congenital or those that are due to aging.

Skin disorders are easy to detect visually; however, determining the cause is usually based on a thorough history, as many skin alterations have a similar appearance. Skin disorders often occur in certain body distributions (e.g., trunk or extremities) while sparing other body areas, so pattern and distribution are also important in diagnosing skin disorders.

Impairments in skin integrity require interventions that are intended to either prevent such impairment, maintain skin integrity, or improve skin integrity. Because the skin provides a barrier to protect the body from invasion, skin disorders are a relevant risk for infection. Interventions to prevent and manage infections are directed at minimizing contamination, such as by hand washing and wound care, and by supporting the immune system, such as by proper nutrition. The skin can also be a window to the body's inside, and an understanding of skin alterations can provide relevant information in regard to potential or actual systemic disorders.

Congenital Integumentary Disorders

Congenital disorders of the integumentary system can vary widely in severity. Many conditions occur because of an error during embryonic development. These errors may occur randomly, due to environmental influences, or because of genetic abnormalities. Congenital disorders usually cause vascular tumors and malformations or pigmentary abnormalities. They may cause minor conditions with only aesthetic problems (e.g., birthmarks) or they may cause life-altering states (e.g., albinism). Occasionally, seemingly benign conditions may be associated with other, more serious problems that warrant further investigation. Treatment is often unnecessary, but when needed, options are usually limited.

Birthmarks

Birthmarks are skin anomalies that are present at birth or shortly after. Most are harmless and may even shrink or disappear with age. Birthmarks vary from barely noticeable to disfiguring. These abnormalities may be flat or raised and may have regular or irregular borders. In addition, they may have different shades of coloring, including brown, tan, black, pale blue, pink, red, or purple. Birthmarks cannot be prevented and are not the result of anything done or not done during pregnancy. Two types of birthmarks are distinguished—vascular and pigmented.

Vascular Birthmarks

Vascular birthmarks can arise from vascular malformations, vessels that are proliferating (i.e., vascular tumors such as hemangiomas), or due to vessel dilation (e.g., nevus simplex). Due to the vascular alterations, these birthmarks are generally red. The various types of vascular birthmarks include nevus simplex, hemangiomas, and port-wine stains.

Macular stains, also called *nevus simplices*, *salmon patches*, *angel kisses*, and *stork bites*—are the most common type of vascular birthmark (**Figure 13-4**). Nevus simplex is due to vessel vasodilation. These faint red patches (flat, nonpalpable >1 cm) are blanchable and often occur on the forehead, eyelids, posterior neck, nose, upper lip, or posterior head (**Table 13-1**). On a baby, these birthmarks may be more noticeable when crying. Most often, these marks fade on their own by age 2 years, but they sometimes last into adulthood (e.g., back-of-neck patches).

Figure 13-4 Macular stain.

Table 13-1 Lesion Description

Lesion	Description
Macule	Flat, nonpalpable, <1 cm (circumscribed area)
Patch	Flat, nonpalpable, >1 cm
Papule	Raised, palpable, <1 cm (circumscribed area)
Nodule	Raised, palpable; deeper in dermis than papule, 1–2 cm (circumscribed area)
Plaque	Raised, palpable, >1 cm (rough surface)
Vesicle	Serous filled "blisters" <1 cm
Bulla	Serous filled "blisters" >1 cm
Pustule	Like vesicle but fluid is pus
Cyst	Raised, palpable, filled with liquid (circumscribed area)

Hemangiomas, also referred to as *strawberries* (although not all look like strawberries), are birthmarks that appear usually as a solitary bright red papule (raised, palpable, <1 cm), a plaque (raised, palpable, >1 cm), or a nodule due to the proliferation of the vascular endothelium in the skin (**Figure 13-5**). They are more common in girls than boys. They may be either superficial or deep. The deep hemangiomas may be bluish because they involve deeper blood vessels and may have a telangiectatic patch in the center.

Hemangiomas proliferate during the first year of life and then usually recede over time. Most involute by age 5 years and the remainder by age 9 years. Some hemangiomas, particularly larger ones, leave scars as they recede; these scars can be corrected by minor plastic surgery. Many hemangiomas are found on the head or neck, although they can appear anywhere on the body. Most are benign and not associated with other medical conditions, but they can ulcerate and cause complications if their location interferes with sight, feeding, breathing, or other bodily functions.

Port-wine stains, also called *nevus flammeus*, are discolorations that look like wine was spilled on an area of the body—hence their name (**Figure 13-6**). These birthmarks most often occur on the face, neck, arms, and legs. Port-wine stains are caused by malformations of the dermis capillaries and venules. Port-wine stains are patches that are blanchable and can be of any size, but they grow only as the child grows. They tend to be unilateral and do not cross the midline (i.e., they stay on one side). They tend to darken

Figure 13-5 Hemangioma.

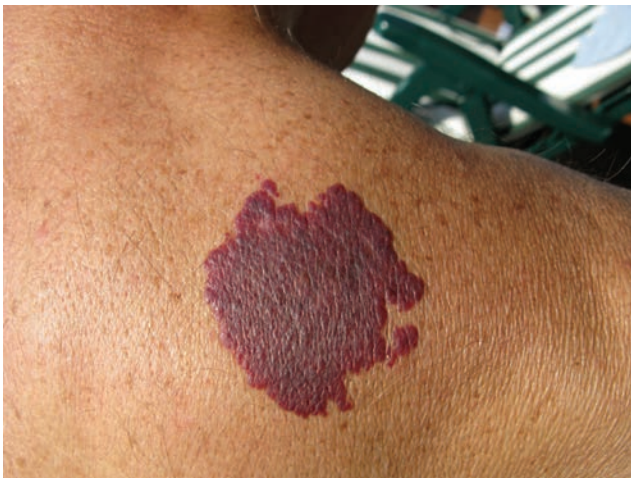


Figure 13-6 Port-wine stain.

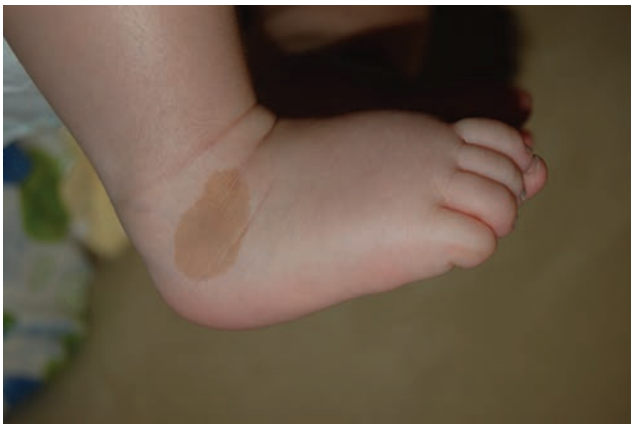


Figure 13-7 Café au lait spot.

Courtesy of Marnie Pasciuto-Wood.

over time and can thicken and have a cobblestone texture in mid-adulthood unless treated. Port-wine stains will not resolve spontaneously, and those occurring near the eye should be assessed for possible complications such as glaucoma, as well as other conditions (e.g., Sturge-Weber syndrome).

Pigmented Birthmarks

Pigmented birthmarks are made of a cluster of pigment cells, which cause color in skin. These birthmarks can be many different colors, from tan to brown, gray to black, or even blue. The most common pigmented birthmarks are café au lait spots, Mongolian spots, and moles.

Café au lait macules or spots are very common birthmarks that are the color of coffee with milk—hence their name (**Figure 13-7**). These birthmarks are macules (flat, nonpalpable, <1 cm) or patches and can appear anywhere on the body. They sometimes increase in number as a child gets older. Café au lait macules are caused by increased melanin production. One café au lait macule alone is not usually a concern, but the child should be further evaluated if he or she has several patches larger than a quarter, which can be a sign of neurofibromatosis (see the cellular function chapter).

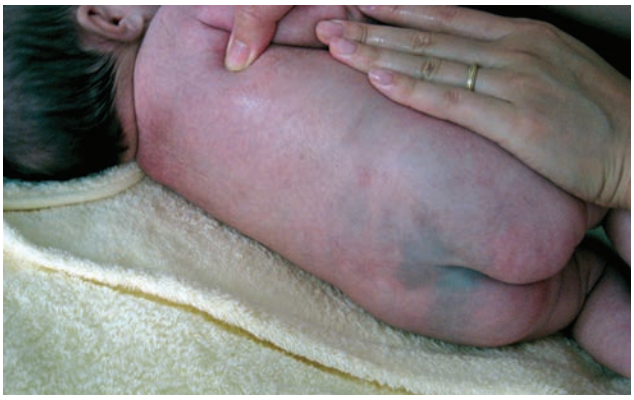


Figure 13-8 Mongolian spot.

Courtesy of Wassa Catlow.

Mongolian spots, also called *congenital dermal melanocytosis*, are flat, bluish-gray macules or patches that are nonblanching and often found on the lower back or buttocks (**Figure 13-8**). Mongolian spots are caused because melanocytes that are not normally in the dermis (only in the basal layers) have not migrated up to the epidermis. These birthmarks are most common on individuals with darker complexions, such as children of Asian, American Indian, Black, Hispanic, and Southern European descent. Mongolian spots are usually benign, and they usually fade, often completely, by school age without treatment. However, some are associated with pediatric disorders (e.g., inborn errors of metabolism).

Mole (congenital nevi, hairy nevi) is a general term for melanocytic nevi or brown nevi (the singular is *nevus*). Most people get moles at some point in life. When present at birth or within a few months after birth, the mole is called a *congenital melanocytic nevus* and will last a lifetime. Large or giant congenital nevi are more likely to develop into skin cancer (melanoma) later in life; however, all moles should be monitored for cancerous changes. Moles can be tan, brown, or black; can be flat or raised; and may have hair growth.

Diagnosis and Treatment. Diagnosis of birthmarks is often made during a physical examination. Treatment strategies vary depending on the type of birthmark, as some birthmarks cannot be treated. Macular stains and Mongolian spots usually fade away on their own.

Hemangiomas are usually left untreated, as they typically shrink back into themselves by age 9. Laser therapy can be used for removal of some hemangiomas, such as small, superficial ones. Larger or more serious hemangiomas are often treated with steroids.

Laser therapy is the treatment of choice for port-wine stains. Most port-wine stains lighten significantly after several laser treatments, although some return and need retreatment. Laser treatment is typically started in infancy when the stain and the blood vessels are smaller. Marks on the head and neck are the most responsive to laser treatment.

Pigmented birthmarks are usually left untreated, except for moles and, occasionally, café au lait macules. Moles (particularly large or giant congenital nevi) are surgically removed. Café au lait macules can be removed with laser treatment but often return.

Some birthmarks can be disfiguring and embarrassing for children. Special opaque makeup can be used to conceal or minimize the appearance of some birthmarks. Additionally, support and coping strategies can be helpful.

Learning Points

Erythema Blanching

When there is erythema or skin discoloration, the lesion should be assessed for blanching. Pressure is applied for a few seconds over an area, which temporarily decreases blood flow, and the area turns pale or white. When the pressure is released, the color should return, and the erythema is described as *blanchable*. In nonblanchable erythema, pallor does not occur, or the redness persists (this may appear differently in darker skin); in other words, the erythema does not fade when pressure is applied. Nonblanchable erythema is an indication of altered perfusion.

Disorders of Melanin

Melanin is a pigment that provides color and protection. Disorders involving melanin result in alterations in skin coloring and can leave the skin vulnerable to the harmful effects of UV light. Melanin disorders include albinism and vitiligo.

Albinism

Albinism is a condition that results in little or no melanin production even though melanocyte numbers are adequate. Most cases are inherited in an autosomal recessive pattern. Melanin deficits cause a lack of pigment in the skin, hair, and the eyes (**Figure 13-9**).

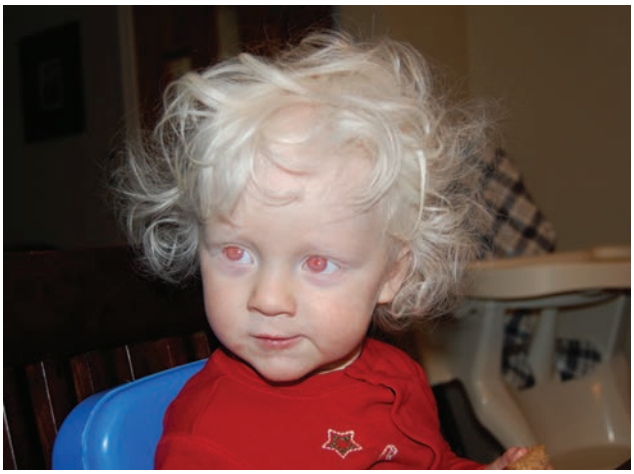


Figure 13-9 Albinism.

Courtesy of Cassandra Hartley.

In addition to coloring and protection, melanin plays a role in the development of certain optical nerves. Therefore, all forms of albinism cause problems with eye development and function. Albinism is generally grouped into oculocutaneous albinism (OCA), which affects the skin, hair, and eyes, or ocular albinism (X-linked recessive), which affects mainly the eyes, and hair and skin color are normal to near-normal coloration. OCA is more common than ocular albinism.

Seven different types of OCA (OCA 1–7) are associated with mutations on different genes. The most common worldwide is OCA type 2, and in the United States, OCA type 1 and type 2 are the most prevalent forms. OCA type 1 is due to a defect in an enzyme, tyrosinase, which is necessary in melanin synthesis (i.e., melanogenesis). Tyrosinase defects can cause no melanin production (i.e., OCA1A) to varying amounts of melanin production (i.e., OCA1B). OCA type 2 is due to a mutation in the *OCA2* gene (formerly called *P gene*). The *OCA2* gene regulates a pH-regulating protein that is necessary for tyrosinase enzyme function. Subtypes 3 through 7 are associated with mutations in genes affecting melanocytes and are found in families in various countries (e.g., China, Pakistan, Japan, and Africa).

Rare OCA mutations can occur in genes also associated with several syndromes such as Hermansky-Pudlak and Chédiak-Higashi syndromes. Hermansky-Pudlak syndrome is very rare (1 in 500,000–1,000,000 people worldwide) and results in OCA, a bleeding disorder, and lung and bowel diseases (National Institutes of Health [NIH], 2020b). Chédiak-Higashi syndrome is also rare (200–500 cases worldwide) and results in OCA, recurrent bacterial infections, neurologic abnormalities, coagulation defects, and lymphoma-like abnormalities (NIH, 2020a).

Clinical Manifestations. Clinical manifestations of albinism are usually—but not always—apparent in a person's skin, hair, and eye color. Regardless of the effect of albinism on appearance, all people with the disorder experience vision impairments. Manifestations may include the following conditions:

- **Skin changes:** Although the most recognizable form of albinism results in milky white skin (OCA 1), skin pigmentation can range from white to nearly the same as relatives without albinism (OCA 2). For some people with albinism, skin pigmentation never changes. For others, melanin production may begin or increase during childhood and adolescence, resulting in slight increases in pigmentation. Some people may synthesize melanin and develop freckles, moles (with or without pigment), or lentigines (large freckle-like spots) with exposure to the sun.
- **Hair changes:** Hair color can range from very white to brown. People who are Black or of Asian descent who have albinism may have hair color that is yellow, reddish, or brown. Hair color may also change by early adulthood.
- **Eye changes:** Eye color can range from very light blue to brown and may change with age. The lack of pigment in the irises makes them somewhat translucent, meaning they cannot completely block light from entering the eye. This translucence can cause very light-colored eyes to appear pink-red in some lighting because of light reflecting off the retina and passing back out through the iris again. The retina also has reduced pigmentation, which makes the back of the eye appear yellowish or orange; this pigmentary change gives a different color eye, similar to the mechanism by which a flash photograph changes eye color.
- **Vision changes:** Multiple vision issues can result from the lack of melanin, including the following problems:
  - Nystagmus (rapid, involuntary back-and-forth eye movement)
  - Strabismus (inability of both eyes to stay directed at the same point or to move in unison, or crossed eyes)
  - Extreme nearsightedness or farsightedness
  - Photophobia (sensitivity to light)
  - Astigmatism (abnormally shaped cornea)
  - Functional blindness

Diagnosis and Treatment. Diagnostic procedures for albinism consist of a history, physical examination (including a thorough ophthalmologic exam), and genetic testing (most accurate). Although there is no cure for albinism, people with this disorder can take steps to improve vision and avoid damage from sun exposure:

- Using sunscreen with a high sun protection factor (SPF) against ultraviolet A and B (UVA and UVB, respectively) rays
- Wearing protective clothing (e.g., long-sleeved shirts, long pants, and hats)
- Limiting time outdoors, especially between 10:00 a.m. and 4:00 p.m., when the sun's UV rays are the most intense
- Wearing sunglasses with UV protection may relieve light sensitivity
- Avoiding or cautiously using medications that increase photosensitivity (e.g., antihistamines, statins)
- Performing routine skin self-examination and obtaining a routine skin examination by a healthcare provider (e.g., dermatologist)
- Wearing glasses to correct vision problems and eye position
- Having eye muscle surgery to correct abnormal eye movements (i.e., nystagmus)

Albinism does not impair intellectual development, although people with albinism often feel socially isolated and may experience discrimination. Coping strategies and support may be beneficial in addressing these issues. The visual issues may lead to educational challenges. Educational strategies may include sitting at the front of the classroom, using large-print books and notes, and printing materials with high-contrast colors (e.g., black and white).

Vitiligo

Vitiligo is a rare condition characterized by areas of hypopigmentation (**Figure 13-10**). This disorder occurs

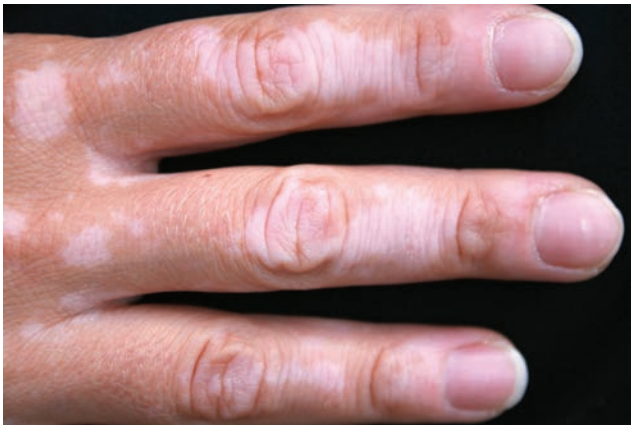


Figure 13-10 Vitiligo.

when melanocytes die or no longer form melanin, causing slowly enlarging hypopigmented macules and patches that are well demarcated. The lesions can vary in size from millimeters to a few centimeters. This condition affects people of all races but may be more noticeable and disfiguring in people with dark skin tones. Vitiligo affects an estimated 1% of the population worldwide (NIH, 2022).

The exact cause is unknown, but potential causes of melanocyte destruction include genetic susceptibility, autoimmunity, and oxidative stress. Vitiligo is associated with combination of mutation in more than 30 genes (e.g., *NLRP1* and *PTPN22*) inherited in a non-Mendelian multifactorial, polygenic inheritance pattern. Vitiligo is associated with autoimmune disorders such as pernicious anemia, autoimmune hypothyroidism (most common), and Addison's disease.

Although any area of the body may be affected, depigmentation usually develops first on sun-exposed areas (e.g., hands, feet, arms, face, and lips). Vitiligo often first appears between ages 10 and 30 years, and in this age group, most report a family or personal history of autoimmune disorder. Onset at a younger age (e.g., younger than 12 years) is often associated with a family history of vitiligo or other depigmenting disorders.

The natural course of vitiligo is difficult to predict. Vitiligo generally develops in one of three patterns. Nonsegmental vitiligo encompasses a focal pattern, meaning depigmentation is limited to one or a few areas of the body. The second nonsegmental pattern is generalized, and depigmentation is widespread across many parts of the body, often symmetrically. The third pattern is considered segmental, and depigmentation occurs on one side of the body. Segmental patterns are not as common as the nonsegmental patterns and usually occur with childhood-onset vitiligo.

Sometimes the patches stop forming without treatment. In most cases, pigment loss spreads and can eventually involve most of the skin's surface. In addition to patchy skin depigmentation, clinical manifestations may include depigmentation of the hair, mucous membranes, and retina.

Diagnosis and Treatment. Diagnosis of vitiligo is usually made clinically with a history and physical examination. Use of a Wood's lamp will reveal depigmented areas that are bright blue–white. Due to the association with autoimmune thyroid disorder, diagnostic evaluation will include a thyroid function panel (e.g., thyroid-stimulating hormone, T_3, T_4) and antithyroperoxidase and antithyroglobulin antibodies. Evaluation for other autoimmune disorders may be necessary. Skin biopsy may be necessary if the diagnosis is questionable. There is no cure for vitiligo.

The goal of treatment is to stop or slow the progression of pigment loss and attempt to return some pigment. Treatments to stabilize rapid progression of vitiligo may include the following measures:

- Intermittent or minipulse therapy (e.g., given two times per week for a few months)
- Low-dose corticosteroids, oral or intramuscular
- Phototherapy

Treatment for repigmentation and coping strategies may include the following measures:

- Phototherapy (controlled exposure to intense UV light in a clinic or hospital)
- Pharmacotherapy, including the following medications:
  - Oral synthetic melanizing agents (e.g., trimethylpsoralen [Trisoralen])
  - Topical corticosteroid agents
  - Topical immunosuppressants, which are also known as calcineurin inhibitors (e.g., pimecrolimus [Elidel] and tacrolimus [Protopic])
  - Topical repigmenting agents (e.g., methoxsalen [Oxsoralen])
  - Oral or topical photochemotherapy (e.g., psoralen plus UVA radiation)
- Skin graft
- Autologous melanocyte transplant (still experimental)
- Permanent depigmentation of the remaining skin (a last resort reserved for extreme cases)
- Sun safeguards (e.g., sunscreen and protective clothing)
- Coping strategies and support:
  - Makeup or skin dyes
  - Tattooing (most effective around the lips)

Integumentary Changes Associated with Aging

The skin undergoes several changes with aging. Sensations of pain, vibration, cold, heat, pressure, and touch usually decrease over the course of the life span. These changes may be related to decreases in blood flow to touch receptors, decreased numbers of mechanoreceptors that sense vibration and fine touch (i.e., Meissner and pacinian corpuscles), or decreased blood flow to the brain that can occur with age. Decreases in these sensations can increase the risk of injury, including falls, pressure injuries,

Learning Points

Sunscreens

UV radiation from the sun includes UVA and UVB rays. UVA are the most prevalent rays that reach the earth (95%) and are mainly responsible for photoaging and skin darkening. UVA also causes burn but less than UVB. UVA rays have a possible role in skin cancer development. UVB rays are the remaining rays (5%) that reach the earth and are mainly responsible for sunburns, inflammation, and darkening. A way to remember the key effects is UV**A** causes **a**ging and UV**B** causes **b**urns.

Sunscreens have ingredients (there are 17 different FDA-approved types) that act as filters to reflect or absorb UVA and UVB rays (i.e., they are broad spectrum). Sun protection factor (SPF) is a measure of the sunscreen's ability to absorb UVB rays. The minimum recommended by the FDA is SPF 15 (it absorbs 93% of UVB rays), and the American Academy of Dermatology recommends SPF 30 (absorbs 97% of UVB rays). There are no sunscreens that block 100%, but slightly higher numbers may block more. Regardless of SPF, sunscreen should be applied to all exposed skin (ears, feet, head, etc.) and applied 15 minutes before sun exposure. Reapplication should occur every 2 hours, after swimming, or sweating. Some sunscreens may be water resistant and last longer with water exposure (up to 80 minutes).

burns, hypothermia, and decreased pain perception (see the sensory function chapter).

In addition to sensory changes, the skin undergoes other aging-related changes. The skin loses elasticity, integrity, and moisture over time. Environmental factors, genetic makeup, and nutrition may all contribute to these changes. The greatest single contributing factor, however, is sun exposure (i.e., photoaging). Every month the epidermis sheds, but this shedding decreases with aging, prolonging the amount of time skin is exposed to carcinogens. There is also a decrease in protective macrophages. Natural pigments seem to provide some protection against sun-induced skin damage. Consequently, blue-eyed, fair-skinned people show more of these aging skin changes than people with darker, more heavily pigmented skin. The combined changes of decreased epidermal shedding and decreased macrophages, along with carcinogen exposure, increase the risk for integumentary cancers.

With aging, the epidermis thins, even though the number of cell layers remains unchanged. The number of melanocytes decreases, but the remaining melanocytes increase in size. Aging skin thus appears thin, pale, and translucent. Large, pigmented spots called

Figure 13-11 Lentigo.

Courtesy of Dean Ducas.

age spots, *liver spots*, or **lentigines** (singular: *lentigo*) occur as a consequence of long-standing sun exposure causing increased melanin production. Lentigines may appear in sun-exposed areas (**Figure 13-11**).

Additionally, aging causes the basement membrane, which is normally undulated, to flatten out, and the epidermal thinning makes the skin vulnerable to injuries (e.g., abrasions, blisters). Removing bandages in the elderly can easily cause tearing of the skin. The flattened basement membrane reduces the surface area for movement of necessary and protective skin nutrients. The decreased protective skin nutrients contribute to drying (i.e., xerosis) of the skin and are a risk for loss of skin integrity.

Seborrheic keratosis is a benign tumor that is usually seen in older age. These tumors are due to immature keratinocyte proliferation in the epidermis. The cause is unknown, but there may be a genetic predisposition. The lesions are well demarcated and can be round or oval (**Figure 13-12**). The lesions appear stuck on, and their surface looks like a wart. They are referred to as *senile warts* or *seborrheic verruca*, and they can have different colors (e.g., yellowish, dark brown). While they are usually asymptomatic because they are elevated, friction or trauma can cause pain and bleeding.

The dermal changes in aging include decreases in collagen and elastin. In sun-exposed areas, these changes in the connective tissue reduce the skin's strength, elasticity, and resilience. These changes further contribute to thin skin and are the cause of wrinkling. Dermis blood vessels become fragile, which can lead to bruising (senile purpura), **cherry angiomas**

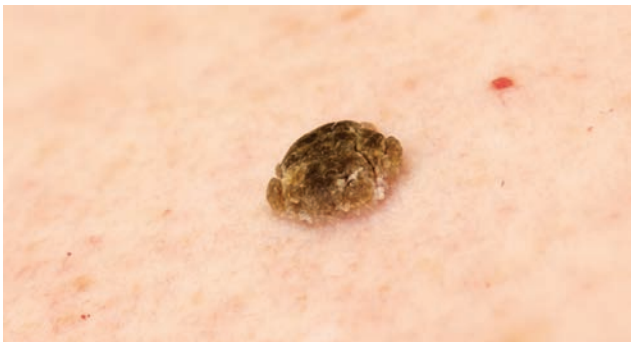


Figure 13-12 Seborrheic keratosis or senile wart.

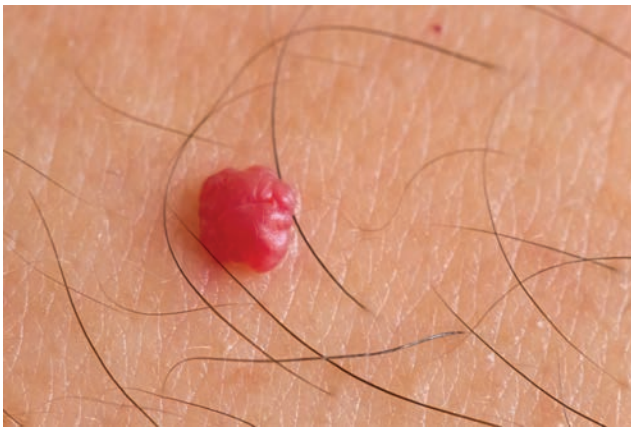


Figure 13-13 Pink senile angioma.

(a benign collection of capillaries; **Figure 13-13**), and other similar conditions.

Sebaceous glands also produce less sebum over time. Men experience a minimal decrease in sebum production, usually after the age of 80, whereas women gradually produce less sebum beginning after menopause. This decrease in sebum can make it difficult to maintain skin moisture, resulting in dryness and itching.

The subcutaneous fat layer, which provides insulation and padding, thins with age. This waning subcutaneous layer increases the risk of skin injury and reduces the ability to maintain body temperature (e.g., heat conservation). Additionally, this fat layer absorbs some medications, so loss of this layer changes the actions of these medications.

The number and functioning of sweat glands decrease, and dermal capillaries are decreased. These two factors reduce the ability to lose heat. These changes make older adults vulnerable to heat stroke.

Aging skin repairs itself more slowly than younger skin. Wound healing may take as much as four times longer to complete. This sluggish repair contributes

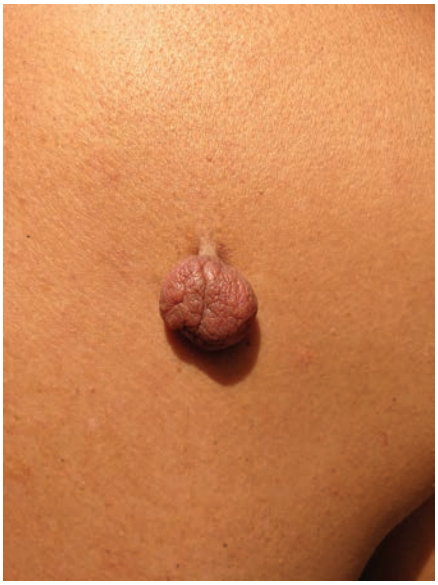


Figure 13-14 Skin tags.

to pressure injury formation and infections. The presence of chronic diseases (e.g., diabetes mellitus and arteriosclerosis) and other aging-related changes (e.g., impaired immunity and circulatory changes) may further delay healing.

The number of hair follicles and the rate of hair growth changes with aging. These changes cause thinning and hair loss. Melanocytes in the hair follicle decrease, so melanin concentration is diminished, causing white hair. Nail changes in older adults include nail plate thickening or thinning and loss of smoothness. These changes may be due to decreased nail bed circulation. The changes in the nail plate cause fissuring and splitting. The nail color can change and become yellow to grayish.

Other skin abnormalities may also develop over time. Abnormalities such as skin tags and other blemishes are more common in older people. **Skin tags** (i.e., acrochordons) are benign, soft brown or flesh-colored masses that usually occur on the neck (**Figure 13-14**). Most skin tags are painless, but they can become inflamed in the presence of constant friction (e.g., from clothing). Skin tags are more common in people who are obese have insulin resistance or type 2 diabetes mellitus.

Acanthosis nigricans is another dermatologic clinical finding associated with insulin resistance. The lesions of acanthosis appear as brown, velvety, hyperkeratotic (i.e., thickened) plaques (**Figure 13-15**). The proposed mechanism for the development of these lesions is that hyperinsulinemia stimulates keratinocytes and fibroblast proliferation by interacting with insulin-like growth factor receptor-1. Skin tags and

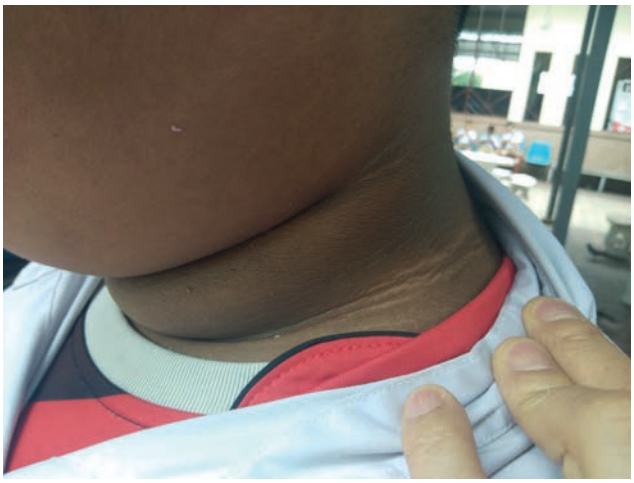


Figure 13-15 Acanthosis.

acanthosis nigricans can be treated by removing the underlying cause (e.g., type 2 diabetes mellitus management). Skin tags can also be removed with surgery, cryotherapy, and cautery. Acanthosis nigricans can be minimized with skin-directed therapy, such as topical retinoids.

Inflammatory Integumentary Disorders

Dermatitis is a general term to describe a broad range of inflammatory skin diseases ranging in severity from mild itching to serious medical complications. *Eczema* is a term often used interchangeably with dermatitis as well as in reference to atopic dermatitis. Dermatitis can be acute with lesions that are characteristically pruritic, erythematous, inflamed, and papulovesicular.

Chronic dermatitis lesions reflect long-term and recurrent skin lesions such as pruritus, xerosis, lichenification (i.e., thickened and roughened), hyperkeratosis, and fissuring (i.e., linear cracks). Dermatitis lesions often cause burning, stinging, and pain. Physical manifestations can be similar, regardless of the cause, so a thorough history is important in identifying the diagnosis. Dermatitises are noncontagious conditions that may occur in isolation or in conjunction with other conditions. Most of these disorders can be resolved or managed easily with treatment; however, chronic disorders can recur frequently and be difficult to treat.

Contact Dermatitis

Contact dermatitis is an acute inflammatory reaction triggered by direct exposure to an irritant or allergen-producing substance (**Figure 13-16**). Contact dermatitis is not contagious or life threatening.

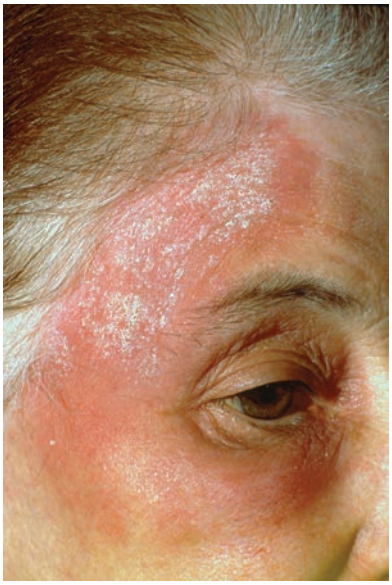


Figure 13-16 Contact dermatitis.

It varies in severity depending on the substance, area affected, exposure extent, and individual sensitivity. There are two main categories of contact dermatitis: irritant and allergic.

Irritant Contact Dermatitis

Irritant contact dermatitis is more common than allergic contact dermatitis. Chemicals, acids, rubber gloves, soaps, and many other environmental substances can cause irritant contact dermatitis. The risk of this type of dermatitis is high in occupations where there is frequent exposure to wetness (e.g., housekeepers, food handlers, healthcare workers).

This type of contact dermatitis does not involve the immune system, but simply triggers the inflammatory response, which causes keratinocytic production of inflammatory cytokines (e.g., tumor necrosis factor). Irritant contact dermatitis produces a reaction similar to a burn. The site of irritant contact dermatitis is often the hands but can be anywhere there has been irritant exposure.

Clinical Manifestations. Manifestations tend to appear soon after exposure to the irritant (from minutes to about 24 hours). Manifestations typically include erythema and edema but may also include pain (e.g., burning, stinging), pruritus, and vesicles (i.e., serous, filled blisters <0.5 cm). The lesions are localized to the area of exposure of the irritant and are well demarcated.

Prolonged exposure to caustic substances can cause erosion, crusting, scaling, and necrosis. Irritant contact dermatitis can become chronic when there is

repeated exposure to irritants (e.g., hairdressers using chemicals, dishwashers using soaps). The lesions with chronic irritant dermatitis can include fissures, xerosis, and crusting.

Allergic Contact Dermatitis

Allergic contact dermatitis results from contact with substances such as metals, chemicals, adhesives, cosmetics, and plants (e.g., poison ivy and poison sumac). Sensitization occurs on the first exposure to the substance, and subsequent exposures to the substance produce manifestations. With exposure, the haptens (allergens) penetrate the epidermis and bind to skin proteins resulting in a type IV hapten-specific T cell–mediated hypersensitivity reaction (see the immunity chapter). The reaction is usually delayed, with manifestations appearing 48 to 72 hours after exposure.

Clinical Manifestations. Typically, manifestations of allergic contact dermatitis include pruritus (a key symptom), erythema, and edema at the site, but vesicles may also be present. In severe cases, bullae (>0.5 cm serous blister) may occur. As with irritant exposure, the lesions in allergic contact dermatitis are localized to the area that comes in contact with the allergen. However, allergic contact dermatitis may spread depending on the allergen or whether there is transfer of the allergen to another area. Lesions with chronicity are similar to other chronic skin dermatitis.

Diagnosis and Treatment. Diagnosis of contact whether irritant or allergic is usually made clinically with a history, physical examination, and allergy testing (for allergic contact dermatitis). Treatment of contact dermatitis centers on identifying and removing the causative agent (e.g., rinsing the affected area). If the offending agent can be avoided, the rash usually resolves in 2 to 4 weeks.

Self-care measures, such as wet compresses or drying agents (e.g., oatmeal compresses), and anti-inflammatory creams (e.g., corticosteroid agents) can help soothe skin and reduce inflammation. Topical calcineurin inhibitors may be used in acute cases that are not responsive to topical steroids or with chronic cases. Calcineurin is an enzyme that activates T cells. Systemic anti-inflammatory agents may be used in severe cases.

Protective strategies may include use of gloves; however, rubber gloves can irritate the dermatitis. Regular use of creams and emollients may offer some protection against irritants. Workplace modifications, such as using milder detergents and cleansers, may be necessary.

Atopic Dermatitis

Atopic dermatitis, often referred to as *eczema*, is a chronic inflammatory condition (**Figure 13-17**). Atopic dermatitis commonly begins in infants but usually resolves by early adulthood. It tends to be characterized by remissions and exacerbations and may be accompanied by asthma and allergic rhinitis. The exact cause is unknown, but atopic dermatitis may result from an immune system malfunction, similar to hypersensitivity (an elevation of immunoglobulin E is usually present).

There is a genetic impairment of the epidermal skin barrier and an increase in water loss that are thought to be caused by several mechanisms: (1) deficiencies in filaggrin (FLG; a protein involved in the maintenance of hydration and water retention), (2) protease imbalances (e.g., kallikrein), (3) disruption of the junctions that keep intercellular spaces sealed, (4) microbial colonization, and (5) an increased immune-mediated response. Epidermal barrier dysfunction is a key feature in atopic dermatitis pathogenesis.

Atopic dermatitis is thought to be the first of a series of allergic diseases that affect the epithelial surfaces (referred to as the *atopic march theory*)—including food allergies, asthma, and allergic rhinitis (Spergel, 2010). It has an inherited tendency, with 70% of patients reporting a positive family history of atopy (i.e., with atopic dermatitis, asthma, or allergic rhinitis). Several genes, such as loss of function mutations in the *FLG* gene, are associated with atopic dermatitis, but it is likely polygenic.

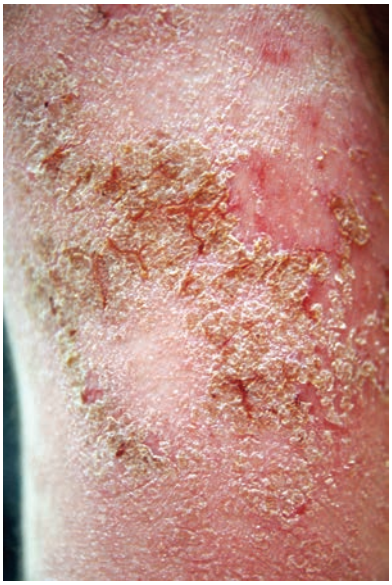


Figure 13-17 Atopic dermatitis.

Courtesy of Paul Matthews.

Complications may include secondary bacterial, viral, or fungal skin infections, neurodermatitis (permanent scarring and discoloration from chronic scratching), and eye problems (e.g., conjunctivitis). Atopic dermatitis may affect any area, but the pattern exhibited tends to be age specific. The skin lesions primarily affect the face, scalp, and extensor surfaces (knees, elbows) in young children (e.g., younger than 2 years). The flexural surfaces of knees (popliteal fossa) and elbows (antecubital space) are the most affected sites in older children and adults.

Clinical Manifestations. Clinical manifestations may be made worse by exposure to allergens (especially to pollen, mold, dust, or animals), cold and dry air, upper respiratory infections, contact with irritants, dry skin, emotional stress, and extreme temperatures. The key manifestations of atopic dermatitis are xerosis and pruritus, which may be severe, especially at night. Acute atopic dermatitis lesions include papules and vesicles that can ooze serous fluid and crusting. Chronic atopic dermatitis lesions include dry, scaly, excoriated erythematous papules, lichenification, and fissuring.

Diagnosis and Treatment. Diagnosis of atopic dermatitis is made clinically based on the history and physical examination. Allergy testing to identify triggers and skin biopsy (to rule out other causes) may be performed. Serum IgE and eosinophil count, while not necessary for diagnosis, may be elevated. In children, the condition usually improves with age (starting around age 5–6 years), but flare-ups may occur.

Treatment focuses on decreasing the inflammatory process. These strategies may include the following measures:

- Avoiding factors that can worsen manifestations, including:
  - Long, hot baths or showers
  - Dry skin
  - Stress
  - Sweating
  - Rapid changes in temperature
  - Low humidity
  - Solvents, cleaners, soaps, or detergents
  - Wool or synthetic fabrics or clothing
  - Dust or sand
  - Cigarette smoke
  - Certain foods (e.g., eggs, milk, fish, soy, and wheat)—it is controversial whether elimination or reduction of certain foods is beneficial
- Avoiding scratching
- Moisturizing the skin by applying ointments (e.g., petroleum jelly) two to three times per day
- Using a humidifier
- Employing the following strategies when washing or bathing:
  - Keeping water contact brief and using gentle soap
  - Not excessively scrubbing or drying the skin
  - After bathing, applying lubricating creams, lotions, or ointment on the skin while it is damp to trap moisture in the skin
- Using the following pharmacologic agents:
  - Antihistamine agents (may be topical or oral)
  - Corticosteroid agents (may be topical or oral)
  - Immunomodulators (topical calcineurin inhibitors or oral agents such dupilumab)
  - Phosphodiesterase 4 inhibitor (topical)
  - Antibiotics (may be topical or oral if infection is present)
  - Allergen-desensitizing injections
- Receiving phototherapy

Urticaria

Urticaria, or hives, consist of raised, erythematous skin lesions (welts; **Figure 13-18**). These lesions are a result of a type I hypersensitivity reaction with mast cell mediation (can be IgE or non–IgE mediated). Urticaria occurs when histamine release is initiated by various

Application to Practice

Irritant, allergic, and atopic dermatitis have similar clinical manifestations. Recognizing similarities and differences may aid in determining diagnosis. In this activity, determine whether the manifestations listed are present in irritant, allergic, or atopic dermatitis and identify the key manifestations for each dermatitis. Also, identify which lesions are associated with acute or chronic conditions. The manifestations may be present in more than one type of dermatitis.

- **General manifestations:** Pain, pruritus, xerosis, erythema, edema, appearance of manifestations immediately after exposure to a trigger, delay of manifestations after exposure to a trigger.
- **Lesion appearance:** Vesicles; papules; crusting, lichenification; fissuring; well-demarcated lesions.
- **Lesion distribution:** Lesions confined to one area, lesions affecting multiple areas.

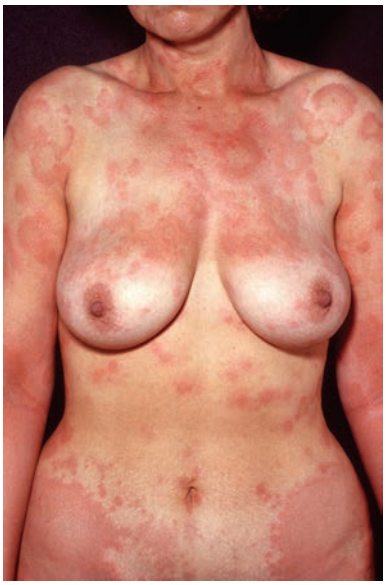


Figure 13-18 Urticaria.

Learning Points

Topical Skin Agents

Topical skin products and medications are made of different types of bases (i.e., the vehicles). The base choice is important in healing response and duration of healing. In general, most drying agents are used for moist, weeping lesions and oil-based bases are used for dry, lichenified lesions. Bases in order of moisturizing (oil) effects to drying effects include ointments, creams, lotions, solutions, and gels. Ointments are semi-occlusive and increase medication absorption but are greasy and messy, decreasing patient acceptance. Ointments are not useful for lesions in the hair. Drying agents such as lotions and solutions are soothing due to the cooling effects. Gels are thin and greaseless bases and are good for use in hair.

substances. This reaction is often triggered by food (e.g., shellfish and nuts) or medicine (e.g., antibiotics) ingestion. Urticaria may also be a result of emotional stress, excessive perspiration, diseases, and infections (e.g., mononucleosis). Other urticarial mechanisms are type II hypersensitivity with tissue-specific cytotoxic cells (e.g., urticarial vasculitis). Type III hypersensitivity urticarial processes involve immune complexes (e.g., autoimmune diseases).

In many cases, no specific trigger can be identified. The resulting skin lesions are usually short-lived and harmless, but breathing can be impaired when swelling occurs around the face (angioedema). Additionally, a type I hypersensitivity reaction can progress to an anaphylactic reaction and shock. The diffuse erythematous plaques (welts) may grow large, spread, and are circumscribed but can fuse together. These plaques are often pale in the center and are blanchable. The plaques are often intensely pruritic. Individual lesions usually resolve within 24 hours, but new ones develop. Urticaria can be acute, lasting fewer than 6 weeks, or chronic, lasting for 6 weeks or longer (see the immunity chapter).

Diagnosis and Treatment. Diagnosis of urticaria is made clinically with a history and physical examination. Diagnostic procedures can include allergy testing and skin biopsy. If there are suspicions of the presence of systemic disorders associated with urticaria, additional procedures may be necessary. Usually, urticarial lesions from systemic disorders are painful, prolonged (e.g., individual lesions last for >24 hours), and recurrent.

Treatment focuses on ceasing the inflammatory reaction and maintaining respiratory status (if appropriate). Mild urticaria may disappear without any treatment. Treatment strategies to reduce itching and swelling include the following measures:

- Avoiding hot baths or showers.
- Avoiding irritating the area (e.g., with tight-fitting clothing or rubbing).
- Taking H_1 antihistamines, usually second generation (e.g., cetirizine, loratadine); however, first-generation agents (e.g., diphenhydramine or hydroxyzine) are available for parenteral administration for a rapid relief.
- Taking H_2 antihistamines (e.g., ranitidine) for acute urticaria, but reports are conflicting as to the efficacy.
- Administering glucocorticoids to inhibit inflammatory response (not mast cell degranulation) for short periods (e.g., <1 week).

Severe reactions, especially if angioedema is present, may require epinephrine (adrenaline) or corticosteroid injections. Additionally, airway maintenance may be necessary, including an artificial airway, oxygen therapy, and mechanical ventilation. Epinephrine (adrenaline) and other bronchodilator agents can also be administered directly into the respiratory tract to improve ventilation.

Psoriasis

Psoriasis is a common, chronic inflammatory condition that affects the life cycle of the skin cells (**Figure 13-19**). Psoriasis is an immune-mediated disease that occurs

in genetically susceptible individuals. The disease features alterations and dysregulation of the innate immune system (e.g., dendritic cells) and adaptive immune system (e.g., T cells), with altered keratinocyte function and vascular dysfunction.

Key innate and adaptive immunologic processes that are thought to occur in psoriasis include:

- Immune cells (e.g., dendritic, macrophages, and neutrophils in the skin) are activated.
- Immune cells, particularly plasmacytoid dendritic cells, produce proinflammatory cytokines such as interferon alfa, which activates other immune cells (e.g., myeloid dendritic cells).
- Immune cells that were activated, particularly myeloid dendritic cells, produce cytokines such as tumor necrosis factor alpha and various interleukins (IL) such as IL-12 and 23. IL-23 plays a major role in creating a T-cell response. Myeloid dendritic cells alter keratinocyte function (via IL-20) and induces vasodilation through nitric oxide production.
- T cells, particularly $CD4^+$ T cells and T helper type 17, also produce cytokines such as IL-17A. Cytokines collaborate and cause keratinocytes to proliferate and further produce proinflammatory cytokines and antimicrobial peptides.
- A vicious loop of cytokine production by immune cells and keratinocytes maintains the inflammatory process.

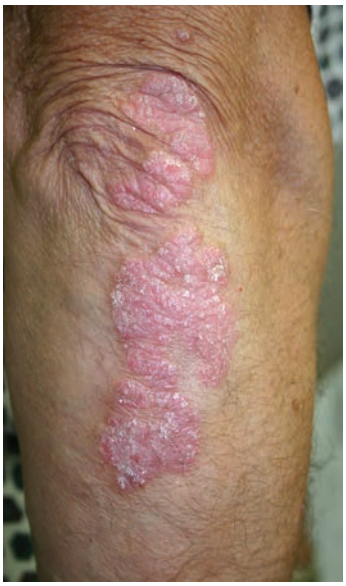


Figure 13-19 Psoriasis.

Courtesy of Yale Residents' Slide Collection, Dermatology Department, Yale University School of Medicine.

Key keratinocyte alterations and processes that are thought to occur in psoriasis include:

- IL, particularly 20 and 22, produced by immune cells, contributes to keratinocyte hyperplasia with resulting epidermal thickening and keratinocyte developmental defects. Cellular proliferation is significantly increased in this disorder, such that cells build up too rapidly on the skin's surface (**Figure 13-20**).
- The process of skin cells growing in the innermost layers of the skin (i.e., basal cell layer) and then rising to the surface (i.e., stratum corneum) normally takes weeks, but with psoriasis, this process occurs over 3 to 4 days. The dead cells cannot be shed fast enough, causing thickening.
- The ILs also increase cellular response of antimicrobial polypeptides (e.g., cathelicidins) produced by keratinocytes that contribute to the integumentary inflammatory response.

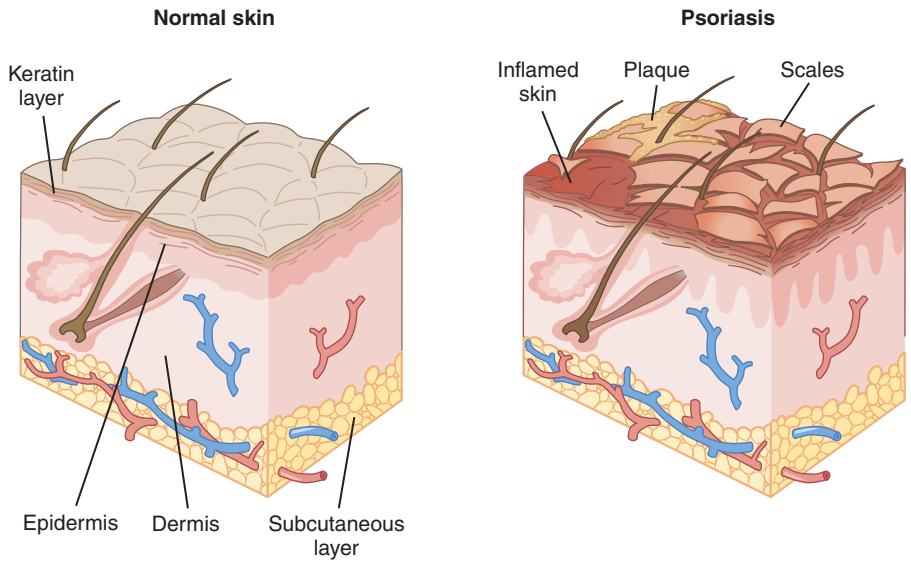


Figure 13-20 Cellular changes associated with psoriasis.

- Keratinocytes produce substances that sustain the inflammatory response by affecting the immune system.

The key vascular change that is thought to occur in psoriasis is as follows:

- Tortuous and leaky blood vessels in the integument as a result of increased endothelial cellular expression of nitric oxide (vasodilator), prostaglandin (vasodilator), and vascular endothelial growth factor (which stimulates angiogenesis).

Psoriasis occurs in families, so there is a hereditary predilection. Several human leukocyte alleles are associated with the development of psoriasis. The mode of transmission is multifactorial.

Environmental factors are known to trigger psoriasis; however, the mechanism by which this occurs is unclear. Psoriasis can affect people of any age, but onset peaks between ages 30 and 39 years and again between ages 50 and 69 years. The onset may be sudden or gradual, and many patients will experience remissions and exacerbations (see the immunity chapter).

The following factors may trigger psoriasis exacerbation or make the condition more difficult to treat:

- Bacterial or viral infections in any location
- Dry air or dry skin
- Skin injuries (e.g., cuts, burns, and insect bites)
- Use of certain medicines (e.g., antimalaria agents, beta blockers, and lithium)
- Stress
- Too little or too much sunlight
- Excessive alcohol consumption

The severity of psoriasis varies widely, from being a mere nuisance to being disabling; as many as 30% of people with psoriasis also have arthritis, a combination referred to as *psoriatic arthritis*. In general, psoriasis may be severe in people who have a weakened immune system (e.g., those with AIDS, those with autoimmune conditions, or those who are receiving chemotherapy).

Clinical Manifestations. There are different forms of psoriasis, and the major different clinical types include the following (**Figure 13-21**):

- **Plaque (chronic):** Thick, red plaques covered by flaky, silver-white scales (the most common type); lifting the scale causes bleeding (i.e., Auspitz sign). The bleeding occurs because of the abnormal blood vessels' proximity to the scales. The lesions are usually symmetrically distributed and sharply defined. The usual location of the lesions is the scalp, extensor surface of elbow, knees, and the gluteal cleft (i.e., "butt crack"). In darker skin tones, lesions may appear purplish or darker.
- **Erythrodermic (acute or chronic):** Intense erythema and scaling that covers a large area usually from head to toe. The lesions are usually pruritic

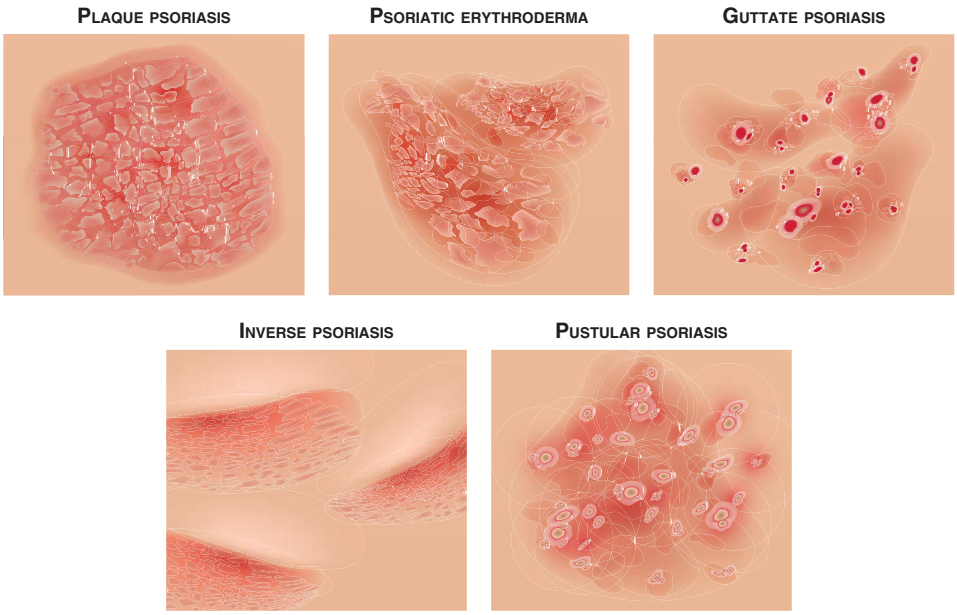


Figure 13-21 Types of psoriasis.

© Net Vector/Shutterstock.

and painful. There is high risk for infections and fluid and electrolyte abnormalities due to the large skin surface area affected.

- **Guttate:** Small, pink-red papules and plaques that usually appear abruptly and acutely with no necessary prior history of psoriasis; may occur postinfection (e.g., streptococcal pharyngitis). Guttate means droplike and refers to the small size of the lesions (<1 cm). The usual location of the lesions is the trunk and proximal extremities. Guttate psoriasis may remit, recur, or progress to plaque psoriasis.
- **Inverse:** Erythema and irritation, usually with no scaling, that occur in the intertriginous areas such as armpits, groin, and skin folds; referred to as *inverse* as the location of the lesions are opposite of the usual extensor surface areas that are affected.
- **Pustular:** Papules or plaques with pustules surrounded by erythema. This type can have an acute onset, and severe forms can be associated with infectious signs such as malaise and fever. Systemic complications can include sepsis and respiratory, renal, or hepatic abnormalities. This type may be present without a prior history of psoriasis. Pustular psoriasis may remit for years and recur after a few years.

Other manifestations may include joint pain or aching (psoriatic arthritis). Nail changes such as thickening, yellow-brown spots, dents (pits) on the nail surface, and separation of the nail from the base (onycholysis) may occur, and the nail changes can occur prior to skin manifestations. At times, only the nails are involved in psoriasis. There is an increased recognition of risk or association with comorbid diseases such as cardiovascular disease, hypertension, inflammatory bowel disease, and other autoimmune disorders. The mechanism for association with these disorders is thought to be due to the inflammatory mediators. Therefore, manifestations of these disorders may be present with the skin lesions.

Diagnosis and Treatment. Diagnosis is made clinically with a history and physical examination. Skin biopsy may be performed to rule out other causes. Other tests may be conducted to rule out conditions that mimic psoriasis (e.g., seborrheic dermatitis and tinea corporis).

No cure exists for psoriasis, but treatment can improve symptoms significantly in most cases. The goal of psoriasis treatment is to interrupt the process that leads to cell buildup and improve manifestations. Treatment is usually multipronged and includes three main approaches—topical treatments, phototherapy, and systemic medications:

- Topical treatments:
  - **Corticosteroid agents:** Slow cell turnover, decrease inflammation by suppressing the immune system
  - **Vitamin D analogues:** Slow down the skin cell growth and immune modulations (inhibit T cells and inflammatory mediators)
  - **Anthralin (Dritho-Scalp):** Normalizes DNA activity in skin cells, removes scales, and smoothes skin
  - **Retinoids:** Normalize DNA activity in skin cells and possibly decrease inflammation
  - **Calcineurin inhibitors:** Disrupt the activation of T lymphocytes, thereby reducing inflammation and plaque buildup
  - **Salicylic acid:** Promotes shedding of dead skin cells and reduces scaling
  - **Coal tar:** Reduces scaling, itching, and inflammation, although the mechanism of action remains unknown
  - **Moisturizers:** Reduce dryness, itching, and scaling (ointment-based moisturizers are the most effective, especially when applied while skin is moist after washing)
  - **Dandruff shampoo:** Reduces cellular turnover
- Phototherapy:
  - **Sunlight:** UV light, whether natural or artificial, causes activated T lymphocytes in the skin to die, slowing cell turnover, reducing scaling, and decreasing inflammation
  - **Broadband UVB phototherapy:** Slows cellular growth
  - **Narrowband UVB phototherapy:** A newer and more effective treatment than broadband UVB treatment
  - **Photochemotherapy, or psoralen plus ultraviolet A:** Psoralen (a light-sensitizing medication) administration before exposure to UVA light to increase the response to the light
  - **Excimer laser:** A controlled beam of UVB light of a specific wavelength that is directed to only the skin involved
- Systemic therapy: Oral or injected pharmacotherapy (primarily reserved for severe or resistant cases and used for brief periods because of the potential serious side effects)
  - **Retinoids:** Related to vitamin A; used in an attempt to reduce the production of skin cells

- **Methotrexate:** Decreases the production of skin cells (alters DNA synthesis) and suppresses inflammation (suppresses T-cell activity)
- **Cyclosporine:** Systemic calcineurin inhibitor; suppresses the immune system (T-cell suppression) similarly to methotrexate
- **Hydroxyurea:** Suppresses the immune system but not as effectively as methotrexate and cyclosporine
- **Phosphodiesterase 4 inhibitor:** Reduces cytokines
- **Immunomodulator drugs:** Biologics; block interactions between certain immune system cells such as tumor necrosis factor alpha inhibitors (e.g., etanercept), anti-IL-17 (e.g., ixekizumab); anti-IL-12 and IL-23 (e.g., ustekinumab)
- **Janus kinase inhibitors:** Protein kinase C inhibitors, selective tyrosine kinase 2 inhibitors; interrupt cellular signaling, thereby reducing the inflammatory response; in clinical trials

In addition to these main strategies, stress management (e.g., coping strategies and support) and avoiding psoriasis triggers may be beneficial. Due to the chronicity and the potentially disfiguring nature of psoriasis, it is important to recognize and address psychosocial distress that may arise. Psoriasis can be difficult to manage and referrals to counseling and support groups such as the National Psoriasis Foundation may be helpful.

Infectious Integumentary Disorders

Skin infections are common and may be caused by several pathogens (e.g., bacteria, viruses, and parasites). These organisms usually gain access through a breach in the skin or mucous membranes. They often trigger the inflammatory response as well. Such infections can occur in any of the skin layers or structures (e.g., hair follicles, nails); they may be acute or chronic, and they vary widely in severity. In most cases, infectious integumentary disorders resolve easily with treatment.

Bacterial Infections

Any number of bacteria present in the body as part of the normal flora or encountered externally may cause bacterial skin infections. These infections can vary in severity from mild to life threatening. Bacteria in the *Staphylococcus* and *Streptococcus* genera are common culprits in integumentary infections. These infections can result in numerous conditions.

Folliculitis

Folliculitis refers to infections involving the hair follicles. Folliculitis is characterized by tender pustules and erythematous papules that form around hair follicles, often on the scalp, neck, buttocks, and face (any area with hair). *Pseudomonas aeruginosa* (gram negative) causes "hot tub" folliculitis, which is due to inadequate chemical treatment of swimming pools, whirlpools, or hot bubs. Pruritus often accompanies folliculitis.

Furuncles

Furuncles, or boils, are infections that begin in the hair follicles and then spread into the surrounding dermis. Furuncles most commonly occur on the face, neck, axillae, groin, buttocks, and back. A furuncle lesion starts as a firm, red, painful nodule that develops into a large, painful mass, which frequently drains large amounts of purulent exudate. **Carbuncles** are clusters of furuncles.

Impetigo

Impetigo (nonbullous and bullous) is a common and highly contagious skin infection, which is most often seen in children. Impetigo is commonly caused by staphylococci and less commonly streptococci. Although it can occur without an apparent breach, this infection typically arises from a break in the skin (especially from animal bites, human bites, insect bites, and trauma). Impetigo spreads easily to others by direct contact with skin or contaminated objects (e.g., eating utensils, towels, clothing, and toys).

Lesions (nonbullous impetigo) usually begin as small vesicles with surrounding erythema that become pustules, enlarge, and rupture, forming the characteristic honey-colored crust (**Figure 13-22**). In bullous impetigo, the vesicles develop into bullae (blisters that are >0.5 cm) with clear yellow fluid that becomes cloudy and darker. Rupture of these bullae causes a thin brown crust to form.

With bullous impetigo, there are fewer lesions than in the nonbullous type, and the trunk is often affected. Bullous impetigo is also caused by staphylococci strains that cause the release of an exfoliating toxin against the desmoglein protein (maintains tissue integrity). In contrast, nonbullous impetigo is a result of the normal response to an infection rather

than a toxin. Impetigo lesions can spread throughout the body through self-transfer of the exudate. Pruritus is common, and lymphadenopathy can occur near the lesions.

Ecthyma is an ulcerative type of impetigo. The ulceration can extend deep into the dermis. Causes are similar to impetigo and can be the result of untreated or poorly treated impetigo.

Cellulitis

Cellulitis refers to an infection deep in the dermis and subcutaneous tissue. It usually results from a direct invasion of the pathogens through a break in the skin, especially those breaches where contamination is likely (e.g., intravenous drug use and bites), or it spreads from an existing skin infection. Cellulitis appears as a swollen, warm, tender area of erythema with or without purulence (**Figure 13-23**).

Cellulitis is also unilateral, smooth, and has indistinct borders. Linear streaks of erythema (lymphangitic streaks) may be seen near the cellulitis along the lymphatic vessels. These streaks occur because of lymphatic inflammation. Additionally, systemic manifestations of infection are usually present (e.g., fever, leukocytosis, malaise, and arthralgia). If left untreated, cellulitis can lead to necrotizing fasciitis, septicemia, and septic shock.

Erysipelas is a type of cellulitis, but the infection is superficial and confined to the upper dermis and the superficial lymphatics. Erysipelas is commonly caused by beta-hemolytic streptococci. While manifestations of erysipelas are similar to cellulitis (e.g., erythema, warmth), erysipelas is nonpurulent.

Necrotizing Fasciitis

Necrotizing fasciitis is a serious infection that is generally rare. One out of four people who develop this infection will die because of it. Also known as flesh-eating bacteria, necrotizing fasciitis can aggressively destroy skin, fat, muscle, and other tissue (**Figure 13-24**). This infection typically involves a highly virulent strain of gram-positive, group A, beta-hemolytic *Streptococcus* that invades through a minor cut or scrape.

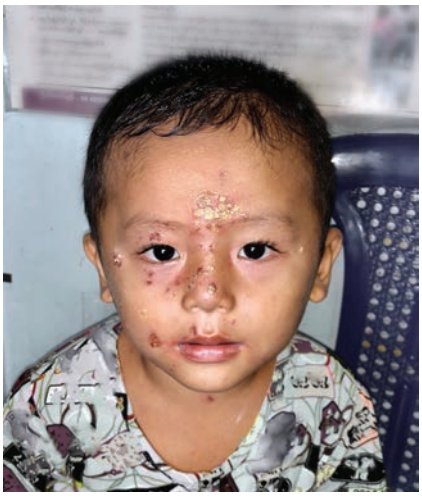


Figure 13-22 Impetigo.

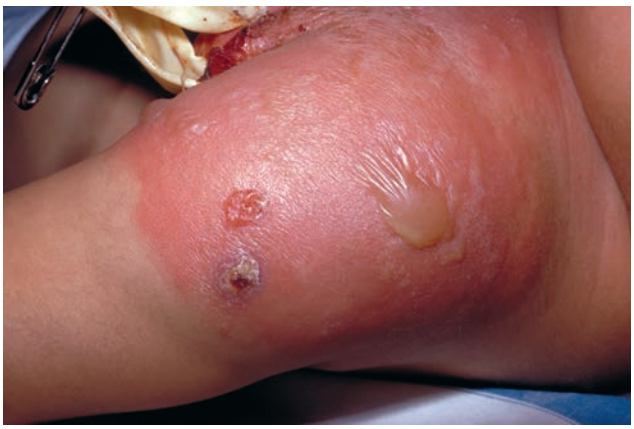


Figure 13-23 Cellulitis.

Application to Practice

Stasis dermatitis is often mistakenly diagnosed as cellulitis. This misdiagnosis leads to unnecessary treatment of stasis dermatitis with antibiotics. The clinical manifestations can be similar, and patients with stasis dermatitis are at risk for developing cellulitis. Both types of dermatitis are erythematous and are commonly present in the lower extremities. Both can present with vesicles and weeping (acute stasis dermatitis).

Some tips to help in distinguishing between cellulitis and stasis dermatitis are as follows:

- **Cellulitis** tends to be unilateral. Systemic symptoms such as fever may be present. Cellulitis tends to be smooth, is generally tender, and progresses rapidly. Inciting factors for cellulitis include immunocompromise, medications, and outdoor activities.
- **Stasis dermatitis** tends to be bilateral. Pitting edema is present. The skin is hyperpigmented, scaly, and edematous. Stasis dermatitis is generally nontender and chronic. Inciting factors for stasis dermatitis include venous insufficiency.

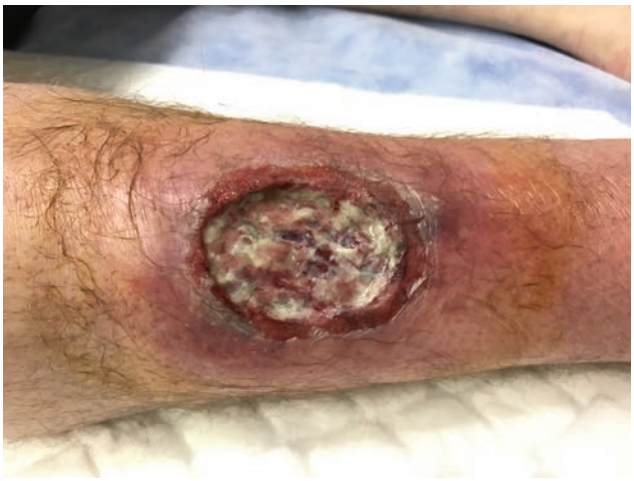


Figure 13-24 Necrotizing fasciitis.

The bacteria begin to grow and release harmful toxins that directly destroy the tissue, disrupt blood flow, and break down material in the tissue. The first sign of infection may be a small, red, painful area on the skin. This area quickly evolves into a painful bronze or purple-colored patch that grows rapidly. The center of the lesion may become black and necrotic. Exudate is often present. The wound may grow quickly—in less than an hour. Systemic manifestations may include fever, tachycardia, hypotension, and confusion. Complications of necrotizing fasciitis include gangrene, multisystem organ failure, and shock.

Diagnosis and Treatment. Diagnostic procedures for all bacterial skin infections center on the identification of the causative organism, usually through cultures. Antibiotics (systemic or oral) are started empirically to avoid systemic complications. Care should be taken when draining any wounds, as this procedure can spread the infection. Other strategies may include maintaining adequate hydration, wound care, surgical debridement, drainage, hyperbaric oxygen therapy, antipyretic agents, and analgesic agents. Hospitalization may be necessary for patients at risk for complications (e.g., elderly or immunocompromised patients), those with evidence of severe infections (e.g., systemic symptoms, hypotension, or tachycardia), or those with an inability to tolerate oral antibiotics. With cellulitis, elevation of the extremity is important to facilitate drainage.

Viral Infections

Several viruses can cause a multitude of skin issues, each with their own manifestations and treatments. These infections can result in numerous conditions, including herpes simplex 1, herpes zoster, verrucae, molluscum contagiosum, and pityriasis rosea.

Herpes Simplex 1

Herpes simplex 1 (HSV-1), or a cold sore, is a viral infection typically affecting the lips, mouth, and face. This common infection usually begins in childhood. HSV-1 can also involve the eyes, leading to conjunctivitis. Herpes keratitis (corneal infection) is a common cause of blindness that results from HSV-1. Additionally, infection with this pathogen can result in meningoencephalitis.

The virus is transmitted by contact with infected saliva. The primary infection may be asymptomatic or present with multiple painful vesicles with surrounding inflammation and erythema. The oral lesions most commonly appear on the lips but can also appear in the mouth or throat. Aphthous stomatitis ulcers (i.e., canker sore) can be confused with HSV-1 that occurs in the mouth. Canker sores, however, are not contagious and appear as small round ulcers (1 or more) with red edges and a gray, white, or yellow center usually in the buccal or labial mucosae. Like HSV-1, canker sores be painful.

With primary infection, fever and malaise may occur. The incubation period (i.e., from exposure to symptoms) is between 1 and 26 days. After the primary infection, the virus remains dormant in the affected sensory nerve ganglion (e.g., trigeminal nerve). Reactivation may be a result of an infection, stress, or sun exposure. When reactivated, HSV-1 causes painful blisters or ulcerations, which are preceded by a burning or tingling sensation or pruritus that can occur about 2 days before the vesicles. Reactivation usually causes lesions around the lips that are not as numerous, and symptoms are not as severe in comparison to the primary infection. The lesions resolve spontaneously within 3 weeks, but healing can be accelerated by administration of oral or topical antiviral agents.

Herpes Zoster

Herpes zoster, or shingles, is caused by the varicella-zoster virus. This condition appears in adulthood after a primary infection of varicella (chickenpox) has occurred in childhood. The virus lies dormant on a cranial nerve or a spinal nerve dermatome until it becomes activated years later. Because the virus affects only a specific nerve, the condition typically presents with unilateral manifestations—for example, pain, paresthesia, and a vesicular rash that develops in a line over the area innervated by the affected nerve (**Figure 13-25**).

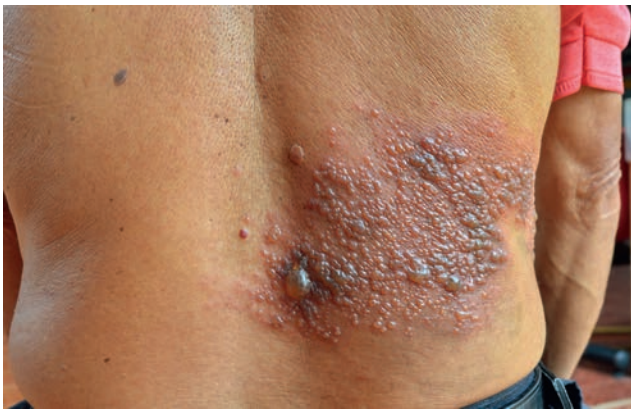


Figure 13-25 Herpes zoster.

The rash may appear red or silvery, and it occurs on one side of the head or torso depending on the nerve affected. The skin often becomes extremely sensitive, and pruritus may be present. The rash may persist for weeks to months. In some cases, especially in older individuals, postherpetic neuralgia or pain may continue long after the rash disappears. Blindness may result if the eye is affected.

Antiviral agents can limit the condition's duration and severity, and antidepressant and anticonvulsant agents have been beneficial in relieving the neuralgia associated with shingles. The Centers for Disease Control and Prevention (CDC, 2019) estimates that up to one in three people in their lifetime may develop herpes zoster, so vaccination with two doses of recombinant zoster vaccine (Shingrix) is recommended starting at the age of 50. A person can receive the herpes zoster vaccine even if they have already had the disease. Vaccines are also available to prevent varicella, and they are usually administered during childhood. Individuals who have been vaccinated with the varicella vaccine (licensed in the United States in 1995) have a lower chance of developing herpes zoster.

Verrucae

Verrucae, or warts, are caused by a number of human papillomaviruses. These skin lesions can develop at any age and often resolve spontaneously. They can be transmitted through direct skin contact between people or within the same person. The incubation period is approximately 2 to 6 months.

The human papillomavirus replicates in the skin cells, causing irregular thickening. Lesions that vary in color, shape, and texture depending on their type can appear. There are three categories of warts: (1) verruca vulgaris (common wart); (2) verruca plana (flat warts because they appear as flat-topped papules); and (3) verruca plantaris (plantar warts because they appear on the plantar surface of the foot; **Figure 13-26**). Plantar surface warts are a common cause of forefoot pain.

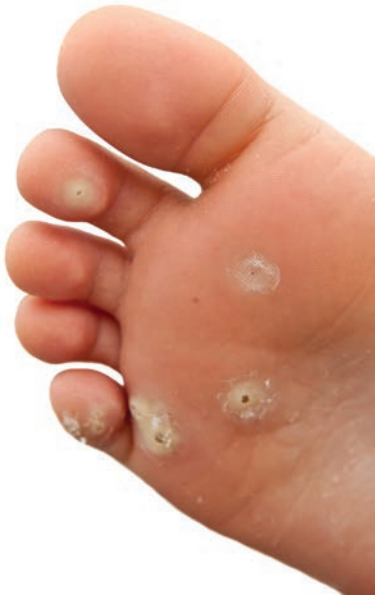


Figure 13-26 Plantar warts.

When warts are pared with a file such as an emery board, small capillaries that are thrombosed may become visible. The presence of the thrombosed capillaries further confirms the diagnosis of a wart, as calluses and corns may look like warts but will not have these vessels. Treatment includes a wide range of local applications such as laser treatments, cryotherapy with liquid nitrogen, electrocautery, and topical medications (e.g., keratolytic, cytotoxic, and antiviral agents), but the verrucae may return after treatment.

Molluscum Contagiosum

Molluscum contagiosum is caused by a poxvirus. Molluscum contagiosum is common in children and is spread by direct skin contact between people and within the same person. The infection can also spread through fomites (inanimate objects that when contaminated with organism can transfer the organism to a host). Examples of fomite transmission would be towels, sponges, or razors. In adolescents and adults, the virus is usually sexually transmitted (lesions are most likely in genital areas) or through contact sports.

The incubation period is 1 week to 6 months. Molluscum lesions are dome-shaped white papules that are shiny, firm, and have a craterlike center (**Figure 13-27**). Lesions can become inflamed. The lesions often resolve without treatment, and it can take up to 1 year for complete resolution. A wide range of treatment is available that includes cryotherapy

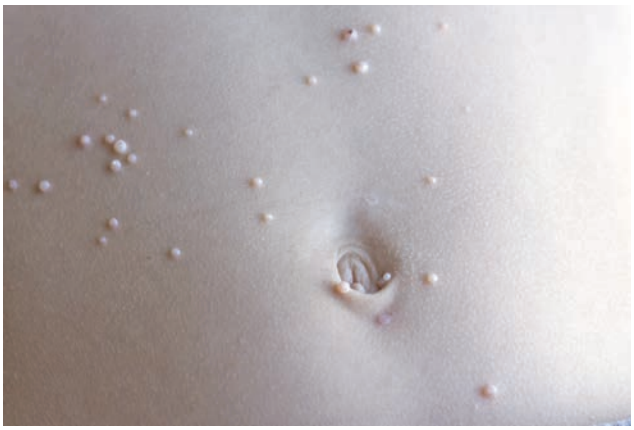


Figure 13-27 Molluscum contagiosum lesions around a person's navel.

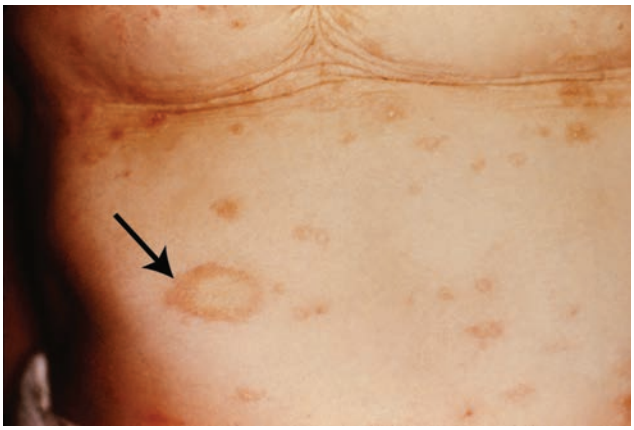


Figure 13-28 Pityriasis rosea and Herald patch.

Courtesy of CDC.

with liquid nitrogen, curettage (physical removal with a curette), and topical medications (e.g., keratolytic agents, cantharidin, and antimitotic agents); however, evidence is lacking to determine effectiveness of these treatments.

Pityriasis Rosea

Pityriasis rosea is a disorder that is thought to be caused by a virus, as the lesions often occur after a viral infection. There is also an absence of association with bacterial and fungal organisms. The human herpes virus and H1N1 influenza virus have been implicated; however, there is inadequate evidence to firmly conclude these organisms as causative. The lesions are benign and noncommunicable. The disorder begins with a lesion termed the *herald patch* that is solitary, salmon-colored, fine, scaly, and clearly demarcated (**Figure 13-28**).

The herald patch usually appears on the chest, neck, or back. After this initial patch, several other similar-appearing patches, scaly papules, and plaques of varying sizes usually start to appear after a few days to 2 weeks. The lesions are on the trunk and proximal areas of the extremities. The distribution of the lesions on the back are said to appear like a Christmas tree. Usually, erythema and pruritus are present, but some cases are asymptomatic. In children, the distribution of lesions is often atypical and appears inversely to adults; lesions appear on the face and distal extremities with sparing of the trunk. In children, the lesions may also be vesicular, pustular, urticarial, or purpuric.

The lesions can take up to 2 months to resolve; however, darker pigmented individuals may have postinflammatory hyperpigmented lesions that last for several months. The diagnosis is made by the characteristics of the lesion, but a potassium hydroxide (KOH) microscopic examination may distinguish the disorder from tinea corporis (i.e., ringworm). The lesions of pityriasis rosea are similar to those caused by secondary syphilis, so serologic testing for syphilis may be indicated in those at risk (e.g., those who are sexually active).

Treatment is not necessary. However, the rash can appear dramatic, so education and reassurance of the benign nature are necessary. If pruritus is present, topical corticosteroids and antipruritics may be used.

Herpangina and Hand-Foot-and-Mouth Disease

Enteroviruses, particularly the coxsackievirus, are implicated in two conditions known as *herpangina* and *hand-foot-and-mouth disease*, which are more common in children. Transmission occurs from contaminated hands, respiratory secretions, and fecal matter. The incubation period is 4 to 6 days. In hand-foot-and-mouth disease, vesicles (small blisters) that can ulcerate appear mainly on the buccal mucosa and tongue. The vesicles can also be on the palate, tonsils, hands, and feet. In herpangina, the lesions are more toward the back of the mouth on the soft palate, uvula, and tonsils. The lesions are grayish-white papulovesicular (raised and blistery) sores that ulcerate. Usually fever, malaise, and odynophagia (painful swallowing) are present. Treatment is geared toward symptom relief, and both diseases are self-limiting and resolve in 7 to 10 days.

Parasitic Infections

Several parasitic infections can occur on the skin, including those caused by fungi. Fungi can include yeasts, of which *Candida albicans* is a common infection-causing pathogen. Fungi can also include

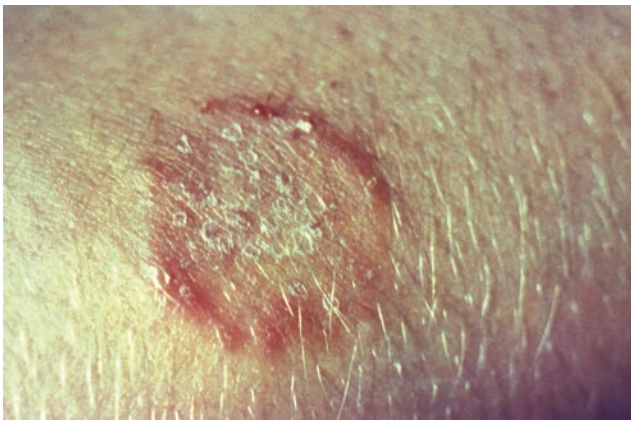
pathogens that cause tinea infections. The tinea pathogens are generally part of three genera: *Microsporum*, *Epidermophyton*, or *Trichophyton*. These conditions are usually diagnosed through microscopic examination of skin scrapings processed with KOH. The presence of hyphae confirms the diagnosis (see the reproductive function chapter).

Many of the causative organisms feed off the dead skin cells of the host and may use the host as a breeding ground. Some of the numerous parasitic skin infections are profiled in the following sections.

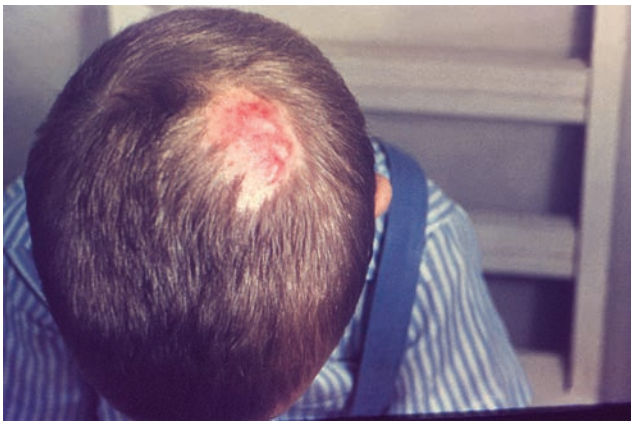
Tinea

Tinea (i.e., dermatophytosis) causes several types of superficial fungal infections. The organisms live and disrupt the keratinized cells of the epidermis. These fungi typically grow in warm, moist places (e.g., showers and locker rooms) and are spread through direct contact. Tineas are described based on the area of the body affected and include:

- **Tinea corporis** is an infection of the body. Tinea corporis typically manifests as a circular, erythematous, scaling patch or plaque. The center of the plaque clears, and there is a circular raised border, giving the common name of ringworm (**Figure 13-29a**).
- **Tinea capitis** is an infection of the scalp commonly encountered in school-age children. Along with the typical rash associated with tinea, hair loss at the site is common (**Figure 13-29b**).
- **Tinea pedis**, also called *athlete's foot*, is the most common dermatophytosis. Tinea pedis involves the feet, especially between the toes. The manifestations of tinea pedis are usually hyperkeratotic and erythematous lesions of the foot. The lesions create the appearance that a person is wearing a moccasin. Tinea pedis between the toes generally causes erythematous erosions and fissures or scales. Tinea in the midfoot area usually causes vesicles or bullae with erythema.
- **Tinea cruris** is an infection of the inner thigh that usually causes tiny vesicles with an erythematous, sharply demarcated border. Tinea cruris can spread around the genital area but usually does not spread to the scrotum. Candidal infections of the groin are differentiated as they cause papules and pustules and can affect the scrotum.
- **Tinea unguium** (i.e., onychomycosis) is an infection of the nails, typically the toenails (in fingernails the fungi is usually *Candida albicans*). This infection begins at the tip of one or two nails and then usually spreads to other nails. The nail initially turns white and then brown, causing it to thicken and crack. Clippings of the nails can be cultured.



A



B

Figure 13-29 Tinea. **(A)** Ringworm on the arm. **(B)** Ringworm on the scalp.
A: Courtesy of Dr. Lucille K. Georg/CDC; **B:** Courtesy of CDC.

Most of the tineas described, regardless of location, can cause pruritus and pain. Several topical and systemic antifungal agents are available to treat tinea infections, but several weeks of treatment may be necessary to resolve the infection.

Tinea versicolor (i.e., pityriasis versicolor) is a superficial, benign, noncommunicable fungal infection. In contrast to other tinea infections, tinea versicolor is not a dermatophytosis as it is caused by yeasts from the *Malassezia* genus (normal skin flora). The disorder has a higher incidence in tropical countries due to the heat and humidity. Excessive sweating also contributes to the development. Tinea versicolor is more prevalent in adolescents and young adults.

The lesions appear as macules and patches and thin plaques that are hyper- or hypopigmented (hence the name *versicolor*). The distribution is usually on the trunk and distal extremities (similar to pityriasis rosea) as the yeast depends on lipids, and sebum

production is greater in the upper body. However, the face is commonly involved in children. The lipid dependency also explains the age distribution as less sebum is produced with aging. Diagnosis is made clinically and confirmed through microscopy with KOH preparation. In some cases, a Wood lamp may reveal yellow-green fluorescence. Treatment includes topical antifungals (e.g., selenium sulfide or azoles). Lesions can take months to clear, even with successful treatment, and recurrence is common.

Scabies

Scabies is a result of a mite (*Sarcoptes scabiei*) infestation. The male mites fertilize the females and then die. The female mites burrow into the epidermis, laying eggs over a period of several weeks in a series of tracts. After laying the eggs, the female mites die. When the larvae subsequently hatch from the eggs, they migrate to the skin's surface. They burrow into the skin in search of nutrients and mature to repeat the cycle. This burrowing appears as small, light brown streaks on the skin (**Figure 13-30**).

The burrowing and fecal matter left behind by the mites triggers the inflammatory process, leading to erythema and intense pruritus, which is worse at night. The lesions are many small erythematous papules. The distribution usually involves the sides and webs of the fingers. Many other body areas such as the wrists, axillae, around the areola, and genitalia (it can include the scrotum) can become affected.

If there are significant numbers of mites (millions as opposed to the usual 15 or so), then thick scales, crusting, and fissuring may be present. The mites can survive for only short periods without a host, so transmission usually results from close contact (between household members or through sexual contact). Several topical treatments (e.g., permethrin) for scabies are available, but multiple applications are usually needed to successfully eradicate the infestation. Clothing, linens, and other fabrics will likely require treatment as well, although transmission is more likely due to close contact.

Pediculosis

Pediculosis refers to lice infestation, which can take three forms—*Pediculus humanus corporis* (body louse), *Phthirus pubis* (pubic louse), and *Pediculus humanus capitis* (head louse). Lice are small, brown, parasitic insects that feed off human blood and cannot survive for long without the human host (**Figure 13-31**). The female lice lay eggs (nits) on the hair shaft close to the scalp (**Figure 13-32**). The nits appear as small white, iridescent shells on the hair. After hatching, the lice bite and suck the host's blood; in turn, the site of the bite develops a highly pruritic macule or papule. Hyperpigmentation can occur and is due to inflammation. The lesions of pediculosis can also occur around seams of clothing as the lice lay eggs in seams and live on clothing. Pediculosis is easily transmitted through close contact. Several topical treatments

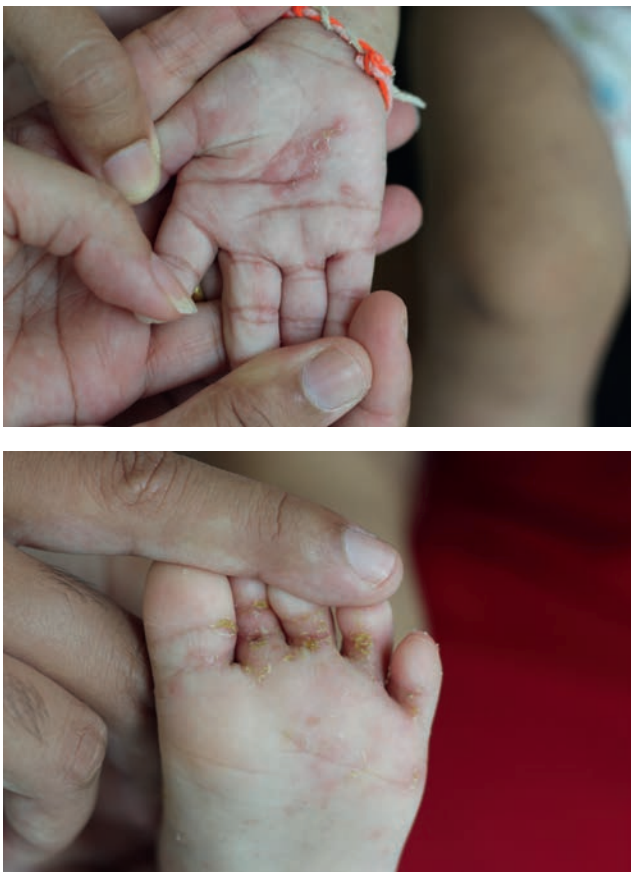


Figure 13-30 Scabies.

Figure 13-31 Louse.

(e.g., permethrin) are available, but multiple applications are usually needed to successfully eradicate the infestation. Clothing, linens, and other fabrics will require washing with hot soapy water.

Traumatic Integumentary Disorders

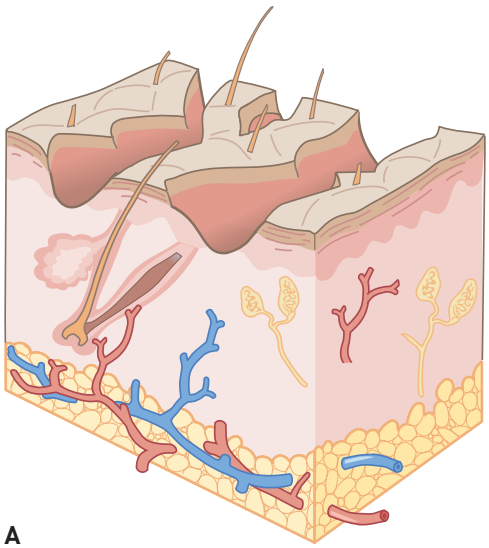
Traumatic integumentary disorders can result from a wide range of injuries. Skin trauma can produce multiple skin conditions, depending on the nature of the injury (**Figure 13-33**). Such injuries may range from mild to life threatening in severity, depending on the location and extent of the injury. Regardless of the nature or extent of the injury, all traumatic skin conditions increase the risk for infection because they create a breach in the body's protective barrier. Although numerous traumatic skin conditions are possible (e.g., lacerations and abrasions), this section will focus on burns.

Figure 13-32 Nits.

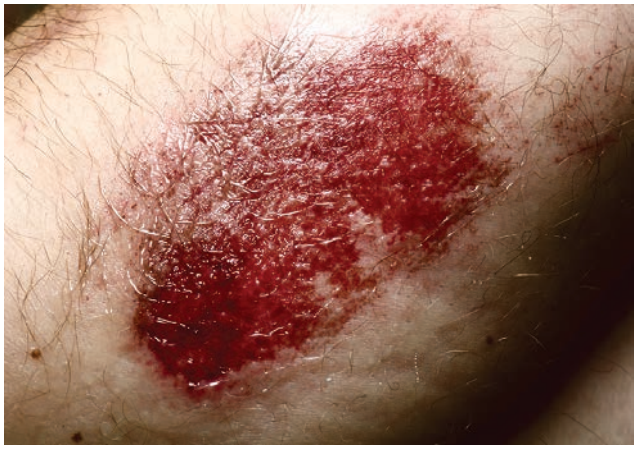
Burns

A burn is a skin injury that results from exposure to either a thermal (heat) or a nonthermal source. Such sources may include dry heat (e.g., fire), wet heat (e.g., steam or hot liquids), radiation, friction, heated objects, natural or artificial UV light, electricity, and chemicals (e.g., acids, alkaline substances, and paint thinner). The burn injury triggers the inflammatory reaction and results in tissue destruction. The severity of the condition varies depending on the location, extent, and nature of the injury. Severity is described, in part, in terms of the levels of the skin that are damaged (**Figure 13-34**):

- **Superficial (first-degree) burns** affect only the epidermis. These burns cause pain, erythema, and edema.
- **Partial-thickness—superficial or deep (second-degree)—burns** affect the epidermis and dermis. Partial-thickness burns cause pain, erythema that blanches, edema, and blistering (usually within a day). Hypopigmentation may remain, but scarring does not occur. Deeper partial-thickness burns will also damage hair follicles and glands and are painful with pressure only. The burn causes blistering and can have a cheesy whitish to red appearance with no blanching. Hypertrophic scarring is common, and resolution can take up to 9 weeks.

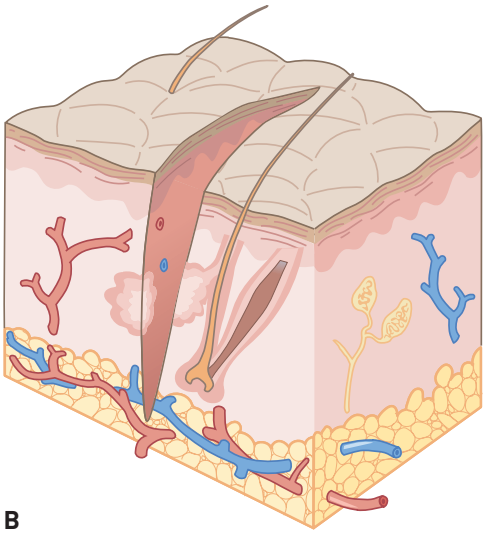


A

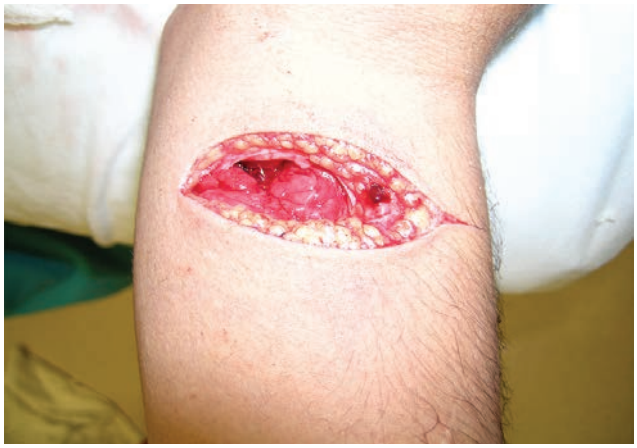


Abrasion

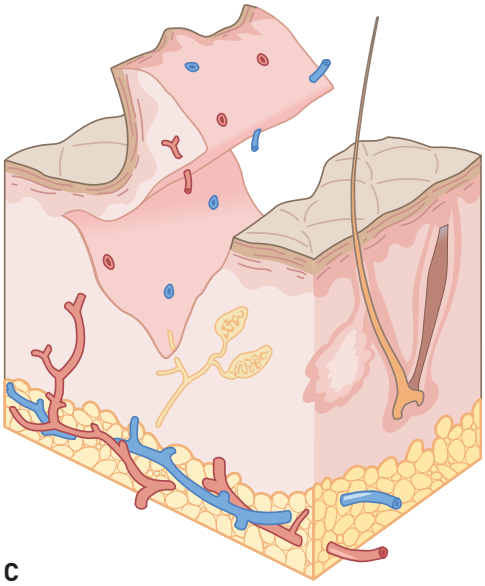
Figure 13-33 Types of wounds.



B

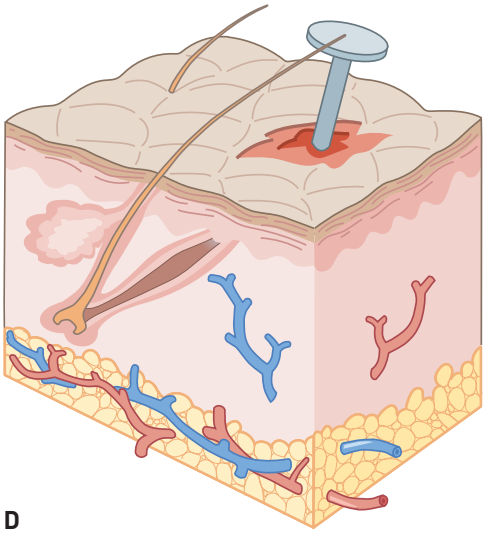


Laceration

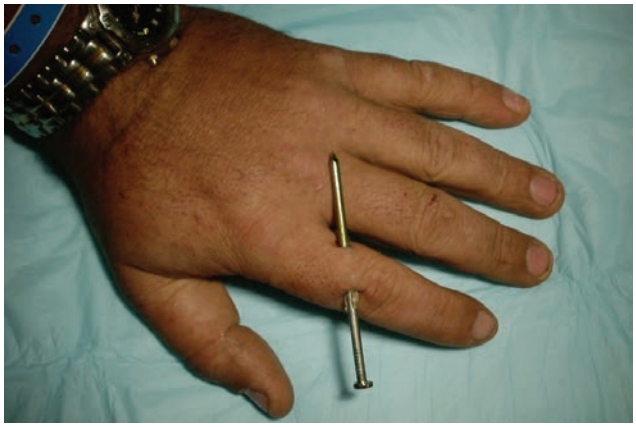


C

Avulsion



D



Penetrating wound

Figure 13-33 Type of Wounds.

B: © E.M. Singletary, M.D. Used with permission; **C:** © NeeooN/Shutterstock; **D:** © E.M. Singletary, MD. Used with permission.

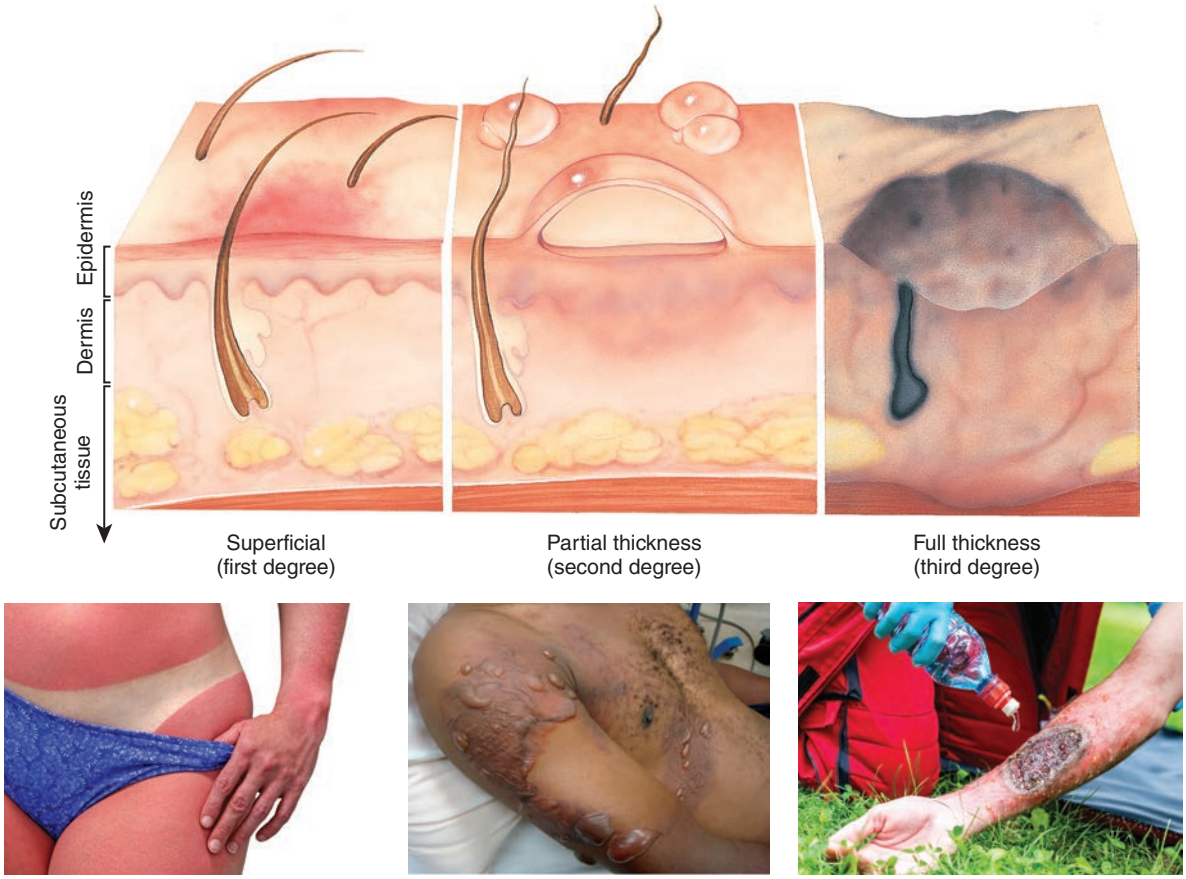


Figure 13-34 Burn classification.

Bottom 1: © Amy Walters/Shutterstock; **Bottom 2:** Courtesy of Rhonda Hunt; **Bottom 3:** © microgen/iStock/Getty Images.

- **Full-thickness (third-degree) burns** extend into deeper tissues (full dermis and subcutaneous). These burns cause white or blackened, charred dead skin (i.e., eschar) that may be numb. Eschar is inelastic, dry (no vesicles), and does not blanch. Scarring is severe and often includes contractures.
- **Deep-tissue-extension (fourth-degree) burns** can extend to underlying muscle and bone.

Clinical Manifestations. Complication development is usually related to burn severity. Burns may result in any of the following complications:

- Local infection (particularly *Staphylococcus* infection)
- Sepsis
- Hypovolemia (burns can damage blood vessels and plasma proteins, causing fluid shifts; see the fluid, electrolyte, and acid–base homeostasis chapter)
- Shock (may result from sepsis or hypovolemia)
- Hypothermia (heat is lost through large injuries)
- Respiratory problems (inhaling hot air or smoke can burn the tissues making up the airway, causing inflammation)
- Scarring
- Contractures

Diagnosis and Treatment. Burns are diagnosed based on a history and physical examination that includes identifying the type of burn and the body surface area of the burn. Treatment varies and is dependent on the location and severity of the burn.

Treatment of a severe burn at the scene of the injury includes the following:

- Removing the source of the burn. If someone is on fire, have the person stop, drop, and roll. Wrap the person in thick material to smother the flames (e.g., a wool or cotton coat, rug, or blanket). Douse the person with water.
- Ensuring the person is breathing. Initiate cardiopulmonary resuscitation if necessary. Continue to monitor the patient's respiratory status—it can become impaired as edema worsens.
- Leaving burned clothing that is stuck to the skin. The clothing may be soaked with sterile water or saline and then removed, and surgical removal may be necessary in severe cases.

At the emergency department, a thorough evaluation of severe burns starts with a primary and secondary survey. The primary assessment proceeds in an ABCDE format, as follows.

ABCDE—Burn Assessment

- **A** **A**irway management
- **B** **B**reathing and ventilation
- **C** **C**irculation and **c**ardiac status
- **D** **D**isability, neurologic **d**eficit, and gross **d**eformity
- **E** **E**xposure (completely disrobe the patient, examine for associated injuries, and maintain a warm environment)

Airway, respiratory, and ventilation compromise should be suspected with inhalation injury. Airway edema can develop rapidly and be life threatening, so maintaining a patent airway is a priority. Suspicions of airway compromise should arise when there is a history indicating smoke, soot, or toxic fume exposure.

Physical signs may include facial burns, singed hair, evidence of soot in oral area, and sputum that appears like soot. Other clinical signs are similar to airway compromise in other conditions (e.g., hoarseness, stridor). Airway management can be accomplished with oral airway devices, endotracheal intubation, or creation of a surgical airway (e.g., tracheotomy). Circumferential burns to the neck or trunk may cause a tourniquet-type effect and impair chest wall movement, so a bedside escharotomy may be necessary.

Circulation and cardiac status evaluation includes focusing on oxygenation (e.g., use of pulse oximeter) and perfusion. Due to the burn injury and subsequent catecholamine response, the heart rate may be slightly tachycardic (e.g., 100–120 beats/minute). Vital signs should be monitored frequently to assess signs of shock (e.g., tachycardia, usually beyond 120 beats/minute) and hypotension.

The administration of intravenous fluids (which may include colloids or crystalloids) is based on specific formulas. Adults with burns greater than 20% of total body surface area (TBSA) and children with burns greater than 10% TBSA will be administered fluids based on body weight and percentage of burn. Fluid bolus administration should be avoided as it may exacerbate edema.

Exposure in the primary survey involves awareness of the altered thermoregulatory abilities. While the patient is being evaluated, all clothing, jewelry, and contact lenses need to be removed. Remove any items that can act as a tourniquet as edema will occur. The environment should be warm, and blankets can be used to prevent hypothermia. If cooling of the burn is necessary, only cool—not cold—water should be used.

The secondary assessment involves evaluating nonburn injuries that could be life threatening and require attention prior to beginning evaluation of the burn injury. At this stage, laboratory evaluation, such as CBC and blood chemistry, can be conducted, and other imaging, such as a chest X-ray can be done as necessary.

After the primary and secondary assessment, attention can be placed on evaluating the burn injury, including depth and body surface area affected. The goal of burn wound care is to promote healing and prevent infection. The general treatment of severe burns includes the following:

- Wound care, which is dependent on the depth of the burn. There are several types of dressings and techniques (e.g., open, closed) available.
- Cleansing the wound with tap water or sterile solutions (there is no evidence for benefit or harm between types). Tap water, if used, should be running (not stored) and meet the standards of the World Health Organization.
- Rupturing blisters only under circumstances where it may be beneficial, such as when a deeper burn may be underneath.
- Protecting the burn area from pressure and friction.
- Limiting the risk for infection and promoting healing by meticulous wound care.
- Providing a clean and dust-free hospital environment, as infections in the hospital usually occur through surface contact, air, and water.

Full-thickness burns will not heal on their own, so excision and grafting are necessary. Excision can be tangential, meaning only the overdevitalized tissue is removed at an angle. Fascial excision involves removal of all layers of the eschar and tissue all the way to the fascia. Fascial excision is usually necessary for deep burns and high-voltage electrical conduction burns. Deep partial-thickness burns (less than full thickness) may also recover quicker with excision and grafts. Skin grafts promote tissue regeneration, prevent scarring, and aid the healing process.

Other treatments will include pain management with oral and intravenous analgesics and sedation. Physical therapy will be necessary to reduce the effects of scar tissue and reduce contractures. Surgery may be necessary for the contractures. An increased dietary intake of protein and carbohydrates is indicated to

promote healing and meet the increased caloric needs. Prophylactic antibiotic administration should not be used as resistance may develop.

Minor and/or smaller surface area burns can be evaluated in the same manner that other nonthermal wounds are assessed. A minor burn is defined by the American Burn Association (2018) as a partial thickness of <10% TBSA for patients ages 10 to 50 years, <5% TBSA in patients younger than age 10 years or older than age 50 years, or a full thickness <2% TBSA without other injuries. Attention should be paid to the depth, as third-degree and deeper burns will not heal on their own and require surgical management.

Circumferential burns, even if in a small area, such as the finger, can act like a tourniquet, cutting off blood supply, so further evaluation and possible surgical management may be required. The burn should be treated in a specialized burn center if a second-degree burn is located on the hands, feet, face, groin, buttocks, or a major joint.

Minor burns can be managed on an outpatient basis. Superficial burns usually heal in less than 2 weeks. Treatment for minor burns includes the following:

- Removing the source of the burn.
- Running cool water over the area or soaking it in a cool water bath (not ice water) if the skin is unbroken. Keep the damaged area submerged for at least 5 minutes. Applying a clean, cold, wet bandage or towel will also help reduce pain.
- Evaluation to determine whether debridement is necessary prior to dressing.
- Debridement can be accomplished with gentle mechanical techniques (e.g., brushing, scraping, or cutting). Enzymatic topical products (e.g., collagenase) can also be used for debridement.
- Coverage with a dry, sterile bandage or clean dressing.
- Protection from pressure and friction.
- Administration of analgesics and nonsteroidal anti-inflammatory drugs to relieve pain and swelling.
- Daily cleansing with mild soap and tap water.
- Application of moisturizing lotion (on intact skin) once the skin has cooled.
- Application of topical antibiotics. Topical antibiotics are usually not necessary if a burn is superficial.
- Tetanus vaccination.

A patient with a minor burn injury will need to be taught how to change their dressing and to watch for signs of infection. The patient should be evaluated soon after the injury and followed closely (e.g., daily). At follow-up, the burn should be reevaluated. Pruritus is common as a burn is healing and can be managed with oral antihistamines. If the minor burn does not heal within 3 weeks, a referral may be necessary to a surgeon as additional measures become necessary. Hypertrophic scarring is more likely the longer it takes for a wound to heal. Hypertrophic scarring can be avoided or minimized with pressure garments, massage, and moisturization.

Pressure Injuries

Pressure injuries are defined as injuries to the skin and soft tissue that have occurred because of unrelieved pressure. The result is damage to the underlying tissue. *Pressure injury* has replaced the common term, *pressure ulcers*, to reflect that deep-tissue injury can occur without ulceration. Pressure injuries occur when an external force is applied to the skin. When the pressure applied is greater than arteriolar pressure of the skin (32 mmHg), then tissue hypoxia can occur. Pressure injuries in vulnerable patients could occur in as short as 1 hour if the pressure applied is high enough and sustained. As an example of applied pressures, there is 300 mmHg of pressure over the ischial tuberosities when a person sits.

Muscle tissue is the most vulnerable to injury caused by pressure, followed by the subcutaneous fat and dermis, so pressure injuries can start deep internally and not be evident on the surface. These deep injuries can then progress to the surface. The hypoxia that can occur from pressure is not the only factor contributing to injury. Compression of small vessels and injury from reperfusion also contribute to hypoxia and damage to the skin.

Shearing forces (such as when a patient is inclined with the head of the bed up) contribute to the injury as muscle and fat are stretched downward. Moisture and friction can contribute to alteration in skin integrity and have been associated with ulcer formation. Moisture alone, such as from urine or perspiration, can lead to skin softening and breakdown (i.e., maceration), and then an ulcer can form. Friction, such as that caused by sliding a patient across a surface, leads to abrasions (see Figure 13-33) and, therefore, leads to a breach in skin integrity and potential ulcer formation. These macerations and abrasions as described are not pressure injuries.

Pressure injuries are usually located over bony prominences such as the sacrum, coccyx, heels, elbows, and trochanters. Pressure injuries can also

occur from pressure caused by medical devices. Risk factors for the development of pressure injuries include immobility (the most significant factor), malnutrition, inadequate tissue perfusion (e.g., hypotension, vasoconstriction), and sensory loss (e.g., neuropathy). Sensory loss is a risk as a patient loses the ability to sense the discomfort or pain from prolonged pressure. Various tools (e.g., Braden scale) can be used to predict risk for pressure injury development.

Clinical Manifestations. Pressure injuries are described based on their stage (e.g., from 1 to 4; **Figure 13-35**). The staging is a reflection of the extent of an injury, the depth, and other injury features. The National Pressure Ulcer Advisory Panel (2016) developed the staging system, and the descriptions are as follows:

- **Stage 1:** Intact skin with nonblanchable erythema. The area is localized and does not include purple or maroon discoloration, which may indicate a deep-tissue pressure injury. Prior to visual changes, blanchable erythema, changes in sensation, temperature, or firmness may be present.
- **Stage 2:** Partial-thickness loss of skin, with exposed dermis. The wound is viable, pink or red, and moist. The wound may present as an intact or ruptured serum-filled blister. Deeper structures, such as adipose (fat) and deeper tissues, are not visible.

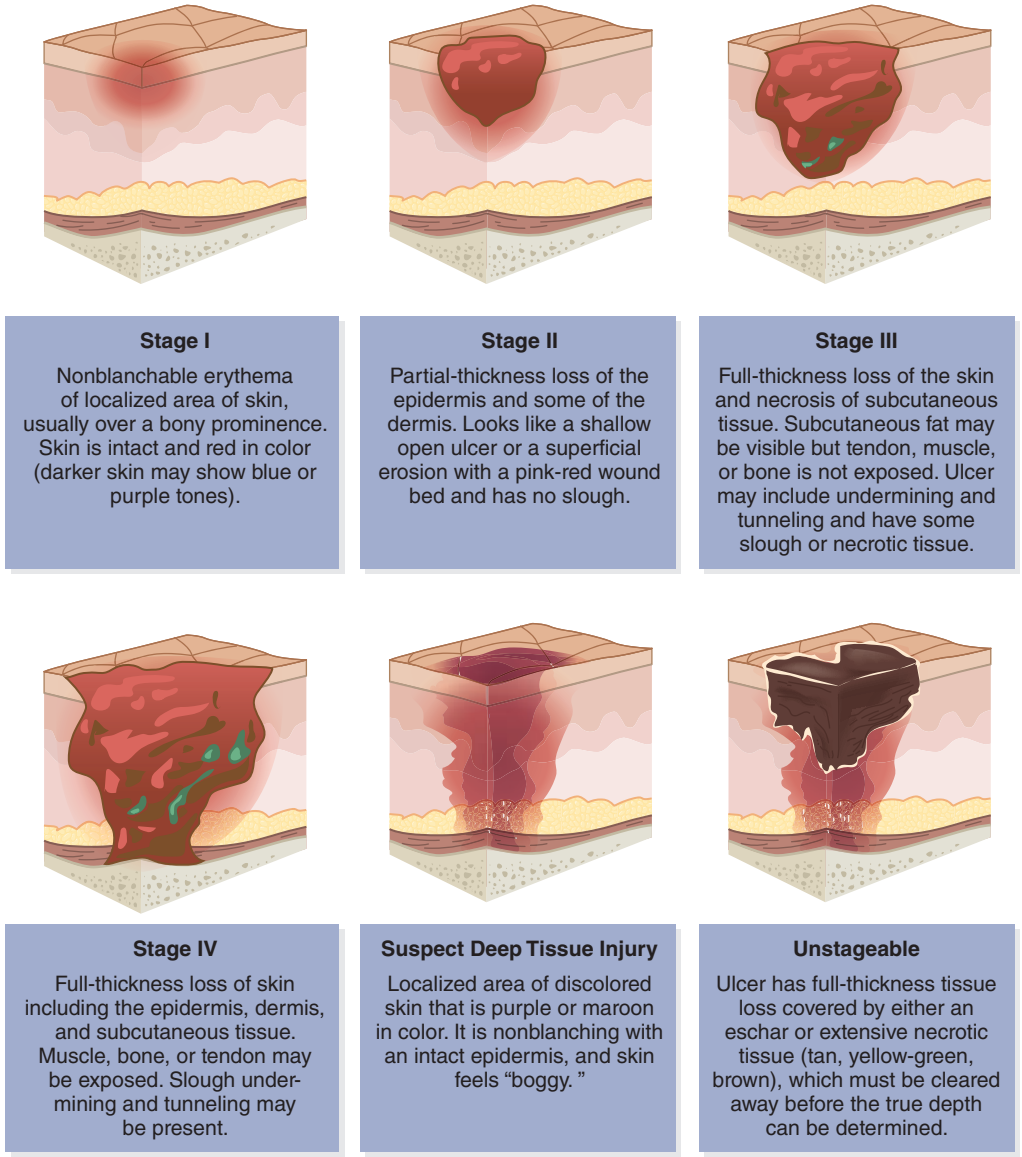


Figure 13-35 The stages of pressure injuries.

There is no granulation tissue (an intermediary to healing and when healthy is shiny red and granular), **slough** (adherent or non-adherent non-viable tissue that is usually yellowish), or **eschar** (necrotic tissue that is leathery, dry, and hard).

- **Stage 3:** Full-thickness skin loss. Adipose is visible in the ulcer and granulation tissue, and an epibole (rolled wound edges) are often present. The depth of tissue damage varies by anatomic location; areas of significant adiposity can develop deep wounds. Undermining (tissue destruction that occurs at the wound perimeter) and tunneling (wound extension in one direction that is deep) may occur. Fascia, muscle, tendon, ligament, cartilage, and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss, it is an unstageable pressure injury.
- **Stage 4:** Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage, or bone in the ulcer. Slough and/or eschar may be visible. Epibole, undermining, and/or tunneling often occur. Depth varies by anatomic location. If slough or eschar obscures the extent of tissue loss, it is an unstageable pressure injury.
- **Unstageable pressure injury:** Obscured full-thickness skin and tissue loss. The extent of the tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a stage 3 or stage 4 pressure injury will be revealed. Stable eschar (i.e., dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.

Deep tissue injuries can occur with intact skin. These injuries at inception do not quite fit into any stages. These injuries may appear as a persistent,

Application to Practice

A 72-year-old Black woman of Haitian descent has a past medical history of hypertension, diabetes mellitus type 2, and osteomyelitis with left great toe amputation. She was discharged from the hospital status post amputation. She does not smoke, does not drink, and eats a regular diet. She is continent of bladder and bowel. On physical exam she is found to have an intact surgical wound on left foot without evidence of infection. On physical exam for the remainder of her skin she is noted to have an area of deep maroon discoloration on her sacrum that is nonblanchable and tender to touch. Answer the following questions:

1. Describe assessment techniques that could be used to evaluate pressure injuries in people with darker skin tones.
2. What pressure injury stage would the sacrum wound be classified as?
   a. Stage 1
   b. Stage 2
   c. Stage 4
   d. Deep tissue injury
3. What age-related skin changes could contribute to the development of this sacral pressure injury? Select all that apply.
   a. Thinning of subcutaneous fat layer
   b. Decreased sensation of pressure and pain
   c. Photo aging
   d. Changes in hair follicles and rate of hair growth
4. What are some next steps that you might take? Select all that apply.
   a. Decrease fluid intake to reduce risk of incontinence
   b. Debride the wound to determine staging
   c. Position patient to offload sacrum
   d. Order specialized bed
5. During a follow-up home visit the following week, the affected area of the sacrum is now an open wound with visible adipose tissue and a non-adherent slough on 15% of the wound bed. What pressure injury stage would the wound be classified as?
   a. Stage 2
   b. Stage 3
   c. Stage 4
   d. Unstageable
6. Define the following possible injury findings: granulation tissue, slough, eschar, and epibole and at what stage each is likely to be found.

Case contributed by: Carmen Victoria Framil, DNP, APRN, ANP-BC

nonblanchable deep red, maroon, or purple discoloration (nonintact or intact skin) or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature changes may precede this type of injury. The wound may evolve rapidly, and the extent will be visible and then can be staged, or resolution may occur without tissue loss.

Diagnosis and Treatment. Diagnosis is based on a history and physical examination using the staging criteria. Impaired skin integrity (e.g., ulcers, abrasions) due to other causes such as diabetes, arterial or venous insufficiency, or chemical irritation (e.g., urine) should be distinguished as treatment will vary (see the cardiovascular function chapter).

Learning Points

Pressure Injuries in Dark Skin Tones

Usual skin tone changes reflective of pressure injuries, such as blanching and erythema, may not be evident in darker skin tones. When evaluating a blanch response in a darker-skinned person, apply pressure and look for a darkened area or a shiny, taut induration. Underlying vasodilatory changes that cause erythema in lighter skin may present as hyperpigmentation or hypopigmentation, and redness may not be visible. Feeling for temperature and texture changes (e.g., increased warmth or soft bogginess) are necessary for pressure injuries. Moisturizing the skin may intensify skin color changes.

Treatment for pressure injuries is focused on reduction or elimination of pressure through redistribution with proper position and use of supportive surfaces. Pressure injury prevention points are as follows:

- Frequently turn and reposition patients in bed or in a chair.
- Turn the patient to a side-lying position, and with a hand, check that the sacrum is off the bed. The head of bed should be no higher than 30° to prevent shearing. If the head of the bed is higher than 30°, then a polyurethane foam dressing should be on the sacrum.
- If pressure injuries are present, do not position the person on the affected areas.
- Ensure that heels are not on the bed.
- Use pressure-redistributing cushions for sitting on chairs or wheelchairs.
- Use breathable dressings or thin foam under medical devices.
- Bed surfaces may be enhanced with nonpowered support devices, such as foam mattresses or overlays.
- Specialized beds may be necessary (e.g., air-fluidized beds, alternating-air mattress).
- Meticulous skin assessment is important in early identification.
- Daily cleansing with pH-balanced solutions will minimize injury.
- Use skin moisturizers daily.
- Ensure adequate nutrition and hydration.

Wound management is dependent on the pressure injury stage. Dressings are used to prevent infection and promote healing. Wounds that are especially moist and exudative will cause further skin breakdown and slow epithelial cell proliferation, while exceptionally dry wounds will slow epithelial cell migration and slow wound healing. The longer the wound is open, the greater the chance of infection and scarring. To maximize wound healing, proper nutrition with adequate protein and smoking cessation is critical.

Absorptive dressings, such as foams, are best for moist injuries, and hydrogels or other dressings that maintain moisture are best for dry wounds. Stage 1 injuries are covered with a transparent film as a protective barrier. Stage 2 injuries require maintenance of a moist environment. Various dressings can be used to maintain the moist environment. These include hydrocolloids or hydrogels, which are both occlusive, and transparent films, which are semi-occlusive. Stage 3 and 4 injuries generally require debridement for the wound to heal. Debridement includes the following: mechanical (e.g., hydrotherapy or wet to dry dressings), autolytic (e.g., synthetic occlusive or semi-occlusive dressings), enzymatic (e.g., collagenase, fibrinolysin), surgical, and biosurgical (e.g., medical maggot therapy) techniques. After debridement, an appropriate moist dressing is applied. Wounds, as in burns, may require skin grafting or mucocutaneous flaps for rapid healing. Adjunctive therapies for wound healing may be useful, but data are limited on their effectiveness. These therapies can include electrical stimulation, negative-pressure wound therapy, therapeutic ultrasound, hyperbaric oxygen or direct oxygen application, and use of topical growth factors.

Chronic Integumentary Disorders

Numerous chronic conditions can affect the integumentary system. Several have been discussed and categorized based on their underlying pathophysiology

(e.g., psoriasis and inflammatory disorders). These conditions vary in severity. Although most of these conditions are not life threatening, many can have a significant impact on an individual's appearance.

Acne Vulgaris

Acne vulgaris is an inflammatory skin disorder of the pilosebaceous unit (i.e., hair follicles and sebaceous glands). Acne vulgaris commonly affects adolescents and young adults, but it can occur at any age. The four key factors in the pathogenesis of acne vulgaris are (1) follicular hyperkeratinization, (2) increased sebum production, (3) bacteria in the follicle, and (4) inflammation (**Figure 13-36**).

The pathogenesis of acne vulgaris occurs when sebum production from the sebaceous gland increases under the influence of androgens. This period occurs during prepuberty and sebum accumulates, hyperkeratinization occurs, and the hair follicle becomes blocked. Microcomedones, which are a mixture of sebum and keratin and are not visible on the surface, develop. The microcomedones are lipid rich and provide an environment for the growth of *Cutibacterium acnes* (formerly known as *Propionibacterium acnes*), an anaerobic bacterial organism that is part of the normal skin flora. The bacteria stimulate the immune response with resulting inflammation. The microcomedones become closed comedones (whiteheads). The follicles start to widen and open to the skin, and open comedones

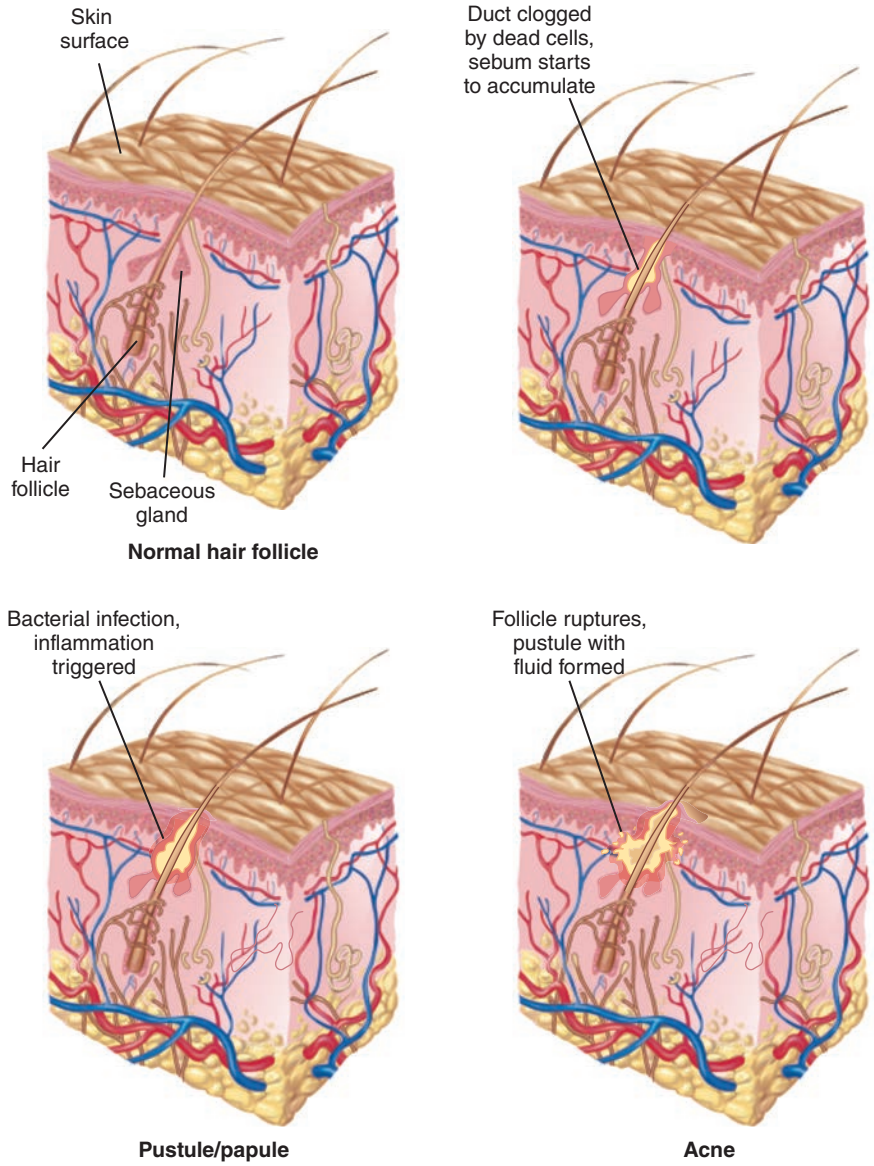


Figure 13-36 Pathogenesis of acne vulgaris.

(blackheads) develop. Blackheads are a mixture of keratinocytes, lipids, and melanin (which gives them a dark color). The follicles eventually rupture with further inflammation ensuing and resulting in inflammatory lesions of papules, pustules, cysts, and nodules (**Figure 13-37**).

Acne vulgaris commonly appears on areas with increased hormone-responsive sebaceous glands such as the face, neck, and shoulders (upper arms), but it may also occur on the trunk, arms, legs, and buttocks. This condition varies widely in severity, with severe cases sometimes resulting in significant scarring. Post-inflammatory hyperpigmentation is more likely to occur in darker skin tones and is the result of increased melanin production due to inflammation. Risk factors for acne vulgaris include the following:

- Family history
- Hormonal changes (e.g., changes that occur with menstrual periods, pregnancy, birth control pill use, and stress)
- Use of oily cosmetic and hair products, which block pilosebaceous follicles and cause comedo formation
- Use of certain medications (e.g., corticosteroids, testosterone, estrogen, and phenytoin)
- High levels of humidity and sweating
- Helmets, bra straps, and shoulder pads, which can cause occlusion of pilosebaceous follicles and comedo formation

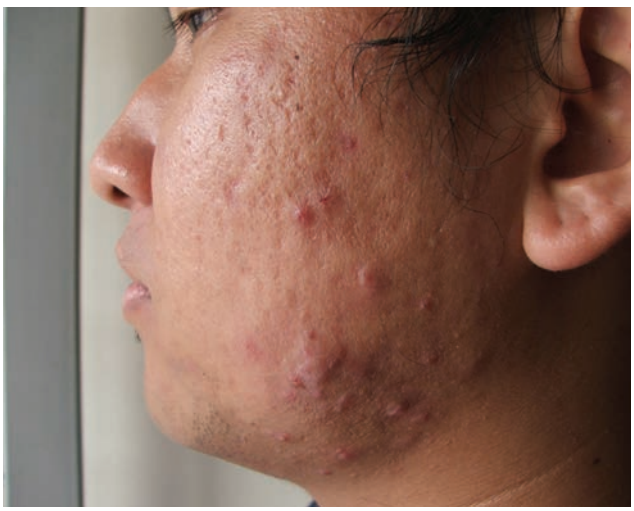


Figure 13-37 Acne vulgaris.

Diagnosis and Treatment. Diagnosis for acne vulgaris is made clinically with a history and physical examination. Diagnostic testing may be necessary if there are underlying disorders causing or contributing to the acne (e.g., polycystic ovarian syndrome).

Treatment varies depending on the severity. Treatment includes behavioral and medical (e.g., medications and techniques such as laser) strategies. Behavioral strategies include the following:

- Cleaning skin gently with a mild, nondrying soap to remove all dirt or makeup once or twice daily, including after exercising, but avoiding excessive or repeated skin washing
- Shampooing hair daily, especially if it is oily
- Combing or pulling hair back to keep it away from the face, but avoiding tight headbands
- Avoiding squeezing, scratching, picking, or rubbing acne because it can lead to skin infections and scarring
- Avoiding touching affected areas
- Avoiding oily cosmetics or creams; using water-based or noncomedogenic formulas instead
- Limiting sun exposure

Pharmacotherapeutic strategies target mechanisms that lead to the pathogenesis (**Table 13-2**). While monotherapy with one agent may be sufficient, there are circumstances where multiple agents—such as a topical retinoid with topical antimicrobial—are used.

Table 13-2 Pharmacotherapeutic Strategies

Pathogenesis	Retinoids	Acids/Benzoyl Peroxide	Antibiotics	Hormonal Therapies
Hyperkeratinization and desquamation (peeling)	+ (topical or oral)	+ Azelaic acid or salicylic acids		+
Increased sebum production	+ (oral)			+
C. acnes proliferation		+ Azelaic acid Benzoyl peroxide	+ (topical or oral)	
Inflammation	+ (topical or oral)	+ Azelaic acid	+ (oral)	

+ indicates effective in targeting underlying pathogenesis.

Hormonal therapies are reserved for women and include oral contraceptives with antiandrogenic ingredients such as drospirenone (spironolactone analogue). Oral isotretinoin (Accutane) is generally used for severe, difficult-to-manage nodular acne. Teratogenicity risk is high with oral isotretinoin, so contraception is important during use. Various medications are available over the counter or may require prescriptions.

Pharmacotherapeutic strategies, in addition to those in Table 13-2, include the following:

- Alternative therapies, including tea tree oil, zinc, guggul, and brewer's yeast
- Photodynamic therapy (laser/light procedure) and photopneumatic therapy (pressure and light)
- Chemical skin peels
- Microdermabrasion or dermabrasion
- Intralesional glucocorticoids for nodular acne
- Soft-tissue fillers (e.g., collagen and fat)

Rosacea

Rosacea is a chronic inflammatory skin condition that typically affects the face. It is poorly understood, but rosacea is prevalent in people who are fair skinned, people who bruise easily, and women. Proposed pathogenesis includes an innate immune dysfunction. The dysfunction causes an abnormal skin response to microorganisms in or on the skin, such as *Demodex* mites. The immune dysfunction leads to the production of chemicals (e.g., cathelicidin) that cause vasoactive and other inflammatory manifestations.

There are four subtypes of rosacea—erythematotelangiectatic, papulopustular (classic presentation), phymatous, and ocular. Rosacea may present as erythema, prominent spiderlike blood vessels (telangiectasia), or swelling (**Figure 13-38**). Acnelike (papules and pustules) eruptions can occur, but unlike acne, there are no comedones. Additional manifestations may include a burning or stinging sensation and red, watery eyes. Further eye examination may reveal infiltrates in the cornea and sclera. A thickening of the skin on the nose (rhinophyma) is more common in men, and the phymatous changes can also occur on the chin, forehead, or cheeks. If left untreated, rosacea is progressive, but most people with this condition experience remissions and exacerbations. Exacerbation triggers are specific to the individual but may include sun or wind exposure, sweating, stress, spicy food, alcohol, hot beverages, hot baths, and cold weather.

Diagnosis and Treatment. Diagnosis of rosacea is made clinically with a history and physical examination. Skin biopsy is indicated if there are suspicions of another disorder. There is no known cure for rosacea. Instead, treatment strategies center on identifying and avoiding possible triggers, so that affected individuals can reduce exacerbations. These strategies may include the following measures:

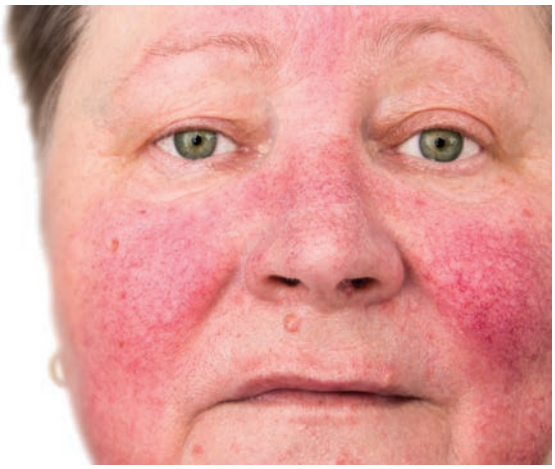


Figure 13-38 Rosacea.

- Avoiding excessive scrubbing when cleaning the skin
- Avoiding sun exposure (e.g., wearing protective hats and clothing, limiting exposure time especially between 10:00 a.m. and 4:00 p.m.) and using sunscreen that protects against both UVA and UVB rays every day
- Avoiding prolonged physical exertion in hot weather
- Managing stress (e.g., through deep breathing and yoga)
- Limiting spicy foods, alcohol, and hot beverages
- Avoiding any other triggers
- Applying topical or oral antibiotics, such as topical metronidazole (antifungal) or ivermectin (antiparasitic) to control skin eruptions (pustules and papules)
- Applying retinoic acid cream or gel (e.g., Retin-A) or oral isotretinoin (Accutane) for pustules and papules
- Administering topical vasoconstrictors (e.g., brimonidine tartrate or oxymetazoline)
- Performing laser/light therapies to help reduce redness
- Performing surgical reduction of enlarged nose tissue
- Applying green- or yellow-tinted prefoundation creams and powders to reduce the appearance of redness

Integumentary Cancers

Skin cancer is an abnormal growth of skin cells. According to the CDC (2021), skin cancer is the most frequently occurring cancer in the United States. Although the number of cases has recently declined slightly, the number of new cases and deaths continue to rise. Prevalence rates are highest in males, Whites, people with fair complexions, and those with a family history.

The overall 5-year survival rate is approximately 92%. UV exposure, either natural or artificial, is by far the most significant risk factor for this type of cancer. For this reason, most skin cancers occur on areas that have the most sun exposure (e.g., the arms and neck).

Three major types of skin cancer are distinguished and include basal cell carcinoma, squamous cell carcinoma, and melanoma. A discussion of each type follows.

- **Basal cell carcinoma** (BCC), the most common type, develops from abnormal growth of the cells in the lowest layer of the epidermis. BCC has a low metastatic potential. However, BCC can cause significant local tissue destruction, and ulceration is common. BCC cells have different clinical manifestations (e.g., nodular, superficial). Nodular is one of the more common types and presents as a flesh-colored papule that appears pearly or translucent (**Figure 13-39**). The lesion often has rolled borders (i.e., the borders are higher than the middle).
- **Squamous cell carcinoma** (SCC) involves changes in the squamous cells, which are found in the middle layer of the epidermis. The lesions have a varied presentation in comparison to BCC. Lesions can be papules, plaques, nodular, smooth, hyperkeratotic, or ulcerative (**Figure 13-40**). There is a higher potential for metastasis in comparison to BCC, but the incidence is low.
- **Actinic keratosis** (i.e., solar keratosis) is a skin lesion that is benign and is a result of proliferation of epidermal keratinocytes. These lesions are considered part of a continuum of SCC. Actinic keratosis lesions have the potential to progress to SCC. Actinic keratosis lesions can be similar to SCC.
- **Melanoma** develops in the melanocytes. It is the least common type but the most serious due to metastasis to other areas. There are several subtypes of melanomas, and the lesions have a varied presentation (**Figure 13-41**). The ABCDE rules were initially developed for early identification of melanoma and are often used in mole evaluation.

Clinical Manifestations. Skin cancers can vary widely in appearance; they can be small, shiny, waxy, scaly, rough, firm, red, crusty, bleeding, and so on (see Figure 13-40). Given the many possible presentations, any suspicious skin lesion should be biopsied. The following features may be considered suspicious:

- Asymmetry—part of the lesion different from the other parts
- Borders that are irregular

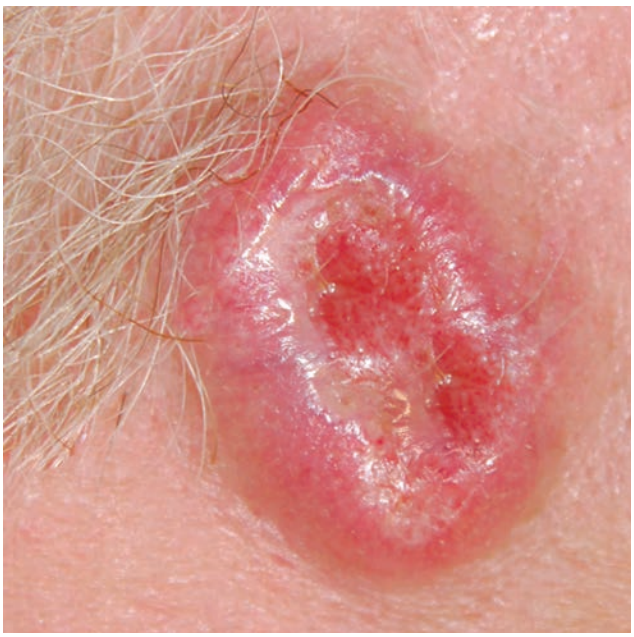


Figure 13-39 Basal cell carcinoma.

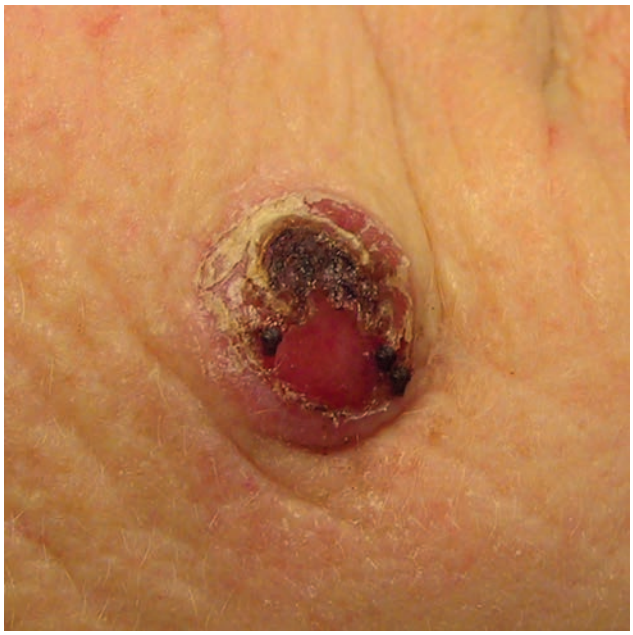


Figure 13-40 Squamous cell carcinoma.

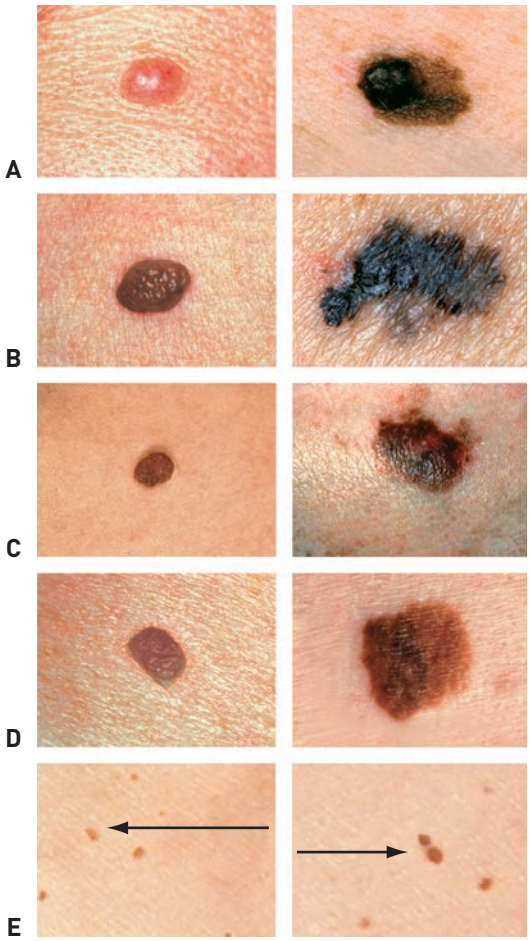


Figure 13-41 Skin cancers.

Courtesy of The Skin Cancer Foundation (www.skincancer.org).

Learning Points

Identifying Skin Cancers

All skin lesions, such as moles, should be monitored for any suspicious changes. These changes can be readily identified—their diagnosis is as easy as A, B, C, D, and E.

Asymmetry—part of the lesion is different from the other
Borders—irregular
Color—varies from one area to another with shades of tan, brown, or black (sometimes white, red, or blue)
Diameter—usually (but not always) larger than 6 mm in size (the diameter of a pencil eraser)
Evolution—a lesion that is changing in size, shape, or color, or a new lesion

- Color that varies from one area to another with shades of tan, brown, or black (sometimes white, red, or blue)
- Diameter that is usually (but not always) larger than 6 mm in size
- Sensory changes
- Any skin growth that bleeds or is crusting or will not heal
- Any skin growth that changes in appearance over time (e.g., shape, color)

Diagnosis and Treatment. Most skin cancers can be prevented by limiting or avoiding exposure to UV light (e.g., by using sunscreen and wearing protective clothing). Early detection is crucial to positive outcomes; with early detection, even the most aggressive forms can be successfully treated. Diagnostic procedures for skin cancer typically include a history, physical examination, and biopsy. Removal of cancerous growths offers the best prognosis. Treatment strategies may include the following measures:

- Cryosurgery
- Excisional surgery
- Laser therapy
- Mohs surgery (the skin growth is removed layer by layer, examining each layer under the microscope, until no abnormal cells remain)
- Curettage and electrodessication (layers of cancer cells are scraped away using a circular blade [curette], and then, an electric needle is used to destroy any remaining cancer cells)
- Radiation therapy
- Chemotherapy

Hair Disorders

Alopecia is defined as hair loss and is associated with several disorders. Hair loss can occur because of growth issues, inflammatory damage to hair follicles, or abnormalities in the hair shaft. There are scarring and nonscarring types of alopecia. The scarring types are termed *cicatricial alopecia* and are due to inflammation of the scalp, resulting in permanent hair loss. Nonscarring alopecia has mild or absent inflammation. The nonscarring alopecia can be further divided by the hair loss distribution with focal hair loss, diffuse hair loss, and patterned hair loss and is described as follows:

- **Alopecia areata** causes focal patches of complete hair loss. Alopecia areata is an autoimmune, T-cell mediated disorder of the hair follicle in genetically susceptible individuals. Alopecia areata in some patients can cause total loss of hair (scalp and body).
- **Telogen effluvium** commonly causes a diffuse hair loss distribution. The cause of telogen effluvium is a premature shift of hair entering the telogen (resting) cycle of growth from the anagen

growth phase. Most hair (about 90%) is in the anagen growing phase, and only a small amount (10%) is in the telogen phase. The hair loss can be acute or chronic, and there is a significant amount of shedding. The scalp is usually visible, but the whole head is not usually affected.

- **Patterned nonscarring alopecia** is the loss of hair in a predictable manner, male pattern hair loss (androgenetic alopecia), female pattern hair loss, and trichotillomania are examples of patterned distribution of hair loss. Male pattern loss usually proceeds in the frontotemporal area where there are androgen-sensitive hair follicles (sides and back of head have androgen-insensitive follicles). Female pattern hair loss usually involves thinning on the frontal and crown areas while the occiput is spared. Both male and female pattern hair loss are more likely to occur in genetically susceptible individuals. Trichotillomania is a mental health disorder characterized by repeatedly pulling hair out. As hair is pulled from different areas the distribution is usually irregular and has different shapes.

Nail Disorders

Nail disorders can cause an alteration in nail growth. Nail disorders can occur with systemic diseases (e.g., splinter hemorrhages with psoriasis), trauma, infection, cancer, or inherited skin/nail disorders. Fungal infections are termed *onychomycosis* and lead to nail thickening, discoloration, and abnormal shaping. Bacterial infections are a common type of infection caused by *S. aureus*.

The nail fold (i.e., around the base or sides of the nail) is often the site of infection and is termed **paronychia**. Viral infections can cause warts due to human papilloma virus around the nail but also in the nail bed. Herpetic whitlow is due to herpes simplex virus that can affect the hand and periungual (around toes/fingers) area. Herpes whitlow often occurs in children who suck their finger and have oral herpes or in healthcare workers through direct contact.

Squamous cell carcinoma and melanoma can occur in the nails. Similar to the ABCDE for skin cancer evaluation, there is an ABCDEF mnemonic for nail changes that should raise suspicion for melanoma.

ABCDEF—Nail Changes

Age of patient (peak age 50–70 years)

Band of dark (brown or black) pigmentation on nail bed; **b**readth greater than 3 mm; border irregular or blurred

Change in the band (e.g., size, rapid growth)

Digit involved (multiple digits are more common)

Extension of the pigmented band (e.g., nail fold)

Family history of melanoma

Nail disorders are diagnosed with a history and physical, and, if necessary (e.g., if melanoma is suspected), a biopsy. Nail infections can additionally be diagnosed with a KOH preparation and sending nail clippings for pathologic evaluation or culture. Nail disorders are treated based on their underlying disorder.

Chapter Summary

The integumentary system plays a vital role in homeostasis and well-being by protecting the body from invasion by pathogens, maintaining water balance, sensing changes in the environment, and regulating body temperature. Conditions affecting this system can cause issues with any or all these functions. Some of these conditions can be prevented through measures such as limiting UV light exposure through using sunscreen that protects against UVA and UVB rays, wearing protective clothing, and limiting time outdoors, especially between 10:00 a.m. and 4:00 p.m., when the sun's rays are the most intense. Early diagnosis and treatment of other conditions can improve prognosis.

References and Resources

American Academy of Orthopaedic Surgeons. (2004). *Paramedic: Anatomy and physiology.* Jones & Bartlett Learning.

American Burn Association. (2018). *Advanced burn life support provider manual.* Author.

Berlowitz, D. (2018). Epidemiology, pathogenesis, and risk assessment of pressure-induced skin and soft tissue injury. In K. A. Collins (Ed.), *Uptodate.* https://www.uptodate.com/contents/epidemiology-pathogenesis-and-risk-assessment-of-pressure-induced-skin-and-soft-tissue-injury

Centers for Disease Control and Prevention (CDC). (2019). *Shingles (herpes zoster).* https://www.cdc.gov/shingles/surveillance.html

Centers for Disease Control and Prevention (CDC). (2021). *Skin cancer.* http://www.cdc.gov/cancer/skin/

Chiras, D. (2019). *Human biology* (9th ed.). Jones & Bartlett Learning.

Elling, B., Elling, K., & Rothenberg, M. (2004). *Anatomy and physiology.* Jones & Bartlett Learning.

Feldman, R. S. (2018). Epidemiology, clinical manifestations, and diagnosis of psoriasis. In A. O. Ofori (Ed.), *Uptodate.* https://www.uptodate.com/contents/epidemiology-clinical-manifestations-and-diagnosis-of-psoriasis

Hubert, R., & VanMeter, K. (2018). *Gould's pathophysiology for the health professions* (6th ed.). Elsevier.

International Society for Burn Care (ISBI). (2016). ISBI practice guidelines for burn care. *Burns, 42*, 953–1021.

Keller, C. E., Tomecki, J. K., & Alraies, C. M. (2012). Distinguishing cellulitis from its mimics. *Cleveland Clinic Journal of Medicine, 79*(8), 547–552.

Lyons, F., & Ousley, L. (2015). *Dermatology for the advanced practice nurse.* Springer.

National Institutes of Health (NIH). (2020a). *Chediak-Higashi syndrome.* https://ghr.nlm.nih.gov/condition/chediak-higashi-syndrome

National Institutes of Health (NIH). (2020b). *Hermansky-Pudlak syndrome.* https://ghr.nlm.nih.gov/condition/hermansky-pudlak-syndrome

National Institutes of Health (NIH). (2022). *Vitiligo.* https://medlineplus.gov/genetics/condition/vitiligo/#frequency

National Pressure Ulcer Advisory Panel. (2016). *NPUAP pressure injury stages.* https://www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages/

Robinett, H. R., & Chop, C. W. (2015). *Gerontology for the health care professional* (3rd ed.). Jones & Bartlett Learning.

Robinett, H. R., Chop, C. W., & Brossoie, N. (2020). *Gerontology for the health care professional* (4th ed.). Jones & Bartlett Learning.

Sommers, S. M. (2011). Color awareness: A must for patient assessment. *American Nurse Today, 6*(1). https://www.americannursetoday.com/color-awareness-a-must-for-patient-assessment/

Spergel, J. (2010). From atopic dermatitis to asthma: The atopic march. *Annuals of Allergy, Asthma, and Immunology, 105*(2), 107–109.

Weston, L. W., & Howe, W. (2019). Atopic dermatitis (eczema): Pathogenesis, clinical manifestations, and diagnosis. In R. Corona (Ed.), *Uptodate.* https://www.uptodate.com/contents/atopic-dermatitis-eczema-pathogenesis-clinical-manifestations-and-diagnosis