

irritant dermatitis, seborrheic dermatitis, nummular eczema, AD, stasis dermatitis).

Eczema or dermatitis has three primary stages. This condition can manifest in any one of the three stages, or the three stages may coexist. *Acute dermatitis* is characterized by extensive erosions with serous exudate or by intensely pruritic, erythematous papules and vesicles on a background of erythema.

Subacute dermatitis is characterized by erythematous, excoriated (scratched or abraded), scaling papules or plaques that are either grouped or scattered over erythematous skin. Often the scaling is so fine and diffuse the skin acquires a silvery sheen.

Chronic dermatitis is characterized by thickened skin and increased skin marking (called *lichenification*) secondary to rubbing and scratching; excoriated papules, fibrotic papules, and nodules (*prurigo nodularis*); and postinflammatory hyperpigmentation and hypopigmentation.

Incidence and Etiologic Factors

Dermatitis is a common skin disorder in older people. It may be caused by hypoproteinemia, venous insufficiency, allergens, irritants, or underlying malignancy, such as leukemia or lymphoma. Because older people often take multiple medications, dermatitis from drug-drug interaction can occur. The normal aging process with the flattened epidermal-dermal junction and loss of dermis results in skin fragility, which contributes to the development of skin tears and dermatitis.

Stasis Dermatitis

Stasis dermatitis is the development of areas of very dry, thin skin and sometimes shallow ulcers of the lower legs primarily as a result of venous insufficiency. The client commonly has a history of varicose veins or deep vein thrombosis (see also the section on Venous Diseases in Chapter 12).

The process of stasis dermatitis begins with edema of the leg as a result of slowed venous return. As the venous insufficiency continues, the tissue becomes hypoxic from inadequate blood supply. This poorly nourished tissue begins to necrose.

The clinical manifestations include itching, a feeling of heaviness in the legs, brown-stained skin, and open shallow lesions (Fig. 10-6). The lesions are very slow to heal because of a lack of oxygenated blood. Gait training is an important part of compression, the gold standard, in the treatment of stasis dermatitis. Compression hose work well in the recumbent position, but ambulation with the muscular contract-relax cycle pushes the venous return within the compressive field.

Environmental Dermatoses

It is well documented that exposure to various environmental chemicals and to physical stimuli (Box 10-6) is capable of inducing adverse cutaneous responses. Common environmental skin diseases seen in a therapy practice may include irritant and allergic dermatitis, acne lesions, pigmentary changes (hyperpigmentation,



Figure 10-6

Stasis dermatitis secondary to venous insufficiency. Hemosiderin staining (dark pigmentation) indicative of venous insufficiency is evident. This staining is caused by the leakage of hemosiderin (an iron-rich pigment, the product of red cell hemolysis) as a result of blood that cannot return due to valvular incompetence. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission)

Box 10-6

ENVIRONMENTAL FACTORS THAT INDUCE SKIN DISEASE

Mechanical Factors

- Superficial
- Friction
- Pressure
- Vibration
- Cuts
- Internal: shearing (skin is forced in opposite direction)

Physical Factors

- Heat
- Cold
- Humidity
- Water
- Sunlight
- Ultraviolet light
- Ionizing radiation

Chemical Agents

- Primary irritants
- Sensitizers
- Photoirritants
- Photosensitizers

Biologic Agents

- Insect and animal parasites
- Bacteria
- Rickettsiae
- Fungi
- Viruses
- Irritant and sensitizing plants and woods

From Brooks SM, Gochfeld M, Herzstein J, et al: *Environmental medicine*, St Louis, 1995, Mosby.

hypopigmentation, absence of pigment), photosensitivity reactions, scleroderma, infectious disorders, and cutaneous malignancy. Each of these environmentally induced skin conditions is discussed separately in this chapter (see also Chapter 4).

Rosacea

Rosacea* is a chronic facial disorder of middle-aged and older people. Although it is a form of acne, it is differentiated by age, the presence of a large vascular component (erythema, telangiectasis), and usually the absence of comedones.

An acneiform rosacea can occur with papules, pustules, and oily skin. No known cause or factor has been identified to explain the pathogenesis of this disorder. A statistically significant incidence of migraine headaches accompanying rosacea has been reported.

Rosacea has often been linked with gastrointestinal (GI) disturbances, and a causal relationship between *Helicobacter pylori* (a bacterium that causes gastritis) and rosacea was reported in the early 1990s. Many studies linking rosacea to *H. pylori* infection were uncontrolled and were performed in areas where the endemic rates of both *H. pylori* infection and rosacea are high.¹⁴⁹ A study done in Iran in 2003, where prevalence is low, also supported some form of relationship between rosacea and *H. pylori* infection.¹⁶⁷ Continued investigation of this issue is required before a causal relationship can be confirmed.

Clinically, the cheeks, nose, and chin (sometimes the entire face) may have a rosy appearance marked by reddened skin. This benign but obvious condition is most common in people with fair skin who flush easily. Sun, hot weather, and humidity can all trigger flare-ups; the condition is worse in the summer.

The affected person reports burning or stinging with episodes of flushing that come and go, but the condition may worsen over time, causing lasting redness, pimples, telangiectasias, or nasal hypertrophy (rhinophyma). Inflammatory papules are prominent, and there may be pustules. It is not uncommon to have associated ophthalmic disease, including blepharitis and keratitis.

Medical management aimed at the inflammatory papules, pustules, and surrounding erythema may include topical or systemic therapy. Rosacea tends to be a persistent condition that can be controlled with drugs. Chronic rosacea has long been treated by pulsed dye lasers. A newer system, the Intense Pulsed Light (IPL) system, allows deeper and wider area treatments.¹³²

Rosacea associated with *H. pylori*-induced gastritis can be effectively treated by addressing the underlying problem. Although therapists do not treat this condition, clients with other diagnoses often present with this condition also. Clients with this condition should see a physician for adequate medical treatment.

SKIN INFECTIONS

Many bacterial, viral, fungal, and other parasitic skin infections encountered by the therapist are not the primary focus of intervention but rather occur in clients who are hospitalized or being treated for some other condition. Many of these skin disorders are contagious (Table 10-3) and require careful handling by all health care professionals to avoid spreading the infection and becoming contaminated themselves.

Sources of infection differ depending on the disease and mode of transmission (see also Chapter 7). Predisposing factors to skin infections include decreased resistance, dehydrated skin, burns or pressure ulcers, decreased blood flow, contamination from nasal discharge, poor hygiene, and crowded living conditions. Only the most common skin infections encountered in the therapy or rehabilitation setting are discussed further in this section.

Bacterial Infections

Normally the skin harbors a variety of bacterial flora, including the major pathogenic varieties of staphylococci and streptococci. The degree of their pathogenicity depends on the invasiveness and toxigenicity of the specific organisms, the integrity of the skin, the barrier of the host, and the immune and cellular defenses of the host. Organisms usually enter the skin through abrasions or puncture wounds of the hands.

In the therapist's practice, periwound care requires cleaning away from the wound opening to avoid introducing bacteria from the surrounding skin into the wound. Clinical infection develops 3 to 7 days after inoculation. Septicemia can develop if treatment is not provided or if the person is immunocompromised.

People at risk for the development of bacterial infections include children and adults who are immunocompromised, such as occurs with acquired or inherited immunodeficiency; anyone in a debilitated physical condition; those receiving immunosuppressive therapy; and those with a generalized malignancy, such as leukemia or lymphoma.

All these factors emphasize the importance of careful handwashing and cleanliness to prevent spread of infection before and after caring for infected people and their lesions.

Some conditions (e.g., impetigo) are easily spread by self-inoculation; therefore the affected person must be cautioned to avoid touching the involved area. Follicular lesions should not be squeezed because this will not hasten the resolution of the infection and may increase the risk of making the lesion worse or spreading the infection.

Impetigo

Definition and Overview. Impetigo is a superficial skin infection commonly caused by staphylococci or streptococci. It is most commonly found in infants, young children 2 to 5 years of age, and older people. Predisposing factors include close contact in schools, overcrowded

*For further information contact the National Rosacea Society, Barrington, Illinois, (847) 382-8971 or www.rosacea.com.

Table 10-3 Infections of the Skin

Type of Infection	Transmission
Bacterial	
Impetigo contagiosa	Contagious
Pyoderma	Contagious
Folliculitis (pimple, boil)	Contagious; minimal chance of spread
Cellulitis	Contagious*
Viral	
Verrucae (warts)	Contagious; autoinoculable†
Verruca plantaris (plantar wart)	Contagious; autoinoculable
Herpes simplex	
Type 1: cold sore, fever blister	Contagious
Type 2: genital lesion	Contagious
Varicella-zoster virus (herpes zoster; shingles)	Contagious; chickenpox can occur in anyone not previously exposed
Fungal	
Tinea corporis (ringworm)	Person to person Animal to person Inanimate object to person
Tinea capitis (affects scalp)	Person to person Animal to person Person to person Animal to person Person to person
Tinea cruris (jock itch)	Transmission to other people rare despite general opinion to the contrary
Tinea pedis (athlete's foot)	Person to person; sexually transmitted during birth from colonized vagina to neonatal oropharynx
Candidiasis	
Other	
Scabies	Person to person; sexually transmitted during birth from colonized vagina to neonatal oropharynx Inanimate object to person
Lice	Same as scabies

*Technically, cellulitis is contagious, but from a practical point of view the chances of this spreading are very low and would require a susceptible host, for example, an open cut on the therapist's hand coming in contact with blood or pus from the client's open wound.

†Capable of spreading infection from one's own body by scratching.

living quarters, poor skin hygiene, anemia, malnutrition, and minor skin trauma. It can be spread by direct contact, environmental contamination, or an arthropod vector. Impetigo often occurs as a secondary infection in conditions characterized by a cutaneous barrier broken to microbes, such as eczema or herpes zoster excoriations.

Clinical Manifestations. Small macules (flat spots) rapidly develop into vesicles (small blisters) that become pustular (pus-filled). When the vesicle breaks, a thick yellow crust forms from the exudate, causing pain, surrounding erythema, regional adenitis (inflammation of gland), cellulitis (inflammation of tissue), and itching.

Scratching spreads infection, a process called *autoinoculation*. Lesions frequently affect the face, heal slowly, and leave depigmented areas. If the infection is extensive, malaise, fever, and lymphadenopathy may also be present. A less common presentation occurs with few isolated bullae.

MEDICAL MANAGEMENT

Single small lesions can often be managed by soaking them for 10 minutes with drying agents (Burow's solution). Oral antibiotics are regularly used to treat impetigo. Rarely, extensive lesions require systemic antibiotics to reduce the risk of glomerulonephritis and to prevent

this contagious condition from spreading. A skin swab culture may be necessary to determine the contaminating organism.

Cellulitis

Cellulitis is a rapidly spreading acute inflammation with infection of the skin and subcutaneous tissue that spreads widely through tissue spaces. *Streptococcus pyogenes* or *Staphylococcus* is the usual cause of this infection in adults and *Haemophilus influenzae* type b in children, although other pathogens may be responsible. Clients at increased risk for cellulitis include older adults and people with lowered resistance from diabetes, malnutrition, steroid therapy, and the presence of wounds or ulcers.

Other predisposing factors include the presence of edema or other cutaneous inflammation or wounds (e.g., tinea, eczema, burns, trauma). Venous insufficiency or stasis, thrombophlebitis, surgery, substance abuse, immunocompromise (e.g., HIV infection, chemotherapy, autoimmune diseases, chronic use of immunosuppressants), and lymphedema also predispose individuals to this condition. There is a tendency for recurrence, especially at sites of lymphatic obstruction. See also the sections on Streptococcal Cellulitis in Chapter 8 and Lymphangitis in Chapter 13.

Cellulitis of the breast can occur following breast conservation therapy for breast cancer. Although only a minority of women who undergo this therapy will develop breast cellulitis, the therapist may be the first to observe signs of this disorder. A definitive pathogen has not been identified, and recurrent breast cellulitis is possible months to years after the procedure is completed. Local breast findings include the skin changes typical of cellulitis with or without fever.³

Cellulitis usually occurs in the loose tissue beneath the skin, but it may also occur in tissues beneath mucous membranes or around muscle bundles. The skin is erythematous, edematous, tender, and sometimes nodular. It can develop under the skin anywhere but affects the extremities most often.

Erysipelas, a surface cellulitis of the skin, affects the upper dermis and is characterized by patches of skin that are red and painful with sharply defined borders and that feel hot to the touch. Red streaks extending from the patch indicate that the lymph vessels have been infected. Facial cellulitis involves the face, especially the cheek or periorbital or orbital tissues; the neck may also be affected. Pelvic cellulitis involves the tissues surrounding the uterus and is called *parametritis*.

Intravenous (IV) antibiotic infusion is the primary treatment. Good nutrition and hydration are advised to help fight infection, repair tissue, and remove bacteria and their by-products. Extensive cellulitis requires surgical debridement of the necrotic tissue. Lymphangitis may occur if cellulitis is untreated, and gangrene, metastatic abscesses, and sepsis can result.

Viral Infections

Viruses are intracellular parasites that produce their effect by using the intracellular substances of the host cells. Viruses are composed only of DNA or ribonucleic acid (RNA), not both, usually enclosed in a protein shell, and are unable to provide for their own metabolic needs or to reproduce themselves.

After a virus penetrates a cell of the host organism, it sheds the outer shell and disappears within the cell, where the nucleic acid core stimulates the host cell to form more virus material from its own intracellular substance. In a viral infection the epidermal cells react with inflammation and vesiculation (as in herpes zoster) or by proliferating to form growths (warts).

Herpes Zoster

Definition, Incidence, and Risk Factors. Herpes zoster, or shingles, is a local disease brought about by the reactivation of the same virus, *varicella-zoster virus* (VZV), that causes a systemic disease called *varicella* (chickenpox). The initial infection with varicella-zoster virus is common during childhood. Shingles may occur and recur at any age, although peak incidence occurs between ages 50 and 70 years.

An estimated 300,000 episodes of zoster occur annually. Of these episodes, 95% are first occurrences and 5% are recurrences. By age 80 years, almost 15% of persons will have experienced at least one episode of zoster.⁴ The disease is usually brought on by an immunocompro-

mised state, such as occurs with stress, advancing age, underlying malignancy, organ transplantation, or acquired immunodeficiency syndrome (AIDS).

Pathogenesis. Herpes zoster results from reactivation of varicella virus that has been dormant in the cerebral ganglia (extramedullary ganglia of the cranial nerves) or the ganglia of posterior nerve roots from a previous episode of chickenpox.

The immunologic mechanism that controls latency of VZV is not well understood. One explanation is that the virus multiplies as it is reactivated and that it is neutralized by antibodies remaining from the initial infection. If effective antibodies are not present, the virus continues to multiply in the ganglia, destroying the host neuron and spreading down the sensory nerves to the skin. Factors associated with recurrent disease include aging, immunosuppression, intrauterine exposure to VZV, and varicella at a young age (less than 18 months).⁴

Clinical Manifestations. The vesicular eruption of zoster generally occurs unilaterally in the distribution of a specific dermatome supplied by a dorsal root or extramedullary cranial nerve sensory ganglion. Most often, this involves the trunk or the area of the fifth cranial nerve. Two to four days before the eruption the affected person may have some warning (prodromal symptoms) that the virus has become reactivated, especially in repeat incidences.

Early symptoms of pain and tingling along the affected spinal or cranial nerve dermatome are usually accompanied by fever, chills, malaise, and GI disturbances. One to three days later red papules are seen along a dermatome (Fig. 10-7). The lesions most commonly spread unilaterally around the thorax or vertically over the arms or legs.

Herpes papules rapidly develop into vesicles that vary in size and may be filled with clear fluid or pus. The vesicles are confined to the distribution of the infected nerve root and begin to dry 5 days after eruption with gradual, progressive healing over the next 2 to 4 weeks.

Postherpetic neuralgia, or pain in the area of the recurrence that persists after the lesions have resolved, is a distressing complication of zoster with no adequate therapy currently available. Incidence of postherpetic neuralgia increases sharply in people over the age of 60 years and may last as long as 1 year after the episode of zoster. Children are unaffected by postherpetic pain.

In the adult, severe neuralgic pain can occur in peripheral areas innervated by the nerves arising in the inflamed root ganglia. The pain may be constant or intermittent and vary from light burning to a deep visceral sensation. The cause of postherpetic neuralgia is not fully understood. Scarring and degenerative changes involving the nerve trunks, ganglia, and skin may be important factors. The incidence of scarring and hyperpigmentation is much higher in older adults.

Occasionally herpes zoster involves the cranial nerves, especially the trigeminal and geniculate ganglia or the oculomotor nerve. Geniculate zoster may cause vesicle formation in the external auditory canal, ipsilateral facial palsy, hearing loss, dizziness, and loss of taste. Trigeminal ganglion involvement causes eye pain and possibly corneal and scleral damage with loss of vision.

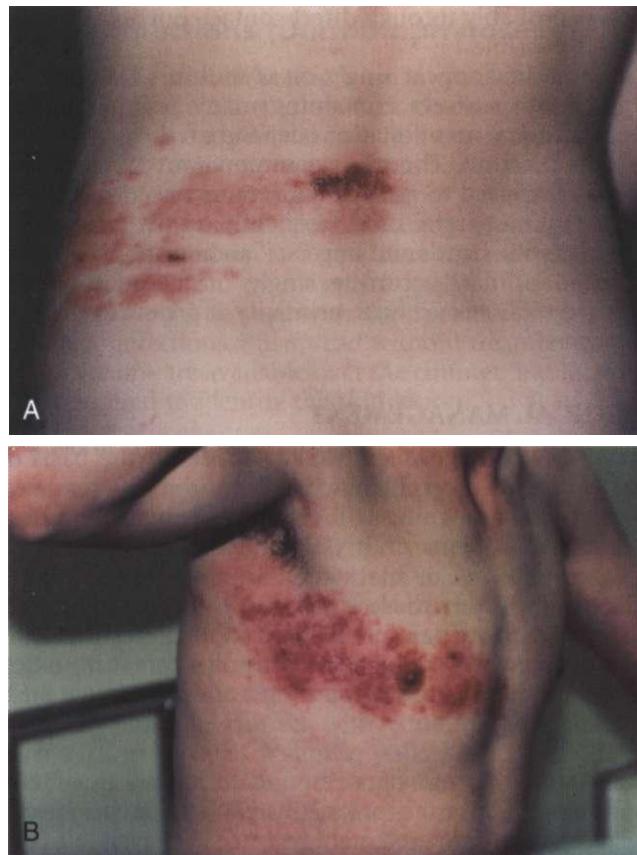


Figure 10-7

Herpes zoster (shingles). **A**, Lesions appear unilaterally along the path of a spinal nerve. **B**, Eruptions involving the T4 dermatome. (**A**, From Callen J, Greer K, Hood H, et al: *Color atlas of dermatology*, Philadelphia, 1993, Saunders. **B**, From Marx J, Hockberger R, Walls R: *Rosen's emergency medicine: concepts and clinical practice*, ed 6, St Louis, 2006, Mosby.)

In rare cases, herpes zoster leads to generalized central nervous system (CNS) infection, muscle atrophy, motor paralysis (usually transient), acute transverse myelitis, and ascending myelitis. More often, generalized infection causes acute retention of urine and unilateral paralysis of the diaphragm.

MEDICAL MANAGEMENT

DIAGNOSIS. Diagnosis is usually based on clinical examination and recognition of the skin lesions with accompanying systemic signs of infection. Laboratory diagnosis may include culture and histologic examination of a skin biopsy specimen.

Differentiation of herpes zoster from localized herpes simplex requires staining of antibodies from vesicular fluid and identification using a fluorescent monoclonal antibody test that is very sensitive and specific. For those individuals who are uncertain whether they have had childhood varicella, a variety of serologic tests for the varicella antibody are available.

TREATMENT. There is no curative agent for shingles, but supportive treatment to relieve itching and neuralgic pain

is provided. The use of systemic corticosteroids within the first week of eruption may abort the attack and appears to reduce both the acute symptoms and the risk of postherpetic neuralgia in older persons.

Acyclovir (Zovirax) seems to stop the progression of the rash and prevents visceral complications. Famciclovir, an oral drug, is comparable to acyclovir in effectiveness but has a longer duration of action and requires less frequent dosing. Valacyclovir (Valtrex) is dosed only three times daily and has been shown in studies to decrease postherpetic neuralgia and shorten duration of symptoms.

Hospitalized clients with varicella-zoster should be placed in isolation rooms, and personnel entering the room should wear gowns, gloves, and masks. Eye involvement in zoster requires ophthalmologic evaluation and treatment.

A live varicella virus vaccine (Varivax) is available for use in persons 12 months of age or older who have not had varicella. The Advisory Committee on Immunization Practices (ACIP) also recommends the vaccine for use in susceptible persons following exposure to varicella. Data from the United States and Japan collected in a variety of settings indicate that varicella vaccine is effective in preventing illness or modifying the severity of illness if used within 3 days, and possibly up to 5 days, of exposure.⁴

The vaccine provides long-lasting (but not lifelong) immunity with an 80% to 85% efficacy. Vaccine breakthrough cases are common but mild. Persons present with fewer lesions (usually less than 50) and lack systemic symptoms (such as fever).

The first shingles vaccine (Zostavax; zoster vaccine live) has been approved for adults age 60 and older. Zostavax has been shown to reduce the incidence of shingles by 51% and the incidence of postherpetic neuralgia by 67% in adults age 60 and older. Among people who get shingles despite being vaccinated, it can reduce its severity. Approval of this drug for use in adults ages 50 to 59 is pending more evidence to support its safety and effectiveness in this age group.¹¹

PROGNOSIS. Overall prognosis is good unless the infection spreads to the brain (rare). Most people recover completely, with the possible exception of scarring and, with corneal damage, visual impairment. Occasionally, intractable pain associated with neuralgia may persist for months or years. Those persons who develop postherpetic neuralgia may require further medical intervention.

SPECIAL IMPLICATIONS FOR THE THERAPIST

10-5

Herpes Zoster (see the discussion on herpesviruses in Chapter 8; also see the discussion on herpes zoster in Chapter 39)

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

Continued.

Adults with shingles are infectious to persons who have not had chickenpox, and the person with shingles can develop shingles more than one time. For this reason, therapists who have never had chickenpox should receive the vaccination; complications and morbidity associated with adult onset of varicella warrant this precaution.

Any female therapist who is pregnant or planning a pregnancy should be tested for immune status if unsure about her previous history of varicella. This is especially important because transmissibility of the virus occurs 2 to 3 days before symptoms develop; immunocompromised clients with shingles are probably contagious during the entire period new lesions are appearing until all lesions are crusted over.

This means anyone receiving intervention by a therapist may be an asymptomatic host during the period of communicability; exposure to self and further transmission to others can occur without the therapist's awareness.

Susceptible health care workers with significant exposure to varicella should be relieved from direct client contact from day 10 to day 21 after exposure. If workers develop chickenpox, varicella lesions must be crusted before they return to direct client contact.

The Centers for Disease Control and Prevention (CDC) has set up guidelines for the care of all clients regarding precautions for the transmission of infectious skin diseases (see Table 10-3; see also Table 8-5). These Standard Precautions (see Appendix A) should be used with all clients regardless of their disease status.

All skin lesions are considered potentially infectious and should be handled as such. The careful use of these precautionary measures severely limits the transmission of any disease. In addition, each hospital has isolation precautions organized according to categories of disease to prevent the spread of infectious disease to others. Every health care professional must be familiar with these procedures and follow them carefully. See also the section on Isolation Procedures in Chapter 8 and Table 8-3.

Neither heat nor ultrasound should be used on a person with shingles because these modalities can increase the severity of the person's symptoms. For the person with severe herpetic pain, relaxation techniques may be useful. In the case of unresolved postherpetic neuralgia, the individual may benefit from a program of chronic pain management.

Warts (Verrucae)

Warts are common, benign viral infections of the skin and adjacent mucous membranes caused by human papillomaviruses (HPVs). There are more than 50 different varieties of these viruses depending on their location on the skin. The incidence of warts is highest in children and young adults, but warts can occur at any age. Transmis-

sion is probably through direct contact, but autoinoculation is possible.

Warts may appear singly or as multiple lesions with thick white surfaces containing many pointed projections. Clinical manifestations depend on the type of wart and its location. The most common wart (*verruca vulgaris*) is referred to as such and appears as a rough, elevated, round surface most frequently on the extremities, especially the hands and fingers. Plantar warts are slightly elevated or flat, occurring singly or in large clusters referred to as *mosaic warts*, primarily at pressure points of the feet.

MEDICAL MANAGEMENT

DIAGNOSIS. Diagnosis is usually made on the basis of visual examination. Plantar warts can be differentiated from corns and calluses by certain distinguishing features. Plantar warts obliterate natural lines of the skin, may contain red or black capillary dots that are easily discernible if the surface of the wart is shaved down with a scalpel, and are painful on application of pressure. Both plantar warts and corns have a soft, pulpy core surrounded by a thick callous ring; plantar warts and calluses are flush with the skin surface.

TREATMENT. Some warts respond to simple treatment, and some disappear spontaneously. Warts can be chronic or recurrent. Many treatment regimens are available. The specific choice of treatment method is influenced by the location of the wart or warts, size and number of warts, presence of secondary infection, amount of tenderness present on palpation, age and gender of the client, history of previous treatment, and individual compliance with treatment. Over-the-counter salicylic acid preparations (e.g., DuoFilm, Wart-Off, Clear Away, or other wart-removing compounds) applied topically may be used to induce peeling of the skin.

Cryotherapy is performed with either liquid nitrogen or solid carbon dioxide. This procedure is widely used as the cosmetically preferred treatment choice, but it is painful. The procedure causes epidermal necrosis; the area dries and peels off together with the wart.

Acids in liquid form or as a paste (salicylic acid, lactic acid) can be painted on warts daily, removed after 24 hours, and reapplied. This treatment choice is not recommended for areas where perspiration is heavy, for areas likely to get wet, or for exposed body parts where patches are cosmetically undesirable. Acid therapy requires a commitment from the client or family to perform it on a daily basis.

Electrodesiccation and **curettage** of warts are widely used for common warts and occasionally for plantar warts. High-frequency electric current destroys the wart and is followed by surgical removal of dead tissue at the base with application of an antibiotic ointment and bandage for 48 hours. Atrophic scarring may occur, and the recurrence rate is 20% to 40%.

The use of mechanical (nonthermal) ultrasound has been advocated by some in the treatment of plantar warts, but this has not been widely accepted by the medical community.

Fungal Infections (Dermatophytoses)

Fungal infections such as ringworm are caused by a group of fungi that invade the stratum corneum, hair, and nails.⁵⁷ These are superficial infections by fungi that live on, not in, the skin and are confined to the dead keratin layers, unable to survive in the deeper layers. Since the keratin is being shed (desquamated) constantly, the fungus must multiply at a rate that equals the rate of keratin production to maintain itself; otherwise the organisms would be shed with the discarded skin cells.

Fungal infections will spread without treatment; antifungal creams are available over the counter, but diagnosis is required to identify the skin lesion.

Ringworm (Tinea Corporis)

Dermatophytoses, or fungal infections of the hair, skin, or nails, are designated by the Latin word *tinea*, with further designation related to the affected area of the body (see Table 10-3). Tinea corporis, or ringworm, has no association with worms but rather is marked by the formation of ring-shaped pigmented patches covered with vesicles or scales that often become itchy (Fig. 10-8). Transmission can occur directly through contact with infected lesions or indirectly through contact with contaminated objects, such as shoes, towels, or shower stalls.

Diagnosis can be made through laboratory examination of the affected skin. Treatment for any type of ringworm requires maintaining clean, dry skin and applying antifungal powder or topical agent as prescribed.

Treatment with the drug griseofulvin may take weeks to months to complete and should be continued throughout the prescribed dosage schedule even if symptoms subside. Possible side effects of this agent include headache, GI upset, fatigue, insomnia, and photosensitivity. Prolonged use of this drug requires monitoring of liver function. Oral medication is reserved for clients with more involved cases.

Occasionally an obese client with tinea corporis is referred to therapy for wound care secondary to skin breakdown. Advanced wound dressings may be applied to areas of moist, denuded skin to optimize healing.

Athlete's Foot (Tinea Pedis)

Tinea pedis, or athlete's foot, causes erythema, skin peeling, and pruritus between the toes that may spread from the interdigital spaces to the sole. Severe infection may result in inflammation, with severe itching and pain on walking. Some individuals develop a strong foot odor as well.

Clean, dry socks and adequate footwear (well-ventilated, properly fitting) are important. After washing the feet and drying thoroughly between the toes, antifungal cream or powder (the latter to absorb perspiration and prevent excoriation) can be applied.

A history of antibiotic use, yeast infections (*candidiasis*, including intestinal yeast), and other risk factors for *candidiasis* may contribute to athlete's foot. If symptomatic treatment including topical preparations does not eradicate the problem, treatment of intestinal yeast may be required.

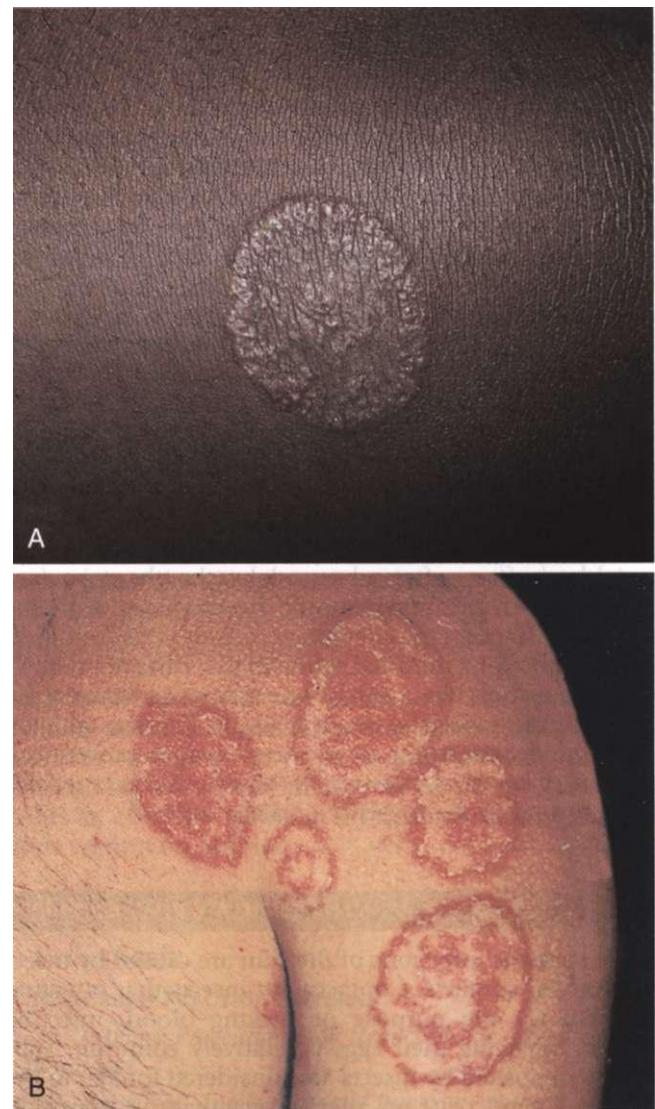


Figure 10-8

Tinea corporis (ringworm). **A**, Scales forming circular lesions with clear centers are characteristic of tinea corporis (ringworm). **B**, Most adults or children present with multiple lesions that are hyperpigmented in Caucasians and depigmented in dark-skinned people. The lesions occur most often on the face, chest, abdomen, and back of arms. **A**, From Zitelli BJ, Davis HW: *Atlas of pediatric physical diagnosis*, St Louis, 2002, Mosby. **B**, From Habif T: *Clinical dermatology*, ed 4, St Louis, 2004, Elsevier.)

SPECIAL IMPLICATIONS FOR THE THERAPIST

10-6

Fungal Infections

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

The infectious nature of fungal infections requires specific hygienic measures common to all infectious conditions. Affected persons should not share hair care products (e.g., combs, brushes, headgear), clothes, or

Continued.

other articles that have been in proximity to the infected area. Affected persons must use their own towels and linens.

Since fungal infections are superficial (living on the skin), the therapist is advised to avoid shaving body hair for the application of electrodes or other adhesives. Cutting the hair closely will avoid providing microscopic nicks that can give entrance for the transmission of surface pathogens.

Ringworm

Since ringworm can be acquired by animal-to-human transmission (see Table 10-3), all household pets must be examined for the presence of ringworm as well. Other sources of infection include seats with headrests (e.g., theater seats, seats on public transportation, or other public seats that can be shared).

Athlete's Foot

Athlete's foot, often observed by the therapist (see previous description), should be discussed with the client. Although the client may consider this condition a nuisance or a minimal problem that does not require medical attention, it can be an entry point for bacterial infections, especially in older adults. Keeping athlete's foot under control is an important way to prevent cellulitis, a bacterial infection in the legs, and is especially important in the presence of diabetes.¹⁹

monly in the interdigital web spaces, flexor aspects of the wrist (volar surface), axillae, waistline, nipples in females, genitalia in males, and the umbilicus. Intense scratching can lead to severe excoriation and secondary bacterial infection. Itching can become generalized secondary to sensitization.

MEDICAL MANAGEMENT

DIAGNOSIS AND TREATMENT. The mite can be excavated from one end of a burrow with a needle or a scalpel blade and examined under a microscope. In longstanding cases, a mite may not be found. At that point treatment is based on a presumptive diagnosis.

Treatment has traditionally been with a scabicide, usually a lotion or cream containing permethrin or lindane, applied to the entire body from the neck down. Single oral-dose therapy of ivermectin (*Stromectol*) is an effective treatment for this infestation. Permethrin is generally the treatment of choice for head lice and scabies because of its residual effect and because toxicity and absorption are minimal. Ivermectin may be reserved for cases where permethrin fails¹⁹; further research is advocated regarding the safety and effectiveness of ivermectin.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-7

Scabies

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

If a hospitalized person has scabies, prevent transmission to self and others by practicing good hand-washing technique and by wearing gloves when touching the affected person and a gown when in close contact. Observe wound and skin precautions for 24 hours after treatment of scabies. Gas-autoclave blood pressure cuffs or other equipment used with the affected person before using them on other people. All linens and toweling used must be isolated after use until the person is noninfectious. If the person is treated anywhere outside the hospital room (e.g., on a plinth or treatment mat), the area must be thoroughly disinfected after each session.

In using a scabicide, the individual must understand that NO area can be missed. After 24 hours, the affected person should bathe. All bed linens and clothes must be laundered in hot water or dry-cleaned. Other household members and those in close contact with the affected person should be treated. A second application of the cream or lotion may need to be applied 7 days later. The same procedure is followed.

Itching may persist for 1 to 2 weeks after treatment until the stratum corneum is replaced, but lesions on the forearms or legs can be occluded with Unna's boots to eliminate the scratch-itch cycle (Fig. 10-9). Widespread bacterial infections require additional treatment with systemic antibiotics.

Other Parasitic Infections

Some parasitic infections of the skin are caused by insect and animal contacts. Contact with insects that puncture the skin for the purpose of sucking blood, injecting venom, or laying their eggs is relatively common. Substances deposited by insects are considered foreign to the host and may create an allergic sensitivity in that individual and produce pruritus, urticaria, or systemic reactions of a greater or lesser degree, depending on the individual's sensitivity.

Scabies

Definition. Scabies (mites) is a highly contagious skin eruption caused by a mite, *Sarcoptes scabiei*. It is a common public health problem with an estimated prevalence of 300 million cases worldwide. The female mite burrows into the skin and deposits eggs that hatch into larvae in a few days.

Scabies is easily transmitted by skin-to-skin contact or by contact with contaminated objects, such as linens or shared inanimate objects. Infections with human T-cell leukemia/lymphoma virus 1 (HTLV-1) and HIV are associated with scabies.¹⁹ Mites can spread rapidly between members of the same household, nursing home, or institution, but the inflammatory response and itching do not occur until approximately 30 to 60 days after initial contact.

Clinical Manifestations. The symptoms include intense pruritus (worse at night), usually excoriated skin, and the burrow, which is a linear ridge with a vesicle at one end. The mite is usually found in the burrow, com-

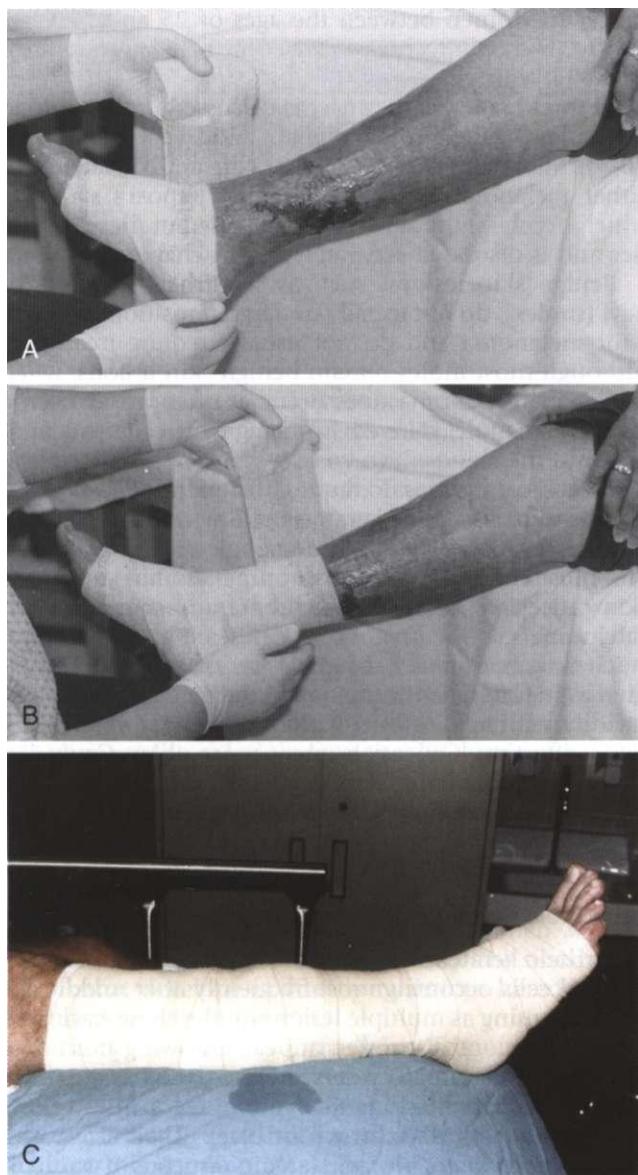


Figure 10-9

A, Unna's boots. Although most often used in cases of venous insufficiency (the client must be ambulatory to enlist the calf muscle pump), the application of Unna's boot to the forearms or legs can be used with a variety of skin lesions to eliminate the scratch-itch cycle. Unna's boot is a dressing made of gauze impregnated with gelatin, zinc oxide, calamine, and glycerin. **B**, The bandage is applied in a spiral fashion and allowed to dry, forming a semi-rigid dressing. This dressing can be allowed to stay intact for 7 days. **C**, A four-layer gradient compression wrap (Profore) provides sustained compression for up to 1 week, for both ambulatory and nonambulatory clients. (**A** and **B**, Courtesy Pam Unger, PT, Community General Hospital, Center for Wound Management, Reading, PA, 1995. **C**, Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

Pediculosis (Lousiness)

Pediculosis is an infestation by *Pediculus humanus*, a very common parasite infecting the head, body, and genital area. Transmission is from one person to another, usually on shared personal items, such as combs, lockers, clothes, or furniture. Lice are not carried or transmitted by pets.

School-age children are easily infected as are people who live in overcrowded surroundings and those older adults who have poor personal hygiene, depend on others for care, or live in a nursing home.

Pediculus humanus var. *capitis*, the head louse, is transmitted through personal contact or through shared hair-brushes or shared head wear. Severe itching accompanied by secondary eczematous changes develops, and small greyish or white nits (eggs) are usually seen attached to the base of the hair shafts.

Pediculus corporis, the body or clothes louse, produces intense itching, which in turn results in severe excoriations from scratching and possible secondary bacterial infections. The lice or nits are generally found in the seams of the affected individual's clothing.

Pediculus pubis (*Phthirus pubis*), the pubic or crab louse, is usually transmitted by sexual contact but can be transferred on clothing or towels. The lice and nits are usually found at the base of the pubic hairs. Sometimes dark brown particles (louse excreta) may be seen on underclothes.

MEDICAL MANAGEMENT

Traditional treatment has been with the appropriate cleaning solution (e.g., shampoo or soap containing permethrin) specific to the type of louse present. As with scabies, single oral-dose therapy of ivermectin (Stromectol) is an effective treatment for this infestation (see previous section on Scabies).

SPECIAL IMPLICATIONS FOR THE THERAPIST

10-8

Pediculosis

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

The therapist must always be conscious of the personal hygiene of all clients. Anyone can get pediculosis regardless of age, socioeconomic status, or status of personal cleanliness. Wear gloves while carefully inspecting the head of any adult or child who scratches excessively. Look for bite marks, redness, and nits or movement that indicates a louse. If exposure to lice occurs, treatment for the client as well as the therapist may be required depending on the exposure level. Use the same precautions outlined earlier in the section on Scabies.

All combs and brushes should be soaked in the cleaning agent, and clothing must be boiled, dry-cleaned, or washed in a machine (hot cycle). The seams of the clothing should be pressed with a hot iron. Carpets, car seats, pillows, stuffed animals, rugs, mattresses, upholstered furniture, and similar objects that come in contact with the affected person must be vacuumed or cleaned thoroughly with hot water and the cleaning agent. Any item that cannot be cleaned can be stored in a sealed plastic bag for 2 to 3 weeks until all lice have been killed.

SKIN CANCER

The American Cancer Society (ACS) estimates that skin cancers are the most prevalent form of cancer, eventually affecting nearly all Caucasian people older than 65 years of age. Skin cancer is the most rapidly increasing cancer in the United States, with over 1 million new cases of nonmelanoma (i.e., basal and squamous cell) skin cancer diagnosed annually in the United States. There is no evidence that this epidemic has peaked.^{70,141}

Solar radiation (exposure to midrange-wavelength ultraviolet B [UVB] radiation) causes most skin cancers, and protection from the sun during the first two decades of life significantly reduces the risk of skin cancer (Box 10-7). The melanoma rate is rising most rapidly in persons younger than 40 years of age and is now the most common

cancer in women between the ages of 25 and 29 years and second only to breast cancer in the age group from 30 to 34 years.

In this chapter, skin cancer is discussed in three broad categories: benign, premalignant, and malignant (Box 10-8). Malignant lesions of the skin are considered as either melanoma or nonmelanoma. Kaposi's sarcoma, which occurs in the skin, is not included in these categories and is discussed separately in this chapter.

Benign skin lesions, such as seborrheic keratosis or nevi (moles), do not usually undergo transition to malignant melanoma and do not usually require treatment. Although most moles remain benign skin lesions, when malignant melanoma does occur, it often arises from a preexisting mole, derived from pigment cells (melanocytes) of the skin.

Keratoacanthomas do require treatment. Precancerous lesions, such as actinic keratosis or Bowen's disease, may progress to malignancy and must be carefully evaluated. The most common types of (nonmelanoma) malignant skin cancer are basal cell carcinoma and squamous cell carcinoma.

These carcinomas occur twice as often in Caucasian men as in Caucasian women, and the incidence increases steadily with age. A third type of malignant skin cancer (also affecting Caucasian men more than Caucasian women), malignant melanoma, is the most serious skin cancer, resulting in early metastasis and possible death.

Benign Tumors

Seborrheic Keratosis

Seborrheic keratosis is a hereditary benign proliferation of basal cells occurring most frequently after middle age and presenting as multiple lesions on the chest, back, and face. The lesions also often appear following hormonal therapy or inflammatory dermatoses. The areas are waxy, smooth, or raised lesions that vary in color from yellow to flesh tones to dark brown or black. Their size varies from barely palpable to large verrucous (wartlike) plaques. These tumors are usually left untreated unless they itch or cause pain. Otherwise, cryotherapy with liquid nitrogen is an effective treatment.

Nevi (Moles)

Nevi are pigmented or nonpigmented lesions that form from aggregations of melanocytes beginning early in life. Most moles are brown, black, or flesh-colored and may appear on any part of the skin. They vary in size and thickness, occurring in groups or singly.

Nevi seldom undergo transition to malignant melanoma, but as previously mentioned, when malignant melanoma does occur, it often arises from a preexisting mole; the chances of cancerous transformation are increased as a result of constant irritation. Any change in size, color, or texture of a mole; bleeding; or any excessive itching should be reported to a physician.

Precancerous Conditions

There are two common premalignant skin lesions: actinic keratosis and Bowen's disease.

Box 10-7

IMPORTANT TRENDS IN SKIN CANCER

Incidence

- More than 1 million cases per year with the majority being the highly curable basal or squamous cell cancers; not as common is the most serious skin cancer (malignant melanoma) with an estimated 62,000 new cases per year.

Mortality

- Total estimated deaths in 2008 were 11,200; 8420 from malignant melanoma and 2780 from other skin cancers.

Risk Factors

- Excessive exposure to ultraviolet radiation from the sun; fair complexion; occupational exposure to coal tar, pitch, creosote, arsenic compounds, and radium; chronic immunosuppression; skin cancer is negligible in blacks because of heavy skin pigmentation.

Warning Signals

- Any unusual skin condition, especially a change in the size or color of a mole or other darkly pigmented growth or spot.

Prevention and Early Detection

- Avoidance of sun when ultraviolet light is strongest (e.g., between 10 AM and 4 PM); use sunscreen preparations; basal and squamous cell skin cancers often form a pale, waxlike, pearly nodule or a red, sharply outlined patch; melanomas are usually dark brown or black; they start as small molelike growths that increase in size, change color, become ulcerated, and bleed easily from a slight injury.

Treatment

- There are four methods of treatment: surgery, electrodesiccation (tissue destruction by heat), radiation therapy, and cryosurgery (tissue destruction by freezing); for malignant melanomas, wide and often deep excisions and removal of nearby lymph nodes are required.

Survival

- For basal cell and squamous cell cancers, cure is virtually ensured with early detection and treatment; malignant melanoma, however, metastasizes quickly; this accounts for a lower 5-year survival rate for white people with this disease.

Box 10-8**TYPES OF SKIN CANCER**

Benign	Premalignant	Nonmelanoma	Melanoma
Seborrheic keratosis Nevi	Actinic keratosis Bowen's disease	Basal cell carcinoma Squamous cell carcinoma	Superficial spreading melanoma Nodular melanoma Lentigo maligna melanoma Acral lentiginous melanoma

Actinic Keratosis

Actinic keratosis (also known as *solar keratosis*) is a skin disease resulting from many years of exposure to the sun's UVA rays. The damage caused by overexposure to sunlight results in abnormal cell growth, causing a well-defined, crusty, or sandpaper-like patch or bump that appears on chronically sun-exposed areas of the body (e.g., face, ears, lower lip, bald scalp, dorsa of hands and forearms).

The base may be light or dark, tan, pink, red, or a combination of these, or it may be the same color as the skin. The scale or crust is horny, dry, and rough; it is often recognized by touch rather than sight. Occasionally it itches or produces a pricking or tender sensation. The skin abnormality or lesion develops slowly to reach a size that is most often 3 to 6 mm. It may disappear only to reappear later. Often there are several actinic keratoses present at one time.

Actinic keratosis affects nearly 100% of the older Caucasian population. It is most common in fair-complexioned, blue- or green-eyed, middle-aged men with a history of sun exposure (solar radiation). The number of lesions that develops is directly related to heredity and lifetime exposure to the sun.

There is a known risk of malignant degeneration and subsequent metastatic potential in neglected lesions. Almost half of the estimated 5 million current cases of skin cancer began as actinic keratosis lesions. It is important that this condition be diagnosed properly, because it is often difficult to distinguish a large or hypertrophic actinic keratosis from a squamous cell carcinoma. A biopsy may be indicated.

Not all keratoses need to be removed. The decision about treatment protocol is based on the nature of the lesion, the number of lesions, and the age and health of the affected person. Treatment may be with 5-fluorouracil (5-FU, Efudex), a topical antimetabolite that inhibits cell division, or masoprolac cream; cryosurgery using liquid nitrogen; or curettage by electrodesiccation (superficial tissue destruction through the use of bursts of electrical current).

These clients should be advised to avoid sun exposure and use a high-potency (sun protection factor [SPF] 15) sunscreen 30 to 60 minutes before going outside. SPF 30 is recommended for people of fair complexion. Sunscreens are not recommended for infants under 6 months of age. Infants should be kept out of the sun or shaded from it. Fabric with a tight weave, such as cotton, is suggested.

Some conditions call for more invasive treatments, such as laser resurfacing (outer layers of the skin are

vaporized) or chemical peels (outer layers are burned off via chemical solution). In June 2000 the U.S. Food and Drug Administration (FDA) approved the use of photodynamic treatment of actinic keratosis of the face and scalp using a topical application (Levulan Kerastick) followed by exposure to a nonlaser blue light source. This is a painful and involved treatment requiring application of Levulan 16 hours prior to exposure to the light source.

Bowen's Disease

Bowen's disease can occur anywhere on the skin (exposed and unexposed areas) or mucous membranes (especially the glans penis in uncircumcised males). It presents as a persistent, brown to reddish brown, scaly plaque with well-defined margins. Often the person has a history of arsenic exposure in youth. Multiple lesions have been associated with an increased number of internal malignancies and therefore require close follow-up. Treatment is with surgical excision and topical 5-FU.

Malignant Neoplasms**Basal Cell Carcinoma**

Definition and Overview. Basal cell carcinoma is a slow-growing surface epithelial skin tumor originating from undifferentiated basal cells contained in the epidermis. This type of carcinoma rarely metastasizes beyond the skin and does not invade blood or lymph vessels but can cause significant local destruction.

Until recently, this tumor rarely appeared before age 40 years and was more prevalent in blond, fair-skinned males. In the age group under 30 years, more women than men develop skin cancer associated with the use of indoor tanning booths with concentrated doses of UV radiation. It is the most common malignant tumor affecting Caucasians, with a reported 100,000 new cases each year; African Americans and Asians are rarely affected.

Etiologic and Risk Factors. Prolonged sun exposure and intermittent sun exposure are the most common causes of basal cell carcinoma; but immunosuppression (e.g., organ transplant recipients, individuals who are HIV positive), genetic predisposition, and rarely, the site of vaccinations are other possible causes. Immunosuppressed organ transplant recipients are more likely to develop squamous cell carcinoma, whereas HIV-infected adults are far more likely to have basal cell carcinoma.

These lesions are seen most frequently in geographic regions with intense sunlight in people with outdoor occupations and on those areas most exposed, the face

and neck. Dark-skinned people are rarely affected because their basal cells contain the pigment melanin, a protective factor against sun exposure. Anyone who has had one basal cell carcinoma is at increased risk of developing others. Recurrences of previously treated lesions are possible, usually within the first 2 years after initial treatment.

Pathogenesis. The pathogenesis of basal cell tumors remains uncertain, and basal cell carcinoma is considered biologically unusual. It is a stable growth characterized by monotonous structure (the same in small as well as large tumors), the absence of progression to metastasis, and a small amount of chromosomal damage (as compared with moderate chromosomal damage associated with squamous cell carcinoma).

To the dismay of investigators seeking to design experiments, basal cell carcinomas are very seldom seen in animals and not found in laboratory rodents at all. Whereas squamous cell carcinoma is often preceded by a precursor (actinic keratosis), there are no known precursors to basal cell carcinoma. This fact suggests that basal cell carcinoma tumors need only a few mutations to induce malignant transformation.¹¹⁴

One theory suggests that these tumors arise as a result of a defect that prevents the cells from being shed by the normal keratinization process. The process of epidermal cell maturation is called *keratinization* because the cells synthesize a fibrous protein called *keratin*. Basal cells that lack the normal keratin proteins form basal cell tumors. Another hypothesis is that undifferentiated basal cells become carcinomatous instead of differentiating into sweat glands, sebum, and hair. See also the section on Squamous Cell Carcinoma: Pathogenesis.

Clinical Manifestations. Basal cell carcinoma (Fig. 10-10) typically has a pearly or ivory appearance, has rolled edges, and is slightly elevated above the skin surface, with small blood vessels on the surface (telangiectasia) (see Fig. 10-3).

The nodule is usually painless and slowly increases in size and may ulcerate centrally. More than 65% of basal cell carcinomas are found on the head and neck. Other locations are the trunk, especially the upper back and chest.

They also can appear similar to Bowen's disease, chronic venous ulcer (Fig. 10-11), or squamous cell carcinoma in a flatter, scaling lesion, usually on the trunk or extremities.

MEDICAL MANAGEMENT

DIAGNOSIS AND TREATMENT. Diagnosis by clinical examination of appearance must be confirmed via biopsy and histologic study. Treatment depends on the size, location, and depth of the lesion and may include curettage and electrodesiccation, chemotherapy, surgical excision, and irradiation.

Mohs' micrographic surgery is the gold standard treatment in which the specimen is excised, frozen-sectioned, and examined for positive margins while the client waits, thus ensuring clean margins before complex repairs are performed. Irradiation is used if the tumor location requires it and in older or debilitated people who cannot tolerate surgery.



Figure 10-10

Basal cell carcinoma. **A**, Skin cancer in the form of basal cell carcinoma can appear as a shiny, pearly, or translucent pink, red, or white bump. There may be a rolled border with an indented center. **B**, This type of skin cancer may also present as a red patch; a crusty, open sore that will not heal; or a scarlike area. (**A**, From Lookingbill D, Marks J: *Principles of dermatology*, ed 3, Philadelphia, 2000, Saunders. **B**, From Townsend C, Beauchamp RD, Evers BM, Mattox K: *Sabiston textbook of surgery*, ed 17, Philadelphia, 2004, Saunders.)



Figure 10-11

Chronic venous ulcer. Basal cell carcinoma can also mimic a chronic venous ulcer potentially causing a delay in diagnosis. Biopsy is required to make the definitive medical diagnosis. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

Radiation therapy is generally contraindicated in persons less than 50 years of age because of the risk of recurrence and the development of secondary radiation-induced tumors of the skin. Radiotherapy can be followed by chronic skin ulcers that are difficult to close, much less heal. Some radiation-induced ulcers open on and off for years, and some just develop 10 to 20 years after the radiation therapy.¹³⁶

If the tumor is identified and treated early, local excision or even nonexcisional destruction is usually curative. Skin grafting may be required in cases where large areas of tissue have been removed. A new experimental treatment called *photodynamic therapy (PDT)* is being investigated in the treatment of superficial nonmelanoma skin cancers. This technique requires the administration of a drug that induces photosensitivity, followed in 48 to 72 hours by exposure to light that helps outline the tumor. The tumor cells concentrate this drug so as to allow selective destruction of the cancer cells when exposed to a laser light of 630 nm.^{75,150}

Clinical trials are under way investigating the use of chemopreventive agents, such as vitamin A analogues called *retinoids*. These topical agents may potentially complement sunscreens and result in decreased incidence, morbidity, and mortality of skin cancer.¹⁴⁵

Tretinoin has proven effective in preventing UV-induced lesions and can be considered for high-risk basal or squamous cell carcinoma patients, as well as those with actinic keratosis, realizing that it is off-label use.¹³¹ Topical imiquimod was approved by the FDA in 2004 for individuals who have superficial basal cell carcinoma.⁴⁹

Cytokine therapy, including interferon and interleukin, is a type of systemic immunotherapy used to treat skin cancer. Both cytokines mentioned here have been FDA approved for metastatic melanoma.¹²⁸

PROGNOSIS. If left untreated, basal cell lesions slowly invade surrounding tissues over months and years, destroying local tissues such as bone and cartilage, especially around the eyes, ears, and nose.

Squamous Cell Carcinoma

Definition and Overview. Squamous cell carcinoma is the second most common skin cancer in whites, usually arising in sun-damaged skin, such as the rim of the ear, the face, the lips and mouth, and the dorsa of the hands (Fig. 10-12). It is a tumor of the epidermal keratinocytes and rarely occurs in dark-skinned people.

Squamous cell tumors may be one of two types: *in situ* (confined to the site of origin) and invasive (infiltrate surrounding tissue). *In situ* squamous cell carcinoma is usually confined to the epidermis but may extend into the dermis. Common premalignant skin lesions associated with *in situ* carcinomas are actinic keratosis and Bowen's disease (see earlier section).

Invasive squamous cell carcinoma can arise from premalignant lesions of the skin, including sun-damaged skin, actinic dermatitis, scars, whitish discolored areas (leukoplakia), radiation-induced keratosis, tar and oil keratosis, and chronic ulcers and sinuses.

Incidence. As with basal cell carcinoma, fair-skinned people have a higher incidence of squamous cell carci-



Figure 10-12

Squamous cell carcinoma can take the form of a persistent scaly, red patch that sometimes crusts or bleeds or an open sore that does not heal. This type of skin cancer may also present as a raised or wartlike growth that may bleed. (From Goldman L: Cecil textbook of medicine, ed 22, Philadelphia, 2004, Saunders.)

noma. This particular type of tumor has a peak incidence at 60 years of age and affects men more than women.

Etiologic and Risk Factors. Predisposing factors associated with squamous cell carcinoma include cumulative overexposure to UV radiation (e.g., outdoor employment or residence in a warm, sunny climate), burns, presence of premalignant lesions such as actinic keratosis or Bowen's disease, radiation therapy, ingestion of herbicides containing arsenic, chronic skin irritation and inflammation, exposure to local carcinogens (tar, oil), and hereditary disease such as xeroderma pigmentosum and albinism.

Organ transplant recipients who are chronically immunosuppressed are at risk for the development of recurring squamous cell carcinoma. Rarely, squamous cell carcinoma may develop on the site of a smallpox vaccination, psoriasis, or chronic discoid lupus erythematosus.

Pathogenesis. UV radiation continues to be one of the most important causes of skin cancer, because the sun's UV rays damage the DNA inside the nuclei of the epidermal cells, triggering enzymes to repair the damage. We differ in our ability to produce repair enzymes, which may explain our differences in tanning ability as well as susceptibility to skin cancer. Not all DNA lesions are properly repaired, increasing the risk of skin cancer.

Newer studies show that when DNA damage occurs, a cell surface molecule (Fas ligand; FasL) belonging to the tumor necrosis factor family binds to its receptor Fas and attaches to the damaged cells, inducing them to die by apoptosis (i.e., programmed cell death).

These suicidal cells known as *keratinocytes* take themselves out of action, and the less damaged ones repair themselves. After many years of cumulative sun exposure, keratinocytes can become malignant. But even then, the

**Figure 10-13**

Marjolin's ulcer. This large lesion constitutes a Marjolin's ulcer. The client had a history of venous ulcers and had been treated for many months by home health nurses with no improvement. She was seen at the Archbold Center for Wound Management; a biopsy was done immediately with a fresh-frozen section, and a diagnosis of Marjolin's was made. The cancer had metastasized to the bone, and a below-knee amputation was required. It was never clear whether the cancer began in a new ulcer or in scar tissue from a previous ulcer. (Courtesy Harriett B. Loehne, PT, DPT, CVWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

cancers (either basal or squamous cell) grow slowly and do not spread easily.

On the other hand, melanocytes, the cells that give rise to melanomas, seem highly resistant to self-destruction. After a person gets badly sunburned, damaged melanocytes continue to replicate, increasing the chance that some will turn malignant. These studies suggest that Fas ligand is a critical defense against the accumulation of mutations caused by sunlight exposure. Its absence or inactivation may be key to the development of skin cancer.^{67,110}

Clinical Manifestations. Squamous cell lesions are more difficult to characterize than basal cell tumors. The squamous cell tumor has poorly defined margins, since the edge blends into the surrounding sun-damaged skin.

This type of carcinoma can present as an ulcer, a flat red area, a cutaneous horn, an indurated plaque, or a nodule. It may be red to flesh-colored and surrounded by scaly tissue. More than 80% of squamous cell carcinomas occur in the head and neck region.

Malignant transformation of any chronic wound can occur (Fig. 10-13). *Marjolin's ulcer* is the term given to aggressive epidermoid tumors that arise from areas of chronic injury and form squamous cell carcinomas. Flealed burn wounds are common sites, but any chronic wound can transform into a malignancy. Dr. Jean Nicolas Marjolin first described the occurrence of ulcerating lesions within scar tissue in 1828. If these lesions are not detected and treated early, they may invade deep tissues and ulcerate (see Prognosis).

Usually lesions on unexposed skin tend to be more invasive and more likely to metastasize, with the exception of lesions on the lower lip and ears. These sites tend to metastasize early, beginning with the process of indu-

Table 10-4 Staging of Squamous Cell Carcinoma (Skin)

Primary Tumor (T)*

TX	Primary tumor cannot be assessed
T0	No evidence of primary tumor
Tis	Carcinoma in situ
T1	Tumor ≤2 cm in greatest dimension
T2	Tumor >2 cm but not >5 cm in greatest dimension
T3	Tumor >5 cm in greatest dimension
T4	Tumor invades deep extradermal structures (i.e., cartilage, skeletal muscle, bone)

Regional Lymph Nodes (N)

NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Regional lymph node metastasis

Distant Metastasis (M)

MX	Presence of distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis

Used with permission of the American Joint Committee on Cancer (AJCC), Chicago. AJCC handbook for staging of cancer, ed 6, New York, 2002, Springer-Verlag.

*In the case of multiple simultaneous tumors, the tumor with the highest T category will be classified and the number of separate tumors will be indicated in parentheses, for example, T2 (5).

ration and inflammation of the lesion. Metastasis can occur to the regional lymph nodes, producing characteristic systemic symptoms of pain, malaise, fatigue, weakness, and anorexia.

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. An excisional biopsy provides definitive diagnosis and staging (Table 10-4) of squamous cell carcinoma. Other laboratory tests may be appropriate depending on the presence of systemic symptoms. The size, shape, location, and invasiveness of a squamous cell tumor and the condition of the underlying tissue determine the treatment method selected (see the section on Basal Cell Carcinoma; see also Box 10-7). A deeply invasive tumor may require a combination of techniques. As with all benign, premalignant, or malignant skin lesions, sun protection is vitally important (see Box 10-4).

All the major treatment methods have excellent rates of cure; generally, the prognosis is better with a well-differentiated lesion in an unusual location.

Malignant Melanoma

Definition and Overview

Malignant melanoma is a neoplasm of the skin originating from melanocytes or cells that synthesize the pigment melanin. The melanomas occur most frequently in the skin but can also be found in the oral cavity, esophagus, anal canal, vagina, or meninges or within the eye. The clinical varieties of cutaneous melanoma are classified into four types (Fig. 10-14)¹⁵:



Figure 10-14

A, Superficial spreading melanoma. An irregular margin with multiple colors of black, blue, pale red, and white may be seen. **B**, Nodular lesion of melanoma. This lesion developed on top of a benign compound nevus. **C**, Lentigo maligna. If left alone, progression to lentigo maligna melanoma occurs. **D**, Acral lentiginous melanoma. This brown to black, flat lesion has irregular borders and variable pigmentation. (From Callen JP, Greer K, Paller A, Swinyer L: *Color atlas of dermatology*, ed 2, Philadelphia, 2000, Saunders. **D**, Courtesy Dr. Neil A. Fenske, Tampa.)

1. *Superficial spreading melanoma (SSM)* is the most common type of melanoma and accounts for 75% of cutaneous melanomas. SSM can occur on any part of the body, especially in areas of chronic irritation, the legs of females between the knees and ankles, or the upper back in both genders. SSM is usually diagnosed in people between 20 and 60 years of age. It usually arises in a preexisting mole and presents as a brown or black, raised patch with an irregular border and variable pigmentation (red, white, and blue; brown-black; black-blue). It is usually asymptomatic. With advanced lesions, itching and bleeding may occur.

2. *Nodular melanoma* can be found on any part of the body with no specific site preference. Men between 20 and 60 years of age are affected more often than women. It is often described as a small, suddenly appearing but quickly enlarging, uniformly and darkly pigmented papule (may be greyish) and accounts for approximately 12% of cutaneous mel-

anomas. This type invades the dermis and metastasizes early.

3. *Lentigo maligna melanoma (LMM)* is a less common type of lesion occurring predominantly on sun-exposed areas, especially the head, neck, and dorsa of hands or under the fingernails, in the 50- to 80-year-old age group, accounting for 6% to 10% of cutaneous melanomas. This lesion looks like a large (3- to 6-cm), flat freckle with an irregular border containing varied pigmentation of brown, black, blue-black, red, and white found in a single lesion. These lesions enlarge and become progressively irregularly pigmented over time. Approximately one third develop into malignant melanoma and therefore bear careful watching.

4. *Acral lentiginous melanoma (AIM)* is a relatively uncommon form of melanoma accounting for 5% of all cutaneous melanomas. It is the most common form of melanoma in dark-skinned people (e.g., Africans, Asians). These lesions usually have flat,

Box 10-9**RISK FACTORS FOR THE DEVELOPMENT OF MALIGNANT MELANOMA**

- Family history of malignant melanoma
- Presence of blond or red hair
- Presence of marked freckling on the upper back
- History of three or more blistering sunburns before age 20 years
- History of 3 or more years of an outdoor summer job during adolescence
- Presence of actinic keratosis (sharply outlined horny growth)

Modified from Friedman RJ, Rigel DS, Silverman MK, et al: Malignant melanoma in the 1990s, *CA Cancer J Clin* 41:201-225, 1991.

dark brown portions with raised bumpy areas that are predominantly brown-black or blue-black. Most common areas include low-pigment sites where hair is absent, such as the palms of the hands, soles of the feet, nail beds of fingers and toes, and mucous membranes.

Incidence

Malignant melanoma accounts for up to 5% of all cancers, currently affecting 1 in 75 people in the course of a lifetime. This has increased dramatically from a 1 in 1500 risk of developing melanoma in the 1930s.

Epidemiologists, who report that the incidence of melanoma is doubling every 10 to 20 years, call this a melanoma epidemic. The ACS estimates 62,190 new cases of malignant melanoma in 2006,⁷⁰ accounting for 7910 deaths, more than from any other skin disorder. The peak incidence is between 40 and 60 years; women are affected more than men but the mortality rate is greater among men. The incidence is rising in younger age groups, but the disorder remains rare in children before adolescence.

Etiologic and Risk Factors

Most people who develop melanoma have blond or red hair, fair skin, and blue eyes; are prone to sunburn; and are of Celtic or Scandinavian ancestry (Box 10-9). These risk factors are believed to be linked to variations in a gene called *MC1R* that assists in producing melanin pigment to help protect the skin against UV rays.^{61,123} Not all UVB radiation (280 to 320 nm) but all UVA radiation (320 to 400 nm), the type produced by sun lamps, may promote skin cancer. For these reasons, tanning parlors should also be considered a significant risk factor for the development of skin cancer.

Melanomas appear to be more prevalent among Caucasians of high socioeconomic status who work indoors and tend to take short vacations with intense sun exposure than in people who are at risk of chronic sun exposure. This correlation appears to be related to education, not income.⁶⁰

Melanoma occurs more often within families and among people who have dysplastic nevus syndrome, also known as the *atypical mole syndrome*. This is a familial disorder that results in a large number of irregular moles

that have an almost 50% chance of developing into melanoma during the person's lifetime. Puberty and pregnancy may enhance growth. Previous history of melanoma places the individual at greater risk of developing a second melanoma.

Other risk factors include excessive exposure to UV radiation through sunlight or tanning booths, especially intense intermittent exposure, and immune suppression from chemotherapy. There are some reports that airline pilots and flight crews exposed to ionizing radiation of cosmic origin have increased rates of malignant melanoma.^{13,83,18}

Increased cancer risk among all flight personnel has been previously noted, including breast cancer among flight attendants and acute myeloid leukemia among pilots. Further studies are needed to identify specific and potentially preventable risk factors.^{7,42}

Although the evidence is clear that flight personnel have the highest incidence ratio of skin cancer, whether this is a result of excessive exposure to cosmic and UV radiation (which can easily penetrate the cockpit), from flying over multiple time zones (disturbance of circadian rhythm), or from factors related to lifestyle (excessive sunbathing) rather than work conditions remains inconclusive.^{58,119} Supportive research shows that an 8% to 10% increase in UVB radiation occurs for every 1000 feet of elevation, suggesting a higher incidence of skin cancer at higher elevations.¹²⁸

Pathogenesis

See the section on **Squamous Cell Carcinoma: Pathogenesis**.

The majority of malignant melanomas appear to be associated with the intensity rather than the duration of sunlight exposure; that is, most people who develop melanoma work indoors and have intense but limited exposure to the sun on weekends or vacations. This accounts for the location of melanomas most commonly on skin that is covered most of the year.

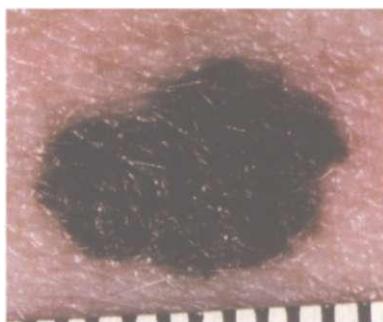
Clinical Manifestations

Melanoma can appear anywhere on the body, not just on sun-exposed areas. Common sites are the head and neck in men, the legs in women, and the backs of people exposed to excessive UV radiation. Up to 70% arise from a preexisting nevus. Any change in a skin lesion or nevus (increased size or elevation; bleeding; soreness or inflammation; changes in color, pigmentation, or texture) must be examined for melanoma (Fig. 10-15).

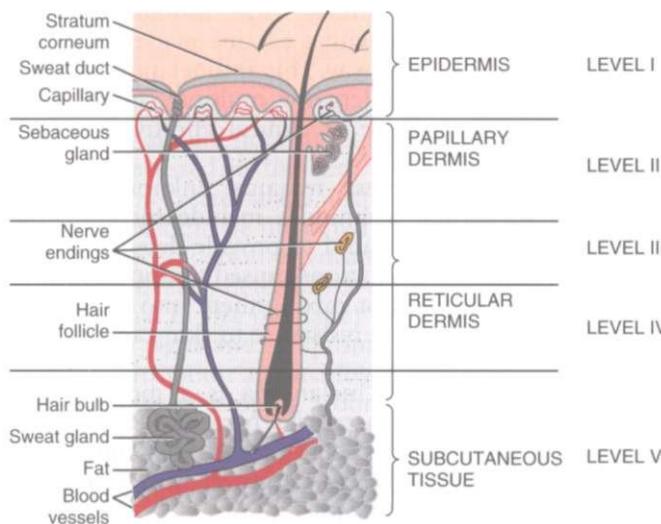
MEDICAL MANAGEMENT

DIAGNOSIS. Early recognition of cutaneous melanomas can have a major impact on the surgical cure of this disease. The ACS suggests a monthly self-examination. A skin biopsy with histologic examination can distinguish malignant melanoma from other lesions, determine tumor thickness, and provide staging.

There are several techniques for staging skin cancer. The Breslow method measures the thickness of the melanoma; the thinner the melanoma, the better the prognosis. Generally, melanomas less than 1 mm in depth have a very small chance of spreading. A second system (Clark's

**Figure 10-15**

Malignant melanoma. Warning signs of malignant melanoma include asymmetry, irregular border, two or more shades of color, and diameter as outlined in Box 10-10. Any other changes in moles or skin lesions (e.g., itchiness, tenderness, swelling, redness, softening, or hardening) should be evaluated by a physician. (From Goldman L: *Cecil textbook of medicine*, ed 22, Philadelphia, 2004, Saunders.)

**Figure 10-16**

Clark's levels, a system of classifying tumor progression according to skin layer penetration. Level I involves only the epidermis, level II has spread to the dermis, level III involves most of the upper dermis, level IV has spread to the lower dermis, and level V indicates the cancer has spread to the subcutis. The higher the level, the greater the chance that metastasis has occurred.

levels) used to determine the appropriate stage evaluates the layers of skin that are invaded by the melanoma (Fig. 10-16).

A third method of staging, TNM, combines both previously described methods (Table 10-5). Depending on the depth of the tumor invasion and metastatic spread, other testing procedures may be used, including baseline laboratory studies, a bone scan for metastasis, or computed tomographic (CT) scan for metastasis to the chest, abdomen, CNS, and brain.

Diagnostic accuracy will continue to improve as digitized images of lesions can be analyzed, enabling the physician to determine whether a biopsy is needed. Computer-aided microscopic examination of biopsy slides

Table 10-5 Staging of Malignant Melanoma***Primary Tumor (T)**

TX	Primary tumor cannot be assessed (e.g., shave biopsy or regressed melanoma)
T0	No evidence of primary tumor
Tis	Melanoma in situ
T1	Melanoma ≤ 1.0 mm in thickness with or without ulceration
T1a	Melanoma ≤ 1.0 mm in thickness and level II or III, no ulceration
T1b	Melanoma ≤ 1.0 mm in thickness and level IV or V or with ulceration
T2	Melanoma 1.01-2 mm in thickness with or without ulceration
T2a	Melanoma 1.01-2.0 mm in thickness, no ulceration
T2b	Melanoma 1.01-2.0 mm in thickness, with ulceration
T3	Melanoma 2.01-4 mm in thickness with or without ulceration
T3a	Melanoma 2.01-4.0 mm in thickness, no ulceration
T3b	Melanoma 2.01-4.0 mm in thickness, with ulceration
T4	Melanoma >4.0 mm in thickness with or without ulceration
T4a	Melanoma >4.0 mm in thickness, no ulceration
T4b	Melanoma >4.0 mm in thickness, with ulceration

Regional Lymph Nodes (N)

NX	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in one lymph node
N1a	Clinically occult (microscopic) metastasis
N1b	Clinically apparent (macroscopic) metastasis
N2	Metastasis in two to three regional nodes or intralymphatic regional metastasis without nodal metastases
N2a	Clinically occult (microscopic) metastasis
N2b	Clinically apparent (macroscopic) metastasis
N2c	Satellite or in-transit metastasis† without nodal metastasis
N3	Metastasis in four or more regional nodes, or matted metastatic nodes, or in-transit metastasis or satellite(s) with metastasis in regional node(s)

Distant Metastasis (M)

MX	Distant metastasis cannot be assessed
M0	No distant metastasis
M1	Distant metastasis
M1a	Metastasis to skin, subcutaneous tissues, or distant lymph node(s)
M1b	Metastasis to lung
M1c	Metastasis to all other visceral sites or distant metastasis at any site associated with an elevated serum lactic dehydrogenase (LDH)

Used with permission of the American Joint Committee on Cancer (AJCC), Chicago. AJCC handbook for staging of cancer, ed 6, New York, 2002, Springer-Verlag.

*Several systems exist for staging malignant melanoma, including the TNM (tumor, node, metastasis) system, developed by the American Joint Committee on Cancer, and Clark's system, which classifies tumor progression according to skin layer penetration (see Fig. 10-16).

†In-transit metastasis involves skin or subcutaneous tissue more than 2 cm from the tumor but not beyond the regional lymph nodes.

may lead to better diagnosis, and teledermatology will add additional assistance in melanoma diagnosis. This technology makes it possible to compress digital images of suspicious lesions and transmit them electronically anywhere in the world, making consultation easier.¹²⁷

Scientists are continuing to seek complementary ways of predicting metastatic potential so that preventive therapies can be initiated earlier. Tests that can detect submicroscopic melanoma cells circulating in the blood vessels and lymphatics could prove invaluable, since melanoma generally metastasizes via these systems.

The recently developed reverse transcriptase polymerase chain reaction (RT-PCR) assay designed to detect genetic markers or indicators of potential metastatic melanoma in the blood has been seriously hampered by false-negative reports. A study in 2005 concluded that RT-PCR does not correlate with traditional indicators for prognosis of melanoma.^{12,115,5}

Additional assays that make use of multiple melanoma markers are being investigated.^{36,52} Adhesion markers have proven valuable¹¹² as well as microtubule-associated protein 2 (MAP2), a marker of neuronal differentiation that induces mitotic defects, inhibits growth of melanoma cells, and predicts metastatic potential of cutaneous melanoma.¹⁴²

TREATMENT. Neither cryosurgery with liquid nitrogen nor electrodesiccation is used to treat melanoma, although they are among the acceptable procedures for squamous cell and basal cell tumors. The treatment of choice for melanoma without evidence of distant metastatic spread is surgical excision. Surgery is combined with postoperative adjuvant radiation therapy and/or chemotherapy when there is evidence of regional spread. Surgery is not usually recommended for tumors that have metastasized to distant sites.¹⁰⁰

Previously, surgical excision of the primary lesion site may have been accompanied by removal of regional lymph nodes (regional lymphadenectomy), but sentinel node biopsy (see Chapter 9) has been shown to be a reliable diagnostic tool for selecting individuals to be submitted to lymph node dissection, thereby reducing the extent of surgery for those who do not need this procedure.^{20,101}

There is considerable debate about the use of sentinel node biopsy in staging melanoma at this time. Some studies report that this type of biopsy is highly reliable in experienced hands but a low-yield procedure in most thin melanomas,^{24,162} whereas others argue against its use except in clinical trials¹⁵¹ and claim that there is not enough evidence to support the current combined use of sentinel node biopsy and systemic interferon (IFN) for melanoma.¹⁰⁹

In 2005 a study indicated that children with high-risk melanoma could be treated with sentinel lymph node biopsy and IFN-alfa-2b with tolerable toxicity.¹⁰³ A more recent study of sentinel lymph node biopsy histology in melanomas less than or equal to 1.0 mm in depth suggests it is a significant predictor of outcome and should be considered for individuals with melanomas 0.75 to 1.0 mm in depth.¹²⁰

Surgeons are now able to use aggressive surgical approaches on a more selective basis and therefore decrease treatment-related complications and disfigurement without compromising surgical goals. This change in treatment approach came as a result of the knowledge that the recurrence rate for people with melanoma clinically localized to the skin correlates directly with tumor thickness or depth of invasion, whereas the prognosis for people whose disease has spread to the regional lymph nodes depends primarily on the number of nodes that have tumors.¹²⁹

Deep primary lesions may warrant adjuvant chemotherapy and biotherapy to eliminate or reduce the number of tumor cells, but there is no role at present for chemotherapy or radiation therapy as the initial treatment. Radiation therapy is used for metastatic disease to reduce tumor size and provide palliative relief from painful symptoms; it does not prolong survival time.

Ongoing research to develop immunomodulation therapy remains a large area of study. In addition to immunotherapy to stimulate the immune system, research to treat people with a vaccine made from their own cancer cells is under way. This antimelanoma immunization is currently undergoing multiple clinical trials and has shown evidence of clinical effectiveness with minimal side effects.^{16,99,152}

Long-term effectiveness remains unknown. Other more investigational techniques include incorporating Fas ligands (a protein found to provide defense against the accumulation of mutations caused by sun exposure) into short-acting drugs or mixing them into sunscreens to boost people's natural resistance to skin cancer.

PROGNOSIS. Malignant melanoma is a more serious problem than other skin cancers because it can spread quickly and insidiously, becoming life-threatening at an earlier stage of development. However, it is essentially 100% curable if detected early.

The prognosis for all types of melanoma depends primarily on the tumor's thickness and depth of invasion, not on the histologic type. The more superficial or thin the tumor, the better the prognosis. For example, melanoma lesions less than 0.76 mm deep have an excellent prognosis (5-year survival rate is 90%), whereas deeper lesions (more than 0.76 mm) carry the risk for metastasis (5-year survival rate is 65% with local metastasis; 30% to 35% when distant metastases are present). Metastases to the brain, lungs, bones, liver, and CNS are universally fatal, usually within 1 year.

Prognosis is better for a tumor on an extremity that is drained by one lymphatic network than for one on the head, neck, or trunk drained by several lymphatic networks. Tumors can recur more than 5 years after primary surgery, requiring close long-term medical follow-up. Local recurrence without metastases may not represent a poor prognosis if the recurrence is simply an outgrowth of residual undetected microscopic cells from the previously excised primary tumor.^{17,168} Education on the effects of UVB exposure can dramatically reduce the incidence and recurrence of skin cancer.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-9

Malignant Melanoma**PREFERRED PRACTICE PATTERNS**

For potential treatment intervention for nonhealing skin graft following excision:

7D: Impaired Integumentary Integrity Associated with Full-Thickness Skin Involvement and Scar Formation

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

During observation and inspection of any client, the therapist should be alert to the potential signs of skin cancer. Therapists should not become overly concerned about small pink spots on the client's skin, since other common skin conditions, such as eczema, psoriasis, and seborrheic dermatitis, are prevalent in more than half of all people at some time in their lives.

Therapists should look for abnormal spots, especially in sun-exposed areas, that are rough in texture, are persistently present, and bleed on minimal contact or with minimal friction. Keep in mind that seborrheic keratosis commonly bleeds; once diagnosed, this bleeding should not cause undue alarm.

As discussed in the text, *any* change in a wart or mole (color, size, shape, texture, ulceration, bleeding, itching) should be inspected by a physician. The Skin Cancer Foundation advocates the use of the ABCD method of early detection of melanoma and dysplastic (abnormal in size or shape) moles (Box 10-10).

Other signs and symptoms that may be important include irritation and itching; tenderness, soreness, or new moles developing around the mole in question; or a sore that keeps crusting and does not heal within 6 weeks. For any client with a previous history of skin cancer, emphasize the need for continued close follow-up to detect recurrence early. Education on the effects of UV radiation and taking precautions (Box 10-11) can dramatically reduce the incidence of skin cancer.

If surgery included lymphadenectomy, the therapist may be involved in minimizing lymphedema or treating residual lymphedema (see the section on Lymphedema in Chapter 13). Wound management may involve care of a skin graft and the associated donor site; the donor site may be as painful as the tumor excision site and just as much at risk for infection. Standard Precautions (see Appendix A) are essential for the postoperative as well as immunosuppressed client.

For the dying client, hospice care may include pain control and management. It is important that pain relief not be delayed until after pain occurs but rather that a schedule of analgesia to prevent pain or to prevent an increase in pain level is instituted. Wound management must include standard of care unless the client declines it.

Box 10-10**ABCD METHOD OF EARLY MELANOMA DETECTION**

- A: Asymmetry: uneven edges, lopsided in shape, one half unlike the other
- B: Border: irregularity, irregular edges, scalloped or poorly defined edges
- C: Color: black, shades of brown, red, white, pink, occasionally blue
- D: Diameter: Larger than a pencil eraser

Box 10-11**GUIDELINES FOR PREVENTION OF SKIN CANCER**

- Avoid peak hours of sunlight.
- Wear close-woven protective clothing.
- Use a sunscreen of sun-protective factor (SPF) 15 or higher.
- Teach children sun protection.
- Do not work on getting a tan.
- Do not patronize tanning salons.
- Examine your skin regularly.
- If you notice any changes, see your physician promptly.

Kaposi's Sarcoma**Definition and Overview**

Kaposi's sarcoma (KS) is a malignancy of vascular tissue that presents as a skin disorder. Until recently, this tumor was most commonly seen in older men of Mediterranean or Eastern European origin, especially men of Jewish or Italian ancestry (now referred to as *classic KS*).

The sudden emergence of this malignancy in the Western world is directly related to AIDS-associated immunodeficiency, and the incidence has risen dramatically along with the incidence of AIDS (*epidemic KS*). KS may also occur in kidney transplant recipients taking immunosuppressive drugs.

Etiologic Factors and Incidence

Research in the mid-1990s confirmed that KS is caused by a herpesvirus infection. The human herpesvirus 8 (HHV-8) or Kaposi's sarcoma-associated herpesvirus (KSHV) is present in all AIDS-related KS and has been linked to all four forms of KS (classic, iatrogenic, endemic [African], HIV associated); however, only iatrogenic KS and HIV-associated KS have been shown to be linked to impairment of the host immune response. This universal detection of KSHV/HHV-8 suggests a central role for the virus in the development of KS and common etiologic factors for all KS types.⁶⁹ Genetic or hereditary predisposition may be a factor in the classic form.

Epidemiologic surveys indicate that the seroprevalence for HHV-8 parallels the risk of developing KS—5% to 10% in the general population of the Western world but ranging up to 20% to 70% in homosexual HIV-infected clients. Among the people who develop AIDS, KS is seen

almost exclusively among homosexual or bisexual men, with the probability of HHV-8 seropositivity directly proportional to the number of previous male sex partners. If a person contracts AIDS as a result of IV drug use or from a transfusion, the chance of developing KS is less than 2%.

Rates are lower (10%) among HIV-infected women, people with hemophilia, and injection drug users. Overall incidence has been reduced among adults with AIDS associated with the use of antiretroviral therapy to control HIV replication and limit the associated immunodeficiency.^{24,53}

Pathogenesis

Although HHV-8 is detectable in saliva and semen, the exact mechanism of transmission is not known and the details of the pathogenesis remain unknown. KS is an angioproliferative tumor. It is suspected that endogenous substances produced by HIV-infected cells and/or a viral-induced tumorigenesis may promote angiogenesis and the growth of KS.^{1,97}

Studies have demonstrated the role of vascular endothelial growth factor A (VEGF-A) and its receptors in the pathogenesis of KS.³ VEGF-A is needed in the pathogenesis of KSHV, due to its ability to mediate angiogenesis.³ In Kaposi's sarcoma, VEGF was also increased in cells from lesions as a result of organ transplants and even more so in normal cells around the Kaposi's sarcoma's lesion.¹⁴³

Clinical Manifestations

This neoplasm involves the skin and mucous membranes as well as other organs and can lead to tumor-associated edema and ulcerations. Classic KS occurs commonly on the lower extremities, and the affected areas are red, purple, or dark blue macules (Fig. 10-17) that slowly enlarge to become nodules or ulcers. Itching and pain in the lesions that impinge on nerves or organs may occur; and as the sarcoma progresses, causing lymphatic obstruction, the legs become edematous. The lesions may spread by metastasis through the upper body to the face and oral mucosa.

Unlike classic forms of the disease, AIDS-associated KS is a multicentric entity that appears on the upper body (including face, chest, and neck) but can occur on the legs. It frequently involves lymph nodes, lung, and the GI tract; it may be the first manifestation of AIDS (Fig. 10-18; see also Fig. 7-18).

Early lesions are faint pink and can easily be mistaken for bruises or nevi and be ignored. Systemic involvement may present with one or more signs and symptoms, including weight loss (10% of body weight), fever of unknown origin that exceeds 100° F (37.8° C) for more than 2 weeks, chills, night sweats, lethargy, anorexia, and diarrhea. Pulmonary involvement may be characterized by dyspnea, cough, chest pain, and hemoptysis (in order of prevalence).

MEDICAL MANAGEMENT

DIAGNOSIS, TREATMENT, AND PROGNOSIS. Diagnosis is by skin biopsy using a highly sensitive and specific test for this neoplasm. A CT scan may be performed to detect



Figure 10-17

Classic Kaposi's sarcoma is present as typical nodular lesions on this ankle and foot. (From Kumar V, Abbas AK, Fausto M: Robbins and Cotran pathologic basis of disease, ed 7, Philadelphia, 2005, Saunders.)



Figure 10-18

Epidemic Kaposi's sarcoma in the plaque stage. Evolving lesions develop into raised papules or thickened plaques that are oval in shape and vary in color from red to brown. (From Swartz MH: Textbook of physical diagnosis, ed 5, Philadelphia, 2006, Saunders.)

and evaluate possible metastasis. Dermatologic manifestations of KS can be alarming, but it is visceral involvement associated with AIDS KS that is most life-threatening.

However, new antiretroviral therapies, in particular the protease inhibitors, appear to be changing the clinical course of KS. It is now possible to see a complete resolution and control of KS with the use of these new agents. As researchers continue to unravel the pathogenesis of KS, new treatment modalities will target its pathogenic pathways.

Chemotherapy remains an integral part of treatment, and new agents are becoming available. Experimental therapies being evaluated in ongoing clinical trials include angiogenesis inhibitors, pregnancy hormone (human chorionic gonadotropin), photodynamic therapy, isotretinoin, antiviral medications ganciclovir and foscarnet, retinoic acid derivatives, and immune modulators, such as interleukin-12.^{48,98,164} See discussion of AIDS in Chapter 7.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-10

Kaposi's Sarcoma

PREFERRED PRACTICE PATTERNS

6H (Lymph Nodes): Impaired Circulation and Anthropometric Dimensions Associated with Lymphatic System Disorders

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7C: Impaired Integumentary Integrity Associated with Partial-Thickness Skin Involvement and Scar Formation

7D: Impaired Integumentary Integrity Associated with Full-Thickness Skin Involvement and Scar Formation

7E (Skin and Soft Tissue): Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

The KS skin lesions in the AIDS client are not contagious, and the health care provider need have no fear of transmission of KS or HIV through daily contact with the client. Standard Precautions must be followed whenever providing care for clients with KS to prevent the spread of infection to the client.

Prevention of skin breakdown and wound management is the usual focus of intervention. Clients receiving radiation therapy must keep the irradiated skin dry to avoid possible breakdown and subsequent infection (see the section on Radiation Injuries: Special Implications for the Therapist in Chapter 5).



Figure 10-19

Deforming arthritis of the hands in a person with psoriasis (psoriatic arthritis). (From Callen JP, Greer K, Paller A, Swinyer L; *Color atlas of dermatology*, ed 2, Philadelphia, 2000, Saunders.)

genetically determined. Researchers have discovered a significantly higher than normal incidence of certain human leukocyte antigens (HLAs) in families with psoriasis, suggesting a possible immune disorder.

In the search for a psoriasis gene, researchers have discovered that, although there may be a primary "gatekeeper" gene, multiple genes seem to be involved. If one parent has psoriasis, a child has a 10% to 15% chance of developing the disease; if both parents have psoriasis, the chances increase to 50%.

Although psoriasis is believed to be genetically linked, it may be triggered by mechanical, UV, and chemical injury; various infections (especially by β -hemolytic streptococci); prescription drug use; psychologic stress; smoking; and pregnancy and other endocrine changes.¹¹³

Cold weather and severe anxiety or emotional stress tend to aggravate psoriasis. Flare-ups are often related to specific systemic and environmental factors but may be unpredictable. New epidemiologic studies present evidence that both smoking and drinking have an influence on psoriasis, suggesting that simple modifications in lifestyle may reduce both the prevalence and the severity of psoriasis.⁶⁶

Pathogenesis

The underlying abnormality in psoriasis has not been definitively identified. It is a disorder of the keratinocytes, which form in the lower epidermis, flatten with age, and move toward the surface as new cells are generated below.

Normally, the life cycle of a skin cell is 26 to 28 days: 14 days to move from the basal layer to the stratum corneum and 14 days of normal wear and tear before the cell is sloughed off. In contrast, the turnover time of psoriatic skin is 3 to 4 days. This shortened cycle does not allow time for the cell to mature; thus cells stick and build up on the skin, resulting in a thick and flaky stratum corneum, which in turn produces the cardinal manifestations of psoriasis.

SKIN DISORDERS ASSOCIATED WITH IMMUNE DYSFUNCTION**Psoriasis****Definition and Incidence**

Psoriasis is a chronic, inherited, recurrent inflammatory but noninfectious dermatosis characterized by well-defined erythematous plaques covered with a silvery scale (Fig. 10-19). There are several types of psoriasis, including plaque, guttate, erythrodermic, and pustular psoriasis.

Psoriasis occurs equally in both genders and most commonly in young adults (mean age of onset is 27 years of age) but can occur at any point in a person's life and, once present, becomes a chronic condition that may go in and out of remission. Although psoriasis can occur in infancy, it is uncommon in children under the age of 6 years. It is uncommon among blacks but affects 1% to 2% of the white population; more than 6 million Americans are affected, with more than 100,000 classified as severe cases.

Etiologic and Risk Factors

The cause of psoriasis is unknown, but it appears to be hereditary; that is, the tendency to develop psoriasis is

A second component in the pathogenesis of psoriasis is the immune system reaction, since T cells appear at the sites of heightened keratinocyte activity, much as they would at the site of an infection or tumor. The accelerated activity also triggers capillary growth supplying blood and nutrients to the tissue at that site.

It remains uncertain whether the accelerating keratinocyte turnover initiates the immune system reaction. One theory is that psoriasis is an autoimmune condition in which T-cell attack is provoked by a protein present in the skin, yet other animal studies suggest the pathogenesis begins in the T lymphocytes and initiates the disease process.¹⁰⁷

Clinical Manifestations

Psoriasis appears as erythematous papules and plaques covered with silvery scales. The lesions in ordinary cases have a predilection for the scalp, chest, nails, elbows, knees, groin, skin folds, lower back, and buttocks. The occurrence may vary from a solitary lesion to countless patches covering large areas of the body in a symmetric pattern. Two clearly distinguishing features are the tendency for this condition to recur and to persist.

Lesions that develop at the site of a previous injury are known as the *Koebner phenomenon*. Flare-ups are more common in the winter as a result of dry skin and lack of sunlight, and, as is true for many skin ailments, the severity of psoriasis varies over time, and its exacerbations and remissions often correlate with stress levels and mental outlook.

The most common subjective complaint is itching and, occasionally, pain from dry, cracked, encrusted lesions. In approximately 30% of cases, psoriasis spreads to the fingernails, producing small indentations and yellow or brown discoloration. In severe cases, the accumulation of thick, crumbly debris under the nail causes it to separate from the nail bed (nail dystrophy).

Approximately 10% of people with psoriasis (usually moderate to severe) develop arthritic symptoms referred to as psoriatic arthritis (see Chapter 27). Psoriatic arthritis usually affects one or more joints of the fingers or toes, or sometimes the sacroiliac joints, and may progress to spondylitis. These clients report morning stiffness that lasts more than 30 minutes.

Joint symptoms show no consistent linkage to the course of the cutaneous manifestations of psoriasis but rather demonstrate remissions and exacerbations similar to those of rheumatoid arthritis. No other systemic effects of psoriasis have been reported, but hyperuricemia (gout) is fairly common in clients, precipitated by treatment with methotrexate and as a result of nucleic acid turnover caused by cellular breakdown in lesions of psoriasis.¹⁰⁸

MEDICAL MANAGEMENT

DIAGNOSIS. Diagnosis depends on the previous history, clinical presentation, and, if needed, skin biopsy to identify psoriatic changes in skin or rule out other causes for the lesions. Typically, the serum uric acid level is elevated because of accelerated nucleic acid degradation but without the corresponding gout usually associated with increased uric acid levels. Psoriasis must be distinguished

from eczema, seborrheic dermatitis, and lichenlike papules.

TREATMENT. In the absence of a cure, the goal of treatment is to maximize remission and lessen outbreaks. Psoriasis therapy is highly individualized and often determined by trial and error because different people respond to different treatments. Psoriasis does not spread, and early treatment does not prevent the condition from progressing.

New options exist that adequately suppress the disease process and help provide better control of the psoriasis through the use of a combination of therapies. Various forms of local or systemic treatment routinely offered fall into five general categories: (1) topical preparations, (2) phototherapy, (3) antimetabolites, (4) oral retinoid therapy, and (5) immunosuppressants. New biologic systemic drugs for moderate to severe psoriasis are now available.¹⁰⁹

Identifying individuals who would be good candidates for systemic therapy is under investigation; use of a formal diagnostic tool called the Koo-Menter Psoriasis Instrument (KMPI) has been advocated in making this treatment decision.⁴¹

Topical treatment of psoriasis is usually the first line of therapy, and therapeutic agents include corticosteroids; synthetic vitamin D₃; vitamin A analogues (retinoids); occlusive ointments (e.g., petroleum jelly, salicylic acid preparations, urea-containing topical ointments); oatmeal baths and emollients to relieve pruritus; and occasionally tar preparations.

Corticosteroids are the most commonly prescribed therapy for psoriasis but should be used sparingly because of the incidence of side effects, which have increased with the use of the superpotent fluorinated preparations. Only weak preparations, such as 0.5% or 1.0% hydrocortisone, should be used on the face, perineum, or other sensitive areas (e.g., the flexor surfaces of the arms, abdomen).

The major concerns with all corticosteroid preparations are dermal atrophy, skin fragility, fast relapse times, and, in rare cases, adrenal suppression resulting from systemic absorption. See also the section on Corticosteroids in Chapter 5.

Crude coal tar, one of the oldest remedies for psoriasis, is assumed to work by an antimitotic effect (helps retard rapid cell production). This treatment consists of the daily application of 2% to 5% crude coal tar combined with a tar bath and UV light. The disadvantages of this treatment are the extended time commitments required by the client and the associated mess. More recently, the use of liquid carbonis detergens (LCD) has replaced the use of crude coal tar.

Exposure to UV light (phototherapy), such as UVB or natural sunlight, also helps retard rapid cell production. Widespread involvement may improve with whole-body irradiation with UV light. PUVA refers to the combination of an orally administered photosensitizing drug plus exposure to 1 to 1½ hours of UVA radiation. It is more effective for the thick plaque type of psoriasis, pustular psoriasis, and generalized erythroderma.

PUVA also has its disadvantages as a treatment option, including premature skin aging, increased risk of

nonmelanoma skin and other cancers, and premature cataract formation. This type of therapy is contraindicated in pregnancy and for anyone with abnormal moles or otherwise at risk for skin cancer.*

Methotrexate, originally an anticancer drug, affects DNA synthesis and inhibits reproduction in rapidly growing cells, such as the prolific keratinocytes in psoriasis. Methotrexate also has an immunosuppressant effect, tempering the inflammatory response. Cyclosporine (Neoral), an immunosuppressant most often used to prevent organ transplant rejection, has also been approved as a psoriasis treatment. These pharmaceuticals have potentially serious side effects and must be monitored closely.

A treatment strategy called *sequential therapy* involving a deliberate sequence to optimize therapeutic outcome is being explored for those who require systemic therapy without methotrexate. The rationale for this strategy in psoriasis is that it is a chronic disease requiring long-term maintenance therapy as well as quick relief of symptoms and that some medical interventions are better suited for rapid clearance whereas others are more appropriate for long-term care. In sequential therapy, an acute exacerbation of psoriasis is brought under control promptly with the use of cyclosporine followed by phototherapy and then acitretin is administered in a maintenance phase.⁷⁸⁻⁷⁹

To minimize side effects and maximize efficacy of rapidly clearing lesions and maintaining remission, topical sequential therapy is widely used. A class I corticosteroid and calcipotriene are applied in three different phases—the clearance, transition, and maintenance phases.⁸¹

PROGNOSIS. Psoriasis usually recurs at intervals and lasts for increasingly longer periods, but treatment advances bring relief during flare-ups in approximately 85% to 90% of cases. Spontaneous cure is uncommon, and the risk of infection is high because of the greater than normal amounts of staphylococci present on psoriatic plaques.

People with psoriasis who are HIV positive are at high risk of infection from self-inoculation. As many as 20% of clients who develop psoriatic arthritis may sustain early and severe joint damage with accompanying deformity and disability.

Finally, psoriasis treatment involving PUVA has been shown to contribute to an increased risk of skin cancer decades after the treatment has stopped.¹⁴⁴ New research shows that it may be possible to reduce the risk of skin cancer from PUVA with meditation and stress reduction techniques that reduced healing time to half, thus reducing UV exposure.⁷¹

Physical therapy and occupational therapy are key components in the treatment of moderate to severe psoriasis, with desired outcomes based on minimizing functional limitations.*

Client instruction and direct intervention to provide skin care should emphasize the following: (1) steroid cream application must be in a thin film, rubbed gently into the skin until all the cream disappears; (2) all topical medications, especially those containing anthralin and tar, should be applied with a downward motion to avoid rubbing them into the hair follicles causing inflammation (folliculitis); (3) medication should be applied only to the affected lesions, avoiding contact with normal surrounding skin; and (4) gloves must be worn when applying the cream since anthralin stains and injures the skin.

After application, the client must dust himself or herself with powder to prevent anthralin from rubbing off on the clothes. Mineral oil followed by soap and water can be used to remove the anthralin; the skin should never be rubbed vigorously, but a soft brush can be used to remove the scales.

Any side effects, especially allergic reactions to anthralin, atrophy and acne from steroids, and burning, itching, and nausea, must be reported to the physician immediately. Squamous cell epithelioma may develop from PUVA. Cytotoxins from methotrexate therapy may cause hepatic or bone marrow toxicity; methotrexate may be teratogenic (harmful to fetal development) and should not be prescribed for women who are pregnant, trying to become pregnant, or breast feeding.

Other immunosuppressants, when used over a long period, have an accumulative effect and therefore the potential to cause serious side effects, such as poor wound healing, high blood pressure, kidney damage, and many other complications (see the section on Immunosuppressants in Chapter 5).

Relaxation techniques and stress management are valuable tools whose use should be encouraged on a daily basis but especially during periods of exacerbation.

Psoriatic Arthritis

Clinically, psoriatic arthritis differs from rheumatoid arthritis in the more frequent involvement of the distal interphalangeal joints, asymmetric distribution of affected joints, presence of spondyloarthropathy (including the presence of both sacroiliitis and spondylitis), and characteristic extraarticular features (e.g., psoriatic skin lesions, iritis, mouth ulcers, urethritis, colitis, aortic valve disease).

Joints are less tender in psoriatic arthritis, which may lead to underestimation of the degree of inflammation. Pain and stiffness of inflamed joints are usually increased by prolonged immobility and alleviated by physical activity. Evidence of inflammation is

Continued.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-11

Psoriasis

PREFERRED PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

*For an interesting and comprehensive article on establishing a practical and effective psoriasis treatment center including phototherapy, debridement, and whirlpool therapy, see reference 6.

pain on stressing the joint, tenderness at the joint line, and the presence of effusions.

The increasing use of nuclear magnetic resonance imaging (MRI) techniques, with their ability to delineate cartilage and ligamentous structures and to identify edema, is providing a radical improvement in ascertainment of musculoskeletal abnormalities associated with this disease. It is expected that in the decade to come new information about this aspect of the disease will offer greater information and improved treatment regimens.¹⁶⁰

Psychologic Considerations

Psoriasis can result in psychologic problems because the skin lesions may cause the person to feel contagious and untouchable. In addition, ongoing treatment may not work, and the smell of some topical preparations and the stain may add to the psychologic reaction. Assure the client that psoriasis is not contagious. Flare-ups can be controlled with treatment, and stress control can help prevent recurrences. Relaxation techniques, group counseling, stress management, and medications to treat depression or anxiety may be suggested.

erythema, and bullous lesions. Association with systemic disease is highest in acute cutaneous LE, with virtually all clients meeting the American College of Rheumatology criteria for SLE (see Chapter 7).

Etiologic and Risk Factors and Pathogenesis. The exact cause of cutaneous LE is unknown, but evidence suggests an autoimmune defect. There appear to be interrelated immunologic, environmental, hormonal, and genetic factors involved. Smoking is considered a risk factor for the development of the discoid lesions associated with chronic cutaneous LE and for resistance to treatment with antimalarial agents in this subgroup.^{46,71}

Just how the sun causes skin rash flare-ups remains unknown. One theory is that the DNA of people with lupus, when exposed to sunlight, becomes more antigenic (able to induce a specific immune response). This antigenicity causes accelerated antigen-antibody reactions and thus more deposition of immune complexes in the skin at the dermal-epidermal junction. The photosensitivity is most commonly associated with LE and not other rheumatologic diseases.

Clinical Manifestations. Discoid lesions (*chronic cutaneous LE*) can develop from the rash typically seen in lupus and become raised, red, smooth plaques with follicular plugging and central atrophy. The raised edges and sunken centers give them a coinlike appearance (Fig. 10-20). Although these lesions can appear anywhere on the body, they usually erupt on the face, scalp, ears, neck, and arms or any part of the body that is exposed to sunlight. Lesions more typical of systemic lupus discussed in Chapter 7 are shown in Fig. 10-21.

Hair tends to become brittle, and scalp lesions can cause localized alopecia (bald patches). Facial plaques sometimes assume the classic butterfly pattern with lesions appearing on the cheeks and the bridge of the nose. The rash may vary in severity from a sunburned appearance to discoid (plaquelike) lesions. These lesions can occur in the absence of other lupus-related symptoms and tend to leave hypopigmented and hyperpigmented scars that can become a cosmetic concern.

The most recognized skin manifestation of SLE (*acute cutaneous LE*) is the classic butterfly rash over the nose, cheeks, and forehead (Fig. 10-22) commonly precipitated by exposure to sunlight (UV rays). This classic rash over the nose and cheeks occurs in a large percentage of affected people, but rash can occur on the scalp, neck, upper chest, shoulders, extensor surface of the arms, and dorsum of the hands. These rashes begin abruptly and last from hours to days. They may be precipitated by sun exposure and often coincide with a flare of systemic disease.¹³⁴

Other skin manifestations may point to the presence of vasculitis (inflammation of cutaneous blood vessels) leading to infarctive lesions in the digits (see Fig. 27-14), necrotic leg ulcers, or digital gangrene.

Acute cutaneous LE is usually accompanied by other symptoms of SLE, commonly including malaise, overwhelming fatigue, arthralgia, fever, arthritis, anemia, hair loss, Raynaud's phenomenon, and urologic symptoms associated with kidney involvement.

Lupus Erythematosus

Lupus erythematosus is a chronic inflammatory disorder of the connective tissues. It appears in several forms, including cutaneous lupus erythematosus primarily affecting the skin and systemic lupus erythematosus (SLE), which affects multiple organ systems (including the skin) with considerably more morbidity and associated mortality (see complete discussion in Chapter 7). *Lupus* is the Latin word for "wolf," referring to the belief in the 1800s that the skin erosion of this disease was caused by a wolf bite. The characteristic rash of lupus is red, hence the term *erythematosus*.

Cutaneous Lupus Erythematosus

Overview and Incidence. The subsets of lupus erythematosus (LE) involving the skin include chronic cutaneous LE, acute cutaneous LE, and subacute cutaneous LE. Only the skin-related components of LE are discussed in this chapter. See also the section on Systemic Lupus Erythematosus in Chapter 7.

Chronic cutaneous LE, formerly known as discoid lupus, is marked by chronic skin eruptions on sun-exposed skin that can lead to scarring and permanent disfigurement if left untreated. Usually a systemic disorder does not develop, but in 5% to 10% of cases SLE does develop later; conversely, discoid lesions occur in 20% of people with SLE.¹³⁴ It is estimated that approximately 60% of persons with chronic cutaneous LE are women in their late twenties or older. The disease is rare in children.

Acute cutaneous LE occurs in 30% to 50% of clients who have SLE and includes malar erythema, widespread



A



B



C

Figure 10-20

Discoid lupus erythematosus. Skin changes associated with discoid lupus erythematosus can present in a variety of ways. **A**, Hypertrophic discoid lupus erythematosus with prominent adherent scale. **B**, Round or oval cutaneous lesions can occur on the face or other parts of the body. **C**, Round or oval cutaneous lesions as they appear on a dark-skinned individual. (From Callen JP, Greer K, Paller A, Swinyer L: *Color atlas of dermatology*, ed 2, Philadelphia, 2000, Saunders.)

MEDICAL MANAGEMENT

DIAGNOSIS AND TREATMENT. The client history and appearance of the rash itself are diagnostic. Skin biopsy of the discoid lesions may be performed. The client must report any changes in the lesions to the attending physician. Drug treatment consists of topical, intralesional, or systemic medication.

**Figure 10-21**

The lesions here are typical of *systemic lupus* found on the lower extremities. They are ulcerated, punched-out wounds with necrotic bases. Discoid lupus lesions (Fig. 10-20) are usually found on the face and scalp, and are raised, flat, coin-shaped wounds. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

Potential side effects of systemic therapy (antimalarial agents) for chronic cutaneous LE include diarrhea, nausea, myopathy, cardiomyopathy, and anemia. The lesions resolve spontaneously in 20% to 40% of affected individuals or may cause hypopigmentation or hyperpigmentation, atrophy, and scarring. Discoid lesions are not life-threatening (unless accompanied by complications of SLE) but are associated with psychologic distress and altered quality of life.

Skin lesions require topical treatment, maintaining an optimal wound environment (moist enough to allow tissue healing but not swamplike) while preventing further deterioration or infection. Most often, topical corticosteroid creams are used. The disease process can cause loss of skin integrity and subsequent loss of function.

Clients with any form of cutaneous lupus should avoid prolonged exposure to the sun, fluorescent lighting, or reflected sunlight. They are encouraged to wear protective clothing, use sun-screening agents, and avoid engaging in outdoor activities during periods of intense sunlight (see Box 10-4).

PROGNOSIS. The survival rate has improved dramatically in recent years, although death can occur from renal failure when there is kidney involvement causing progressive changes in the glomeruli; cardiac involvement with deposition of immune complexes in the coronary vessels, myocardium, and pericardium; or cerebral infarct.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-12

Cutaneous Lupus Erythematosus

PREFERRED PRACTICE PATTERNS

4A: Primary Prevention/Risk Reduction for Skeletal Demoralization

4B: Impaired Posture

6B: Impaired Aerobic Capacity/Endurance Associated with Deconditioning

Continued.

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

Clients with lupus erythema with skin involvement require careful assessment, supportive measures, and emotional support. Skin lesions should be checked thoroughly at each visit. The client should be urged to get plenty of rest, follow energy conservation guidelines, and practice good nutrition.

The therapist can be instrumental in teaching and assisting with skin care and prevention of skin breakdown, range-of-motion (ROM) exercises, prevention of orthopedic deformities, ergonomic and postural training, and relief of joint pain associated with SLE. Persons with LE exposed to the long-term effects of corticosteroids should be followed carefully. See specific side effects and the section on Corticosteroids: Special Implications for the Therapist in Chapter 5. See also the section on SLE: Special Implications for the Therapist in Chapter 7.

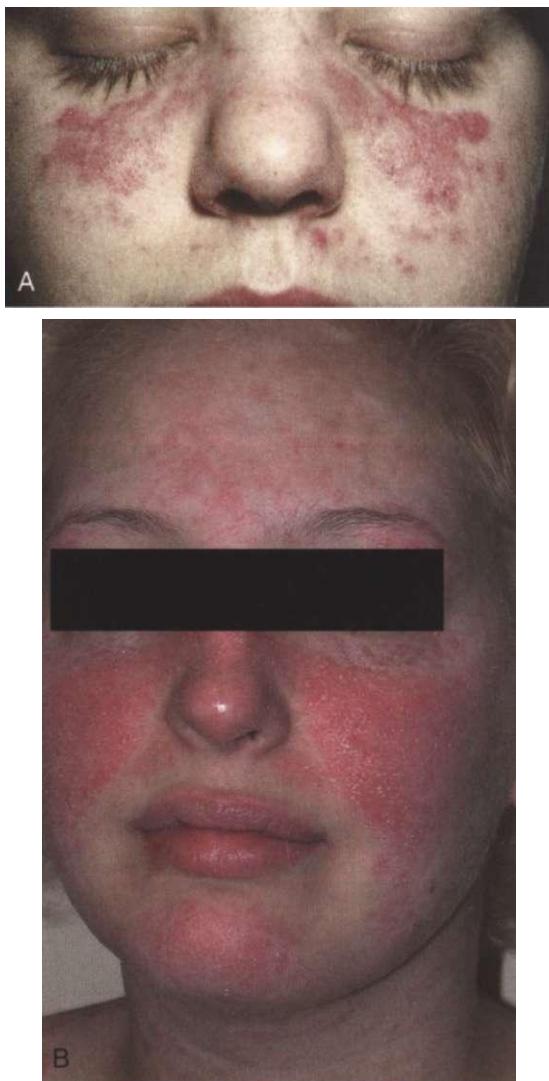


Figure 10-22

A, Butterfly rash of systemic lupus erythematosus across the bridge of the nose and the cheeks. **B,** In some cases the rash covers a larger portion of the face, including the lips and chin. [A, From Goldstein BJ, Goldstein AO: *Practical dermatology*, ed 2, St Louis, 1997, Mosby. Courtesy Department of Dermatology, University of North Carolina at Chapel Hill. Used with permission. B, From Bolognia JL, Jorizzo JL, Rapini RP: *Dermatology*, 2 volume set, St Louis, 2003, Mosby.]

within the first 5 years. The severity of the disease depends on the number of organs affected and the extent of the effect.

Localized scleroderma affects primarily the skin in one or many different areas without visceral organ involvement and is therefore a benign form of this disease. Localized scleroderma should not be confused with limited cutaneous scleroderma. The latter is a form of systemic rather than localized disease.

There is further differentiation of localized scleroderma: *morphea* is characterized by hard, oval-shaped patches on the skin, generally on the trunk. These patches are usually white with a purple ring around them. *Linear* refers to the bandlike lesions that occur in the areas of the arms, legs, and forehead. The bones and muscles beneath these areas may also be affected. Ultimately,

Systemic Sclerosis

Definition and Overview

Systemic sclerosis (SSc, progressive systemic sclerosis [PSS], scleroderma) is a diffuse connective tissue disease that causes fibrosis of the skin, joints, blood vessels, and internal organs. SSc is a chronic disease, lasting for months, years, or a lifetime, and is classified according to the degree and extent of skin thickening.

The presence of a distinctive, widespread vascular lesion characterized by endothelial abnormalities as well as by proliferative reaction of the vascular intima was a significant factor in changing terminology from *scleroderma* to *systemic sclerosis*. General clinical vernacular still refers to this condition as *scleroderma*, although that term simply refers to thickening or hardening of the skin.

There are two distinct subtypes: systemic scleroderma and localized scleroderma (Box 10-12). *Systemic scleroderma* can take one of three forms: limited (ISSc), diffuse (dSSc), and an overlap form with either diffuse or limited skin thickening.

Limited cutaneous SSc was previously known as the *CREST syndrome* from its manifestations (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia). Persons with this form of SSc have a much lower incidence of serious internal organ involvement, although pulmonary hypertension and esophageal disease are not uncommon. Skin tightness is limited to the hands and face (excluding the trunk).

Although the diffuse form is less common than the limited form, it is by far the more debilitating because of the more frequent renal and pulmonary involvement. Some measurable degree of heart, lung, or kidney involvement, or any combination of these, can be found in the majority of people with SSc.

Diffuse scleroderma is characterized by involvement of all body parts, including the skin. In most people, this involvement tends to progress slowly, if at all, but if involvement is to become severe, it tends to do so early,

Box 10-12**CLASSIFICATION OF SCLERODERMA****Systemic Sclerosis**

Limited (75%-80%)

- Symmetric skin thickening
- Restricted to distal extremities and face
- Slow progression of skin changes
- Late development of visceral involvement
- CREST syndrome
- Relatively good prognosis ($\geq 70\%$ survival at 10 years)

Diffuse (15%-20%)

- Symmetric skin thickening
 - Widespread, affecting distal and proximal extremities, face, trunk
 - Rapid progression of skin changes
 - Early appearance of visceral involvement (GI tract, lungs, heart, kidneys)
 - Overall poor prognosis (40%-60% survival at 10 years)
- Overlap (5%-10%)
- Either diffuse or limited skin thickening
 - Associated with one or more connective tissue diseases (e.g., systemic lupus erythematosus, dermatomyositis)

CREST, Calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, telangiectasia; GI, gastrointestinal.

ROM and a child's growth are greatly affected. Linear scleroderma often occurs in childhood.

Other forms include *chemically induced localized scleroderma*, *eosinophilic myalgia syndrome* (previously associated with ingestion of L-tryptophan), toxic oil syndrome (associated with ingestion of contaminated rapeseed oil), and *graft-versus-host disease*.

Incidence

The annual incidence of SSc based on epidemiologic studies of hospital records and death certificates is 10 to 20 cases per 1 million, affecting approximately 400,000 Americans. Until the 1980s, SSc was considered a rare (1 per 2000 individuals) disease, but studies at that time reported a prevalence as much as five times greater than the highest prevalence rate previously reported. Some evidence suggests continued rising incidence rates among adults as well as children, but whether this reflects worldwide incidence or merely regional differences remains unknown.^{14,40}

SSc affects women two to three times more often than men, with the female/male ratio peaking at 15:1 during the childbearing years. Preliminary studies show that fetal cells persist in maternal blood for as long as 27 years postpartum even if the pregnancy does not go to full term. This phenomenon, referred to as *fetomaternal cell trafficking*, may provide an explanation for the increased prevalence of autoimmune disorders such as SSc in adult women following childbearing.¹²

Etiologic and Risk Factors

The cause of scleroderma is reportedly unknown. However, several groups suggest that scientific evidence accumulated over the last 50 years strongly points to SSc as an acquired disease triggered by bacteria (mycoplasma).^{22,86} It has also been suggested that an autoimmune mechanism is the underlying cause, because specific

Localized

Morphea

- Single or multiple plaques of skin fibrosis without systemic disease
- Linear
- Single or multiple fibrotic bands involving skin and deeper tissues

autoantibodies occur in the sera of these clients. Other possible triggers suggested include cytomegalovirus (CMV; increased levels of anti-CMV antibodies present in scleroderma) or immune reactions to viral or environmental factors.⁷⁴

The potential role of placental transfer of fetal cells in the pathogenesis of autoimmune diseases has been mentioned (see Incidence). In childbearing women with scleroderma, fetal cell-derived DNA is detected more frequently in the peripheral blood than in controls. This finding of a limited number of fetal cells in maternal tissues leading to microchimerism (see explanation of chimerism in Chapter 21) has been proposed to have a role in the induction of scleroderma.¹⁴⁸

The occasional onset after trauma suggests the possibility of trophoneurosis (a trophic disorder consequent to disease or injury to nerves). The onset of scleroderma immediately following a severe emotional shock is in some cases a manifestation and result of a psychosomatic disturbance that causes vascular spasm.

Chemicals, especially from occupational exposure to silica, vinyl chloride, or various organic solvents (whether through direct contact or by inhalation), may also induce scleroderma-like changes. Exposure to organic solvents such as benzene and trichloroethane, chemicals common in paint thinners, stains, epoxy resins, and degreasers, associated with recreational hobby or occupation causes the body to produce an antibody called *Scl-70* associated with scleroderma-like illnesses.^{105,106} The toxic oil syndrome and eosinophilia-myalgia syndrome are best-known examples of chemically induced localized forms of scleroderma.³³

Pathogenesis

Widespread small-vessel vasculopathy and fibrosis set SSc apart from other connective tissue diseases. The relentless deposition of extracellular matrix (collagen) in the intima

of blood vessels, the pericapillary space, and the interstitium of the skin is distinctive for SSc and distinguishes it from other autoimmune disorders.

Endothelial injury, obliterative microvascular lesions, and increased vascular wall thickness preferentially affecting small arteries, arterioles, and capillaries are present in all involved organs. Autonomic nerve dysfunction (parasympathetic impairment and marked sympathetic overactivity) seems to be linked to the development of microvascular, cardiac, and GI alterations.⁵⁰

The vascular pathologic condition is characterized by altered vascular function with increased vasospasm, reduced vasodilatory capacity, and increased adhesiveness of the blood vessels to platelets and lymphocytes. The connection between the vascular pathologic condition and development of tissue fibrosis remains unknown, but it is hypothesized that SSc modifies the activity of both the endothelium and the peripheral nervous system, eventually leading to the clinical manifestations of this condition.⁵¹ The extent of injury and dysfunction is reflected by changes in the circulating levels of vascular markers.⁷⁴

Clinical Manifestations

The three stages in the clinical development of scleroderma are the *edematous stage*, the *sclerotic stage*, and the *atrophic stage*. In the edematous stage, bilateral nonpitting edema is present in the fingers and hands and, rarely, in the feet. The edema can progress to the forearms, arms, upper chest, abdomen, back, and face. After a few weeks to several months, edema is replaced by thick, hard skin.

The replacement of edema takes place in the sclerotic stage, when the skin becomes tight, smooth, and waxy and seems bound down to underlying structures. Accompanying changes include a loss of normal skin folds, decreased flexibility, and skin hyperpigmentation and hypopigmentation.

The skin changes may stabilize for periods (years) and may then either progress to the third stage or soften and return to normal. Actual atrophy of skin may occur, particularly over joints at sites of flexion contractures, such as the proximal interphalangeal joints and the elbows. Such thinning of the skin contributes to the development of ulcerations at these sites. Softening and return to normal of the skin may occur to some extent. Improvement typically begins centrally, so that the last areas to become classically involved are the first to show regression.

Not all people pass through all the stages. Subcutaneous calcification (calcinosis) is a late-developing complication that is considerably more frequent in IESSc. Sites of trauma are often affected, such as the fingers, forearms, elbows, and knees. These calcifications vary in size from tiny deposits to large masses ulcerating the overlying skin.

Raynaud's Phenomenon. Scleroderma affects everyone in a different fashion. Each previously mentioned form affects the body in different ways (Box 10-13). Raynaud's phenomenon is very often the first manifestation of SSc, preceding the onset of all the other signs and symptoms of the disease by months or years.⁵¹ It appears

Box 10-13

CHARACTERISTICS LIKELY TO BE SEEN IN CLIENTS WITH SYSTEMIC SCLEROSIS EARLY AND LATE IN THE DISEASE COURSE

Early (≤ 5 Years)

Limited Disease

- Rapidly progressive
 - Renal crisis (5%)
 - Interstitial lung disease (severe in 10%-15%)
- Slowly progressive
 - Raynaud's phenomenon
 - Cutaneous ulceration
 - Esophageal dysmotility

Diffuse Disease

- Rapidly progressive
 - Skin thickening
 - Heart involvement (severe in 10%-15%)
 - Interstitial lung disease (severe in 15%)
- Renal crisis (15%-20%)
 - Contractures, joint pain
 - Cutaneous ulcerations
 - Esophageal dysmotility
 - Gastrointestinal complications

Late (> 5 Years)

Limited Disease

- Slowly progressive
 - Raynaud's phenomenon
 - Cutaneous ulcerations
 - Esophageal dysmotility
 - Gastrointestinal complications
- Very late
 - Pulmonary artery hypertension
 - Biliary cirrhosis

Diffuse Disease

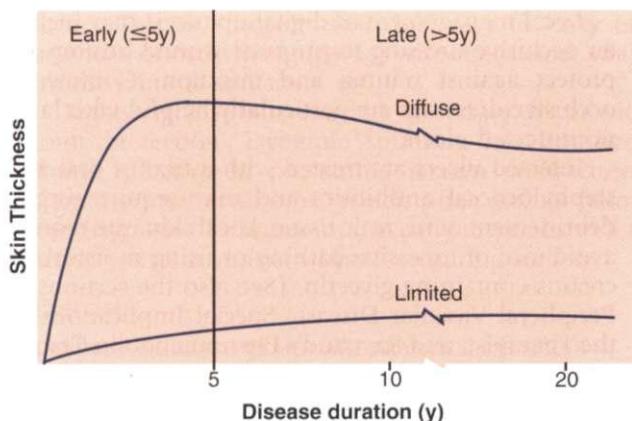
- Improvement
 - Skin thickening
 - Musculoskeletal pain
- Slowly progressive
 - Heart, lung, kidney involvement
 - Raynaud's phenomenon
 - Esophageal dysmotility
 - Gastrointestinal complications

Modified from Clements PJ: Systemic sclerosis: natural history and management strategies, *J Musculoskel Med* 11(1):43-50, 1994.

almost universally in IESSc and in approximately 75% of cases of dSSc.

Raynaud's phenomenon is characterized by sudden blanching, cyanosis, and erythema of the fingers and toes as the walls of the blood vessels that supply the hands and feet become narrowed, making it difficult for the blood to pass through. Closure of the muscular digital arteries, precapillary arterioles, and arteriovenous shunts of the skin causes the hands or feet to become white and numb and then bluish in color as blood flow remains blocked.

As the spasm eases and blood flow returns (approximately 10 to 15 minutes after the triggering stimulus has

**Figure 10-23**

The progression of skin thickening in systemic sclerosis is greatest in the early period (first 5 years), especially in those with diffuse disease. (Modified from Clements PJ: Systemic sclerosis: natural history and management strategies, *J Musculoskel Med* 11[11]:43-50, 1994.)

ended), rewarming occurs and the fingers or toes become red and painful. This cycle is initiated in response to stress or exposure to cold. Progressive phalangeal resorption may shorten the fingers, and compromised circulation resulting from abnormal thickening of the arterial intima may cause slowly healing ulcerations on the tips of the fingers or toes that may lead to gangrene.¹⁵⁸

Skin. Other symptoms include pain, stiffness, and swelling of the fingers and joints. Skin thickening produces taut, shiny skin over the entire hand and forearm. As tightening progresses, contractures may develop. Flexion contractures are especially severe in people with dSSc (Fig. 10-23).

Facial skin may also become tight and inelastic, and the face takes on a stretched and masklike appearance, with thin lips and a pinched nose. Peripheral nervous system involvement affects nerve terminals, reducing sensory fibers in SSc skin. Neuropeptides released by sensory nerve endings are reduced, resulting in vasoconstriction in the skin.

Neuromusculoskeletal System. Most persons with dSSc have disuse atrophy of muscle because of limited joint motion secondary to skin, joint, or tendon involvement. A small percentage of people may have overlap syndromes and demonstrate marked weakness and inflammatory myopathy indistinguishable from polymyositis or dermatomyositis.

Some individuals develop myositis or erosive arthropathy that complicates the joint retraction induced by skin fibrosis. SSc also targets the peripheral nervous system with distal mononeuropathy of the median nerve as a frequent and early feature.¹⁵⁹ Neuropathy from carpal tunnel syndrome is also common.

Polyarthralgias affect both small and large joints and are especially frequent early in dSSc; polyarthritis is unusual. Tenosynovial involvement with inflammation and fibrosis of the tendon sheath or adjacent tissues is characterized by the presence of carpal tunnel syndrome and by coarse, leathery friction rubs palpated during motion over the extensor and flexor tendons of the

fingers, distal forearms, knees, ankles, and other sites. These friction rubs are found almost exclusively in persons with dSSc, and their presence signifies a poorer overall clinical outcome.¹⁵⁸

Viscera. GI motility dysfunction affects the esophagus and anorectal regions, causing frequent reflux, heartburn, dysphagia, and bloating after meals. Other effects include abdominal distention, diarrhea, constipation, and malodorous, floating stools. In advanced disease, cardiac and pulmonary fibrosis develops.

Cardiac involvement can be manifested as myocardial disease, pericardial disease, conduction system disease, or arrhythmias. Pulmonary involvement is characterized by impaired diffusing capacity for carbon monoxide. Kidney involvement and scleroderma renal crisis are now considered rare because of the introduction of angiotensin-converting enzyme (ACE) inhibitors.¹⁶⁰

Other. Nearly 50% of adults with scleroderma report other symptoms, such as major depression, sexual dysfunction, trigeminal neuralgia, hypothyroidism, dental involvement, and corneal tears.

MEDICAL MANAGEMENT

DIAGNOSIS. Early diagnosis and accurate staging of visceral involvement are fundamental for appropriate management and therapeutic approach to this disease. However, diagnosis can be delayed because there is no single laboratory test diagnostic for SSc. A thorough physical examination and history are the first steps to a definitive diagnosis.

Laboratory tests (skin biopsy, urinalysis, blood studies, including erythrocyte sedimentation rate [ESR], presence of rheumatoid factor [results found to be positive in 30% of SSc cases], presence of antinuclear antibodies) are used to determine the extent of involvement and rule out other disease processes. Distinctive serum autoantibodies are found in more than 90% of cases.¹⁶¹ Other tests may include chest films and pulmonary function studies, GI series, and electroencephalogram (EEG).

TREATMENT. Presently there is no cure for SSc. A global vision of SSc is necessary for this multisystem disease, and each treatment program is individualized to manage the specific disease process. Treatment ranges from merely symptomatic for a person with only limited skin involvement after 5 years to aggressive treatment for a person with early, diffuse skin involvement.

When organ involvement occurs, it most often develops early in the disease course, and in the acute phase it requires aggressive management. The program may include medications (e.g., immunosuppressants, penicillamine, antiinflammatory drugs), exercises, joint protection techniques, skin protection techniques, and stress management. See also the section on Raynaud's Phenomenon in Chapter 12.

Penicillamine, a disease-modifying drug, is a penicillamine-derivative immunomodulating agent that has been shown to improve the skin by interfering with cross linking of collagen and to prolong survival in people with early, rapidly progressive SSc. Oral tetracyclines have been found to be the most effective and have the fewest side effects, with minocycline or doxycycline being the

antibiotic of choice. Brand-name drugs (Minocin, Vibramycin) are preferred because some generics are ineffective.¹⁵³

Treatment of the pulmonary complications (pulmonary hypertension, interstitial lung disease) remains difficult. Home blood pressure monitoring can screen for acute hypertension signaling a renal crisis; treatment with ACE inhibitors (see Chapter 12) may be lifesaving.

Research remains ongoing to investigate various treatment regimens for scleroderma, including the use of recombinant human relaxin to reduce skin thickening, improve mobility, and improve function in people with moderate to severe diffuse scleroderma.¹³⁷ The use of antibiotics such as minocycline without the use of any disease-modifying drugs or steroids has shown improvement in the disease, reduction of pain and severity of condition, and better quality of life.^{25,84}

PROGNOSIS. The prognosis in SSc principally depends on early diagnosis; the intensity and rapidity of involvement of the lungs, heart, gut, and kidneys; and appropriate medical management. A model to predict mortality based on a combination of three factors (proteinuria, elevated ESR, low carbon monoxide diffusing capacity) has been reported to have an accuracy of more than 80% in predicting mortality. The absence of these three factors is associated with 93% survival.¹⁹

Spontaneous recovery is common in children, but approximately 30% of clients with SSc die within 5 years of onset. Persons with dSSc who have lived beyond the 5-year mark with no significant visceral involvement are unlikely to experience such organ involvement. Those in whom significant visceral disease developed early can expect a slowing in its progression or at least a stabilization of its course. This 5-year mark is also a time when skin softening begins and musculoskeletal aches and pains begin to ease.

Treatment with ACE inhibitors, started early, now prevents previously fatal complications (acute hypertension, renal failure). Aggressive treatment of early interstitial lung disease may further survival.⁵⁶ Localized scleroderma may reach an end point beyond which the disease does not progress.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-13

Systemic Sclerosis

Skin (Pruritus and Ulcers)

INTEGUMENTARY PRACTICE PATTERNS

7A: Primary Prevention/Risk Reduction for Integumentary Disorders

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7C: Impaired Integumentary Integrity Associated with Partial-Thickness Skin Involvement and Scar Formation

Itching can be a major problem in this condition, and excoriation from scratching can cause open wounds susceptible to infection. The therapist can offer some simple suggestions to soothe skin, ease the itching, and prevent skin damage (see Box 10-4).

Local management of digital tip ulcers may include an occlusive dressing to promote wound healing and protect against trauma and infection. Commercial occlusive dressings are particularly helpful with larger noninfected ulcers.

Infected ulcers are treated with a trial of oral anti-staphylococcal antibiotics and may require surgical debridement of necrotic tissue. Local skin care requires avoidance of excessive bathing or using moisturizing creams containing glycerin. (See also the sections on Peripheral Vascular Disease; Special Implications for the Therapist, and Raynaud's Phenomenon in Chapter 12).

Muscle

INTEGUMENTARY PRACTICE PATTERN

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

MUSCULOSKELETAL PRACTICE PATTERNS

4B: Impaired Posture

4C: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated with Connective Tissue Disorders

Myositis (muscle inflammation) is treated with corticosteroids and sometimes requires the addition of immunosuppressive drugs, whereas fibrotic myopathy (fibrotic tissue laid down within the muscle) is best managed with strengthening and ROM exercises. The efficacy of using soft tissue mobilization or similar techniques has not been investigated. Caution must be used when attempting such treatment, because the skin of these people is usually very sclerosed and sensitive to pressure. Aquatic therapy is an excellent choice for clients with this condition.

Joints and Tendons

INTEGUMENTARY PRACTICE PATTERN

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

MUSCULOSKELETAL PRACTICE PATTERN

4E: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated with Localized Inflammation

Joint and tendon sheath involvement is common and may be treated successfully with nonsteroidal antiinflammatory drugs (NSAIDs). In early dSSc, tenosynovitis can be very painful, limiting joint movement. In addition to NSAIDs, early aggressive therapy is important in preventing or minimizing contractures.

For clients with scleroderma, regular exercise will assist with keeping the skin and joints flexible, maintaining better blood flow, and preventing contractures. Active and passive stretching exercises are necessary but difficult in the presence of extreme pain.

Analgesia is required to optimize participation in an exercise program. Protecting swollen and painful joints from stresses and strains is also an important

factor. This may require teaching ways to carry out activities of daily living (ADLs) without causing strain on the joint or joints.

Lightweight splints may be necessary to provide joint protection. Dynamic splinting has not been found effective in preventing flexion contractures. Carpal tunnel syndrome, which often occurs before the diagnosis of scleroderma, usually responds well to conservative treatment without requiring surgery.

Exercise

CARDIOVASCULAR/PULMONARY PRACTICE PATTERNS

6B: Impaired Aerobic Capacity/Endurance Associated with Deconditioning

6D: Impaired Aerobic Capacity/Endurance Associated with Cardiovascular Pump Dysfunction or Failure

Practice patterns in this area will vary depending on the form of cardiovascular or pulmonary involvement manifested. When cardiopulmonary involvement occurs, intervention must take into consideration effects of this disease on the individual's activity and lifestyle. The client's primary diagnosis and primary intervention may be integument or orthopedics related, but functional limitations may be present secondary to systemic involvement (e.g., decreased aerobic capacity, endurance, and overall general physical condition secondary to cardiovascular and/or pulmonary involvement).

Psychologic Considerations

Persons with early dSSc with or without organ involvement are often anxious because their bodies are changing rapidly and in unexpected ways. They may not understand the grave nature of the disease.

Since persons with dSSc are at greatest risk for early visceral disease and early mortality, education about the disease is important, as is identifying where they are in the natural history of SSc. They should be encouraged to take their blood pressure at home at least three times per week, since this is the best method of screening for acute hypertension. The therapist may screen blood pressure as well.

Box 10-14

TYPES OF INFLAMMATORY MYOPATHIES*

- Primary idiopathic polymyositis
- Primary idiopathic dermatomyositis
- Dermatomyositis or polymyositis associated with malignancy (lung, breast, ovarian, gastric, colon)
- Juvenile polymyositis or dermatomyositis
- Polymyositis associated with other connective tissue diseases (overlap):
 - Sjögren's syndrome
 - Mixed connective tissue disease
 - Rheumatoid arthritis
 - Systemic lupus erythematosus
 - Scleroderma

*Listed in descending order of frequency. Primary idiopathic polymyositis and dermatomyositis account for nearly three fourths of all cases.

Incidence

Polymyositis and dermatomyositis are not very common in the United States, affecting approximately 5 to 10 persons per 1 million; the incidence appears to be increasing. Myositis can affect people of any age, but mostly adults between 45 and 65 years and children between 5 and 15 years are affected. Twice as many women as men are affected with the exception of dermatomyositis associated with malignancy, which is most common in men over age 40 years.

Etiologic Factors

The cause of these conditions remains unknown, although there appears to be some autoimmune mechanism whereby the T cells inappropriately recognize muscle fiber antigens as foreign and attack muscle tissue. Autoantibodies are present in most cases. Polymyositis and dermatomyositis may be drug induced; possibly triggered by a virus; or associated with other disorders as listed in Box 10-14. The association of dermatomyositis with malignancy, particularly ovarian, gastric, and colonic malignancies, may suggest that the neoplasm may stimulate the dermatomyositis.¹⁷

Pathogenesis

If these conditions are caused by an autoimmune reaction, diffuse or focal muscle fiber degeneration is followed by regeneration of new muscle cells, producing remission. Muscle biopsy reveals focal or diffuse inflammatory infiltrates consisting primarily of lymphocytes and macrophages surrounding muscle fibers and small blood vessels. Muscle cells show evidence of degeneration and regeneration, and fiber atrophy is often most severe at the periphery of the muscle bundle. Extensive interstitial fibrosis and fatty replacement are common in longstanding cases.

Clinical Manifestations

Symmetric proximal muscle weakness is the dominant feature of these diseases, although it is variable in its onset, progression, and severity. In some people, symptoms appear suddenly, progress rapidly, and quickly

Polymyositis and Dermatomyositis

Definition and Overview

Polymyositis and dermatomyositis are the two most common idiopathic inflammatory diseases of muscle (Box 10-14). Other types of inflammatory muscle disease have been distinguished, but no satisfactory classification of the idiopathic inflammatory myopathies exists; however, histologic analysis allows differentiation among the types of dermatomyositis.¹⁷ They are diffuse, inflammatory myopathies that produce symmetric weakness of striated muscle, primarily the proximal muscles of the shoulder and pelvic girdles, neck, and pharynx. These related illnesses belong to the family of rheumatic diseases. These diseases often progress slowly, with frequent exacerbations and remissions.

result in a bedridden state, sometimes requiring ventilatory assistance and tube feeding.

More typically, malaise and weight loss develop insidiously over months or even years, with some people either unable to identify the onset of the disease or unaware of the gradual disability developing. Fatigue, rather than weakness, is a commonly reported symptom, but close questioning usually reveals functional losses indicating weakness as well. Pain is not a key feature of these diseases in the adult population, although aching muscles are not uncommon. Muscle wasting is observed in long-standing or severe cases.

Cardiac involvement is not uncommon and contributes significantly to mortality. Nearly half of all people with polymyositis or dermatomyositis have arrhythmias, congestive heart failure, conduction defects, ventricular hypertrophy, or pericarditis.

Pulmonary disease (progressive pulmonary fibrosis) can result from weakness of the respiratory muscles, intrinsic lung pathologic conditions, or aspiration. Swallowing difficulties, nasal regurgitation, and esophageal dysphagia and reflux are common, especially in severe cases.

Polymyositis. Polymyositis begins acutely or insidiously with muscle weakness, tenderness, and discomfort. The proximal muscles of the shoulder and pelvic girdle are affected more often than the distal muscles, usually in a symmetric pattern, but asymmetry is common.

The legs are affected more often than the arms, and the anterior thigh is more frequently involved than the posterior thigh. Initially, the muscles may be slightly swollen, but as the disease progresses muscular atrophy and induration become more noticeable, reflecting the deposition of fibrous tissue. Some persons have a mild peripheral neuropathy with loss of deep tendon reflexes.

Early signs of muscle weakness may include impaired functional status, such as difficulty climbing stairs, getting up from a chair, reaching into an overhead cupboard, combing the hair, or lifting the head from a pillow; difficulty with balance; or a tendency to fall, often resulting in a fracture.

Other muscular effects may include decreased deep tendon reflexes, contractures, arthralgias, arthritis, an inability to move against resistance (e.g., pushing open a heavy door, opening a car door), proximal dysphagia (difficulty swallowing), and dysphonia (difficulty speaking).

Dermatomyositis. When a rash is associated with polymyositis, it is referred to as *dermatomyositis*. A characteristic purplish rash appears on the eyelids (heliotrope erythema), accompanied by periorbital edema (puffy eyelids). The rash may progress to the anterior neck, upper chest and back, shoulders, and arms and may appear around the nail beds. Gottron's papules (red or violet, smooth or scaly patches) may appear on the knuckles, elbows, knees, or medial malleoli (Fig. 10-24).

Although the disease usually begins with erythema and swelling of the face and eyelids, cutaneous manifestations can develop concomitantly with muscle involvement or even afterward. The cutaneous lesions of



Figure 10-24

Gottron's papules or Gottron's sign. Typical lesions over bony prominences on the extensor surfaces of the hand. (From Bolognia JL, Jorizzo JL, Rapini RP: Dermatology, 2 volume set, St Louis, 2003, Mosby.)

dermatomyositis are nearly always present by the time proximal muscle weakness manifests itself. In some persons, muscle involvement is minimal, whereas in others it may progress to wasting and contractures associated with extreme disability.¹⁶³

MEDICAL MANAGEMENT

DIAGNOSIS. The diagnosis of myositis is often difficult because it resembles closely several other diseases and the pathologic manifestation can be localized, sometimes resulting in nondiagnostic biopsies. The physician must rule out internal malignancy first, requiring appropriate medical testing.

Laboratory studies to evaluate muscle enzymes, biopsy to assess muscle fibers, and electromyography (EMG) to measure the electrical activity of the muscles are all necessary to properly diagnose myositis.

Most people with these diseases have an elevated creatine kinase (CK) level at presentation. The CK represents striated muscle involvement, although in people with chronic disease CK may be of the cardiac MB isotype (see Table 40-15). MRI can reveal muscle inflammation and may help to select the site on which to do a biopsy in difficult cases.

TREATMENT. The treatment must be individualized; the components include medication, exercise, and rest. High-

dose daily oral systemic corticosteroid therapy is the usual initial pharmacologic treatment for polymyositis or dermatomyositis. Steroids reduce the inflammation, shorten the time to normalization of muscle enzymes, and reduce morbidity. Persons who do not respond well to steroids or who are unable to tolerate the high dosages required may be treated with immunosuppressive drugs.

PROGNOSIS. The adult prognosis varies depending on age and progression of the disease process, but overall prognosis has improved with the introduction of systemic glucocorticoid therapy. At present, 85% of people with dermatomyositis can be expected to survive. Approximately 50% are left with residual weakness and have persistently elevated serum CK levels or experience a relapse when corticosteroids are reduced, and 20% are substantially disabled.

Generally, the prognosis is worse with visceral organ involvement, and death occurs from associated malignancy, respiratory disease, or heart failure. Side effects of therapy (corticosteroids, immunosuppressants) contribute to long-term morbidity. The prognosis for children is guarded if the disease is left untreated; it progresses rapidly to disabling contractures and muscular atrophy.

graded exercise program (i.e., when muscle enzyme levels fall to acceptable levels indicating effective medical intervention). Often heat, whirlpools, and massages are very effective adjunctive treatments. Pool therapy may be initiated sooner than other forms of exercise.

If the person is confined to bed, protection from foot drop and contractures and prevention of pressure ulcers are essential. If the client has a skin rash, the therapist should caution about the possibility of infection from scratching. If antipruritic medications do not relieve severe itching, tepid sponges or compresses can be applied (see also Box 10-4). If the client is receiving corticosteroids, observe for side effects (weight gain, acne, edema, hypertension, purplish stretch marks [striae], easy bruising).

Long-term use of steroids lowers resistance to infection, may induce diabetes, causes myopathy and/or neuropathy, and is associated with loss of potassium in the urine and gastric irritation (see Table 5-4 and the section on Corticosteroids in Chapter 5). If side effects are marked, advise against abruptly discontinuing corticosteroids until the client consults the physician first. A low-sodium diet will help prevent fluid retention.

Progressive pulmonary fibrosis complicates dermatomyositis and polymyositis in 10% of adults. During the acute phase of illness, clients must be closely monitored for signs of respiratory weakness that requires ventilatory assistance and for overwhelming infection that can lead to circulatory collapse.¹⁷

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-14

Polymyositis and Dermatomyositis

The abrupt onset of any of the cutaneous lesions associated with polymyositis or dermatomyositis could also be a sign of underlying malignancy, particularly genitourinary or GI. The differential diagnosis requires medical evaluation before proceeding with therapy intervention.

PREFERRED PRACTICE PATTERNS

4D: Impaired Joint Mobility, Motor Function, Muscle Performance, and Range of Motion Associated with Connective Tissue Dysfunction

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

The therapist plays a pivotal role in the management of myositis. Manual muscle testing and tests of functional abilities are useful tools in following disease progression and therapeutic response over a long period. The individualized exercise program can help improve muscle strength and function.¹² Aerobic exercise testing may be a useful functional assessment tool in some cases.¹⁵⁷

It is suggested that the medication regimen be well established before beginning exercise. In the early stages of treating myositis, the muscle fibers are fragile and could be damaged further, causing rhabdomyolysis (disintegration of muscle fibers) from exercises and other forms of therapy.

The therapist treating a client with myositis should keep in close contact with the physician, who will be using physical examination and laboratory tests to determine the most opportune time for initiating a

THERMAL INJURIES

Cold Injuries

Cold injuries result from overexposure to cold air or water and occur in two major forms: localized injuries (e.g., frostbite) and systemic injuries (e.g., hypothermia). Untreated or improperly treated frostbite can lead to gangrene and may necessitate amputation requiring therapy and rehabilitation. Hypothermia is a medical emergency and is not discussed in detail here.

Incidence and Etiologic and Risk Factors

Cold injuries, once almost exclusively a military problem, are becoming more prevalent among the general population, especially in athletes using localized cryotherapy or participating in outdoor sports. Frostbite results from prolonged exposure to dry temperatures far below freezing.

The risk of serious cold injuries is increased by lack of insulating body fat, old age, homelessness, drug or alcohol use, cardiac disease, psychiatric illness, motor vehicle problems, or smoking when combined with unplanned circumstances leading to cold exposure without adequate protective clothing.¹⁴⁶

Research is ongoing to reduce the risk of hypothermia and frostbite through the use of nuclear, biologic, and chemical (NBC) protective clothing combined with the

Extreme Cold Weather Clothing System (ECWCS) for those individuals engaging in cold weather outdoor activities. It is undetermined yet whether wearing protective clothing may increase the risk of hypothermia during periods of strenuous activity followed by subsequent periods of inactivity accompanied by sweat accumulation in clothing, which may compromise insulation.¹⁶⁵

Pathogenesis and Clinical Manifestations

Cold-induced injuries can be local or systemic. Severe cold affects all organ systems and especially the central nervous and cardiovascular systems. Many biologic reactions and pathways become distorted or slowed at low body core temperatures. Low body shell temperature can interfere with athletic ability by weakening and slowing muscle contractions, by delaying nerve conduction time, and by facilitating injury.¹³⁵

Typically, an initial vasoconstriction in the skin will protect body parts from a drop in core temperature, but when tissue temperature drops to 35.6° F (2° C), ice crystals form in the tissues and expand extracellular spaces, resulting in localized cold injuries. With compression of cells, cell membranes rupture, interrupting enzymatic and metabolic activities.

Additional injury occurs with thawing when increased capillary permeability accompanies the release of histamine, resulting in aggregation of red blood cells and microvascular occlusion. Research into the pathophysiology of cold injuries has revealed marked similarities in inflammatory processes to those seen in thermal burns and ischemia/reperfusion injury.¹⁰²

Frostbite may be deep or superficial. Superficial frostbite affects the skin and subcutaneous tissue, especially of the face, ears, extremities, and other exposed body areas. Although it may go unnoticed at first, after return to a warm place, frostbite produces burning, tingling, numbness, swelling, and a mottled, blue-grey skin color.

When the affected area begins to rewarm, the person will feel pain and numbness followed by hypoesthesia. Deep frostbite extends beyond subcutaneous tissue and usually affects the hands or feet. The skin becomes white until it has thawed and then turns purplish blue. Deep frostbite produces pain, blisters, tissue necrosis, and gangrene (Fig. 10-25).

MEDICAL MANAGEMENT

DIAGNOSIS AND TREATMENT. Diagnosis is usually made based on the history and presenting symptoms; measures to prevent and treat general hypothermia are taken before managing the local frostbite injuries. Evidence of the role of thromboxanes and prostaglandins in cold injuries has resulted in more active approaches in the medical treatment of frostbite wounds, including the use of vasodilators, thrombolysis, and hyperbaric oxygen.¹⁰²

Triple-phase bone scans can be used to distinguish between tissue that is irreversibly destined for necrosis and tissue that is at risk for necrosis but potentially salvageable. These improvements in radiologic assessment have led to earlier surgical intervention to provide at-risk tissue with a new blood supply and preserve both function and length in an extremity.¹⁴⁶



Figure 10-25

Frostbite of the feet. Blackened areas in the photo show tissue necrosis and gangrene, the result of deep frostbite that extends beyond the subcutaneous tissue. [From Auerbach PS: *Wilderness medicine: management of wilderness and environmental emergencies*, ed 4, St Louis, 2001, Mosby.]

In a localized cold injury, treatment consists of rewarming the injured part without rubbing or massaging the area to avoid further tissue damage, and supportive measures (e.g., analgesics for pain [200 mg of ibuprofen every 6 hours]¹²² and proper positioning to avoid weight bearing with gauze between the toes to prevent maceration). More severe and deeper injuries should not be thawed until medical treatment can be given in a hospital.

The management of blisters is still somewhat controversial, but current practice indicates that clear blisters (shallow injury) should be aspirated; hemorrhagic blisters (deep injury) should not be debrided to avoid desiccation and infection of underlying deep tissue²⁸ (see the section on Skin Lesions: Special Implications for the Therapist).

All frostbitten areas should be treated with topical aloe vera cream. Foam dressings may be applied to maintain a moist wound bed, absorb drainage, and provide protection. A bed cradle may be needed to keep the weight of bedcovers off the affected part or parts.

In the case of a developing compartment syndrome a fasciotomy may be performed to increase circulation by lowering edematous tissue pressure. If gangrene occurs, amputation may be necessary. Smoking causes vasoconstriction and slows healing; the client should be advised to quit smoking, at least during the recovery period.

PROGNOSIS. The prognosis depends on the extent of localized cold injury and development of any complications, such as compartment syndrome, necrosis, or gangrene. Rapid triage and treatment of frostbite can lead to dramatic improvements in outcome and prognosis.¹²² Long-term effects may include increased sensitivity to cold, burning and tingling on reexposure to cold, and increased sweating of the affected area.

Future cold injuries may be prevented through the use of wind-proof, water-resistant, many-layered clothing; moisture-wicking socks; a head covering; mittens instead of gloves; and heat-generating devices (except for those with peripheral neuropathy) in pockets or battery-operated socks.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-15**Cold Injuries****PREFERRED PRACTICE PATTERNS**

4J: Impaired Motor Function, Muscle Performance, Range of Motion, Gait, Locomotion, and Balance Associated with Amputation

7D: Impaired Integumentary Integrity Associated with Full-Thickness Skin Involvement and Scar Formation

7E: Impaired Integumentary Integrity Associated with Full-Thickness Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

Local cold injury subsequent to prolonged exposure may not be seen in a therapy practice until complications such as necrosis and gangrene result in amputation. Whirlpool with gentle agitation directed away from the affected area may be prescribed as part of the rewarming procedure. Water temperature is based on tissue temperature and should be determined in conjunction with the medical staff.

Use of cryotherapy as a modality among the general population can result in localized tissue damage requiring documentation (e.g., filing an accident report) and possible medical evaluation and treatment. Massage may cause further tissue damage and should not be carried out until local tissue has healed.

Burns**Definition and Overview**

Injuries that result from direct contact with or exposure to any thermal, chemical, electrical, or radiation source are termed *burns*. Burn injuries occur when energy from a heat source is transferred to the tissues of the body. The depth of injury is a function of temperature or source of energy (e.g., radiation) and duration of exposure.

The severity of burn injury is assessed with respect to the risk of infection, mortality, and cosmetic or functional disability.¹¹⁷ Factors that influence injury severity include burn depth, burn size (percentage of total body surface area [TBSA]), burn location, age, general health, and mechanism of injury. Burn depth can be divided into categories based on the elements of the skin that are damaged (Fig. 10-26). Most burn wounds that require medical intervention are a combination of partial- and full-thickness burns.

Burn size is determined by one of two techniques: the rule of nines (Fig. 10-27) and the Lund-Browder method (Figs. 10-28 and 10-29). The rule of nines is based on the division of the body into anatomic sections, each of which represents 9% or a multiple of 9% of the TBSA. This is an easy method to quickly assess the percentage of TBSA injured and is most commonly used in emergency departments where the initial evaluation takes place.

The Lund-Browder method modifies the percentages for body segments and provides a more accurate estimate

of burn size according to age. For the most accurate estimate of burn size, the burn diagram should be confirmed following the initial wound debridement.⁹⁰

Incidence

In the United States, approximately 1.4 to 2 million burn injuries occur each year; 70,000 people are hospitalized with severe injuries; and 7500 are fatalities. Extensive autografts are required in over 1500 third-degree burns every year and 40,000 second-degree burns.

Burn injuries are the third leading cause of accidental death in all age groups. Males tend to be injured more frequently than females, except for the older population (older than 70 years).¹¹⁸ The incidence of burn injuries is expected to increase as an aging society characterized by a striving for independence becomes more apparent.⁹⁴

Etiologic Factors

Burn injuries are categorized according to their mechanism of injury: thermal, chemical, electrical, or radiation. Thermal burns are caused by exposure to or contact with sources such as flames, hot liquids, steam, semisolids (tar), or hot objects.

Chemical burns are caused by tissue contact with or ingestion, inhalation, or injection of strong acids, alkalis, or organic compounds. Chemical burns can result from contact with certain household cleaning agents and various chemicals used in industry, agriculture, and the military.

Electrical burns are caused by heat that is generated by the electrical energy as it passes through the body. Electrical burns can result from contact with exposed or faulty electrical wiring, high-voltage power lines, or lightning.

Radiation burns are the least common burn injury and are caused by exposure to a radioactive source. These types of injuries have been associated with the use of ionizing radiation in industry or with therapeutic radiation sources in medicine. A sunburn from prolonged exposure to UV rays is also considered a type of radiation burn.

Risk Factors

Data collected from the National Burn Information Exchange indicate that 75% of all burn injuries result from the actions of the injured person, occurring most often in the home. Children under 3 years and adults over 70 years are at the highest risk for burn injury.

Risk factors include inadequate adult supervision (in the case of children), psychomotor disorders (e.g., impaired judgment, impaired mobility, drug or alcohol use), rural location, mobile home residence, occupation, lack of smoke detectors, fireworks, and misuse of cigarettes.^{27,94,95}

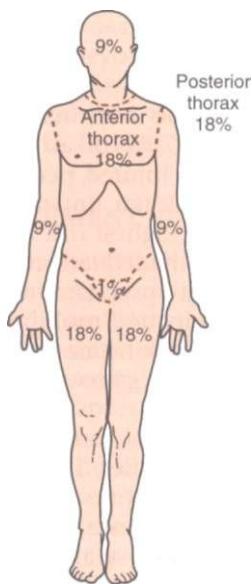
New data demonstrate a change in the epidemiology of burns from previous studies. These data point out the relationship between epileptic seizures and domestic scald injuries. Scald injuries are now the major cause of burns in people with epilepsy and account for approximately 2% of all burn admissions.⁷²

Safety recommendations for prevention of burn injuries while showering have been made, including nonlever water handles, limited-temperature devices on water

		CAUSE	APPEARANCE	SENSATION	COURSE
EPIDERMIS	SUPERFICIAL BURN First-degree burn	Sunburn Ultraviolet exposure Brief exposure to flash, flame, or hot liquids	Mild to severe erythema; skin blanches with pressure; dry, no blisters; edema variable amount	Painful Hyperesthetic Tingling Pain eased by cooling	Discomfort lasts about 48 hours Desquamation in 3-7 days
DERMIS	PARTIAL-THICKNESS BURN Second-degree burn	Superficial: Scalding liquids, semiliquids (oil, tar), or solids Deep: Immersion scald, flame	Large thick-walled blisters covering extensive area (vesiculation) Edema; mottled red base; broken epidermis; wet, shiny, weeping surface	Painful Sensitive to cold air	Superficial partial-thickness burn heals in 14-21 days Deep partial-thickness burn requires 21-28 days for healing Healing rate varies with burn depth and presence or absence of infection
SUBCUTANEOUS TISSUE	FULL-THICKNESS BURN Third-degree or fourth-degree burn	Prolonged exposure to: Chemical, electrical, flame, scalding liquids, steam	Variable (e.g., deep red, black, white, brown) Dry surface Edema Fat exposed Tissue disrupted	Little or no pain Insensate	Full-thickness dead skin suppures and liquefies after 2-3 weeks Spontaneous healing may be impossible but small areas may be left alone to form scarring without grafting (called secondary intent) Requires removal of eschar and subsequent split- or full-thickness skin grafting Hypertrophic scarring and wound contractures likely to develop without preventive measures

Figure 10-26

Burn injury classification according to depth of injury. This information is important to review because it will help determine the practice pattern to use when making a physical therapy diagnosis. A *partial-thickness* burn involves loss of epidermis and/or a portion of the dermis. Because part of the dermis is intact and that is where the regenerating elements are, a partial-thickness wound has the ability to heal via epithelialization. A *full-thickness* burn involves total destruction of the epidermis and dermis and cannot heal independently without granulation and contraction, sometimes requiring a flap or skin graft procedure.³⁸

**Figure 10-27**

The rule of nines provides a quick method for estimating the extent of a burn injury.

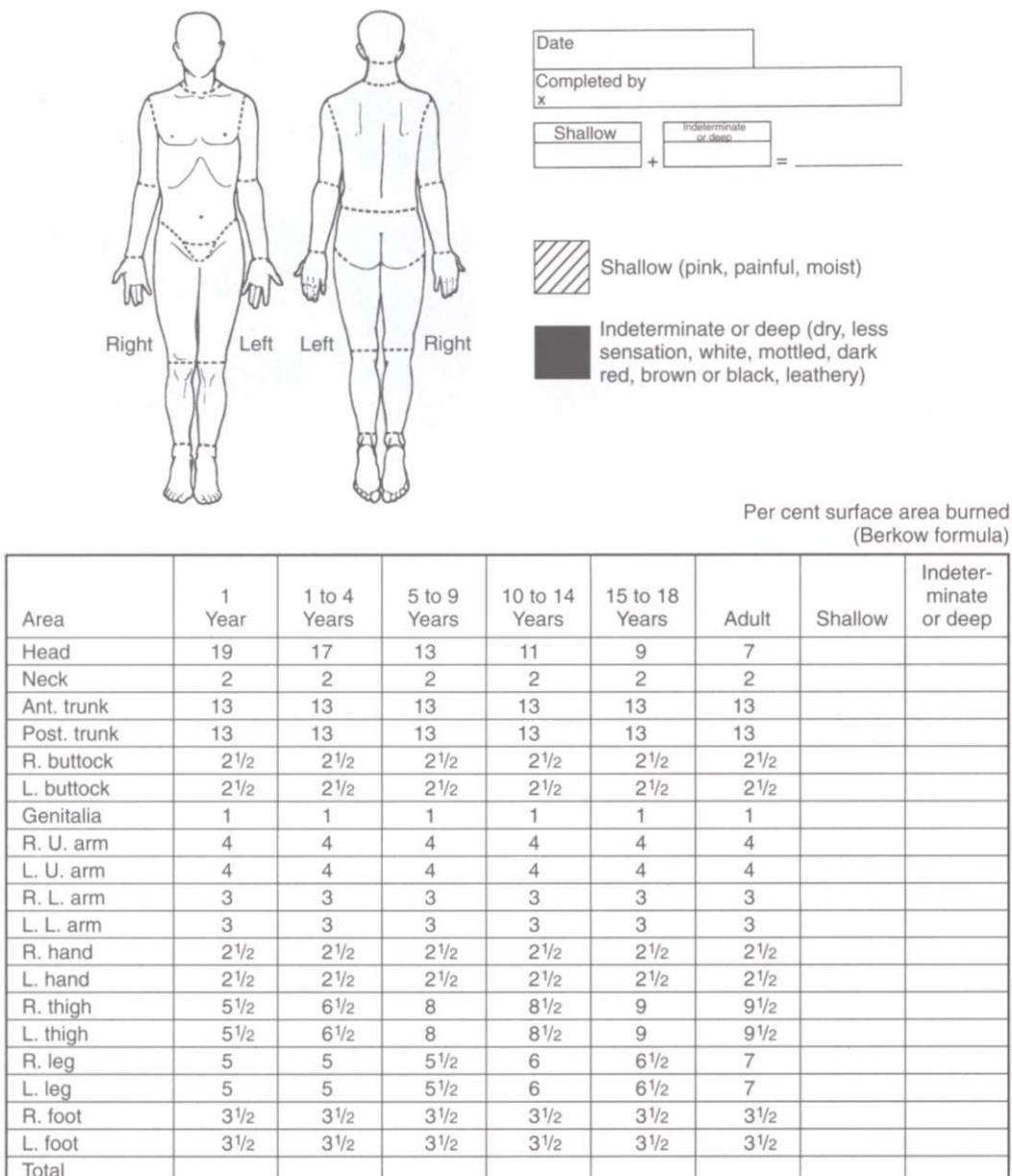
heaters, and curtains rather than cubicles for easy escape.¹⁵⁴

Pathogenesis

Cutaneous Burns. The pathophysiologic changes that occur immediately following a cutaneous burn injury depend on the extent or size of the burn. For smaller burns, the body's response to injury is localized to the injured area. With more extensive burns (25% or more of the TBSA), the response is systemic, potentially affecting all major systems of the body. The systems more obviously affected include the cardiovascular, renal, GI, immune, and respiratory systems.

Cardiovascular changes occur immediately following a burn injury as vasoactive substances (catecholamines, histamine, serotonin, leukotrienes, prostaglandins) are released from the injured tissue, causing an increase in capillary permeability.

Extensive burns result in generalized body edema in both burned and nonburned tissues and a decrease in circulating intravascular blood volume. Heart rate increases in response to catecholamine release and to the

**Figure 10-28**

A sample chart for recording the extent and depth of a burn injury using the Lund-Browder formula.

hypovolemia, but overall cardiac output falls. If the intravascular space is not replenished with IV fluids, hypovolemic (burn) shock and death may result.

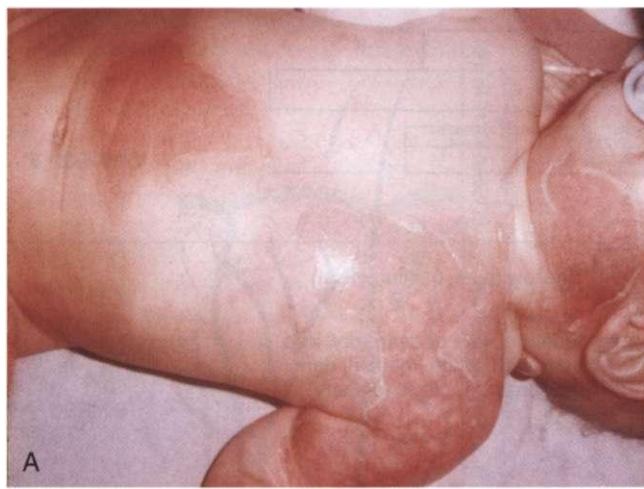
Within 18 to 36 hours after the burn, capillary permeability decreases and continues to return to normal for several weeks following the injury. Cardiac output returns to normal and then increases approximately 24 hours after the injury to meet the hypermetabolic needs of the body. The body begins to reabsorb the edema fluid and excretes the excess fluid over the ensuing days and weeks. See also the section on Common Causes of Fluid and Electrolyte Imbalances in Chapter 5.

The renal and GI systems are affected as the body responds initially by shunting blood from the kidneys and intestines, leading to oliguria (decreased urine

output) and intestinal dysfunction, respectively, in clients with burns of greater than 25% of TBSA. Immune system function is depressed, increasing the risk of infection and life-threatening sepsis. The respiratory system may respond with pulmonary artery hypertension and decreased lung compliance, even when there has been no inhalation injury.

Smoke Inhalation. Smoke inhalation may result in injury secondary to inhalation of carbon monoxide, smoke poisoning from the inhalation of by-products of combustion, or direct thermal burns to the pulmonary airways. See the section on Noxious Gases, Fumes, and Smoke Inhalation in Chapter 15.

Electrical and Chemical Burns. In electrical burns, heat is generated as the electricity travels through the



A

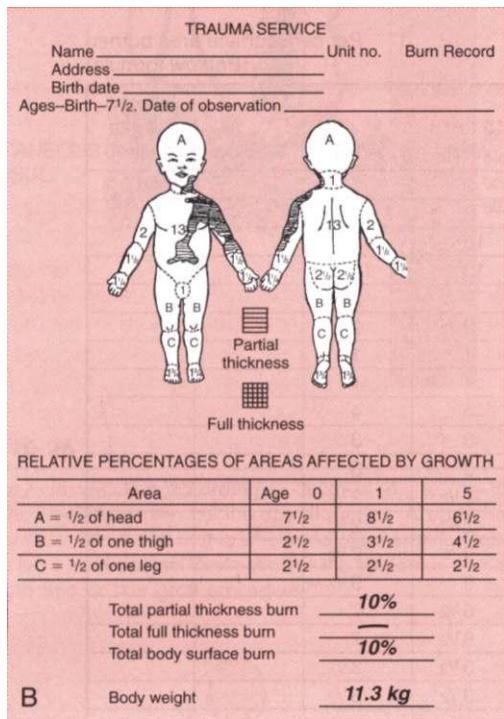


Figure 10-29

A, Pediatric scald burn. **B**, Corresponding Lund-Browder chart. (Courtesy Katherine S. Biggs, PT, Yale New Haven Hospital, New Haven, Conn.)

body, resulting in internal tissue damage. However, entrance and exit wounds may be significant, distracting medical personnel from internal injuries.

Cutaneous burn injuries associated with electrical burns may be negligible, but soft tissue and muscle damage may be extensive, particularly in high-voltage electrical injuries. However, it is possible for electrical sources to ignite the person's clothes, causing thermal burns as well.

The voltage, type of current (direct or alternating), contact site, and duration of contact are important factors in the amount and type of damage sustained. Alternating current is more dangerous than direct and is often associated with cardiopulmonary arrest, ventricular fibrillation, and tetanic muscle contractions.



Figure 10-30

Pediatric burn of the hand. This 2-year-old grabbed a hot iron, sustaining a partial-thickness burn to her hand. An intact blister remains on her middle finger. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

Other significant injuries, such as long-bone or vertebral compression fractures, spinal cord injury, or traumatic brain injury, can occur if the victim falls on electrical contact. Chemical burns are associated with systemic toxicity from cutaneous absorption. See also Chapter 4.

Clinical Manifestations

Appearance, sensation, and course of injury of superficial, partial-thickness, and full-thickness burns are outlined in Fig. 10-26. Burn location influences injury severity in that burns of certain areas of the body are commonly associated with specific complications. For example, burns of the head, neck, and chest frequently have associated pulmonary complications.

Burns involving the face may have associated corneal abrasions. Burns of the hands (Fig. 10-30) and joints can result in permanent physical and vocational disability requiring extensive therapy and rehabilitation. Circumferential burns of extremities may produce a tourniquet-like effect and lead to total occlusion of circulation (Fig. 10-31).

Theoretically, with a full-thickness burn the nerve endings have been destroyed and no pain should be associated with this type of injury. However, most full-thickness burns occur with superficial and partial-thickness burns in which nerve endings are intact and exposed. Excised eschar (dead tissue) and donor sites expose nerve fibers as well. As peripheral nerves regenerate, painful sensation returns. Consequently, people with burn injuries often experience severe pain that is related to the size and depth of the burn.

The clinical course of the (major) burn client can be divided into three phases: the emergent and resuscitation phase, the acute phase, and the rehabilitation phase. The *emergent period* begins at the time of injury and concludes with the restoration of capillary permeability, usually 48 to 72 hours following injury.



Figure 10-31

Circumferential burns of extremities may produce a tourniquet-like effect and lead to total occlusion of circulation. This 3-year-old child reached into the microwave oven and spilled macaroni in boiling water, resulting in a partial-thickness scald burn. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

The *resuscitation period* begins with initiation of fluid resuscitation measures and ends when capillary integrity returns to near-normal levels and the large fluid shifts have decreased. The *acute phase* of recovery begins when the person is hemodynamically stable, capillary permeability is restored, and diuresis has begun, usually 48 to 72 hours after the initial injury occurred. The acute phase continues until wound closure is achieved.

The *rehabilitation phase* represents the final phase of burn care, often overlaps the acute care phase, and lasts well beyond the period of hospitalization. This phase focuses on gaining independence through achievement of maximal functional recovery.

Infection is the most common and life-threatening complication of burn injuries. Burn wound infections can be classified on the basis of the causative organism, the depth of invasion, and the tissue response.

Individuals with extensive burns and in whom wound closure is difficult to achieve are at greatest risk for infection and other complications. Inhalation injury with major burns and added staphylococcal septicemia are often fatal.⁴⁷ The multiple organ system response that occurs following a burn injury may result in the multiple organ dysfunction syndrome and death (see Chapter 5).

Hypertrophic scarring is a second complication that although not life-threatening is associated with considerable morbidity and potential lifelong disfigurement. Children and African Americans are at greatest risk for hypertrophic scarring, presumably because of the abundance of collagen in these groups. Aging Caucasian adults with wrinkled, loose skin have little to no hypertrophic scarring because of the absence of collagen.

MEDICAL MANAGEMENT

TREATMENT. The therapist may be involved in wound care for minor burns consisting of cleansing; removal of any damaging agents (e.g., chemicals, tar); debridement of loose, nonviable tissue; and application of topical antimicrobial creams or ointment and a sterile dressing.

Blister management usually includes debridement of the blister (see the section on Skin Lesions: Special Implications for the Therapist 10-1). Although the blister fluid is theoretically sterile, most blisters break, and the fluid is an ideal medium for bacteria.¹³⁰

Instructions for home care include observation for clinical manifestations of infection and active ROM exercises to maintain normal joint function, decrease edema formation, and decrease possible scar formation.

Treatment of major burns includes lifesaving measures (ABCs: airway, breathing, circulation) immediately after the injury followed by restorative care (e.g., infection control, wound care, skin grafts, pain management) during the acute phase until wound closure is achieved. Therapists are closely involved early in the acute phase of recovery to maximize functional recovery and cosmetic outcome.

Therapeutic interventions include wound management—irrigation, debridement, advanced wound dressings, positioning and immobilization following skin grafting to prevent unwanted movement and shearing of grafts, scar and contracture prevention and management, exercise, ambulation, and ADLs. Elasticized garments help reduce scar hypertrophy and may be worn for months to 2 years after hospitalization.

Bioengineered temporary biologic dressings may be used to minimize fluid and protein loss from the burn surface, prevent infection, and reduce pain. Types of temporary grafts include *allografts* (homografts), which are usually cadaver skin; *xenografts* (heterografts), which are typically pigskin; and *biosynthetic grafts*, which are a combination of collagen and synthetics. To treat a full-thickness burn, an *autograft* (the person's own skin) may be required.

The transplanted skin graft will be used intact over areas where appearance or joint movement is important, but the graft may be meshed (fenestrated) to cover up to three times its original size. Several new permanent skin substitutes are being utilized to aid in replacing dermal thickness and to assist in coverage of large surface area injuries.¹¹⁵ Cultured skin is usually used in conjunction with allograft dermis. See the section on Skin Transplantation in Chapter 21.

PROGNOSIS. Burn care has improved in recent decades, resulting in a lower mortality rate for victims of burn injuries. Current techniques of burn wound management, such as effective topical antimicrobials and early burn wound excision, have significantly reduced the overall occurrence of invasive burn wound infections.¹⁷

The client's age affects the severity and outcome of the burn. Mortality rates are higher for children less than 4 years of age and for clients over 65 years, although survival rates after burns have improved significantly for children. At present most children, even children with large burns, should survive.¹³⁸ Survival rate for older clients is 70%, with at least 60% of those individuals becoming fully functional 6 months after hospital discharge.⁹⁴

Factors such as obesity, alcoholism, and cardiac disorders affecting general health, especially disorders that impair peripheral circulation, such as peripheral vascular

disease, increase the complication and mortality rates for adults with burns.

Delay in amputation results in prolonged hospital stay, delayed rehabilitation, and a higher mortality rate. Early amputation is associated with a 14% mortality rate compared with a 50% mortality rate for cases of delayed amputation. Earlier identification of nonsalvageable limbs may decrease infectious complications and improve chances of survival.¹⁶⁶

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-16

Burns

PREFERRED PRACTICE PATTERNS

6C: Impaired Ventilation, Respiration/Gas Exchange, and Aerobic Capacity/Endurance Associated with Airway Clearance Dysfunction

6E: Impaired Ventilation and Respiration/Gas Exchange Associated with Ventilatory Pump Dysfunction or Failure

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7C: Impaired Integumentary Integrity Associated with Partial-Thickness Skin Involvement and Scar Formation

7D: Impaired Integumentary Integrity Associated with Full-Thickness Skin Involvement and Scar Formation

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

In light of statistics showing that the population over 70 years old is at highest risk of burn injury, prevention of burn accidents, especially in this population, is an important part of client education. Additional resources for the therapist are available.^{23,108,125,161}

Reviewing simple cooking precautions may be helpful; for example, do not leave burners in use unattended, do not use high heat, do not wear clothing with loose sleeves or belts (especially bathrobes), use front burners whenever possible, and avoid leaning over front burners when using back burners.

Unless employed in an emergency department, most therapists do not begin to treat the burn client until the acute phase (as soon as the person is physiologically stable), continuing intervention through much of the rehabilitation phase. However, initiating bedside intervention before the person is medically stable is ideal in reducing morbidity and functional loss.

The therapist will direct treatment intervention to encourage deep breathing and facilitate lung expansion; promote wound healing; reduce dependent edema formation and promote venous return; prevent or minimize deformities and hypertrophic scarring; increase ROM, strength, and function; increase independence in daily activities and self-care; and encourage emotional and psychologic well-being. Specific compression, lymphatic movement, debridement, and wound care procedures are beyond the scope of this book; the reader is referred to other texts for more detailed information.^{23,63,93,125,147}

Table 10-6 Assessing Medical Complications in the Burn-Injured Adult

System	Complications
Urinary	Visible red or dark brown urine (catheter)
Respiratory	Signs of respiratory distress Restlessness Confusion Labored breathing Tachypnea (>24 respirations/min) Dyspnea $\text{PaO}_2 <90 \text{ mm Hg}$; $\text{O}_2 <95\%$
Peripheral vascular	Pulses absent on palpation Capillary refill (unburned area) >2 sec Numbness or tingling Increased pain with active range-of-motion exercises Increased edema, changes in skin color
Infection	Discoloration of wounds or drainage, odor, delayed healing Headache, chills, anorexia, nausea Increased pain Change in vital signs Paralytic ileus, confusion, restlessness, hallucinations
Gastrointestinal	Paralytic ileus (painful, distended abdomen) Stress-induced gastric ulcer (epigastric pain, abdominal distention, loss of appetite, nausea)

Throughout the acute and rehabilitation phases of burn care, the therapist must remain alert to the development of medical complications, such as ileus, gastric ulcers, respiratory distress, infection, and impaired circulation. Monitor vital signs (e.g., heart rate, blood pressure, oxygen saturation levels) to ensure the person can tolerate therapy. Notify the nursing staff of any new or unusual findings observed during assessment and intervention (Table 10-6).

Laboratory values will change with a burn, especially a full-thickness burn. It is important for the therapist to review the values prior to treatment interventions to be aware of response to treatment and possible mental status changes. Lab values listed in Table 10-7 should be reviewed.

Clients with burns with acute renal failure and abnormal sodium, potassium, chloride, and magnesium values are candidates for hemodialysis. Abnormal BUN (blood urea nitrogen) can be a reflection of decreased renal function or fluid intake. A client may respond to physical therapy treatment with a decreased mental status. Because of the increased metabolism and catabolic state, clients will experience increased weakness.

Wounds will not heal optimally without addressing protein status, reflected in prealbumin, which will be affected by the catabolic state. Glucose must be monitored due to the same metabolic situation.

Regular inspection of the wound must be made; any change in wound appearance must be reported. The amount of body surface area exposed during wound

Table 10-7 Laboratory Values Associated with Burns

	Normal	Optimal (Usually Midrange Values)	Burn
Sodium	135-146 mEq/L	140.5 mEq/L	Decreases (first 36 hr) Increases (36 hr-6 days) Depends on fluid resuscitation*
Potassium	3.5-5.5 mEq/L	4.5 mEq/L	Increases (first 36 hr) Decreases (36 hr-6 days)
Chloride	95-112 mEq/L	103 mEq/L	Decreases
Glucose	60-109 mg/dL	80-109 mg/dL	Increases
Blood urea nitrogen	7-25 mg/dL	16 mg/dL	Increases
Creatinine	0.8-1.1 mg/dL	1.05 mg/dL	Increases
Calcium	8.5-10.3 mEq/L	9.4 mEq/L	Decreases
Phosphorus	2.5-4.5 mEq/L adult 3.6 mEq/L child	3.5 mEq/L adult 4.5 mEq/L child	Decreases
Magnesium	2-3 mg/dL	2-3 mg/dL	Decreases
Prealbumin	17.0-40 g/dL	25-35 g/dL	Decreases

From Demling RH: The Burn Nutrition Module: what are standard clinical monitors used to assess adequate nutrition? Available on-line at <http://www.burnsurgery.com/Modules/burnnutrition/sec8.htm>. Accessed October 25, 2006.

Data from Ramos CG: Management of fluid and electrolyte disturbances in the burn patient, *Annals of Burns and Fire Disasters*, 8(4), 2000 [electronic version]. Available on-line at http://www.medbc.com/annals/review/vol_13/num_4/text/vol13n4p201.htm. Accessed October 25, 2006.

*Hyponatremia (sodium imbalance) is seen initially due to extracellular changes following changes in cellular permeability. Fluid replacement is initiated to manage both fluid and electrolyte imbalances; the use of a number of solutions can effectively manage this deficit. Hypernatremia can occur as a result of dehydration, which explains why some patients can have hyponatremia and then develop hypernatremia.

care must be minimized to prevent hypothermia, because heat is lost in open wounds and after hydrotherapy by evaporation. Hydrotherapy treatment must be limited to 30 minutes or less with water temperature in the 98° to 102° F (36.7° to 38.9° C) range if whirlpool is used. External heat shields or radiant heat lamps can provide a source of external heat.

Clients excluded from hydrotherapy are generally those who are hemodynamically unstable and those with new grafts. In recent years, hydrotherapy has been challenged, and alternative methods are being advocated (e.g., shower versus tub).⁸⁷ Pulsed lavage with suction (PLWS) (Fig. 10-32) is an ideal intervention for irrigation and debridement, allowing treatment in the Burn Unit of appropriate areas without disturbing new grafts.⁸⁷

People with burns are at high risk of infection because of the significant loss of skin barrier and impaired immune response. Infection control techniques must be practiced carefully at all times (see Appendix A). Skin donor sites require the same care and precautions as other partial-thickness wounds in order to promote healing and prevent infection.

Arrange any therapy likely to elicit a painful response to coincide with medications (allow 30 minutes for oral, 10 minutes for intramuscular [IM], 3 to 5 minutes for IV administration). Combining relaxation techniques, music therapy, distraction, and other techniques for pain modulation may be helpful. Burned areas must be maintained in positions of physiologic function within the limits imposed by associated injuries, grafting, and other therapeutic devices (see Table 10-8 for positioning recommendations).

Burned areas are prone to develop contractures requiring close assessment of ROM and muscle strength. Encourage active ROM exercises at least every



Figure 10-32

Personal protective equipment (PPE) worn during treatment with pulsed lavage with suction (PLWS). (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

2 hours while the person is awake unless this is contraindicated by a recent grafting procedure.

Prolonged stretching is sometimes combined with splinting or orthoses to maintain motion. Splinting is sometimes controversial due to the lack of evidence validation, although most clinicians employ splints successfully to prevent contractures.¹²⁶

Provide honest, positive reinforcement throughout intervention, being aware that each individual will progress through stages of denial, grief, and acceptance of injury and recovery. During the rehabilitation phase, chronic pain protocols may be helpful (see Box 3-15).

Table 10-8 Therapeutic Positioning for the Burn-Injured Client

Burned Area	Therapeutic Position	Positioning Techniques
Neck Anterior	Extension	No pillow; small towel roll beneath cervical spine to promote neck extension
Circumferential Posterior or asymmetric	Neutral toward extension Neutral	No pillow No pillow
Shoulder, axilla	Arm abduction to 90-110 degrees	Splinting; arms positioned away from body and supported on arm troughs; elbow splint
Elbow	Arm extension	Elbow splint; elbow(s) positioned in extension with slight bend at elbow (≤ 10 degrees of elbow flexion) Arms supported on arm troughs with the forearm in slight pronation
Hand		
Wrist	Wrist extension	Hand splint
Metacarpophalangeal (MCP) joints	MCP flexion at 90 degrees	Hand splint
Proximal or distal interphalangeal (PIP/DIP) joints	PIP/DIP extension	Hand splint
Thumb	Thumb abduction	Hand splint with thumb abduction
Web spaces	Finger abduction	Web spacers of gauze, foam, or thermoplastics to decrease webbing formation
Hip	Hip extension	Supine with the head of bed flat and legs extended Trochanter roll to maintain neutral rotation (toes pointing toward ceiling) Prone positioning
Knee	Knee extension	Supine with knees extended and toes pointing toward ceiling Prone with feet extended over end of mattress Sitting with legs extended and elevated Knee splint
Ankle	Neutral	Padded footboard Ankle positioning devices (avoid position of ankle inversion or eversion) Suspend heels (lying and sitting) to prevent pressure ulcer

Modified from Carrougher GJ: Nursing care of clients with burns. In Black JM, Matassarin-Jacobs E, eds: *Medical-surgical nursing*, ed 5, Philadelphia, 1997, Saunders, p 2260.

MISCELLANEOUS INTEGUMENTARY DISORDERS

Integumentary Ulcers

Integumentary ulcers can be caused by a variety of underlying disorders, including diabetes, arterial insufficiency, radiation damage, SSc, vasculitis, and prolonged pressure. In keeping with the focus of this text of recognizing the underlying pathology for various conditions, integumentary ulcers are discussed in individual sections according to the pathogenesis (e.g., diabetic ulcers, see the section on Diabetes Mellitus in Chapter 11; arterial insufficiency ulcers, see the section on Peripheral Vascular Disease in Chapter 12).

Pressure Ulcers

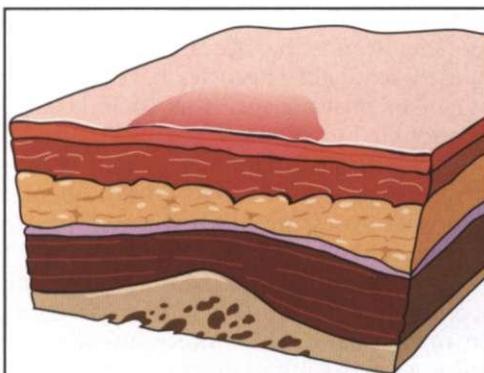
Definition and Overview

A pressure ulcer (formerly called *bed sore*, *decubitus ulcer*) is a lesion caused by unrelieved pressure resulting in damage to underlying tissue. Pressure ulcers usually occur over bony prominences, such as the heels, sacrum, ischial tuberosities, greater trochanters, elbows, and scapula, and

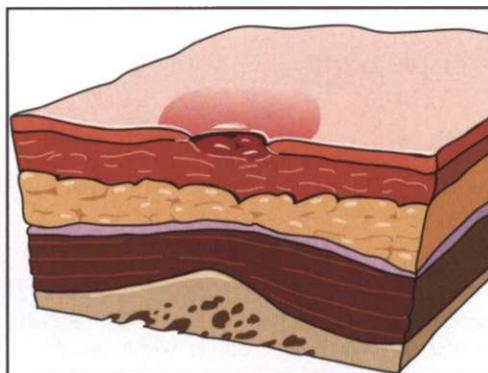
are staged to classify the degree of tissue damage observed (Fig. 10-33; Box 10-15).

In 1975 a landmark paper was published describing a method of classifying pressure ulcers, defined by the anatomic depth of soft tissue damage^{1,37a} (see Fig. 10-33). Since that time the original staging system has been modified and developed into the current staging system adopted by the Agency for Health Care Policy and Research (AHCPR) Pressure Ulcer Guideline Panels and published in both sets of Pressure Ulcer Clinical Practice Guidelines.^{9,65} The revised stage I definition adopted by the National Pressure Ulcer Advisory Panel (NPUAP) in 1998 that is more inclusive of the range of skin pigmentation is included in Box 10-15.

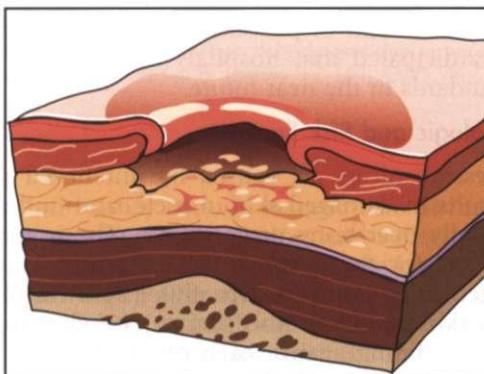
Wounds cannot be backstaged. Once a pressure ulcer is designated as stage II, III, or IV, it will always remain classified the same for documentation. As the lesion fills with granulation tissue and closes with epithelial tissue, grafts, or flaps, it should be documented as *healing* stage II, III, or IV (still using the original deepest level noted). Some agencies require backstaging for reimbursement purposes; hopefully this will be changed in the future so that no backstaging will exist in any form.



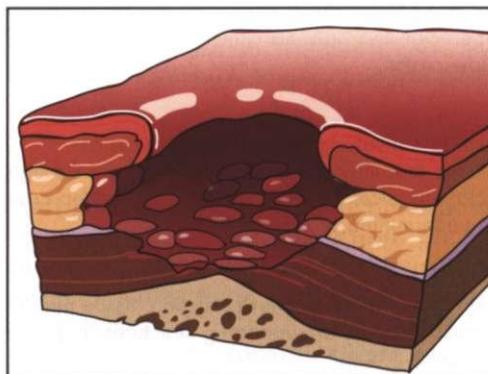
Stage I. Skin remains intact but with observable local changes in temperature (warmth or coolness), texture (firm or boggy feel), color (red in light skin; red, blue, or purple in darker skin), or sensation (pain or itching).



Stage II. Partial-thickness skin loss. The ulcer involves the epidermis, dermis, or both and is considered a partial-thickness skin loss. It is superficial and may look like an abrasion, blister, or shallow crater.



Step III. Full-thickness skin loss. The ulcer forms a deep crater. The adjacent tissue may be involved. There is damage to or necrosis of the subcutaneous tissue, which may extend down to the underlying fascia. The fascia is not affected.



Step IV. Full-thickness skin loss accompanied by tissue necrosis or damage to muscle, bone, or supporting structures, such as tendon or joint capsule. There is extensive tissue destruction; sinus tracts may be present.

Figure 10-33

Staging pressure ulcers is based on the depth and type of tissue damage. This system was developed by the National Pressure Ulcer Advisory Panel (NPUAP).

It should be noted that this staging classification is only for pressure ulcers. Other types of ulcers, such as vascular (arterial, venous) are designated partial or full thickness. Neuropathic ulcers (see Table 12-20) are staged using Wagner's classifications (Table 10-9). The term *neuropathic ulcer* is used interchangeably with *diabetic ulcer*, but a diabetic ulcer is really a neuropathic ulcer in someone with diabetes. Neuropathic ulcers can occur in anyone with loss of sensation (e.g., alcoholic neuropathy, peripheral neuropathy).

Incidence

Consistent data concerning incidence of pressure ulcers in the United States are difficult to find. Studies differ in a number of significant ways, and methodologies vary considerably.^{10,11} Given these limitations it is estimated that 1.8 million¹⁴ people develop pressure ulcers each year, including 500,000 people in nursing homes and another 400,000 people with diabetic foot ulcers.

Table 10-9 Wagner's Ulcer Grade Classification

Grade	Characteristics
0	Preulcerative lesions; healed ulcers; presence of bony deformity
1	Superficial ulcer without subcutaneous tissue involvement
2	Penetration through the subcutaneous tissue; may expose bone, tendon, ligament, or joint capsule
3	Osteitis, abscess, or osteomyelitis
4	Gangrene of digit
5	Gangrene of foot requiring disarticulation

This classification scheme for ulceration is used for neuropathic ulcers and does not represent pressure ulcers. It is included here for comparison with the stages of pressure ulcers (see Box 10-15).

From Wagner REW: The dysvascular foot: a system for diagnosis and treatment, *Foot Ankle* 2:64-122, 1981.

Box 10-15**STAGES OF PRESSURE ULCERS****Stage I***

An observable pressure-related alteration of intact skin that, when compared to an adjacent or opposite area on the body, shows changes in one or more of the following:

- Skin temperature (warmth or coolness)
- Tissue consistency (firm or boggy feel)
- Sensation (pain, itching)

The ulcer appears as a defined area of persistent redness in lightly pigmented skin, whereas in darker skin tones, the ulcer may appear with persistent red, blue, grey, or purple hues.

Note: Reactive hyperemia normally can be expected to be present for one half to three fourths as long as when the pressure-occluded blood flow to the area is restored; it should not be confused with a stage I pressure ulcer.

Stage II

Partial-thickness skin loss involving epidermis and/or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage III

Full-thickness skin loss involving damage or necrosis of subcutaneous tissue that may extend down to, but not through, underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of adjacent tissue.

Stage IV

Full-thickness skin loss with extensive destruction, tissue necrosis, or damage to muscle, bone, or supporting structures (e.g., tendon, joint capsule).

Note: Undermining and sinus tracts may also be associated with stage IV pressure ulcers.

Unstageable

The depth of a wound with eschar or more than 50% necrotic tissue cannot be determined until the necrotic tissue is debrided; it cannot be staged. Wounds that qualify as unstageable have eschar, are covered with necrotic tissue such that the base cannot be determined, or are filled with granulation tissue. These wounds are at least stage III or stage IV.

Proposed New Stage: Deep Tissue Injury

The National Pressure Ulcer Advisory Panel (NPUAP) has added a new stage (deep tissue injury, or DTI) for pressure ulcers with the skin intact, a purple color, and the appearance of a deep bruise (Fig. 10-34). It is a pressure-related injury to the subcutaneous tissues under intact skin. These lesions may herald the subsequent development of a stage III or IV pressure ulcer even with optimal treatment (NPUAP, 2006).

Data from U.S. Department of Health and Human Services: *Pressure ulcers in adults: prediction and prevention*. Clinical practice guideline no. 3. AHCPR publication no. 92-0047, Rockville, MD, 1992, The Department.

National Pressure Ulcer Advisory Panel: 2006. Updated staging system. Available on-line at www.npuap.org. Accessed April 19, 2008.

*Revised 1998 stage I pressure ulcer definition; the NPUAP has not changed the definitions of stages II to IV pressure ulcers.

Note: Wounds cannot be backstaged (see text for explanation).

Pressure ulcers are viewed as high-volume, high-risk problems in most health care settings. In long-term care facilities, regulatory agencies have designated the development of pressure ulcers as an indicator of quality of care provided to clients.⁴⁴

Healthy People 2010 has set as an objective to reduce the proportion of nursing home residents with current diagnosis of pressure ulcers from 16 per 1000 residents to no more than 9 per 1000 residents.

The target-setting method used by *Healthy People 2010* in conjunction with the NPUAP is based on a 50% reduction in prevalence and improvement over the baseline. *Incidence* refers to the rate at which new cases occur in a population over a given period of time, whereas *prevalence* refers to the number of both new and old cases at any one time in the population.

In 2005 the Centers for Medicare and Medicaid Services (CMS) issued new guidelines for nursing homes, giving surveyors specific criteria for inspections. Included is guidance for F Tag 314 for the prevention, assessment, and treatment of pressure ulcers in nursing homes.²⁶ It is anticipated that hospitals will be held to the same standards in the near future.

Etiologic and Risk Factors

Pressure ulcers are caused by unrelieved pressure that results in damaged skin, muscle, and underlying tissue, usually over bony prominences. The primary causative factors for the development of pressure ulcers are (1) interface pressure (externally); (2) friction (rubbing of the skin against another surface); (3) shearing forces (two layers sliding against each other in opposite directions causing damage to the underlying tissues); (4) maceration (softening caused by excessive moisture); (5) decreased skin resilience (e.g., dehydration); (6) malnutrition; and (7) decreased circulation.

Pressure contributes to other types of ulcers (e.g., arterial, venous, neuropathic), and likewise, the underlying cause of the other types of ulcers can contribute to the development of pressure ulcers (see Table 12-20). However, pressure ulcers are a separate entity from these other types of ulcers. A systemic risk assessment evaluating both sensation and physiologic risk of pressure ulcers can be made using a validated risk assessment tool, such as the Braden Scale (Table 10-10) or the Norton Scale (Table 10-11).

Intrinsic factors most commonly associated with pressure ulcer development include decreased sensation, impaired mobility or activity levels, incontinence, diaphoresis, impaired nutritional status, and altered levels of consciousness. Extrinsic factors include pressure, shear, friction, and moisture.

Bed- and chair-bound clients and those with impaired ability to reposition themselves should be assessed for additional factors that increase the risk of developing pressure ulcers. These factors include decreased mobility or immobility; hip or femoral fractures; contractures; increased muscle tone; loss of sensation; incontinence; obesity; nutritional factors; chronic disease accompanied by anemia, edema, renal failure, or sepsis; and altered level of consciousness.



Figure 10-34

Deep tissue injury (DTI). A DTI pressure ulcer presents suddenly as a discolored/bruised-appearing area that quickly progresses to a deep wound regardless of any intervention. It is an unavoidable ulcer and usually occurs when other organs are shutting down. Because the skin is the largest organ in the body, necrosis of the skin will occur when circulation is significantly impaired. **A**, The DTI was discovered when an unstable patient in the intensive care unit was finally able to be turned over after 2 days. He had kidney failure, was on a ventilator, and had tenuous blood pressure. The wound presented as a discolored area with intact skin (formerly required to be staged as a stage I pressure ulcer). **B**, One week later it was solid eschar, which required sharp debridement due to purulent drainage at the margins and was necrosed to bone. The patient expired several days later. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

Nutritional factors may include malnutrition or inadequate nutrition leading to weight loss and subsequent reduction of subcutaneous tissue and muscle bulk. The Agency for Health Care Policy and Research guidelines⁹ for clinically significant malnutrition impairing wound healing include serum albumin less than 3.5 mg/dl and total lymphocyte count less than 1800/mm³ (see Tables 40-5 and 10-11).

Prealbumin, which determines protein over the previous 48 hours, rather than over the previous 3 weeks as with albumin, is a better indicator of the current nutritional status of the client. Prealbumin should be more than 20 for optimal wound healing. It is considered the gold standard for monitoring nutritional progress, allowing for documentation and appropriate interventions.³¹

Pathogenesis

Pressure is the external factor causing ischemia and tissue necrosis. Continuous pressure on soft tissues between bony prominences and hard or unyielding surfaces compresses capillaries and occludes blood flow. Normal capillary blood pressure at the arterial end of the vascular bed averages 32 mm Hg.

When tissues are externally compressed, that pressure may be exceeded, reducing blood supply to, and lymphatic drainage of, the affected area.^{64,65} Shearing (when the skin layers move in opposite directions) is the intrinsic factor that contributes to ripping or tearing of blood vessels, further damaging the integument.

If the pressure is relieved, a brief period of rebound capillary dilation (called *reactive hyperemia*) occurs and no tissue damage develops. If the pressure is not relieved, the endothelial cells lining the capillaries become disrupted by platelet aggregation, forming microthrombi that occlude blood flow and cause anoxic necrosis of sur-

rounding tissues. Necrotic tissue predisposes to bacterial invasion and subsequent infection, preventing healthy granulation. Muscle and tendon tissue can tolerate less pressure loading than skin before incurring ischemic damage (Fig. 10-35).⁹¹

In the case of neuropathic ulcers associated with diabetes, the primary pathogenesis is the absence of protective sensation combined with high pressure. The absence of protective sensation indicates a high risk for pressure ulcers on the feet; diabetic ulcers are typically present on the soles of the feet (see the section on Diabetes Mellitus: Ulceration in Chapter 11).

Clinical Manifestations

Pressure ulcers usually occur over bony prominences and often in a circular pattern shaped like an inverted volcano with the greatest tissue ischemia at the apex next to the bone, or they may assume the shape of objects causing the pressure, such as tubing or clamps. Irregular patterns indicate additional shearing forces or other contributing factors.

Sacral ulcers are often large, undermined, and deep to the bone since the tissue mass over the sacrum is thin and erodes easily to the deep tissues. Pressure ulcers are manifested at the surface as the deeper tissues die, so that a stage I ulcer can become a stage III or IV quickly without further injury.

The wounds (Fig. 10-36) can be described, measured, and categorized with respect to surface area, exudates, and type of wound tissue. Therapists may want to utilize the PUSH Tool to assess and document pressure ulcers. This tool is available from the National Pressure Ulcer Advisory Panel (NPUAP): www.npuap.org. When present, infection can be localized and self-limiting or can progress to sepsis. Proteolytic enzymes from bacteria and

Table 10-10 The Braden Scale for Predicting Ulcer Risk

Sensory perception (ability to respond to discomfort)	1. Completely limited: Unresponsive to painful stimuli, either because of unconsciousness or severe sensory impairment, which limits ability to feel pain over most of body surface.	2. Very limited: Responds only to painful stimuli (but not verbal commands) by opening eyes or flexing extremities. Cannot communicate discomfort verbally OR has a sensory impairment that limits the ability to feel pain or discomfort over $\frac{1}{2}$ body surface.	3. Slightly limited: Responds to verbal commands by opening eyes and obeying some commands but cannot communicate discomfort or needs OR has some sensory impairment that limits ability to feel pain or discomfort in one or two extremities.	4. No impairment: Responds to verbal commands by obeying. Can communicate needs accurately. Has no sensory deficit that would limit ability to feel pain or discomfort.
Moisture (degree to which skin is exposed to moisture)	1. Very moist: Skin is kept moist almost constantly by perspiration and urine. Dampness is detected every time patient is moved or turned. Linen must be changed more than one time each shift.	2. Occasionally moist: Skin is frequently, but not always, kept moist; linen must be changed 2 or 3 times over 24 hr.	3. Rarely moist: Skin is rarely moist more than 3 or 4 times per week, but linen does require changing at that time.	4. Never moist: Perspiration and incontinence are never a problem; linen changed at routine intervals only.
Activity (degree of physical activity)	1. Bed-fast: Confined to bed.	2. Chair-fast: Ability to walk severely impaired or nonexistent and must be assisted into chair or wheelchair. Is confined to chair or wheelchair when not in bed.	3. Walks occasionally: Walks occasionally during day but for very short distances, with or without assistance. Spends majority of each shift in bed or chair.	4. Walks frequently: Walks a moderate distance at least once every 1-2 hr during waking hours.
Mobility (ability to change and control body position)	1. Completely immobile: Unable to make even slight changes in position without assistance.	2. Very limited: Makes occasional slight changes in position without help but unable to make frequent or significant changes in position independently.	3. Slightly limited: Makes frequent although slight changes in position without assistance but unable to make or maintain major changes in position independently.	4. No limitations: Makes major and frequent changes in position without assistance.
Nutrition (usual food intake pattern)	1. Very poor: Never eats a complete meal. Rarely eats more than $\frac{1}{3}$ of any food offered. Intake of protein is negligible. Takes even fluids poorly. Does not take a liquid dietary supplement OR is NPO or maintained on clear liquids or IV feeding for more than 5 days.	2. Probably inadequate: Rarely eats a complete meal and generally eats only about $\frac{1}{2}$ of any food offered. Protein intake is poor. Occasionally will take a liquid dietary supplement OR receiving less than optimum amount of liquid diet or tube feeding.	3. Adequate: Eats over $\frac{1}{2}$ of most meals. Eats moderate amount of protein source 1 or 2 times daily. Occasionally will refuse a meal. Will usually take a dietary supplement if offered OR is on tube feeding or TPN, which probably meets most nutritional needs.	4. Excellent: Eats most of every meal. Never refuses a meal. Frequently eats between meals. Does not require a dietary supplement.
Friction and shear	1. Problems: Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Either spasticity, contractures, or agitation leads to almost constant friction.	2. Potential problem: Moves freely independently or requires minimum assistance. Skin probably slides against bed sheets or chair to some extent when movement occurs. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. No apparent problem: Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.	

Copyright Barbara Braden and Nancy Bergstrom, 1988. Used with permission of Braden B, Frantz R: Selecting a tool to measure skin integrity. In

Stromberg M, ed: *Instruments for clinical nursing research*, ed 2, Norwalk, CT, 1997, Appleton and Lange.

Key: A score of 15 to 16 (15 to 18 if >75 yr) indicates minimum risk; 13 to 14, moderate risk; ≤12, high risk.

/IV, Intravenous; NPO, nothing by mouth; TPN, total parenteral nutrition.

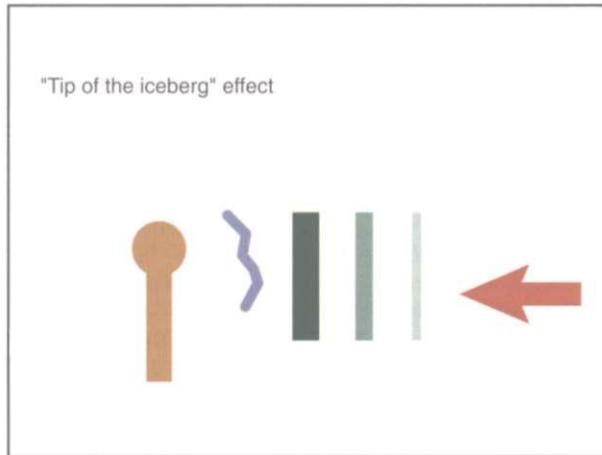
Table 10-11 Norton Scale

Name _____ Date _____		Physical Condition	Mental Condition	Activity	Mobility	Incontinent	Total Score
Good	Fair	Poor	Very bad	Alert	Apathetic	Confused	Stupor
4	3	2	1	4	3	2	1
Fair	Apathetic	Confused	Stupor	Walk/help	Slightly limited	Very limited	Immobile
Poor				Chairbound			
Very bad				Bed			

From Norton D, McLaren R, Exton-Smith AN: *An investigation of geriatric nursing problems in the hospital*, London, 1962, National Corporation for the Care of Old People (now the Centre for Policy on Ageing).

Key: The Norton Scale is a summated rating scale made up of five subscales scored from 1-4 (1 for low level of functioning and 4 for highest level functioning), for total scores that range from 5-20. The subscales measure functional capabilities of the person that contribute to their risk of developing pressure ulcers. A lower Norton Scale score indicates lower level of functioning and, therefore, higher level of risk for pressure ulcer development. A score of 5 to 14 rates the client "at risk."

TISSUE LOAD MANAGEMENT

**Figure 10-35**

Evolving stages of a stage IV pressure ulcer. This diagram represents a trochanter partially surrounded by tendon, with subcutaneous, dermis, and epidermis layers [vertical lines left to right]. As pressure occurs from the outside (arrow), the tendon becomes ischemic first, and then the subcutaneous layer is affected because it is less vascular than the dermis. The last tissue to become ischemic is the epidermis. The observer would initially identify the epidermal tissue change as a stage I pressure ulcer. Initially, the ischemic inner tissue layers would not be known. Only as the necrosis moves superficially will the full impact of tissue damage become observable, identifying the area as a stage IV pressure ulcer with extensive damage to the bone. (Courtesy Karen Kendall, PT, CWS, Medical Center for Continuing Education, Gulf Breeze, FL, 2000.)

macrophages dissolve necrotic tissues and cause a foul-smelling discharge that appears like, but is not, pus.

Necrosis associated with pressure ulcers is not painful, but the surrounding tissue is often painful in individuals who do not have loss of sensation from spinal cord trauma or neuropathy. Trauma to the tissues produces an acute inflammatory response with hyperemia, fever, and increased white blood cell count.

Many individuals never initiate a significant acute inflammatory response because of the heavy bioburden from large amounts of necrotic tissue, but develop an unresolved chronic inflammation. Individuals who are

**Figure 10-36**

Unstageable (due to eschar) trochanteric pressure ulcer. (Courtesy Harriett B. Loehne, PT, DPT, CWS, FCCWS, Archbold Center for Wound Management, Thomasville, GA, 2006. Used with permission.)

immunosuppressed or who have diabetes mellitus are often unable to mount a sufficient inflammatory response to start the healing cascade and thus are at greater risk for infection.

MEDICAL MANAGEMENT

DIAGNOSIS. Prevention is the key to this condition (Box 10-16), starting with assessment of people at high risk for the development of pressure ulcers. In fact, risk prediction should be an ongoing assessment carried out by all health care professionals. In addition to the Braden Scale (see Table 10-10) or Norton Scale (see Table 10-11), laboratory data on hemoglobin, hematocrit, prealbumin, total protein, and lymphocytes should be assessed by all health care professionals involved.

Box 10-16**GUIDELINES FOR PREVENTION OF PRESSURE ULCERS IN ADULTS**

- All clients at risk should have a systematic skin inspection at least once each day, paying particular attention to the bony prominences. Results of skin inspection should be documented (see Box 10-5).
- Clean skin at the time of soiling and at routine intervals. Individualize the frequency of skin cleaning according to need and client preference. Avoid hot water, and use a mild cleaning agent that minimizes irritation and dryness of the skin. During the cleaning or wound care process, minimize the force and friction applied to the skin. Ideal is the use of disposable perineal cloths, impregnated with dimethicone.
- Minimize environmental factors leading to skin drying, such as low humidity (<40%) and exposure to cold. Treat dry skin with moisturizers.
- Do NOT perform massage over reddened areas. Perform indirect soft tissue mobilization techniques or massaging the tissue around and toward the area with caution.
- Minimize skin injury caused by friction and shear forces through proper positioning, transferring, and turning techniques. Reduce friction injuries by the use of moisturizers, transparent film dressings, skin sealants, and protective padding.
- Maintain current activity level, mobility, and range of motion. Evaluate the potential for improving the person's mobility and activity status, and institute rehabilitation efforts.
- Monitor and document interventions and outcomes.
- If abnormal (<20 g/dl), order prealbumins one to two times weekly and have dietitian follow to optimize nutrition.
- Reposition any person in bed who is assessed to be at risk of developing pressure ulcers at least every 2 hours if consistent with overall treatment goals.
- For persons in bed, use positioning devices such as pillows or foam wedges to keep bony prominences (e.g., knees, ankles) from direct contact with one another.
- Provide persons in bed who are completely immobile with devices that completely relieve pressure on the heels (i.e., suspend the heels).
- Do not use doughnut-type devices.
- When side-lying position is used in bed, avoid positioning directly on the greater trochanter.
- Maintain the head of the bed at the lowest degree of elevation (30- to 35-degree lateral incline; see Fig. 10-38) consistent with medical conditions and other restrictions, except at mealtimes. Limit the amount of time the head of the bed is elevated.
- Use lifting devices, such as a trapeze, hydraulic lift, slide board, or linen, to move (rather than drag) persons in bed who cannot assist during transfers and position changes.
- Place any person assessed to be at risk of developing pressure ulcers, when lying in bed, on a pressure-redistribution surface, such as foam, air loss, gel, or water mattress.
- Avoid uninterrupted sitting in any chair or wheelchair for any person at risk of developing a pressure ulcer. Reposition the person, shifting the points under pressure at least every hour, or put him or her back to bed if consistent with overall management goals. Persons who are able should be taught to shift weight every 15 minutes.
- For chair-bound persons, use a pressure-redistribution device. Do not use doughnut-type devices.

Modified from Panel on the Prediction and Prevention of Pressure Ulcers in Adults: *Pressure ulcers in adults: prediction and prevention*. Clinical practice guidelines. CPR publication no. 92-0050, Rockville, MD, 1992, Agency for Health Care Policy and Research, Public Health Service.

The diagnosis is reached by looking at the location of the wound and the type of tissue response. The pressure ulcer is then staged (see Box 10-15 and Table 10-10). If there is evidence of infection, the wound is cleaned with isotonic saline and debrided if necrosis is present, and then viable tissue is cultured (not a swab specimen of the exudates or necrotic tissue).

The definition of infection is invasion into viable tissue. Cultures of the organisms that have invaded the tissue causing the infection must be determined following these procedures. Clinical practice of wound cultures must be careful to avoid culturing wound exudate contaminants, of which there are usually a minimum of three per wound.⁷⁶

Sensitivity testing to identify infecting organisms and to help determine appropriate topical or systemic antibiotics may be needed. The AHCPR (No. 15) recommends blood cultures for ulcer-related sepsis to determine appropriate systemic antibiotics.

TREATMENT. Prevention and removing the causative factor are the first step in the treatment intervention for pressure ulcers. Preventing shear and friction forces requires education of the client and primary caretakers. The pressure ulcer is cleansed thoroughly. Healing will occur optimally when the ulcer is kept moist.

Topical antibiotics (e.g., Polysporin, Neosporin, bacitracin, Bactroban, MetroGel) can be effective on local infections without systemic involvement to control bacterial concentration, being mindful of allergic reactions, especially to neomycin and bacitracin. Antiseptics are not recommended because these are cytotoxic.

Some physicians continue to advocate the initial use of wet-to-dry dressing for debridement (application of open wet dressing, allowing it to dry on the ulcer, and mechanically debriding exudate by removal of the dressing). Because there is a risk of removing viable tissue, damaging new granulation tissue, as well as bleeding with this procedure, it is not acceptable for debridement if any viable tissue is evident and should be used only rarely. Wet-dry is not permissible as a dressing change order—only for debridement.^{8,75}

The use of antiseptics such as hydrogen peroxide or povidone iodine (cadexomer iodine is an excellent antimicrobial dressing choice) is not recommended because these are cytotoxic and can be damaging to granulation tissue. Hyperbaric oxygen therapy (HOT) has not been approved for pressure ulcers except when osteomyelitis is present that has failed systemic antibiotic treatment or there are complications from a flap or graft.

Successful healing requires continued adequate redistribution of pressure (e.g., turning, positioning, support surfaces) and absence of infection. The presence of necrotic tissue in a wound may provide an optimal environment for bacteria to grow, hence the importance of removing necrotic material from a wound as rapidly as possible.

Therapeutic intervention may include hydrotherapy (e.g., PLWS; see Fig. 10-32), electrical stimulation, ultrasound, debridement (autolytic, enzymatic, mechanical, sharp), or any combination of these. An appropriate wound dressing is then applied to provide an optimal wound environment.

Large deep pressure ulcers may require sharp or surgical debridement of necrotic tissue and opening of deep pockets for drainage. A slower method of debridement is the use of proteolytic enzymes. A variety of skin-grafting techniques may be used if the wound requires surgical closure.

In stage III ulcers, undamaged tissue near the wound is rotated to cover the ulcer. In stage IV ulcers, musculoskeletal flaps (a single unit of skin with its underlying muscle and vasculature), as well as a variety of other skin-grafting techniques, may be used effectively to close the wound.

Bioactive human dermal tissue capable of interacting with the wound bed is now available commercially for use in pressure and neuropathic ulcer wound management.

These skin substitutes derived from living human tissue (human fibroblasts) represent an important advance in the treatment of burns and skin ulcers, including neuropathic foot ulcers, venous ulcers, and pressure ulcers. See the extensive section on Skin Transplantation in Chapter 21.

PROGNOSIS. Most clients have multiple complicating medical factors that contribute to poor wound closure. Each client responds differently to a course of therapy. Provided there is no infection, there is a good blood supply, the pressure has been eliminated or redistributed, and the client is not malnourished and has no medical complications, the wound should heal successfully. The presence of any of these factors alters the prognosis negatively.

SPECIAL IMPLICATIONS FOR THE THERAPIST 10-17

Pressure Ulcers

PREFERRED PRACTICE PATTERNS

7B: Impaired Integumentary Integrity Associated with Superficial Skin Involvement

7C: Impaired Integumentary Integrity Associated with Partial-Thickness Skin Involvement and Scar Formation

7D: Impaired Integumentary Integrity Associated with Full-Thickness Skin Involvement and Scar Formation

7E: Impaired Integumentary Integrity Associated with Skin Involvement Extending into Fascia, Muscle, or Bone and Scar Formation

The therapist plays a pivotal role in the prevention and management of pressure ulcers. Not only is the



Figure 10-37

Following the "rule of 30s" (head position at 30 degrees; see Fig. 10-38) and bed positioning should include side-lying at oblique angles, usually described as a 30- to 45-degree side-lying position to either side. (Courtesy National Pressure Ulcer Advisory Panel, 2000.)

therapist an expert in the delivery of therapeutic modalities, but also appropriate positioning, management of tissue load (mechanical factors acting on the tissues), and good mobility are essential to the success of the intervention.

High-risk clients should be identified using the Braden Scale or Norton Scale (see Tables 10-10 and 10-11), but all clients in the health care delivery system should be evaluated for risk levels and reassessed at least every 3 months for changes in status (or when there is a change in medical status).

Anyone with a history of previous pressure ulcer is considered high risk requiring a prevention protocol immediately. Acute care clients should be reassessed daily or at least weekly, on transfer to another unit/floor, and with changes in medical status.

The high-risk client will need frequent position changes, at least every 2 hours in bed, and at least every hour while sitting, every 15 minutes if the client can move himself or herself. Utilizing all turning surfaces, position the client at a 35-degree oblique angle when side lying (Fig. 10-37).

Elevate the head of the bed to no greater than 30 degrees when the client is supine (Fig. 10-38); if the head of the bed is elevated beyond 30 degrees (e.g., for eating, watching television, nursing care, or therapy intervention) the duration of this position needs to be limited to minimize both pressure and shear forces. A trapeze bar, turning sheet, or transfer board can be used to prevent shearing injury to the skin during movement or position change. Frequent shifting of body weight prevents ischemia by redistributing the weight and allowing blood to recirculate.

Static or dynamic pressure-redistribution devices using air, gel or water, foam, or other substances are commercially available, but the therapist must be aware that the material covering these devices can also create heat and friction contributing to pressure. Redistributing pressure on the skin must be accompanied by adequate fluid and nutrition intake (see also Box 10-16). Doughnut cushions should never be used, because they can cause tissue ischemia and new pressure ulcers.

Continued.

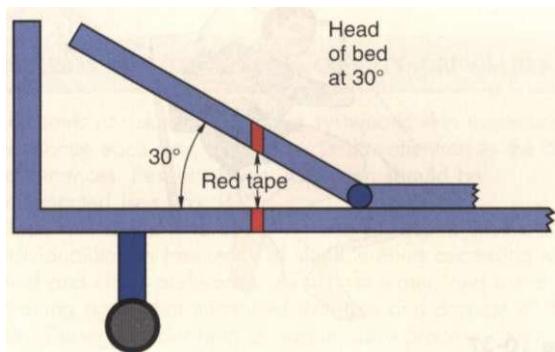


Figure 10-38

Head of bed at 30 degrees. Using a goniometer to determine a 30-degree angle, the therapist can mark the bed frame and raised headboard with colored tape to signify maximum inclination for pressure ulcer prevention. Client, family, and staff education and cooperation are vitally important and remain key intervention strategies. (Courtesy Anthony Stabler, Byron Health Center, Fort Wayne, IN. National Pressure Ulcer Advisory Panel, 2000.)

The person who is incontinent presents an additional challenge to keeping the skin clean and dry. Stool or urine becomes an irritant and places the person at additional risk for skin breakdown. Contamination of an already existing wound by wound drainage, perspiration, urine, or feces is also a concern for the incontinent and immobile population.

Fecal containment products are available for use in the case of acute diarrhea or fecal incontinence when these conditions contribute to the development of ulcers. New products for urinary or fecal incontinence are available to help prevent skin maceration from backflow of urine or feces. These include skin barriers, ointments, and fecal incontinence systems.

Cleaning should be carried out using a mild agent that minimizes irritation and dryness of the skin. Avoid harsh alkali soaps, alcohol-based products that can cause vasoconstriction, tincture of benzoin (may cause painful erosions), and hexachlorophene (may irritate the CNS). During cleaning or wound care, the force and friction applied to the skin should be minimized. Ideal is the use of a disposable, no-rinse, perineal cloth that is impregnated with a barrier ingredient.

Anyone performing PLWS (see Fig. 10-32) must be aware of the potential for aerosolization of microorganisms from a wound during this intervention. Therapists and others in the room during PLWS should wear appropriate personal protective equipment to limit contact with infectious agents. To prevent possible exposure of other clients, PLWS should be performed in a private room or in a treatment room with walls and doors that close, not curtains.⁸⁸

In 2004 and 2005 the FDA and CDC made recommendations for infection control that included the aforementioned plus cover any exposed supplies or client items in the room, cover any open areas not being treated (tubes, ports, etc.), consider masking the patient, observe Standard Precautions, dispose of disposables in appropriate waste stream, discard suction

canister or liner after each treatment, do not reuse single-use-only items, and after treatment disinfect thoroughly all environmental surfaces.^{45,92}

Concerns that high-pressure (output pressure of 70 psi) lavage may disseminate contaminants to surrounding tissues have not been substantiated. Until further research establishes safe levels of irrigation pressure in wound cleansing, therapists are advised to use the AHCPR guideline of irrigation pressures between 4 and 15 psi.⁸⁹ All wound management PLWS products provide a psi of 15 or less.

Pigmentary Disorders

Definition and Overview

Skin color or pigmentation is determined by the deposition of melanin, a dark polymer found in the skin, as well as in the hair, ciliary body, choroid of the eye, pigment layer of the retina, and certain nerve cells.

Melanin is formed in the melanocytes in the basal layer of the epidermis and is regulated (dispersion and aggregation) through the release of melatonin, a pineal hormone.

Hyperpigmentation is the abnormally increased pigmentation resulting from increased melanin production. *Hypopigmentation* is the abnormally decreased pigmentation resulting from decreased melanin production.

Pigmentary disorders (either hyperpigmentation or hypopigmentation) may be primary or secondary. Secondary pigmentary changes occur as a result of damage to the skin, such as irritation, allergy, infection, excoriation, burns, or dermatologic therapy, such as curettage, dermabrasion, chemical peels, or freezing with liquid nitrogen.

Etiologic and Risk Factors

The formation and deposition of melanin can be affected by external influences such as exposure to heat, trauma, solar or ionizing radiation, heavy metals, and changes in oxygen potential. These influences can result in hyperpigmentation, hypopigmentation, or both. Local trauma may destroy melanocytes temporarily or permanently, causing hypopigmentation, sometimes with hyperpigmentation in surrounding skin.

Other pigmentary disorders may occur from exposure to exogenous pigments, such as carotene, certain metals, and tattooing inks. Carotenemia occurs as a result of excessive carotene in the blood, usually from ingesting certain foods (e.g., carrots, yellow fruit, egg yolk). It may also occur in diabetes mellitus and in hypothyroidism. Exposure to metals such as silver can cause argyria, a poisoning marked by a permanent ashen grey discoloration of the skin, conjunctivae, and internal organs. Gold, when given long term for rheumatoid arthritis, can also cause pigmentary changes.

Clinical Manifestations

Hyperpigmentation. Primary disorders in the hyperpigmentation category include pigmented nevi, mongolian