:
Title: *Professional Guide to Diseases, 9th Edition*

14

Skin disorders

Introduction

Skin is the body's front-line protective barrier between internal structures and the external environment. It's tough, resilient, and virtually impermeable to aqueous solutions, bacteria, or toxic compounds. It also performs many vital functions. Skin protects against trauma, regulates body temperature, serves as an organ of excretion and sensation, and synthesizes vitamin D in the presence of ultraviolet light. Skin varies in thickness and other qualities from one part of the body to another, which often accounts for the distribution of skin diseases.

Skin has three primary layers: epidermis, dermis, and subcutaneous tissue. The epidermis (the outermost layer) produces keratin as its primary function. This layer is generally thin but is thicker in areas subject to constant pressure or friction, such as the soles and palms. The epidermis contains two sublayers: the stratum corneum, an outer horny layer of keratin that protects the body against harmful environmental substances and restricts water loss, and the cellular stratum, where keratin cells are synthesized. The basement membrane lies beneath the cellular stratum and serves to attach the epidermis to the dermis.

The cellular stratum, the deepest layer of the epidermis, consists of the basal layer, where mitosis takes place; the stratum spinosum, where cells begin to flatten, and fibrils—precursors of keratin—start to appear; and the stratum granulosum, made up of cells containing deeply staining granules of keratohyalin, which are generally thought to become the keratin that forms the stratum corneum. A skin cell moves from the basal layer of the cellular stratum to the stratum corneum in about 14 days. After another 14 days, normal wear and tear on the skin causes it to

slough off. The epidermis also contains melanocytes, which produce the melanin that gives the skin its color, and Langerhans cells, which are involved in a variety of immunologic reactions.

The dermis, the second primary layer of the skin, consists of two fibrous proteins, fibroblasts, and an intervening ground substance. The proteins are collagen, which strengthens the skin to prevent it from tearing, and elastin to give it resilience. The ground substance, which makes the skin soft and compressible, contains primarily jellylike mucopolysaccharides. Two distinct layers constitute the dermis: the papillary dermis (top layer) and the reticular dermis (bottom layer).

Subcutaneous tissue, the third primary layer of the skin, consists mainly of fat (containing mostly triglycerides), which provides heat, insulation, shock absorption, and a reserve of calories. Both sensory and motor nerves (autonomic fibers) are found in the dermis and the subcutaneous tissue.

Nails, glands, and hair

Nails are epidermal cells converted to hard keratin. The bed on which the nail rests is highly vascular, making the nail appear pink; the whitish, crescent-shaped area extending beyond the proximal nail fold, called the *lunula*—most visible in the thumbnail—marks the end of the matrix, the site of mitosis and of nail growth.

Sebaceous glands, found everywhere on the body (but mostly on the face and scalp) except the palms and soles, serve as appendages of the dermis. These glands generally excrete sebum into hair follicles, but in some cases, they empty directly onto the skin surface. Sebum is an oily substance that helps keep the skin and hair from drying out and prevents water and heat loss. Sebaceous glands abound on the scalp, forehead, cheeks, chin, back, and genitalia, and may be stimulated by sex hormones — primarily testosterone.

The dermis and subcutaneous tissue contain eccrine and apocrine glands, and hair. Eccrine sweat glands open directly onto the skin and regulate body temperature. Innervated by sympathetic nerves, these sweat glands are distributed throughout the body, except for the lips, ears, and parts of the genitalia. They secrete a hypertonic solution made up mostly of water and sodium chloride; the prime stimulus for eccrine gland secretion is heat. Other stimuli include muscular exertion and emotional stress.

Apocrine sweat glands appear chiefly in the axillae and genitalia; they're responsible

for producing body odor and are stimulated by emotional stress. The sweat produced is sterile but undergoes bacterial decomposition on the skin surface. These glands become functional after puberty. (Ceruminous glands, located in the external ear canal, appear to be modified sweat glands and secrete a waxy substance known as *cerumen*.)

Hair grows on most of the body, except for the palms, the soles, and parts of the genitalia. An individual hair consists of a shaft (a column of keratinized cells), a root (embedded in the dermis), the hair follicle (the root and its covering), and the hair papilla (a loop of capillaries at the base of the follicle). Mitosis at the base of the follicle causes the hair to grow; the papilla provides nourishment for mitosis. Small bundles of involuntary muscles known as *arrectores pilorum* cling to hair follicles. When these muscles contract, usually during moments of cold, fear, or shock, the hairs stand on end, and the person is said to have goose bumps or gooseflesh. Melanocytes in the matrix (inner core) of the hair bulb produce melanin, which passes into the innermost layers of the hair and is responsible for hair color. Dark hair contains mostly true melanin. Blond and red hair contains variants of melanin that have iron and more sulfur. Gray hair results from pigment loss due to a decline of tyrosinase, which is required for melanin synthesis. White hair occurs when air bubbles accumulate in the center of the hair shaft.

Vascular influence

The skin is served by a vast arteriovenous network, extending from subcutaneous tissue to the dermis. These blood vessels provide oxygen and nutrients to sensory nerves (which control touch, temperature, and pain), motor nerves (which control the activities of sweat glands, the arterioles, and smooth muscles of the skin), and skin appendages. Blood flow also influences skin coloring because the amount of oxygen carried to capillaries in the dermis can produce transient changes in color. For example, decreased oxygen supply can turn the skin pale or bluish; increased oxygen can turn it pink or ruddy.

Assessing skin disorders

Assessment begins with a thorough patient history to determine whether a skin disorder is an acute flare-up, a recurrent problem, or a chronic condition. Ask the patient how long he has had the disorder; how a typical flare-up or attack begins; whether or not it itches; and what medications — systemic or topical—have been used to treat it. Also, find out if any of his family members, friends, or contacts have the same disorder, and if he lives or works in an environment that could cause the condition. Also ask about hobbies.

When examining a patient with a skin disorder, be sure to look everywhere— mucous membranes, hair, scalp, axillae, groin, palms, soles, and nails. Note moisture, temperature, texture, thickness, mobility, edema, turgor, and any irregularities in skin color. Look for skin lesions; if you find a lesion, record its color, shape, size, and location. (See *Differentiating among skin lesions*.) Try to determine which is the primary lesion—the one that appeared first—which always starts in normal skin. The patient might be able to point it out.

If more than one lesion is in evidence, note the pattern of distribution. Lesions can be localized (isolated), regional, general, or universal (total), involving the entire skin, hair, and nails. Also, observe whether the lesions are unilateral or bilateral and symmetrical or asymmetrical; also note the arrangement of the lesions (clustered or linear configuration, for example).

Diagnostic aids

After simple observation, and examination of the affected area of the skin with a dermatoscope for morphologic detail, the following clinical diagnostic techniques may help to identify skin disorders:

- Biopsy determines histology of cells, and may be diagnostic, confirmatory, or inconclusive, depending on the disease.
- Diascopy, in which a lesion is covered with a microscopic slide or piece of clear plastic, helps determine whether dilated capillaries or extravasated blood is causing the redness of a lesion.
- Gram's stains and exudate cultures help identify the organism responsible for an underlying infection.
- Microscopic immunofluorescence identifies immunoglobulins and elastic tissue in detecting skin manifestations of immunologically mediated

disease.

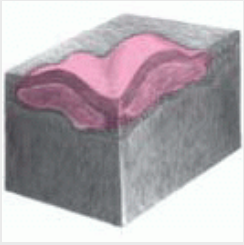
- Patch tests identify contact sensitivity (usually with dermatitis).
- Potassium hydroxide preparations permit examination for mycelia in fungal infections.
- Side-lighting shows minor elevations or depressions in lesions; it also helps determine the configuration and degree of eruption.
- Subdued lighting highlights the difference between normal skin and circumscribed lesions that are hypopigmented or hyperpigmented.
- Wood's light examination reveals yellow, green, or blue-green fluorescence when an area is infected with certain dermatophytes (fungi).

DIFFERENTIATING AMONG SKIN LESIONS

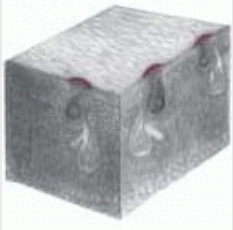
The illustrations below depict the most common primary and secondary skin lesions.

Primary Lesions

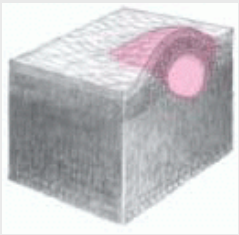
Bulla: Fluid-filled lesions more than 2 cm in diameter (also called blister, as occurs in severe poison oak or ivy dermatitis, bullous pemphigoid, and second-degree burns



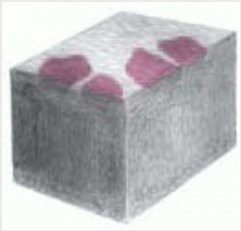
Comedo: Plugged, exfoliative pilosebaceous duct formed from sebum and keratin— for example, blackhead (open comedo) and whitehead (closed comedo)



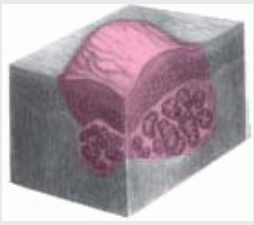
Cyst: Semisolid or fluid-filled encapsulated mass extending deep into dermis—for example, acne



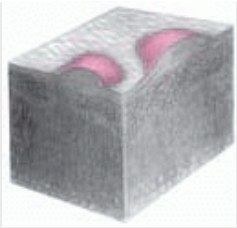
Macule: Flat, pigmented, circumscribed area less than 1 cm in diameter— for example, freckle or rash that occurs in rubella



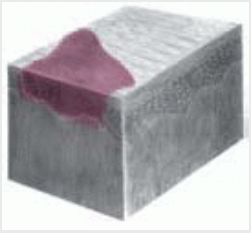
Nodule: Firm, raised lesion, 0.5 to 2 cm in diameter that's deeper than a papule and extends into dermal layer —for example, intradermal nevus



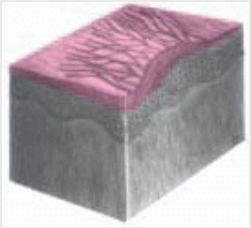
Papule: Firm, inflammatory raised lesion up to 0.5 cm in diameter that may be the same color as skin or pigmented —for example, acne papule and lichen planus



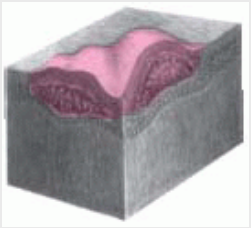
Patch: Flat, pigmented, circumscribed area more than 1 cm in diameter —for example, herald patch (pityriasis rosea)



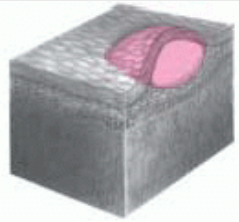
Plaque: Circumscribed, solid, elevated lesion more than 1 cm in diameter that is elevated above skin surface and that occupies larger surface area in comparison with height, as occurs in psoriasis



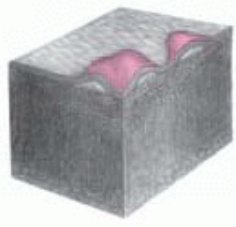
Pustule: Raised, circumscribed lesion, usually less than 1 cm diameter, containing purulent material, making it a yellow-white color— for example, acne or impetiginous pustule and furuncle



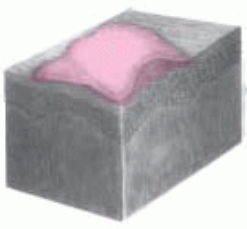
Tumor: Elevated, solid lesion larger than 2 cm in diameter that extends into dermal and subcutaneous layers— for example, dermatofi-broma



Vesicle: Raised, circumscribed, fluid-filled lesion less than 0.5 cm in diameter, as occurs in chickenpox or herpes simplex infection

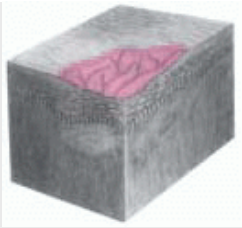


Wheal: Raised, firm lesion with intense localized skin edema that varies in size, shape, and color (from pale pink to red) and that disappears in hours— for example hives and insect bites

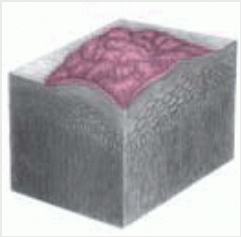


Secondary Lesions

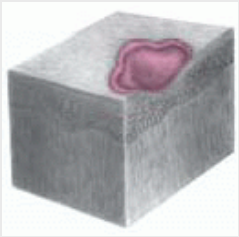
Atrophy: Thinning of skin surface at site of disorder—for example, striae and aging skin



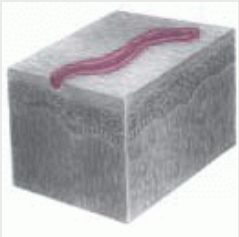
Crust: Dried sebum or serous, sanguineous, or purulent exudate, overlying an erosion or a weeping vesicle, bulla, or pustule, as occurs in impetigo



Erosion: Circumscribed lesion that involves loss of superficial epidermis—for example, abrasion

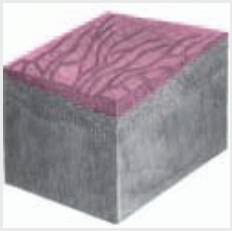


Excoriation: Linear scratched or abraded areas, often self induced —for example, abraded acne lesions or eczema

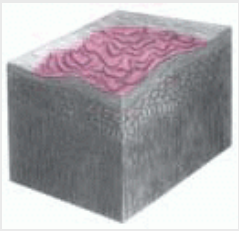


Fissure: Linear cracking of the skin that extends into the dermal layer—for example, hand dermatitis (chapped skin)

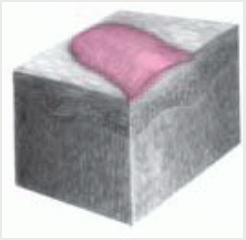
Lichenification: Thickened, prominent skin markings caused by constant rubbing, as occurs in chronic dermatitis



Scale: Thin, dry flakes of shedding skin, occurs in psoriasis, dry skin, or neonatal desquamation

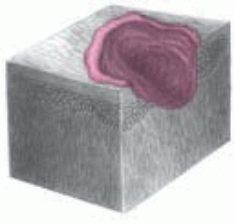


Scar: Fibrous tissue caused by trauma, deep inflammation, or surgical incision, which can be red and raised (recent) pink and flat (6 weeks), or pale and depressed (old)—for example, a healed surgical incision



Ulcer: Epidermal and dermal destruction that may extend into subcutaneous tissue and that usually heals with

scarring —for example, pressure ulcer or stasis ulcer



Special considerations

When assessing a skin disorder, keep in mind its distressing social and psychological implications. Unlike internal disorders, such as cardiac disease or diabetes mellitus, a skin condition is usually obvious and disfiguring. Understandably, the psychological implications are most acute when skin disorders affect the face—especially during adolescence, an emotionally turbulent time of life. However, such disorders can also create tremendous psychological problems for adults. A skin disease usually interferes with a person's ability to work because the condition affects the hands or because it distresses the patient to such an extent that he can't function.

For these reasons, be empathetic and accepting. Above all, don't be afraid to touch such a patient; most skin disorders aren't contagious. Touching the patient naturally and without hesitation helps show your acceptance of the dermatologic condition. Such acceptance is no less important than your patient-teaching about the disease and your guidance and help with carrying out prescribed treatment.

BACTERIAL INFECTIONS

Impetigo

A contagious, superficial skin infection, impetigo occurs in nonbullous and bullous forms. This vesiculopustular eruptive disorder spreads most easily among infants, young children, and elderly people. Predisposing factors, such as poor hygiene, anemia, malnutrition, and a warm climate, favor outbreaks of this infection, most of which occur during the late summer

and early fall. Impetigo can complicate chickenpox, eczema, or other skin conditions marked by open lesions.

Causes and incidence

Coagulase-positive *Staphylococcus aureus* and, less commonly, group A beta-hemolytic streptococci usually produce nonbullous impetigo; *S. aureus* (especially phage type 71) generally causes bullous impetigo.

In the United States, impetigo occurs most often in southern states. It often causes deeper dermal inflammation in blacks than in whites and may result in postin-flammatory hypopigmentation or hyperpigmentation.

Complications

- Ecthyma
- Glomerulonephritis
- Permanent scarring

Signs and symptoms

Common nonbullous impetigo typically begins with a small red macule that turns into a vesicle or pustule. When the vesicle breaks, a thick yellow crust forms from the exudate. (See *Recognizing impetigo*, page 736.) Autoinoculation may cause satellite lesions. Although it can occur anywhere, impetigo usually occurs around the mouth and nose and on the knees and elbows. Other features include pruritus, burning, and regional lymphadenopathy.

A rare but serious complication of streptococcal impetigo is glomerulonephritis, which is more likely to occur when many members of the same family have impetigo.

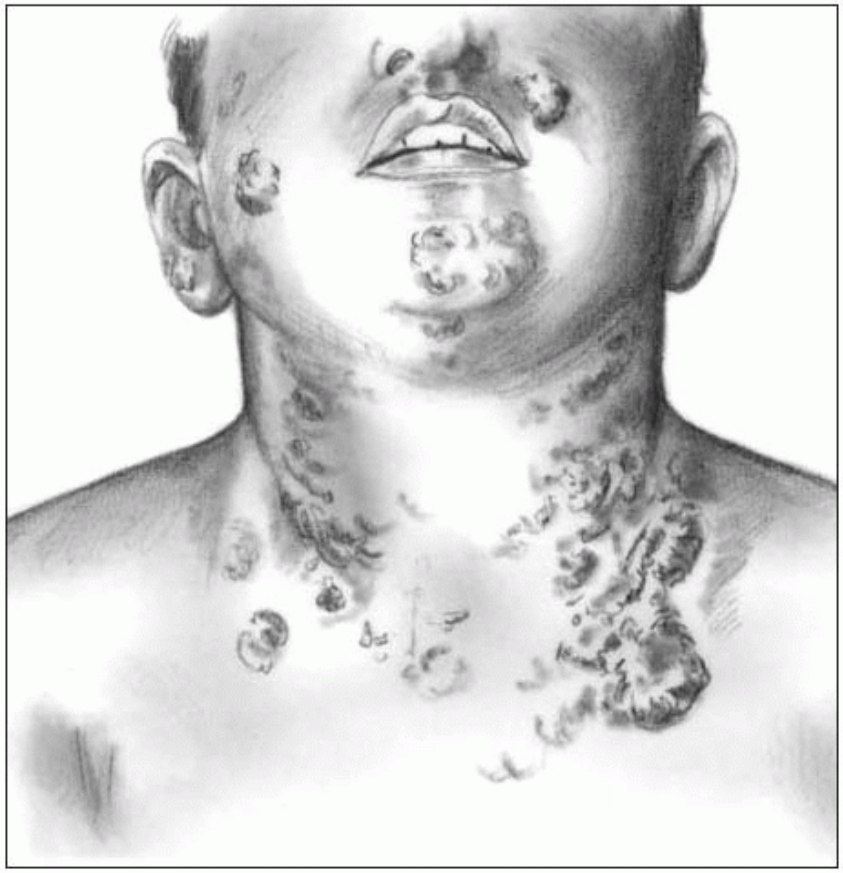
PEDIATRIC TIP

Infants and young children may develop aural impetigo or otitis externa; the lesions usually clear without treatment in 2 to 3 weeks, unless an underlying disorder such as eczema is present. Scarlet fever also may occur.

In bullous impetigo, a thin-walled vesicle opens, and a thin, clear crust forms from the exudate. The lesion consists of a central clearing, circumscribed by an outer ri—much like a ringworm lesion—and commonly appears on the face or other exposed areas. Both forms usually produce painless itching; they may appear simultaneously and be clinically indistinguishable.

RECOGNIZING IMPETIGO

In impetigo, when the vesicles break, crust forms from the exudate. This infection is especially contagious among young children.



Ecthyma is a skin infection that resembles impetigo but extends into the dermis and takes longer to resolve. These lesions are painful and more common on distal extremities. (See *Ecthyma*.)

Diagnosis

Culture and sensitivity testing of fluid or denuded skin may indicate the most appropriate antibiotic, but therapy shouldn't be delayed for laboratory results, which can take 3 days. White blood cell count may be elevated in the presence of infection.

Treatment

Topical mupirocin (Bactroban)and retapamulin (Altabax) are the treatments of choice if the lesions aren't too extensive. These drugs are highly effective against group A beta-hemolytic streptococci and *Staphylococcus aureus,* including methicillin-resistant *S. aureus.* Mupirocin also eliminates nasal carriers of these organisms. Extensive or nonresolving lesions require systemic antibiotics.

Therapy may also include removal of the exudate by washing the lesions two or three times a day with soap and water (or antibacterial soap) or, for stubborn crusts, warm soaks or compresses of normal saline or a diluted soap solution.

Special considerations

- Urge the patient not to scratch, because this spreads impetigo.

PEDIATRIC TIP

Advise parents to cut their child's fingernails and cover his hands with socks or mittens to prevent scratching.

- Give medications as indicated. Remember to check for medication allergy. Stress the need to continue prescribed medications for 7 to 10 days, even after lesions have healed.
- Teach the patient or his family how to care for impetiginous lesions.

ECTHYMA

Ecthyma is a superficial skin infection that usually causes scarring. It commonly occurs in persons with poor hygiene or living in crowded conditions and generally results from

infection by *Staphylococcus aureus* or group A beta-hemolytic streptococci. Ecthyma differs from impetigo in that its characteristic ulcer results from deeper penetration of the skin by the infecting organism (involving the lower epidermis and dermis), and the overlying crust tends to be piled high (1 to 3 cm). These lesions are usually found on the legs after a scratch or bug bite. Autoinoculation can transmit ecthyma to other parts of the body, especially to sites that have been scratched open. Therapy is basically the same as for impetigo, but response may be slower. Parenteral antibiotics (usually a penicillinase-resistant penicillin) are also used.

PREVENTION

- *To prevent further spread of this highly contagious infection, encourage frequent bathing using a bactericidal soap. Tell the patient not to share towels, washcloths, or bed linens with family members. Emphasize the importance of following proper hand-washing technique.*
- *Check family members for impetigo. If this infection is present in a school-age child, notify his school.*

Folliculitis, furunculosis, and carbunculosis

Folliculitis is a bacterial infection of the hair follicle that causes the formation of a pustule. The infection can be superficial (follicular impetigo or Bockhart's impetigo) or deep (sycosis barbae). Folliculitis may also lead to the development of furuncles (furunculosis), commonly known as *boils*, or *carbuncles* (carbunculosis), which involve multiple contiguous hair follicles. The prognosis depends on the severity of the infection and on the patient's physical condition and ability to resist infection.

Causes and incidence

The most common cause of folliculitis, furunculosis, or carbunculosis is coagulase-positive *Staphylococcus aureus*. Predisposing factors include an infected wound, poor hygiene, debilitation, diabetes, alcoholism, occlusive cosmetics, tight clothes, friction, chafing, exposure to chemicals, and treatment for skin lesions with tar or with occlusive therapy, using steroids. Furunculosis often follows folliculitis exacerbated by irritation, pressure, friction, or perspiration. Carbunculosis follows persistent *S. aureus* infection and furunculosis.

Complications

- Cellulitis
- Septicemia
- Scarring

Signs and symptoms

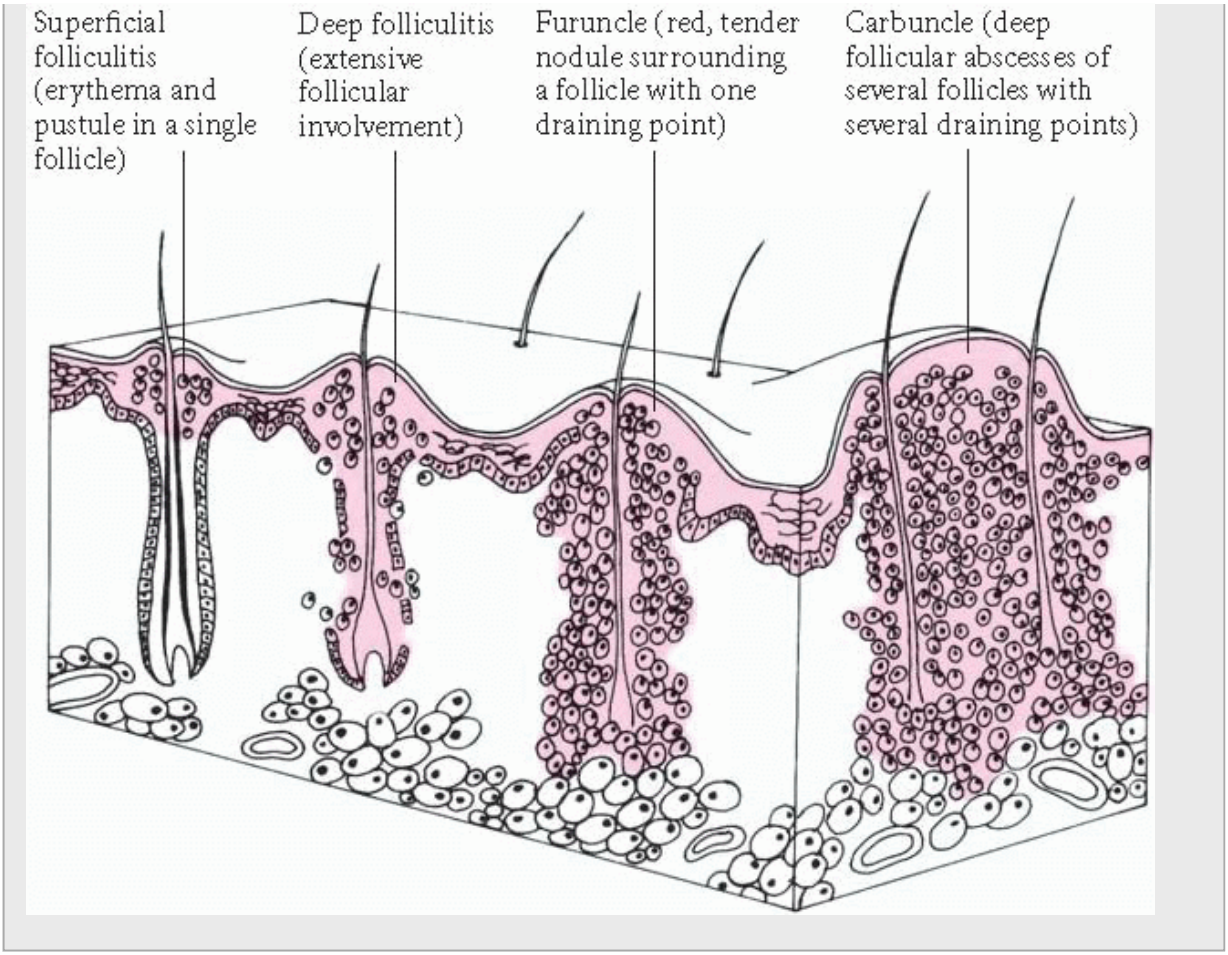
Pustules of folliculitis usually appear in a hair follicle on the scalp, arms, and legs in children; on the face of bearded men (sycosis barbae); and on the eyelids (styes). Deep folliculitis may be painful.

Folliculitis may progress to the hard, painful nodules of furunculosis, which commonly develop on the neck, face, axillae, and buttocks. For several days these nodules enlarge, and then rupture, discharging pus and necrotic material. After the nodules rupture, pain subsides, but erythema and edema may persist for days or weeks.

Carbunculosis is marked by extremely painful, deep abscesses that drain through multiple openings onto the skin surface, usually around several hair follicles. Fever and malaise may accompany these lesions. (See *Follicular skin infections*, page 738.)

FOLLICULAR SKIN INFECTIONS

Degree of hair follicle involvement in bacterial skin infection ranges from superficial erythema and pustule of a single follicle to deep abscesses (carbuncles) involving several follicles.



Diagnosis

CONFIRMING DIAGNOSIS

The obvious skin lesion confirms folliculitis, furunculosis, or carbunculosis. Wound culture shows S. aureus; *sensitivity will help guide antibiotic therapy.*

In carbunculosis, patient history reveals preexistent furunculosis. A complete blood count may reveal an elevated white blood cell count (leukocytosis).

Treatment

Treatment for folliculitis consists of cleaning the infected area thoroughly with antibacterial soap and water; applying warm, wet compresses to promote vasodilation and drainage from the lesions; topical antibiotics such as mupirocin ointment and, in extensive infection or if a furuncle or

carbuncle has developed, systemic antibiotics. Use sensitivity results to guide therapy, but begin treatment before receiving results.

Furunculosis and carbunculosis may also require incision and drainage of ripe lesions if the lesions don't drain after the application of warm, wet compresses. They may also require topical antibiotics after drainage.

Special considerations

Care for patients with folliculitis, furunculosis, and carbunculosis is basically supportive and emphasizes teaching the patient scrupulous personal and family hygiene measures. Taking the necessary precautions to prevent spreading infection is also an important part of care.

- Caution the patient never to squeeze a boil because this may cause it to rupture into the surrounding area.
- Advise the patient with recurrent furunculosis to have a physical examination because an underlying disease, such as diabetes or human immunodeficiency virus, may be present.

In blacks, trauma resulting from such hairstyles as cornrowing (gathering the hair into tight braids or tufts) can cause folliculitis.

PREVENTION

To avoid spreading bacteria to family members, urge the patient not to share towels and washcloths. Tell him that these items should be laundered in hot water before being reused. The patient should change his clothes and bedsheets daily, and these also should be washed in hot water. Encourage the patient to change dressings frequently and to discard them promptly in paper bags.

Staphylococcal scalded skin syndrome

Staphylococcal scalded skin syndrome (SSSS), also known as *Ritter's disease* or *Ritter-Lyell syndrome,* is marked by epidermal erythema, peeling, and necrosis that give the skin a scalded appearance. This severe skin disorder follows a consistent pattern of progression, and most patients recover fully. Mortality is 2% to 3%.

Causes and incidence

The causative organism in SSSS is group 2 *Staphylococcus aureus*, primarily phage type 71, which produces exotoxins that cause detachment of the epidermis. Predisposing factors may include impaired immunity and renal insufficiency—present to some extent in the normal neonate because of immature development of these systems.

SSSS is most prevalent in infants age 1 to 3 months but may develop in children. It's uncommon in adults.

Complications

- Fluid and electrolyte loss
- Sepsis

Signs and symptoms

SSSS can usually be traced to a prodromal upper respiratory tract infection, possibly with concomitant purulent conjunctivitis. Cutaneous changes progress through three stages:

- Erythema: Erythema, which may begin diffusely or as a scarlatiniform rash, usually becomes visible around the mouth and other orifices and may spread in widening circles over the entire body surface. The skin becomes tender; Nikolsky's sign (sloughing of the skin when friction is applied) may appear.
- Exfoliation (24 to 48 hours later): In the more common, localized form of this disease, superficial erosions with a red, moist base and minimal crusting occur, generally around body orifices, and may spread to exposed areas of the skin. (See *Identifying staphylococcal scalded skin syndrome*, page 740.) In the more severe forms of this disease, large, flaccid bullae erupt and may spread to cover extensive areas of the body. These bullae eventually rupture, revealing sections of denuded skin; mucous membranes are spared.
- Desquamation: In this final stage, affected areas dry up, and powdery scales form. Normal skin replaces these scales in 5 to 7 days.

Diagnosis

Diagnosis requires careful observation of the three-stage progression of this disease. Results of exfoliative cytology and biopsy aid in differential diagnosis, ruling out erythema multiforme and drug-induced toxic epidermal necrolysis, both of which are similar to SSSS.

CONFIRMING DIAGNOSIS

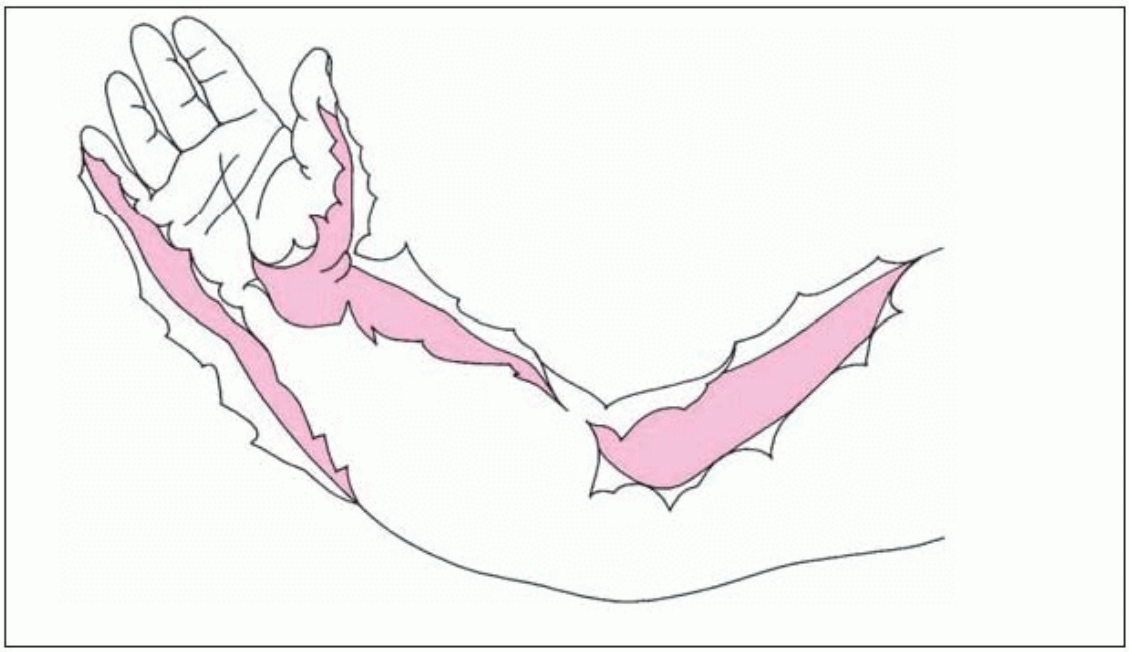
Isolation of group 2 S. aureus *on cultures of skin lesions confirms the diagnosis. However, skin lesions sometimes appear sterile.*

Treatment

Treatment includes systemic antibiotics, usually penicillinase-resistant penicillin. Severe cases require hospitalization and I.V. antibiotics. Oral antibiotics should be adequate for milder cases. Skin lubrication with a non-alcohol-based preparation is beneficial. Washing or bathing should be done sparingly. Replacement measures to maintain fluid and electrolyte balance are necessary.

IDENTIFYING STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Staphylococcal scalded skin syndrome is a severe skin disorder that commonly affects infants and children. The illustration below shows the typical scalded skin appearance, with areas of denuded skin found in an infant.



PEDIATRIC TIP

Hospital admission is appropriate for neonates and young children with extensive sloughing.

Special considerations

- Carefully monitor intake and output to assess fluid and electrolyte balance. In severe cases, I.V. fluid replacement may be necessary.
- Check vital signs. Be especially alert for a sudden rise in temperature, indicating sepsis, which requires prompt, aggressive treatment.
- Maintain skin integrity. Use strict sterile technique to preclude secondary infection, especially during the exfoliative stage, because of open lesions. To prevent friction and sloughing of the skin, leave affected areas uncovered or loosely covered. Place cotton between severely affected fingers and toes to prevent webbing.
- Gently débride exfoliated areas, especially those that have become necrotic.
- Reassure the parents that complications are rare and residual scars are unlikely.

PEDIATRIC TIP

Provide special care for the neonate, if required, including placement in a warming infant incubator to maintain body temperature and provide isolation.

FUNGAL INFECTIONS

Tinea versicolor

A chronic, superficial, fungal infection, tinea versicolor (also known as *pityriasis versicolor*) may produce a multicolored rash, commonly on the upper trunk. Recurrence is common.

Causes and incidence

The agent that causes tinea versicolor is *Pityrosporum orbiculare,* also known as *P. ovale* and *Malassezia furfur.* Whether this condition is infectious or merely a proliferation of normal skin fungi is uncertain. Tinea versicolor is more common in hot

climates—tropical countries or in those with high humidity—and associated with increased sweating. It usually affects adolescents and young men when sebaceous gland activity is at its highest.

Complication

- Secondary bacterial infections

Signs and symptoms

Tinea versicolor typically produces raised or macular, round or oval, slightly scaly lesions on the upper trunk, which may extend to the lower abdomen, neck, arms, groin, thigh, genitalia and, rarely, the face. These lesions are usually tawny but may range from hypopigmented (white) patches in dark-skinned patients to hyperpigmented (brown) patches in fair-skinned patients. Some areas don't tan when exposed to sunlight, causing the cosmetic defect for which most people seek medical help. Inflammation, burning, and itching are possible but usually absent.

Diagnosis

Visualization of blue-green fluorescent lesions during Wood's light examination strongly suggests tinea versicolor. However, if the patient has recently showered, this fluorescence may not show because the chemical that causes fluorescence is water-soluble.

CONFIRMING DIAGNOSIS

Microscopic examination of skin scrapings prepared in potassium hydroxide solution confirms the disorder by showing hyphae, clusters of yeast, and large numbers of variously sized spores (a combination referred to as "spaghetti and meatballs").

Treatment

The most economical and effective treatment is selenium sulfide lotion 2.5% applied once a day for seven days. It's left on the skin for 10 minutes, then rinsed off thoroughly. In persistent cases, therapy may require a single 12- or 24-hour application of this lotion, repeated once a week for 4 weeks. Either treatment may cause temporary redness and irritation.

Other treatments include sodium thiosulfate 25% solution, applied twice daily to affected areas for 2 to 4 weeks; sulfur salicylic shampoo applied as a lotion at bedtime each night and washed off each morning for 2 weeks; zinc pyrithione shampoo 1% lathered into affected areas for 5 minutes before showering, and repeated every day for 2 weeks; or imidazole antifungal agents applied twice daily for 2 weeks.

Ketoconazole and other azole-based creams, such as topical ketoconazole, may be applied once or twice daily for 2 weeks. Oral ketoconazole or another oral azole-based medication, such as oral ketoconazole, may be used if the patient has extensive disease that fails to respond to other therapies.

Special considerations

- Instruct the patient to apply selenium sulfide lotion as ordered. Tell him that this medication may cause temporary adverse effects.
- Assure the patient that once his fungal infection is cured, discolored areas will gradually blend in after exposure to the sun or ultraviolet

light.

- Because recurrence of tinea versicolor is common, advise the patient to watch for new areas of discoloration.
- Teach the patient proper hand-washing technique, and encourage good personal hygiene.
- Provide written instructions for using prescribed medications. Tell the patient to contact the physician if adverse reactions occur.

PREVENTION

- *Stress the importance of not scratching or picking lesions to avoid the risk of skin breaks and secondary bacterial infections.*
- *Encourage the patient to avoid overexposure to heat and humidity.*

Dermatophytosis

Dermatophytosis, commonly called *tinea,* may affect the scalp (tinea capitis), body (tinea corporis), nails (tinea unguium), hands (tinea manuum), feet (tinea pedis), groin (tinea cruris), and bearded skin (tinea barbae). With effective treatment, the cure rate is very high, although about

20% of infected people develop chronic conditions.

Causes and incidence

Tinea infections (except for tinea versicolor) result from dermatophytes (fungi) of the genera *Trichophyton, Microsporum,* and *Epidermophyton.* Transmission can occur through contact with infected lesions, household cats and dogs, and soiled or contaminated articles, such as shoes, towels, or shower stalls.

Tinea infections are prevalent in the United States. They're more common in males than in females.

Complications

- Hair or nail loss
- Secondary bacterial or candidal infections

Signs and symptoms

Lesions vary in appearance depending on the site of invasion (inside or outside the hair shaft), duration of infection, level of host resistance, and amount of inflammatory response. Tinea capitis ranges in appearance from broken-off hairs with little scaling to severe painful, inflammatory, pus-filled masses (kerions) covering the entire scalp. Partial hair loss occurs in all cases. The cardinal clue is broken-off hairs.

Tinea corporis produces flat lesions on the skin at any site except the scalp, bearded skin, hands, or feet. These lesions may be dry and scaly or moist and crusty; as they enlarge, their centers heal, causing the classic ring-shaped appearance. In tinea unguium (onychomycosis), infection typically starts at the tip of one or more toenails (fingernail infection is less common) and produces gradual thickening, discoloration, and crumbling of the nail, with accumulation of subungual debris. Eventually, the nail may be destroyed completely.

Tinea pedis, or *athlete's foot*, causes scaling and blisters between the toes. Severe infection may result in inflammation, with severe itching and pain on walking. A dry, squamous inflammation may affect the entire sole. (See *Athlete's foot*.) Tinea manuum produces scaling patches and hyperkeratosis on the palmar surface. It's usually unilateral and associated with tinea pedis. Tinea cruris (jock itch) produces red, raised, sharply defined, itchy or burning lesions in the groin that may extend to the buttocks, inner thighs, and the external genitalia. Warm weather, obesity, and tight clothing encourage fungus growth. Tinea barbae is an uncommon infection that affects the bearded facial area of men.

Diagnosis

CONFIRMING DIAGNOSIS

Microscopic examination of lesion scrapings prepared in potassium hydroxide solution will reveal branching fungal hyphae. Gently heating the slide helps separate epithelial cells and hyphae. Lowering the microscope condenser and

dimming the light make hyphae easier to identify, as does adding a drop of ink to the potassium hydroxide.

Other diagnostic procedures include Wood's light examination (useful in only about 5% of cases of tinea capitis) and culture of the infecting organism, which is important for identifying hair and nail fungal infections.

Treatment

Tinea infections respond to a wide variety of medications. Typically, infections of the skin (hands, body, feet, and groin) require only topical therapy. Infections of the hair and nails, skin infections causing chronic thickening of the skin, and other unresolving infections require oral antifungal therapy.

Topical preparations are commonly azole-based, though other preparations are available. Oral therapy includes azole-based medications and terbinafine (Lamisil). Griseofulvin is falling out of favor because newer products are easier to use and have a shorter duration of therapy.

ALERT

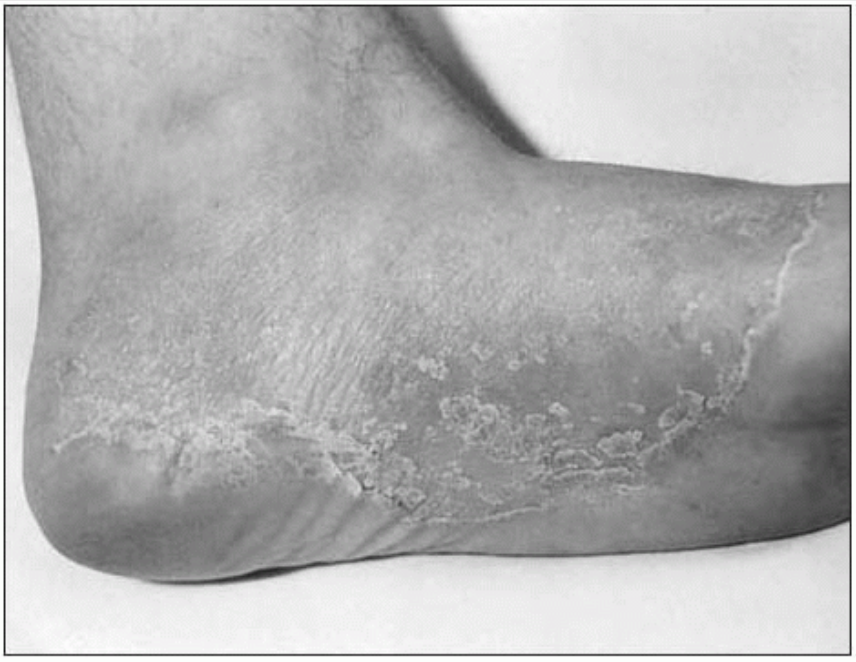
Caution must be taken when using systemic antifungals: liver enzyme levels must be monitored before and throughout treatment if therapy is expected to extend more than 2 months, and chronic medications must be monitored because of the antifungal's potential effect on blood levels.

Treatment should continue from several days to 2 weeks after lesions have resolved. Topical agents with soothing and cooling effects may be used with systemic therapy for infections with severe itching and burning; they may be discontinued when the immediate discomfort resolves.

ATHLETE'S FOOT

Dermatophytosis of the feet (tinea pedis) is popularly called athlete's foot. This infection causes macerated, scaling lesions, which may spread from the interdigital

spaces to the sole. Diagnosis must rule out other possible causes, such as eczema, psoriasis, contact dermatitis, and maceration by tight, illfitting shoes.



Special considerations

Management of tinea infections requires medication compliance, observation for sensitivity reactions, observation for secondary bacterial infections, and patient teaching. Specific care varies by site of infection.

- For tinea barbae—Suggest that the patient let his beard grow (whiskers may be trimmed with scissors, not a razor). If the patient insists that he must shave, advise him to use an electric razor instead of a blade.
- For tinea capitis—If the condition worsens, discontinue medications and notify the physician. Use good hand-washing technique, and teach the patient to do the same. Spores of tinea capitis are shed in the air around an infected patient or may spread on contaminated clothing and other personal articles. To prevent spread of infection to others, advise him to wash his towels, bedclothes, and combs frequently in hot water and to avoid sharing them. Suggest that family members be checked for tinea capitis.

- For tinea corporis—Use abdominal pads between skin folds for the patient with excessive abdominal girth; change pads frequently. Check the patient daily for excoriated, newly denuded areas of skin. Apply wet Burow's compresses two or three times daily to decrease inflammation and help remove scales.
- For tinea cruris—Instruct the patient to dry the affected area thoroughly after bathing and to evenly apply antifungal powder after applying the topical antifungal agent. Advise him to wear loose-fitting clothing, which should be changed frequently and washed in hot water.
- For tinea pedis—Encourage the patient to expose his feet to air whenever possible, and to wear sandals or leather shoes and clean, white cotton socks. Instruct the patient to wash his feet twice daily and, after drying them thoroughly, to apply antifungal cream followed by antifungal powder to absorb perspiration and prevent excoriation. Tell him to allow his shoes to dry out by alternating pairs every other daily. Also

  tell him to wear shower shoes when using public facilities.
- For tinea unguium—Keep nails short and straight. Gently remove debris under the nails with an emery board. Prepare the patient for prolonged therapy.

PARASITIC INFESTATIONS

Scabies

A common skin infection, scabies results from infestation with *Sarcoptes scabiei var. hominis* (itch mite), which provokes a sensitivity reaction. It's transmitted through skin or sexual contact.

Causes and incidence

Mites can live their entire life cycles in the skin of humans, causing chronic infection. (The adult mite can survive without a human host for only 2 or 3 days.) The female mite burrows into the skin to lay her eggs, from which larvae emerge to copulate and then reburrow under the skin. (See *Scabies: Cause and effect*.)

Scabies occurs worldwide, primarily in environments marked by overcrowding and poor hygiene, and can be endemic.

Complications

- Excoriation
- Tissue trauma
- Secondary bacterial infection

Signs and symptoms

Typically, scabies causes itching, which intensifies at night. Characteristic lesions are usually excoriated and may appear as erythematous nodules. These threadlike lesions are approximately 1 cm long and generally occur between fingers, on flexor surfaces of the wrists, on elbows, in axillary folds, at the waistline, on nipples and buttocks in females, and on genitalia in males.

PEDIATRIC TIP

In infants, the burrows (lesions) may appear on the head and neck.

Diagnosis

CONFIRMING DIAGNOSIS

Visual examination of the contents of the scabietic burrow may reveal the itch mite. If not, a drop of mineral oil placed over the burrow, followed by superficial scraping and examination of expressed material under a low-power microscope, may reveal ova, or mite feces. However, excoriation or inflammation of the burrow often makes such identification difficult. If diagnostic tests offer no positive identification of the mite and if scabies is still suspected (for example, if family members and close contacts of the patient also report itching), skin clearing that occurs after a therapeutic trial of a pediculicide confirms the diagnosis.

Treatment

Generally, treatment for scabies consists of application of a pediculicide—permethrin, lindane cream, or crotamiton—in a thin layer over the entire skin surface from the neck down. Lindane and permethrin are left on the skin for 8 to 12 hours. Crotamiton is applied nightly for 2 consecutive nights and washed off 24 hours after the second application. To make certain that all areas have been treated, this application should be repeated in about 1 week.

Lindane is an effective scabicide and when used properly may be applied safely to children, but shouldn't be used in children younger than age 2 or pregnant or nursing mothers because of potential neurologic toxicity. It also shouldn't be applied immediately after a shower. A 6% to 10% solution of sulfur in petrolatum may be used if patients object to using lindane, but they should be advised that sulfur is messy and odorous.

Persistent pruritus (from mite sensitization or contact dermatitis) may develop from repeated use of pediculicides rather than from continued infection. An antipruritic emollient, topical steroid, or oral antihistamine can reduce itching; intralesional steroids may resolve erythematous nodules.

Special considerations

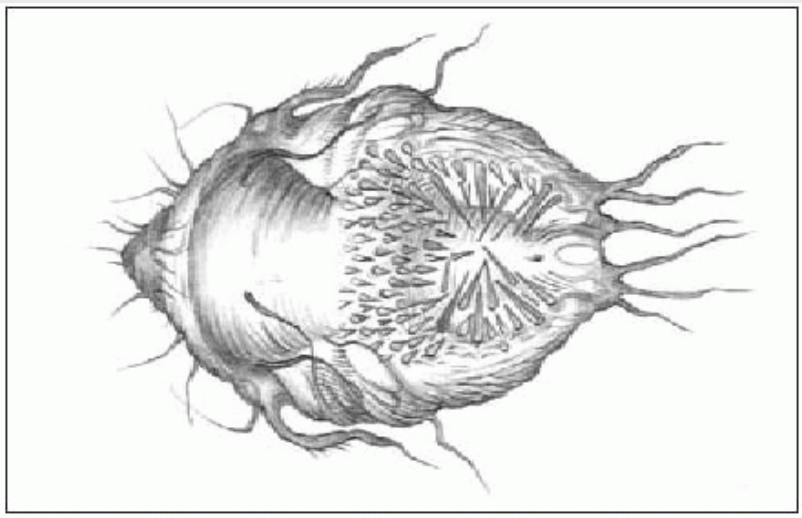
- Instruct the patient to apply permethrin, crotamiton, or lindane cream or lotion

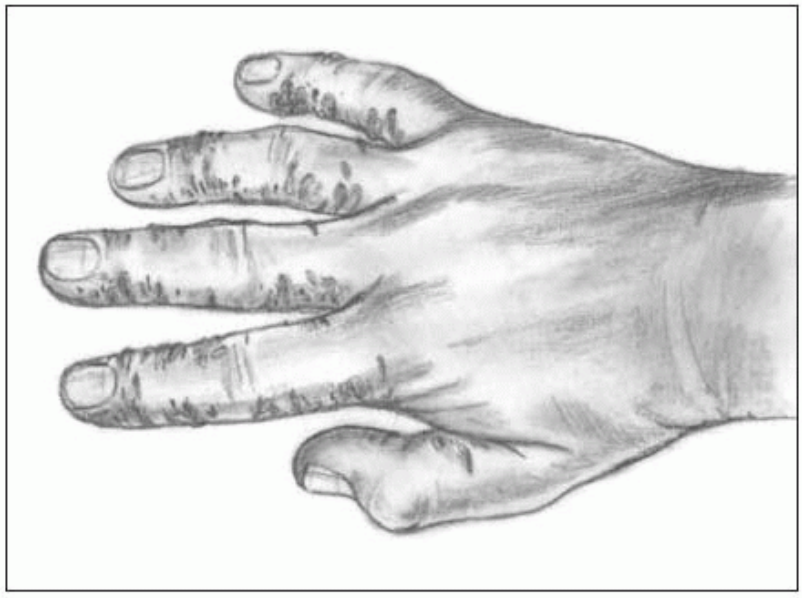
  from the neck down, covering his entire body. He must wait 15 minutes before dressing and avoid bathing for 8 to 12 hours or longer, depending on the treatment used. Contaminated clothing and linens must be washed in hot water or dry cleaned.

SCABIES: CAUSE AND EFFECT

Infestation with *Sarcoptes scabiei*—the itch mite—causes scabies. This mite (shown enlarged at right) has a hard shell and measures a microscopic 0.1 mm. The illustration below shows the erythematous nodules with excoriation

that appear in patients with scabies. These lesions are usually highly pruritic.





- Tell the patient not to apply lindane cream if his skin is raw or inflamed. Advise him that if skin irritation or hypersensitivity reaction develops, he should notify the physician immediately, discontinue using the drug, and thoroughly wash it off his skin.
- Suggest to the patient that his family members and other close personal contacts be checked for possible symptoms.

PREVENTION

If a hospitalized patient has scabies, prevent transmission to other patients. Practice good hand-washing technique or wear gloves when touching the patient; observe wound and skin precautions for 24 hours after treatment with a pediculicide; gas autoclave blood pressure cuffs before using them on other patients; isolate linens until the patient is noninfectious; and thoroughly disinfect the patient's room after discharge.

Cutaneous larva migrans

Cutaneous larva migrans (CLM), also known as *creeping eruption,* is a skin reaction to infestation by nematodes (hookworms or roundworms) that usually infect dogs and cats. Eruptions associated with cutaneous larva migrans clear completely with treatment.

Causes and incidence

Under favorable conditions—warmth, moisture, sandy soil—hookworm or roundworm ova present in feces of affected animals (such as dogs and cats) and hatch into larvae, which can then burrow into human skin on contact. After penetrating its host, the larva becomes trapped under the skin, unable to reach the intestines to complete its normal life cycle.

The parasite then begins to move, producing the peculiar, tunnel-like lesions that are alternately meandering and linear, reflecting the nematode's persistent and unsuccessful attempts to escape its host.

In the United States, CLM is second to pinworm in infestation.

Complications

- Cellulitis
- Excoriation
- Secondary bacterial infection

Signs and symptoms

A transient rash, tingling, or, possibly, a small vesicle appears at the point of penetration, usually on an exposed area that has come in contact with the ground, such as the feet, legs, or buttocks. The incubation period is typically 1 to 6 days. The parasite may be active almost as soon as it enters the skin. Local pruritus begins within hours following penetration.

As the parasite migrates, it etches a noticeable thin, raised, red line on the skin, which may become vesicular and encrusted. Pruritus quickly develops, often with crusting and secondary infection following excoriation. Onset is usually characterized by slight itching that develops into intermittent stinging pain as the thin, red lines develop. The larva's apparently random path can cover from 1 mm to 1 cm a day. Penetration of more than one larva may involve a much larger area of the skin, marking it with many tracks.

Diagnosis

Characteristic migratory lesions strongly suggest cutaneous larva migrans. A thorough patient history usually reveals contact with warm, moist soil within the past several months. A skin biopsy may reveal larva in subbasal layer.

Treatment

Topical application of thiabendazole, ivermectin, or albendazole is effective. The suspension is applied to lesions and the immediate surrounding areas four times daily for 1 week. Oral thiabendazole given in two divided doses for 3 to 5 days is effective. Oral ivermectin and albendazole are equally effective. Tell the patient that adverse effects of systemic thiabendazole include nausea, vomiting, abdominal pain, and dizziness.

Special considerations

- Reassure the patient, especially if he's sensitive about his appearance, that larva migrans lesions usually clear 1 to 2 weeks after treatment. Stress the importance of adhering to the treatment regimen exactly as ordered.
- Have the patient's nails cut short to prevent skin breaks and secondary bacterial infection from scratching. Apply cool, moist compresses to

alleviate itching.

- Be alert for possible adverse reactions associated with systemic treatment, including nausea, vomiting, abdominal pain, and dizziness.
- Encourage the patient to verbalize feelings about the infestation, including embarrassment, fear of rejection by others, and body image disturbance.

PREVENTION

- *Teach patient that these parasites exist and about sanitation of beaches and sandboxes and about proper pet care.*
- *Instruct the patient and his family in good hand-washing technique, and stress the importance of preventing the spread of the infection among family members.*

Pediculosis

Pediculosis is caused by parasitic forms of lice: *Pediculus humanus capitis* causes pediculosis capitis (head lice); *Pediculus humanus corporis* causes pediculosis corporis (body lice); and *Phthirus pubis* causes pediculosis pubis (crab lice). (See *Types of lice*, page 748.) These lice feed on human blood and lay their eggs (nits) on body hairs or clothing fibers. After the nits hatch, the lice must feed within 24 hours or die; they mature in about 2 to 3 weeks. When a louse bites, it injects a toxin into the skin that produces mild irritation and a purpuric spot. Repeated bites cause sensitization to the toxin, leading to more serious inflammation. Treatment can effectively eliminate lice.

Causes and incidence

P. humanus capitis (most common species) feeds on the scalp and, rarely, in the eyebrows, eyelashes, and beard. It's most commonly seen on the back of the head and neck and behind the ears. This form of pediculosis is caused by overcrowded conditions and poor personal hygiene, and commonly affects children, especially girls. It spreads through shared clothing, hats, combs, and hairbrushes.

P. humanus corporis lives in the seams of clothing, next to the skin, leaving only to feed on blood. Common causes include prolonged wearing of the same clothing (which might occur in cold climates), overcrowding, and poor personal hygiene. It spreads through shared clothing and bedsheets.

P. pubis is primarily found in pubic hairs, but this species may extend to the eyebrows, eyelashes, and axillary or body hair. Pediculosis pubis is transmitted through sexual intercourse or by contact with clothes, bedsheets, or towels harboring lice.

In the United States, 6 to 12 million people are affected each year by pediculosis.

Complications

- Excoriation
- Secondary bacterial infection

Signs and symptoms

Clinical features of pediculosis capitis include itching; excoriation (with severe itching); matted, foul-smelling, lusterless hair (in severe cases); occipital and cervical lymphadenopathy (posterior cervical lymphadenopathy without obvious disease is characteristic); and a rash on the trunk, probably due to sensitization. Adult lice migrate from the scalp and deposit oval, gray-white nits on the proximal one-third of hair shafts.

Pediculosis corporis initially produces small, red papules (usually on the shoulders, trunk, or buttocks). Later, wheals (probably a sensitivity reaction) may develop. Untreated pediculosis corporis may lead to vertical excoriations and ultimately to dry, discolored, thickly encrusted, scaly skin, with bacterial infection and scarring. In severe cases, headache, fever, and malaise may accompany cutaneous symptoms.

Pediculosis pubis causes skin irritation from scratching, which is usually more obvious than the bites. Small gray-blue spots (maculae caeruleae) may appear on the thighs or upper body. Small red spots are often seen in the underclothing.

Diagnosis

Pediculosis is visible on physical examination.

- In pediculosis capitis—oval, grayish nits that can't be shaken loose like dandruff (The closer the nits are to the end of the hair shaft, the longer the infection has been present, because the ova are laid close to the scalp.)
- In pediculosis corporis—characteristic skin lesions; nits found on clothing
- In pediculosis pubis—nits attached to pubic hairs, which feel coarse and grainy to the touch.

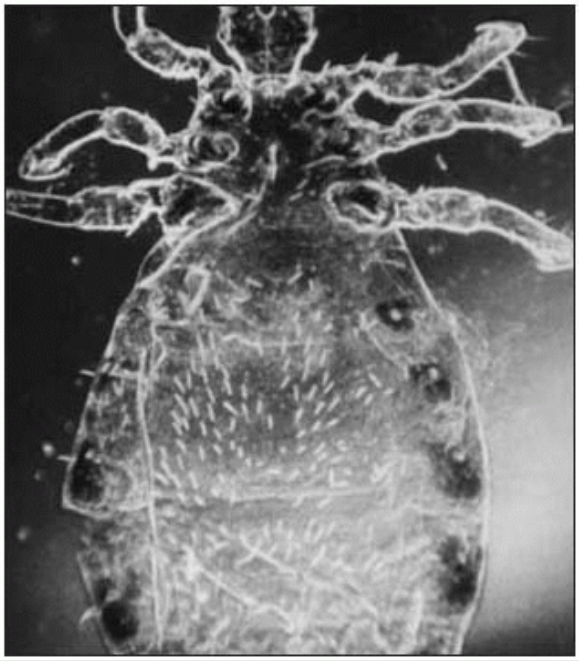
Treatment

Lindane, pyrethrin, permethrin, and malathion, in shampoo or lotion preparations, are all effective against lice. Shampoos should be applied to the infected skin or hair, lathered, then washed off in 5 minutes. Lotions should be applied over the entire affected area, then washed off after 10 minutes. Treatments should be repeated in 7 to 10 days.

TYPES OF LICE

Head louse

Pediculus humanus capitis (head louse) is similar in appearance to *Pediculus humanus corporis*.



Body louse

Pediculus humanus corporis (body louse) has a long abdomen and all its legs are about the same length.

Pubic louse

Phthirus pubis (pubic or “crab lice”) is slightly translucent; its first set of legs is shorter than its second or third.

Permethrin may be used for treating head lice. Saturate the hair and scalp and rinse after 10 minutes. Malathion lotion is also effective when applied to dry hair and washed out in 8 to 10 hours. After treatment, all nits (louse eggs) should be combed out of the hair with a metal nit comb. Nit removal may be aided by pre-rinsing with a pre-rinse solution containing formic acid, or dipping the comb in vinegar. Normal laundering of clothes and bedclothes in hot water after treatment is sufficient to remove adult lice as well as nits.

Special considerations

- Teach the patient how to use the creams, ointments, powders, and shampoos that eliminate lice.
- Ask the patient with pediculosis pubis for a history of recent sexual contacts, so that they can be examined and treated.
- The patient should be tested for other sexually transmitted diseases, including human immunodeficiency virus.

PREVENTION

- *To prevent the spread of pediculosis to other hospitalized persons, examine all high-risk patients on*

admission, especially elderly people who depend on others for care, those admitted from nursing homes, and people who live in crowded conditions.

- *To prevent your own infestation, avoid prolonged contact with the patient's hair, clothing, and bedsheets.*

FOLLICULAR AND GLANDULAR DISORDERS

Acne vulgaris

Acne vulgaris is an inflammatory disease of the sebaceous follicles. The prognosis is good with treatment.

Causes and incidence

The cause of acne is multifactorial, but theories regarding dietary influences appear to be groundless. Predisposing factors include heredity; hormonal contraceptives (many females experience an acne flare-up during their first few menstrual cycles after starting or discontinuing hormonal contraceptives); androgen stimulation; certain drugs, including corticosteroids, corticotropin, androgens, iodides, bromides, trimethadione, phenytoin, isoniazid, lithium, and halothane; cobalt irradiation; and hyperalimentation. Other possible factors are exposure to heavy oils, greases, or tars; trauma or rubbing from tight clothing; cosmetics; emotional stress; and unfavorable climate.

More is known about the pathogenesis of acne. (See *What happens in acne*, page 750.) Androgens stimulate sebaceous gland growth and production of sebum, which is secreted into dilated hair follicles that contain bacteria. The bacteria, usually *Propionibacterium acnes* and *Staphylococcus epidermidis* (which are normal skin flora), secrete lipase. This enzyme interacts with sebum to produce free fatty acids, which provoke inflammation. Also, the hair follicles produce more keratin, which joins with the sebum to form a plug in the dilated follicle.

Acne vulgaris primarily affects adolescents (usually between ages 15 and 18), although lesions can appear as early as age 8. Although acne strikes

boys more often and more severely than girls, it usually occurs in girls at an earlier age and tends to last longer, sometimes into adulthood.

Complications

- Abscess formation
- Permanent scarring
- Secondary bacterial infection

Signs and symptoms

The acne plug may appear as a closed comedo, or whitehead (if it doesn't protrude from the follicle and is covered by the epidermis), or as an open comedo, or blackhead (if it does protrude and isn't covered by the epidermis). The black coloration is caused by the melanin or pigment of the follicle. Rupture or leakage of an enlarged plug into the dermis produces inflammation and characteristic acne pustules, papules or, in severe forms, acne cysts or abscesses.

Diagnosis

CONFIRMING DIAGNOSIS

The appearance of characteristic acne lesions, especially in an adolescent patient, confirms the presence of acne vulgaris.

Treatment

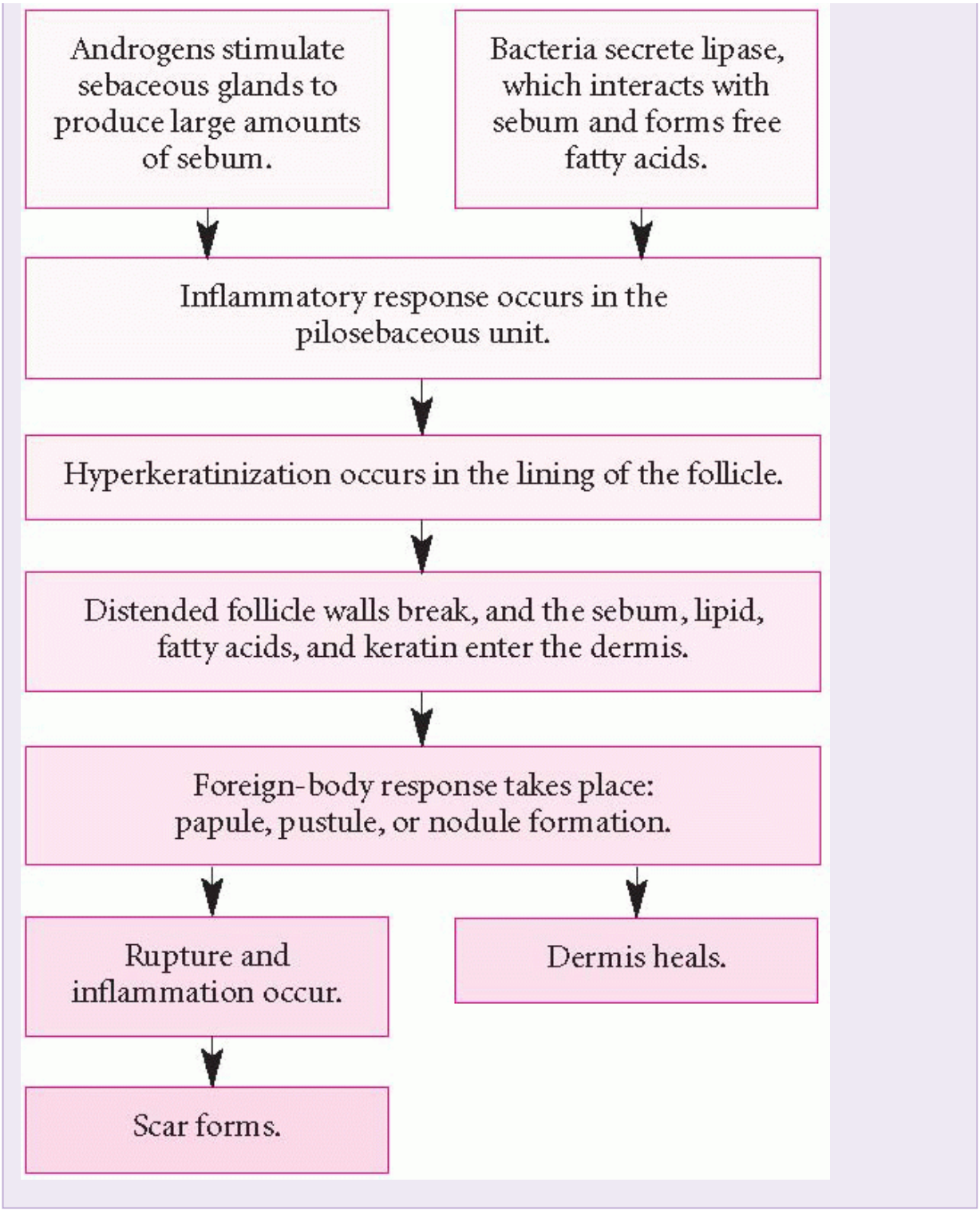
Current therapy for acne includes topical and oral agents. Topical retinoic acid (tretinoin) is the treatment of choice for noninflammatory acne consisting of open and closed comedones. Benzoyl peroxide is antibacterial and is used primarily for inflammatory acne, including papules, pustules, and cysts. Topical antibiotics are effective for mild pustular and comedone acne. Tetracycline, erythromycin, clindamycin, meclocycline, and benzamycin are all available in topical forms. Systemic antibiotics, such as tetracycline, minocycline, clindamycin, erythromycin, ampicillin,

cephalosporins, co-trimoxazole, and systemic retinoids may help reduce the effects of acne.

PATHOPHYSIOLOGY

WHAT HAPPENS IN ACNE

In acne vulgaris, hormonal, bacterial, and inflammatory responses in the skin interact to produce the disorder.



Systemic therapy consists primarily of antibiotics, usually tetracycline (which also exhibits an anti-inflammatory effect), to decrease bacterial growth until the patient is in remission; then a lower dosage is used for long-term maintenance.

ALERT

Tetracycline is contraindicated during pregnancy because it discolors the teeth of the fetus. Erythromycin and ampicillin are alternatives for these patients. Exacerbation of pustules or abscesses during either type of antibiotic therapy requires a culture to identify a possible secondary bacterial infection.

Oral isotretinoin combats acne by inhibiting sebaceous gland function and keratinization.

ALERT

Because of its severe adverse effects, the 16- to 20-week course of isotretinoin is limited to those with severe papulopustular or cystic acne who don't respond to conventional therapy. Because this drug is known to cause birth defects, the manufacturer, with Food and Drug Administration approval, recommends the following precautions: pregnancy testing before dispensing; dispensing of only a 30-day supply; repeat pregnancy testing throughout the treatment period; effective contraception during treatment; and informed consent of the patient or parents regarding the drug's adverse effects.

A serum triglyceride level should be measured before therapy with isotretinoin begins and at intervals throughout its course.

Females may benefit from the administration of estrogens to inhibit androgen activity. Improvement rarely occurs before 2 to 4 months, and exacerbations may follow its discontinuation. Unfortunately, the high estrogen doses that are required present a major risk of severe adverse effects.

Other treatments for acne vulgaris include intralesional or oral corticosteroids, vitamin A and zinc supplements, exposure to ultraviolet

light (but never when a photosensitizing agent such as tretinoin is being used), cryotherapy, and surgery.

Special considerations

The main focus of care is teaching about the disorder as well as its treatment and prevention.

- Check the patient's drug history because certain medications such as hormonal contraceptives may cause an acne flare-up.
- Explain the causes of acne to the patient and his family. Make sure they understand that the prescribed treatment is more likely to improve acne than a strict diet and fanatical scrubbing with soap and water. Provide written instructions regarding treatment.
- Instruct the patient receiving tretinoin to apply it at least 30 minutes after washing his face and at least 1 hour before bedtime. Warn against using it around the eyes or lips. After treatments, the skin should look pink and dry. If it appears red or starts to peel, the preparation may have to be weakened or applied less often. Advise the patient to avoid exposure to sunlight or to use a sunscreening agent. If the prescribed regimen includes tretinoin and benzoyl peroxide, avoid skin irritation by using one preparation in the morning and the other at night.
- Instruct the patient to take tetracycline on an empty stomach and not to take it with antacids or milk because it interacts with their metallic ions and is then poorly absorbed.
- Tell the patient who's taking isotretinoin to avoid vitamin A supplements, which can worsen any adverse effects. Also, teach how to deal with the dry skin and mucous membranes that usually occur during treatment. Tell the female patient about the severe risk of teratogenicity. Monitor liver function and lipid levels.
- Inform the patient that acne takes a long time to clear—even years for complete resolution. Encourage continued local skin care even after acne clears. Explain the adverse effects of all drugs.
- Pay special attention to the patient's perception of his physical appearance, and offer emotional support.

PREVENTION

- *Try to identify predisposing factors that may be eliminated or modified.*
- *Teach the patient and his family techniques to maintain a well-balanced diet, get adequate rest, and manage stress.*

Hirsutism

Usually found in women and children, hirsutism is the excessive growth of body hair, typically in an adult male distribution pattern. This condition commonly occurs spontaneously but may also develop as a secondary disorder of various underlying diseases. It must always be distinguished from hypertrichosis. The prognosis varies with the cause and effectiveness of treatment.

Causes and incidence

Idiopathic hirsutism probably stems from a hereditary trait because the patient usually has a family history of the disorder. Causes of secondary hirsutism include endocrine abnormalities related to pituitary dysfunction (acromegaly or precocious puberty), adrenal dysfunction (Cushing's disease, congenital adrenal hyperplasia, or Cushing's syndrome), or ovarian lesions (such as polycystic ovary syndrome or ovarian neoplasm); prolactinoma; and iatrogenic factors (such as the use of minoxidil, androgenic steroids, testosterone, diazoxide, glucocorticoids, and hormonal contraceptives). Other kinds of hirsutism have been reported. (See *Hypertrichosis,* page 752.)

HYPERTRICHOSIS

Hypertrichosis is a localized or generalized condition in males and females that's marked by excessive hair growth in areas that aren't androgen-sensitive. Localized hypertrichosis usually results from local trauma, chemical irritation, or hormonal stimulation; pigmented nevi (Becker's nevus, for example) may also contain hairs. Generalized hypertrichosis results from neurologic or psychiatric disorders, such as encephalitis, multiple

sclerosis, concussion, anorexia nervosa, or schizophrenia; contributing factors include juvenile hypothyroidism, porphyria cutanea tarda, and the use of drugs such as phenytoin.

Hypertrichosis lanuginosa is a generalized proliferation of fine, lanugo-type hair (sometimes called *down* or *woolly hair).* Such hair may be present at birth but generally disappears shortly thereafter. This condition may become chronic, with persistent lanugo-type hair growing over the entire body, or may develop suddenly later in life; it is very rare and usually results from malignancy.

In the United States, hirsutism occurs in 1 in 20 women of childbearing age.

Complication

- Varies depending on the cause

Signs and symptoms

Hirsutism typically produces enlarged hair follicles as well as enlargement and hyperpigmentation of the hairs themselves. Excessive facial hair growth is the complaint for which most patients seek medical help. Generally, hirsutism involves appearance of thick, pigmented hair in the beard area, upper back, shoulders, sternum, axillae, and pubic area. Frontotemporal scalp hair recession is often a coexisting condition. Patterns of hirsutism vary widely, depending on the patient's race and age.

ELDER TIP

Elderly women commonly show increased hair growth on the chin and upper lip.

In secondary hirsutism, signs of masculinization may appear—eepening of the voice, increased muscle mass, increased size of genitalia, menstrual irregularity, and decreased breast size.

Diagnosis

A family history of hirsutism, absence of menstrual abnormalities or signs of masculinization, and a normal pelvic examination strongly suggest idiopathic hirsutism. Tests for secondary hirsutism depend on associated symptoms that suggest an underlying disorder. About 90% of women with hirsutism have an elevated free testosterone level.

Treatment

At the patient's request, treatment for idiopathic hirsutism consists of eliminating excess hair by scissors, shaving, or depilatory creams, or removal of the entire hair shaft with tweezers or wax. However, removal with laser is the most effective method. Bleaching with hydrogen peroxide may also be satisfactory. Electrolysis can destroy hair bulbs permanently, but it works best when only a few hairs need to be removed. (A history of keloid formation contraindicates this procedure.) Hirsutism due to elevated androgen levels may require low-dose dexamethasone or prednisone, hormonal contraceptives, or androgen receptor-competitive inhibitors—such as spironolactone, cyproterone acetate, or cimetidine—however, these drugs vary in effectiveness.

Treatment for secondary hirsutism varies, depending on the nature of the underlying disorder.

Special considerations

Care for patients with idiopathic hirsutism focuses on emotional support and patient teaching; care for patients with secondary hirsutism depends on the treatment for the underlying disease.

- Provide emotional support by being sensitive to the patient's feelings about her appearance.
- Watch for signs of contact dermatitis in patients being treated with depilatory creams, especially elderly people. Also, watch for infection of hair follicles after hair removal with tweezers or wax.
- Suggest consulting a cosmetologist about makeup or bleaching agents.

Alopecia

Alopecia, or hair loss, usually occurs on the scalp but can also occur on bearded areas, eyebrows, and eyelashes. Hair loss elsewhere on the body is less common and less conspicuous. In the nonscarring form of this disorder (noncicatricial alopecia), the hair follicle can generally regrow hair. However, scarring alopecia involves tissue destruction, such as inflammation, scarring, and atrophy, and usually destroys the hair follicle, making hair loss irreversible.

Causes and incidence

The most common form of nonscarring alopecia is male-pattern alopecia, which appears to be related to androgen levels and to aging. Genetic predisposition commonly influences the time of onset, degree of baldness, speed with which it spreads, and pattern of hair loss. Women may experience diffuse thinning over the top of the scalp.

Other forms of nonscarring alopecia include:

- physiologic alopecia (usually temporary): sudden hair loss in infants, loss of straight hairline in adolescents, and diffuse hair loss after childbirth
- alopecia areata (idiopathic form): generally reversible and self-limiting; occurs most frequently in young and middle-age adults of both sexes (See *Alopecia areata*, page 754.)
- trichotillomania: compulsive pulling out of one's own hair; most common in children
- traction alopecia: localized areas of hair loss due to chronic use of tight braids (such as cornrows) or other hair styles. This condition may also result in scarring alopecia.

Predisposing factors of nonscarring alopecia also include radiation, many types of drug therapies and drug reactions, bacterial and fungal infections, psoriasis, seborrhea, and endocrine disorders, such as thyroid, parathyroid, and pituitary dysfunctions.

Scarring alopecia causes irreversible hair loss. It may result from physical or chemical trauma and chronic tension on a hair shaft, as occurs in braiding. Diseases that produce alopecia include destructive skin tumors,

granulomas, lupus erythematosus, scleroderma, follicular lichen planus, and severe fungal, bacterial, or viral infections, such as kerion, folliculitis, or herpes simplex.

Signs and symptoms

In male-pattern alopecia, hair loss is gradual and usually affects the thinner, shorter, and less pigmented hairs of the frontal and parietal portions of the scalp. In women, hair loss is generally more diffuse; completely bald areas are uncommon but may occur.

Alopecia areata affects small patches of the scalp but may also occur as alopecia totalis, which involves the entire scalp and eyebrows, or as alopecia universalis, which involves the entire body. Although mild erythema may occur initially, affected areas of scalp or skin appear normal. “Exclamation point” hairs (loose hairs with dark, rough, brushlike tips on narrow, less pigmented shafts) occur at the periphery of new patches. Regrowth hairs are thin and may be white or gray. They're usually replaced by normal hair.

In trichotillomania, patchy, incomplete areas of hair loss with many broken hairs appear on the scalp but may occur on other areas such as the eyebrows.

Diagnosis

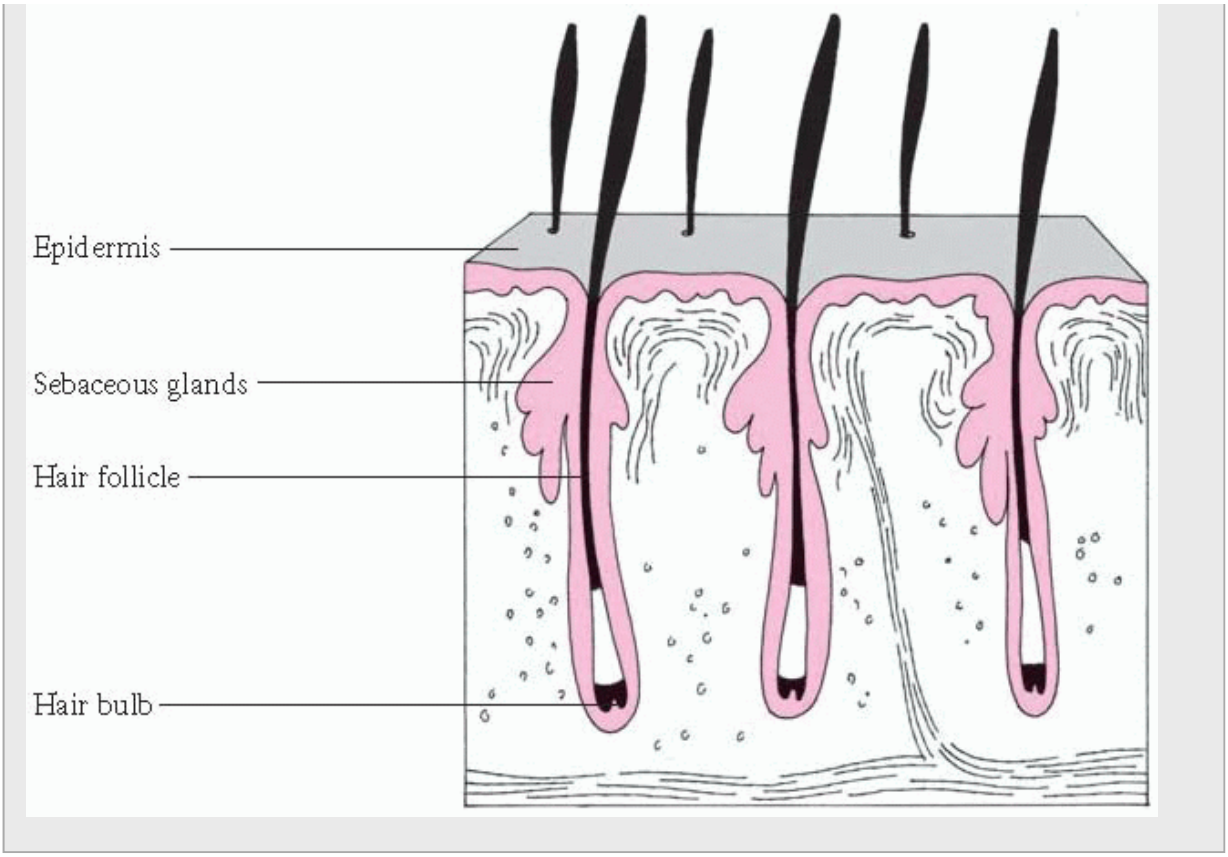
CONFIRMING DIAGNOSIS

Physical examination is usually sufficient to confirm alopecia. In trichotillomania, an occlusive dressing can establish the diagnosis by allowing new hair to grow, revealing that the hair is being pulled out.

Diagnosis must also identify any underlying disorder.

ALOPECIA AREATA

“Exclamation point” hairs often border new patches of alopecia areata. Not seen in any other type of alopecia, these hairs indicate the patch is expanding.



Treatment

Topical application of minoxidil, a peripheral vasodilator more typically used as an oral antihypertensive, has limited success in treating male-pattern alopecia. An alternative treatment is surgical redistribution of hair follicles by autografting. Oral finasteride has been shown to reverse androgenic loss, but it's approved only for use in men.

In alopecia areata, minoxidil is effective, although treatment is often unnecessary because spontaneous regrowth is common. Intralesional corticosteroid injections are beneficial for small patches and may produce regrowth in 4 to 6 weeks. Anthralin, topical high-potency corticosteroids, systemic corticosteroids, topical cyclosporine, oral inosiplex, and topical nitrogen mustard all have been used in treating alopecia areata. Hair loss that persists for more than a year has a poor prognosis for regrowth.

In trichotillomania, an occlusive dressing encourages normal hair growth, simply by identifying the cause of hair loss; clomipramine may be effective for short-term treatment. Treatment for other types of alopecia varies according to the underlying cause.

Special considerations

- Reassure a woman with female-pattern alopecia that it doesn't lead to total baldness. Suggest that she wear a wig.
- If the patient has alopecia areata, explain the disorder and give reassurance that complete regrowth is possible.

Rosacea

A chronic skin eruption, rosacea produces flushing and dilation of the small blood vessels in the face, especially the nose and cheeks. Papules and pustules may also occur, but without the characteristic comedones of acne vulgaris. Ocular involvement

may result in blepharitis, conjunctivitis, uveitis, or keratitis. Rosacea usually spreads slowly and rarely subsides spontaneously.

Causes and incidence

Although the cause of rosacea is unknown, stress, infection, vitamin deficiency, menopause, and endocrine abnormalities can aggravate this condition. Anything that produces flushing—for example, hot beverages, such as tea or coffee; tobacco; alcohol; spicy foods; physical activity; sunlight; and extreme heat or cold—can also aggravate rosacea.

Rosacea is most common in white women between ages 30 and 50. When it occurs in men, however, it's usually more severe and often associated with rhinophyma, which is characterized by dilated follicles and thickened, bulbous skin on the nose.

Complications

- Blepharitis
- Conjunctivitis
- Keratitis
- Uveitis

Signs and symptoms

Rosacea generally begins with periodic flushing across the central oval of the face, accompanied later by telangiectasia, papules, pustules, and nodules. Rhinophyma is commonly associated with severe untreated rosacea but may occur alone. Rhinophyma usually appears first on the lower half of the nose, and produces red, thickened skin and follicular enlargement. It's found almost exclusively in men older than age 40. Related ocular lesions are uncommon.

Diagnosis

CONFIRMING DIAGNOSIS

Typical vascular and acneiform lesions— without the comedones characteristically associated with acne vulgaris —and rhinophyma in severe cases confirm rosacea.

Treatment

Treatment for the acneiform component of rosacea consists of oral tetracycline or erythromycin in gradually decreasing doses over 1 to 2 months as symptoms subside. Resistant cases can be treated with oral minocycline or doxycycline. Isotretinoin is also effective, but its use is limited to those with severe disease. Topical metronidazole gel helps the papules, pustules, and erythema. Sulfacet-R lotion, available in flesh tones, controls pustules and hides redness. It can be used alone or together with oral antibiotics. Other treatments include electrolysis to destroy large, dilated blood vessels and removal of excess tissue in patients with rhinophyma. Topical hydrocortisone preparations worsen the condition.

Special considerations

Assess the effect of rosacea on body image. Because it's always apparent on the face, support is essential.

PREVENTION

- *Instruct the patient to avoid spicy foods, hot beverages, alcohol, extended sun exposure, and other possible causes of flushing.*

- *Encourage the use of sunscreen.*

PIGMENTATION DISORDERS

Vitiligo

Marked by stark-white skin patches that may cause a serious cosmetic problem, vitiligo results from the destruction and loss of pigment cells. It shows no racial preference, but the distinctive patches are most noticeable in blacks. Repigmentation therapy, which is widely used in treating vitiligo, may necessitate several summers of exposure to sunlight; the effects of this treatment may not be permanent.

Causes and incidence

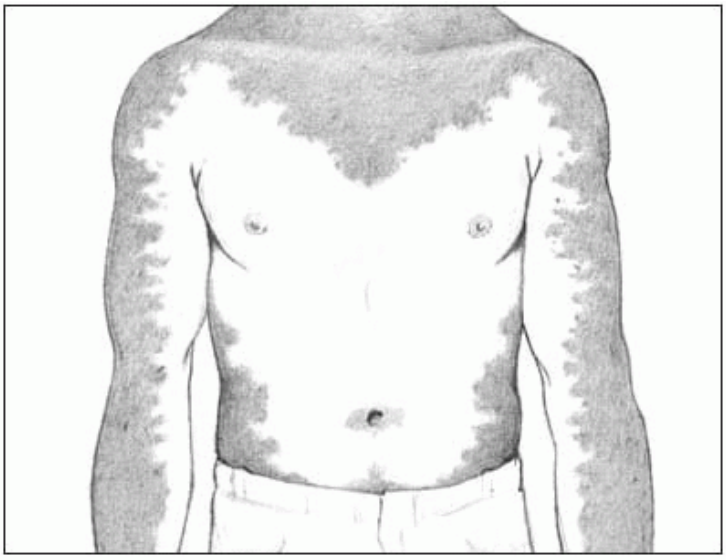
Although the cause of vitiligo is unknown, inheritance seems to be a definite etiologic factor because about 30% of patients with vitiligo have family members with the same condition. Other theories implicate enzymatic self-destructing mechanisms, autoimmune mechanisms, and abnormal neurogenic stimuli.

Some link exists between vitiligo and many other disorders that it often accompanies—thyroid

dysfunction, pernicious anemia, Addison's disease, aseptic meningitis, diabetes mellitus, photophobia, hearing defects, alopecia areata, uveitis, chronic mucocutaneous candidiasis, and halo nevi.

RECOGNIZING VITILIGO

This illustration shows characteristic depigmented skin patches in vitiligo. These patches are usually bilaterally symmetrical, with distinct borders.



The most frequently reported precipitating factor is a stressful physical or psychological event—severe sunburn, surgery, pregnancy, loss of a job, bereavement, or some other source of distress. Chemical agents, such as phenols and catechols, may also cause this condition.

Vitiligo affects about 1 to 2 million people in the United States, usually people between ages 10 and 30, with peak incidence around age 20. It affects men and women equally, but women are more likely to seek treatment.

Complications

- Sunburn
- Skin cancer

Signs and symptoms

Vitiligo produces depigmented or stark-white patches on the skin; on fair-skinned whites, these are almost imperceptible. Lesions are usually bilaterally symmetrical with sharp borders, which occasionally are hyperpigmented. Lesions that are small initially can enlarge and even progress to total depigmentation (universal vitiligo).

These unique patches generally appear over bony prominences on the back of the hands; on the face, the axillae, genitalia, nipples, or umbilicus; around orifices (such as the eyes, mouth, and anus); within

body folds; and at sites of trauma. The hair within these lesions may also turn white. Because hair follicles and certain parts of the eyes also contain pigment cells, vitiligo may be associated with premature gray hair and ocular pigmentary changes. (See *Recognizing vitiligo*.)

Diagnosis

Diagnosis requires an accurate history of onset and of associated illnesses, a family history, and observation of characteristic lesions. Other skin disorders such as tinea versicolor must be ruled out.

CONFIRMING DIAGNOSIS

In fair-skinned patients, Wood's light examination in a darkened room detects vitiliginous patches; depigmented skin reflects the light, and pigmented skin absorbs it. Biopsy will show normal skin except for the absence of melanocytes. If autoimmune or endocrine disturbances are suspected, laboratory studies (such as thyroid studies) are appropriate.

Treatment

Repigmentation therapy combines systemic or topical psoralen compounds (trimethylpsoralen or 8-methoxypsoralen) with exposure to sunlight or artificial ultraviolet light, wavelength A (UVA). New pigment rises from hair follicles and appears on the skin as small freckles, which gradually enlarge and coalesce. Body parts containing few hair follicles (such as the fingertips) may resist this therapy.

Because psoralens and UVA affect the entire skin surface, systemic therapy enhances the contrast between normal skin, which turns darker than usual, and white, vitiliginous skin. Use of sunscreen on normal skin may minimize contrast while preventing sunburn.

Topical class I glucosteroid ointments may be used for single or small macules. Monitor patients on this therapy for skin atrophy or telangiectasia development.

Depigmentation therapy is suggested for patients with vitiligo affecting more than 50% of the body surface. A cream containing 20% monobenzone

permanently destroys pigment cells in unaffected areas of the skin and produces a uniform skin tone. This medication is applied initially to a small area of normal skin once daily to test for unfavorable reactions such as contact dermatitis. In the absence of adverse effects, the patient begins applying the cream twice daily to those areas he wishes to depigment first. Eventually, the entire skin may be depigmented to achieve a uniform color. *Note:* Depigmentation is permanent and results in extreme photosensitivity. Patients may wish to take daily B-carotene to impart an off-white color to the chalkwhite skin.

Commercial cosmetics may also help deemphasize vitiliginous skin. Some patients prefer dyes because these remain on the skin for several days, although the results aren't always satisfactory. Although often impractical, complete avoidance of exposure to sunlight through the use of screening agents and protective clothing may minimize vitiliginous lesions in whites.

Special considerations

- Instruct the patient to use psoralen medications three or four times weekly. (*Note:* Systemic psoralens should be taken 2 hours before exposure to sun; topical solutions should be applied 30 to 60 minutes before exposure.) Warn him to use a sunscreen (sun protection factor [SPF] 8 to 10) to protect both affected and normal skin during exposure and to wear sunglasses after taking the medication. If periorbital areas require exposure, tell the patient to keep his eyes closed during treatment.
- Suggest that the patient receiving depigmentation therapy wear protective clothing and use a sunscreen (SPF 15). Explain the therapy thoroughly, and allow the patient plenty of time to decide whether to undergo this treatment. Make sure he understands that the results of depigmentation are permanent and that he must thereafter protect his skin from the adverse effects of sunlight.
- Caution the patient about buying commercial cosmetics or dyes without trying them first because some may not be suitable.

PEDIATRIC TIP

For the child with vitiligo, modify repigmentation therapy to avoid unnecessary restrictions. Tell parents to give the

initial dose of psoralen medication at 1 p.m. and then let the child go out to play as usual. After this, medication should be given 30 minutes earlier each day of treatment, provided the child's skin doesn't turn more than slightly pink from exposure. If marked erythema develops, parents should discontinue treatment and notify the physician. Eventually, the child should be able to take the medication at 9:30 a.m. and play outdoors the rest of the day without adverse effects. Tell the parents the child should wear clothing that permits maximum exposure of vitiliginous areas to the sun.

- Remind patients undergoing repigmentation therapy that exposure to sunlight also darkens normal skin. After being exposed to UVA for the prescribed amount of time, the patient should apply a sunscreen if he plans to be exposed to sunlight also. If sunburn occurs, advise the patient to discontinue therapy temporarily and to apply open wet dressings (using thin sheeting) to affected areas for 15 to 20 minutes, four or five times daily or as necessary for comfort. After application of wet dressings, allow the skin to air dry. Suggest application of a soothing lubricating cream or lotion while the skin is still slightly moist.

- Reinforce patient teaching with written instructions.
- Be sensitive to the patient's emotional needs, but avoid promoting unrealistic hope for a total cure.

Melasma

A patchy, hypermelanotic skin disorder, melasma (also known as *chloasma* or *mask of pregnancy*) can pose a serious cosmetic problem. It may be chronic but is never life-threatening.

Causes and incidence

The cause of melasma is unknown, but onset is most common in young adults. Histologically, hyperpigmentation results from increased melanin production, although the number of melanocytes remains normal.

Melasma may be related to the increased hormonal levels associated with pregnancy, menopause, ovarian cancer, and the use of hormonal contraceptives. Progestational agents, phenytoin, and mephenytoin may also contribute to this disorder. Exposure to sunlight stimulates melasma, but it may develop without any apparent predisposing factor. Patients with acquired immunodeficiency syndrome have an increased incidence of similar hyperpigmentation.

Melasma affects females more commonly than males. Although it tends to occur equally in all races, the light-brown color characteristic of melasma is most evident in dark-skinned whites.

Signs and symptoms

Typically, melasma produces large, brown, irregular patches, symmetrically distributed on the forehead, cheeks, and sides of the nose. Less commonly, these patches may occur on the neck, upper lip, temples and, occasionally, on the dorsa of the forearms.

Diagnosis

CONFIRMING DIAGNOSIS

Observation of characteristic dark patches on the face usually confirms melasma. The patient history may reveal predisposingfactors. Wood's lamp examination accentuates the hyperpigmentation.

Treatment

Treatment consists primarily of application of bleaching agents containing 2% to 4% hydroquinone in combination with tretinoin or glycolic acid to inhibit melanin synthesis. This medication is applied twice daily for up to 8 weeks. Adjunctive measures include avoidance of exposure to sunlight, use of opaque sunscreens, and discontinuation of hormonal contraceptives.

Special considerations

- Tell the patient that melasma associated with pregnancy usually clears within a few months after delivery and may not return with subsequent

pregnancies.

- Bleaching agents may help but may require repeated treatments to maintain the desired effect. Cosmetics may help mask deep pigmentation.
- Reassure the patient that melasma is treatable. It may fade spontaneously with protection from sunlight, postpartum, and after discontinuing hormonal contraceptives. Serial photographs help show the patient that patches are improving.

PREVENTION

Advise the patient to avoid sun exposure as much as possible by using sunscreens and wearing protective clothing.

Photosensitivity reactions

A photosensitivity reaction is a skin eruption that can be a toxic or allergic response to light alone or to light and chemicals. A phototoxic reaction is a dose-related primary response. A photoallergic reaction is an uncommon, acquired immune response that isn't dose-related—even slight exposure can cause a severe reaction.

Causes and incidence

Certain chemicals can cause a photosensitivity reaction, including dyes, coal tar, and furocoumarin compounds found in plants. The list of drugs that can cause photosensitivity reactions is extensive and includes many drugs within each of the following general categories: antibiotics (especially tetracycline), antidepressants, antihistamines, anticancer agents, antiparasitic agents, antipsychotic agents, diuretics, hypoglycemics, nonsteroidal anti-inflammatories, sunscreens, and miscellaneous agents, such as cardiac glycosides, hormonal contraceptives, and acne medications.

Berlock dermatitis, a specific photosensitivity reaction, results from the use of oil of bergamot—a common component of perfumes, colognes, and pomades.

Complications

- Premature skin aging
- Skin cancer

Signs and symptoms

Immediately after sun exposure, a phototoxic reaction causes a burning sensation followed by erythema (sunburn-type reaction), edema, desquamation, and hyperpigmentation. Berlock dermatitis produces an acute reaction with erythematous vesicles that later become hyperpigmented.

Photoallergic reactions may take one of two forms. Developing 2 hours to 5 days after light exposure, polymorphous light eruption (PMLE) produces erythema, papules, vesicles, urticaria, and eczematous lesions on exposed areas; pruritus may persist for 1 to 2 weeks. Solar urticaria begins minutes after exposure and lasts about an hour; erythema and wheals follow itching and burning sensations.

Diagnosis

Characteristic skin eruptions in sunexposed areas and a patient history of recent exposure to light or certain chemicals suggest a photosensitivity reaction. A photopatch test for ultraviolet A and B (UVA and UVB) done while the patient is on the drug may aid diagnosis and identify the causative light wavelength. Other studies must rule out connective tissue disease, such as lupus erythematosus and porphyrias.

Treatment

For many patients, treatment involves a sunscreen, protective clothing, and minimal exposure to sunlight while the patient continues on the drug. For others, progressive exposure to sunlight can thicken the skin and produce a tan that interferes with photoallergens and prevents further eruptions.

Withdrawal of the causative agent and treatment with oral steroids usually provides relief. The patient should be advised not to use the

causative agent again if it's known, even though this may limit the patient's treatment options.

Antimalarial drugs, beta-carotene, and PUVA (psoralen and UVA) may be used to treat PMLE. Treatment for solar urticaria may also require PUVA. Although hyperpigmentation usually fades in several months, hydroquinone preparations can hasten the process.

Special considerations

Tell the patient to inform his physician about sensitivity to any drugs.

PREVENTION

To prevent reactions, advise the patient to avoid prolonged exposure to light.

INFLAMMATORY REACTIONS

Dermatitis

Inflammation of the skin, dermatitis occurs in several forms: atopic (discussed here), seborrheic, nummular, contact, chronic, localized neurodermatitis, exfoliative, and stasis. (See *Types of dermatitis,* pages 760 to 763.) Atopic dermatitis (atopic or infantile eczema, neurodermatitis constitutionalis, or Besnier's prurigo) is a chronic inflammatory response often associated with other atopic diseases, such as bronchial asthma and allergic rhinitis.

Causes and incidence

The cause of atopic dermatitis is unknown, but a genetic predisposition may be exacerbated by such factors as food allergies, infections, irritating chemicals, temperature and humidity, and emotions. Approximately 10% of childhood cases are due to allergy to certain foods, particularly eggs, peanuts, milk, fish, soy, and wheat. Atopic dermatitis tends to flare up in response to extremes in temperature and humidity. Other causes of flare-ups are sweating and psychological stress.

TYPES OF DERMATITIS

Type	Causes	Signs and symptoms	Diagnosis	Treatment and intervention
Seborrheic dermatitis				
An acute or subacute skin disease that affects areas where sebaceous glands are most active—such as the scalp and face—and occasionally other areas, and is characterized by lesions covered with yellow or brownish gray scales	▪ Unknown; stress and neurologic conditions may be predisposing factors; may be related to the yeast Pityrosporum ovale	▪ Eruptions in areas with many sebaceous glands (usually scalp, face, and trunk) and in skin folds ▪ Itching, redness, and inflammation of affected areas; lesions may appear greasy; fissures may occur ▪ Indistinct, occasionally yellowish, scaly patches from excess stratum corneum (dandruff may be mild seborrheic dermatitis) ▪ Generally worse in winter	▪ Patient history and physical findings, especially distribution of lesions in sebaceous gland areas, confirm seborrheic dermatitis. ▪ Diagnosis must rule out psoriasis.	▪ Removal of scales with frequent washing and shampooing with selenium sulfide suspension (most effective), zinc pyrithione, or tar and salicylic acid shampoo ▪ Application of atopical steroid and an antifungal to nonhairy areas
Nummular dermatitis (discoid eczema, nummular eczema)				
A chronic form of dermatitis characterized by	▪ Possibly precipitated by stress, skin	▪ Round, nummular (coinshaped)	▪ Physical findings and patient	▪ Elimination of known irritants

inflammation of coin-shaped, vesicular, crusted scales and, possibly, pruritic lesions	dryness, irritants, scratching, or bathing with hot water	lesions, usually on arms and legs, with distinct borders of crusts and scales ▪ Possible oozing and severe itching ▪ Summertime remissions common, with wintertime recurrence	history confirm nummular dermatitis; a middle-age or older patient may have a history of atopic dermatitis. ▪ Diagnosis must rule out fungal infections, atopic or contact dermatitis, and psoriasis.	▪ Measures to relieve dry skin: increased humidification, limited frequency of baths and use of mild soap and bath oils, and application of emollients ▪ Application of wet dressings in acute phase ▪ Topical steroids (occlusive dressings or intralesional injections) for persistent lesions ▪ Tar preparations and antihistamines to control itching ▪ Antibiotics for secondary infection ▪ Other interventions as for atopic dermatitis
Contact dermatitis				
Often sharply demarcated inflammation and irritation of the	▪ Mild irritants: chronic exposure to detergents or solvents	▪ Mild irritants and allergens: erythema and small vesicles	▪ Patient history, patch testing to identify	▪ Elimination of known allergens and decreased

skin caused by contact with substances to which the skin is sensitive, such as perfumes, soaps, plants, or chemicals	▪ Strong irritants: damage on contact with acids or alkalis ▪ Allergens: sensitization after repeated exposure	that ooze, scale, and itch ▪ Strong irritants: blisters and ulcerations ▪ Classic allergic response: clearly defined lesions, with straight lines following points of contact ▪ Severe allergic reaction: marked edema of affected areas	allergens, and shape and distribution of lesions suggest contact dermatitis.	exposure to irritants, wearing protective clothing such as gloves, and washing immediately after contact with irritants or allergens ▪ Topical anti-inflammatory agents (including steroids); systemic corticosteroids for edema, bullae, or very extensive outbreaks; antihistamines; and local applications of Burow's solution (for blisters) ▪ Sensitization to topical medications may occur ▪ Other interventions as for atopic dermatitis
Chronic dermatitis Characterized by inflammatory	▪ Usually unknown but may result from	▪ Thick, lichenified, single or multiple	▪ No characteristic pattern or	▪ Antibiotics for secondary infection

eruptions of the hands and feet	progressive contact dermatitis ▪ Secondary (possibly perpetuating) factors: trauma, infections, redistribution of normal flora, photosensitivity, and food sensitivity	lesions on any part of body (often on hands) ▪ Inflammation and scaling ▪ Recurrence following long remissions	course; diagnosis relies on detailed patient history and physical findings.	▪ Avoidance of excessive washing and drying of hands and of accumulation of soaps and detergents under rings ▪ Use of emollients with topical steroids ▪ Elimination of known allergens and decreased exposure to irritants, wear protective clothing, and wash immediately after contact with irrntants or allergens.
Localized neurodermatitis (lichen simplex chronicus, essential pruritus)				
Superficial inflammation of the skin characterized by itching and papular eruptions that appear on thickened, hyperpigmented skin	▪ Chronic scratching or rubbing of primary lesion or insect bite, or other skin irritation	▪ Intense, sometimes continual scratching ▪ Thick, sharp-bordered, possibly dry, scaly lesions with raised papules ▪ Usually affects easily reached areas, such as ankles, lower	▪ Physical findings confirm diagnosis.	▪ Scratching must stop; then lesions will disappear in about 2 weeks ▪ Fixed dressing or Unna's boot to cover affected area ▪ Topical steroids under

		legs, anogenital area, back of neck, and ears		occlusion or by intralesional injection ▪ Antihistamines and open wet dressings ▪ Emollients ▪ Inform patient about underlying cause
Exfoliative dermatitis				
Severe, chronic skin inflammation characterized by redness and widespread erythema and scaling	▪ Usually, preexisting skin lesions progress to exfoliative stage, such as in contact dermatitis, drug reaction, lymphoma, or leukemia	▪ Generalized dermatitis, with acute loss of stratum corneum, and erythema and scaling ▪ Sensation of tight skin ▪ Hair loss ▪ Possible fever, sensitivity to cold, shivering, gynecomastia, and lymphadenopathy	▪ Diagnosis requires identification of the underlying cause.	▪ Severe cases: may require hospitalization with protective isolation and hygienic measures to prevent secondary bacterial infection ▪ Open wet dressings, with colloidal baths ▪ Mild lotions over topical steroids ▪ Maintenance of constant environmental temperature to prevent chilling or overheating ▪ Careful monitoring of

				renal and cardiac status ▪ Systemic antibiotics and steroids ▪ Other interventions as for atopic dermatitis
Stasis dermatitis A condition caused by impaired venous circulation and characterized by eczema of the legs with edema, hyperpigmentation, and persistent inflammation	▪ Secondary to peripheral vascular diseases affecting legs, such as recurrent thrombophlebitis and resultant chronic venous insufficiency	▪ Varicosities and edema common, but obvious vascular insufficiency not always present ▪ Usually affects the lower leg, just above internal malleolus, or sites of trauma or irritation ▪ Early signs: dusky red deposits of hemosiderin in skin, with itching and dimpling of subcutaneous tissue; later signs: edema, redness, and scaling of large area of legs ▪ Possible fissures, crusts, and ulcers	▪ Diagnosis requires positive history of venous insufficiency and physical findings such as varicosities.	▪ Measures to prevent venous stasis: avoidance of prolonged sitting or standing, use of support stockings, weight reduction in obese patients, and increasing of activity ▪ Corrective surgery for underlying cause ▪ After ulcer develops, encourage rest periods, with legs elevated; open wet dressings; Unna's boot (zinc gelatin dressing provides continuous

				pressure to affected areas); and antibiotics for secondary infection after wound culture

An important secondary cause of atopic dermatitis is irritation, which seems to change the epidermal structure, allowing immunoglobulin (Ig) E activity to increase. Consequently, chronic skin irritation usually continues even after exposure to the allergen has ended or after the irritation has been systemically controlled.

PEDIATRIC TIP

Atopic dermatitis is most common in infants, usually developing between ages 1 month and 1 year, commonly in those with strong

family histories of atopic disease. At least half of those cases clear by age 36 months. These children often acquire other atopic disorders as they grow older. Typically, this form of dermatitis flares and subsides repeatedly before finally resolving during adolescence. However, it can persist into adulthood.

In adults, atopic dermatitis is generally chronic or recurring.

Complications

- Altered pigmentation
- Lichenification
- Scarring

Signs and symptoms

Atopic skin lesions generally begin as erythematous areas on excessively dry skin.

PEDIATRIC TIP

In children, lesions typically appear on the forehead, cheeks, and extensor surfaces of the arms and legs.

In adults, lesions appear at flexion points (antecubital fossa, popliteal area, and neck).

During flare-ups, pruritus and scratching cause edema, crusting, and scaling. Eventually, chronic atopic lesions lead to multiple areas of dry, scaly skin, with white dermatographia, blanching, and lichenification.

Common secondary conditions associated with atopic dermatitis include viral, fungal, or bacterial infections, and ocular disorders.

Because of intense pruritus, the upper eyelid is commonly hyperpigmented and swollen, and a double fold occurs under the lower lid (Morgan-Dennie folds, Morgan folds, Dennie pleats, or Mongolian lines). Atopic cataracts are unusual but may develop between ages 20 and 40.

Kaposi's varicelliform eruption, a potentially fatal, generalized viral infection, may develop if the patient with atopic dermatitis comes in contact with a person who's infected with herpes simplex.

Diagnosis

A family history of allergy and chronic inflammation suggests atopic dermatitis. Typical distribution of skin lesions rules out other inflammatory skin lesions, such as diaper rash (lesions are confined to the diapered area), seborrheic dermatitis (no pigmentation changes, or lichenification occurs in chronic lesions), and chronic contact dermatitis (lesions affect hands and forearms, sparing antecubital and popliteal areas). Serum IgE levels are usually elevated.

Treatment

Effective treatment for atopic lesions consists of eliminating allergens and avoiding irritants, extreme temperature and humidity changes, and other precipitating factors; local and systemic measures relieve itching and

inflammation. Antihistamines relieve itching and induce more restful sleep. Topical application of a corticosteroid ointment, especially after bathing, often alleviates inflammation. Between steroid doses, application of a moisturizing cream can help retain moisture. Systemic corticosteroid therapy should be used only during extreme exacerbations. Topical tacrolimus and pimecrolimus (an immunosuppressant known as a *topical immunomodulator*) are new agents used in patients older than age 2 who are intolerant of or unresponsive to conventional therapy. Weak tar preparations and ultraviolet B light therapy are used to increase the thickness of the stratum corneum. Antibiotics are appropriate if a bacterial agent has been cultured.

Special considerations

- Warn the patient that drowsiness is possible with the use of antihistamines to relieve daytime itching. If nocturnal itching interferes with sleep, suggest methods for inducing natural sleep, such as drinking a glass of warm milk, to prevent overuse of sedatives.
- Complement medical treatment by helping the patient set up an individual schedule and plan for daily skin care. Instruct the patient to bathe in plain water, according to the severity of the lesions, and to bathe with a special nonfatty soap and tepid water (96° F [35.6° C]) but to avoid using any soap when lesions are acutely inflamed. Advise the patient to shampoo frequently and apply corticosteroid solution to the scalp afterward, to keep fingernails short to limit excoriation and secondary infections caused by scratching, and to lubricate his skin after a tub bath. Advise the patient to avoid using any perfume or makeup that causes burning or itching.
- To help clear lichenified skin, apply occlusive dressings (such as plastic film) intermittently. This treatment requires a physician's order, experience in dermatologic treatment, and can't be used in all treatment modalities.
- Inform the patient that irritants, such as detergents and wool, and emotional stress, exacerbate atopic dermatitis.
- Be careful not to show any anxiety or revulsion when touching the lesions during treatment. Help the patient accept his altered body image, and encourage him to

verbalize his feelings. Remember, coping with disfigurement is extremely difficult, especially for children and adolescents. Arrange for counseling, if necessary, to help the patient deal with this distressing condition more effectively.

MISCELLANEOUS DISORDERS

Toxic epidermal necrolysis

Toxic epidermal necrolysis (TEN) is a rare, severe skin disorder that causes epidermal erythema, superficial necrosis, and skin erosions. Mortality is high (30%), especially among debilitated and elderly patients. Reepithelialization is slow, and residual scarring is common. TEN primarily affects adults. Some experts consider TEN to be a maximal form of Stevens-Johnson syndrome (SJS), with SJS being a maximal variant of erythema multiforme major.

Causes and incidence

In 80% of cases, TEN is determined to result from a drug reaction—most commonly to sulfonamides, penicillins, barbiturates, hydantoins, procainamide, isoniazid, nonsteroidal anti-inflammatory drugs, or allopurinol. Numerous other drugs have also been implicated, although 5% of patients with TEN report no drug use. It may also result from chemical exposure, viral infection, mycoplasma pneumonia, or immunization.

TEN may reflect an immune response, or it may be related to overwhelming physiologic stress (coexisting sepsis, neoplastic diseases, and drug treatment).

The annual worldwide incidence of TENS is 1 to 3 cases for every 1 million people.

Complications

- Bronchopneumonia
- Pulmonary edema
- GI and esophageal hemorrhage

- Shock
- Renal failure
- Sepsis
- Disseminated intravascular coagulation

Signs and symptoms

Early symptoms include inflammation of the mucous membranes, a burning sensation in the conjunctivae, malaise, fever, and generalized skin tenderness. After such prodromal symptoms, TEN erupts in three phases:

- diffuse, erythematous rash
- vesiculation and blistering
- large-scale epidermal necrolysis and desquamation.

Large, flaccid bullae that rupture easily expose extensive areas of denuded skin, permitting both loss of tissue fluids and electrolytes and widespread systemic involvement.

Diagnosis

CONFIRMING DIAGNOSIS

Early diagnosis is very important and is based on the patient's clinical status at the peak stage of the disease. Nikolsky's sign (skin sloughs off with slight friction) is present in erythematous areas. Culture and Gram stain of lesions determine whether infection is present. Supportive findings include leukocytosis, elevated levels of alanine aminotransferase and aspartate aminotransferase, albuminuria, and fluid and electrolyte imbalances.

Exfoliative cytology and biopsy aid in ruling out erythema multiforme and exfoliative dermatitis.

Treatment

Treatment consists of transferring the patient to a burn center or an intensive care unit and providing I.V. fluid replacement to maintain fluid and electrolyte balance. Xenografts should be used to prevent pain and infection and to provide the framework for reepithelialization. High doses of I.V. immunoglobulins may halt progression if given early in the course of illness. Steroids may be appropriate initially, but should be discontinued as soon as healing occurs. Use of steroids may decrease survival rates only secondary to increased incidence

of infections and other complications. Necrotic skin should be débrided. The patient also should stop using suspected drugs.

Special considerations

- Frequently assess hematocrit and hemoglobin, electrolyte, serum protein, and blood gas levels.
- Monitor vital signs, central venous pressure, and urine output. Watch for signs of renal failure (decreased urine output) and bleeding. Report fever immediately, and obtain blood cultures and sensitivity tests promptly to detect and treat septic infection.
- Maintain skin integrity as much as possible. The patient shouldn't wear clothing and should be covered loosely to prevent friction and sloughing of skin. A low air-loss or air-fluidized bed is helpful.
- Administer analgesics as needed. Wounds will be virtually pain-free after the dermis is covered by the xenograft.
- Provide eye care hourly to remove exudate. Because ocular lesions are common, the ophthalmologist should examine the patent's eyes daily.
- Encourage the patient to wear a medical alert bracelet.

PREVENTION

- *Prevent secondary infection with appropriate precautions. Use systemic antibiotics for specific identified infections only.*
- *Ensure that suspected drugs are never administered.*

Warts

Warts, also known as *verrucae,* are common, benign, viral infections of the skin and adjacent mucous membranes. The prognosis varies: Some warts disappear readily with treatment; others necessitate more vigorous and prolonged treatment. Some warts demonstrate spontaneous resolution.

Causes and incidence

Warts are caused by infection with the human papillomavirus, a group of etherresistant, deoxyribonucleic acid-containing papovaviruses. Mode of transmission is probably through direct contact, but autoinoculation is possible.

Although their incidence is highest in children and young adults, warts may occur at any age.

Complications

- Scarring
- Secondary infection

Signs and symptoms

Clinical manifestations depend on the type of wart and its location:

- common (verruca vulgaris): rough, elevated, rounded surface; appears most frequently on extremities, particularly hands and fingers; most prevalent in children and young adults
- condyloma acuminatum (moist wart or genital wart): usually small, pink to red, moist, and soft; may occur singly or in large cauliflower-like clusters on the penis, scrotum, vulva, cervix, vagina, and anus; can also occur on oral mucosa following oral-genital exposure; considered a sexually transmitted disease
- digitate: fingerlike, horny projection arising from a pea-shaped base; occurs on scalp or near hairline
- filiform: single, thin, threadlike projection; commonly occurs around the face and neck

- flat (also known as *juvenile* or *verruca plana):* multiple groupings of up to several hundred slightly raised lesions with smooth, flat, or slightly rounded tops; common on the face, neck, chest, knees, dorsa of hands, wrists, and flexor surfaces of the forearms; usually occur in children but can affect adults; often linear distribution because of spread from scratching or shaving
- periungual: rough, irregularly shaped, elevated surface; occurs around edges of fingernails and toenails; when severe, may extend under nail and lift it off nail bed, causing pain
- plantar: slightly elevated or flat; occur singly or in large clusters (mosaic warts), primarily at pressure points of feet.

Diagnosis

CONFIRMING DIAGNOSIS

Visual examination usually confirms the diagnosis. 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.

Anal warts require anoscopy or sigmoidoscopy to rule out internal involvement, which may necessitate surgery. Women with vulvar lesions require examination of the vagina and cervix, including a Papanicolaou smear.

Treatment

Treatment for warts varies according to the location, size, number, pain level (present and projected), history of therapy, the patient's age, and compliance with treatment. Most persons eventually develop an immune response that causes warts to disappear spontaneously and require no treatment.

Treatment may include:

- Electrodesiccation and curettage—High-frequency electric current destroys the wart and is followed by surgical removal of dead tissue at the base and application of an antibiotic ointment (such as polysporin), covered with a bandage, for 48 hours. This method is effective for common, filiform and, occasionally, plantar warts. (See *Removing warts by electrosurgery*, page 768.)
- Cryotherapy—Liquid nitrogen kills the wart; the resulting dried blister is peeled off several days later. If initial treatment isn't successful, it can be repeated at 2- to 4-week intervals. This method is useful either for periungual warts or for common warts on the face, extremities, penis, vagina, or anus.
- Acid therapy (primary or adjunctive)— The patient applies plaster patches impregnated with acid (such as 40% salicylic acid plasters) or acid drops (such as 5% to 16.7% salicylic acid in flexible collodion or trichloroacetic or dichloroacetic acids), every 12 to 24 hours for 2 to 4 weeks. This method isn't recommended for areas where perspiration is heavy, for those parts that are likely to get wet, or for exposed body parts where patches are cosmetically undesirable.
- 25% podophyllin in compound with tincture of benzoin (for venereal warts)— The podophyllin solution is applied on moist warts. The patient must lie still while it dries, leave it on for 4 hours, and then wash it off with soap and water. Treatment may be repeated every 3 to 4 days and, in some cases, must be left on a maximum of 24 hours, depending on the patient's tolerance. Avoid using this drug on pregnant patients.

During acid or podophyllin therapy, the patient should protect the surrounding area with petroleum jelly or sodium bicarbonate (baking soda). A small amount of 25% to 50% trichloroacetic acid (for venereal warts) is applied to the wart. After the wart turns white, the acid is neutralized with baking soda or water.

- Carbon dioxide laser therapy—This treatment has successfully treated genital warts.

The use of antiviral drugs is under investigation; suggestion and hypnosis are occasionally successful, especially with children. Patients can apply topical imiquimod cream to sites that aren't thickly keratinized. It's

applied at bedtime three times per week. Imiquimod can be used alternately with a topical retinoid such as tazarotene, which may increase effectiveness.

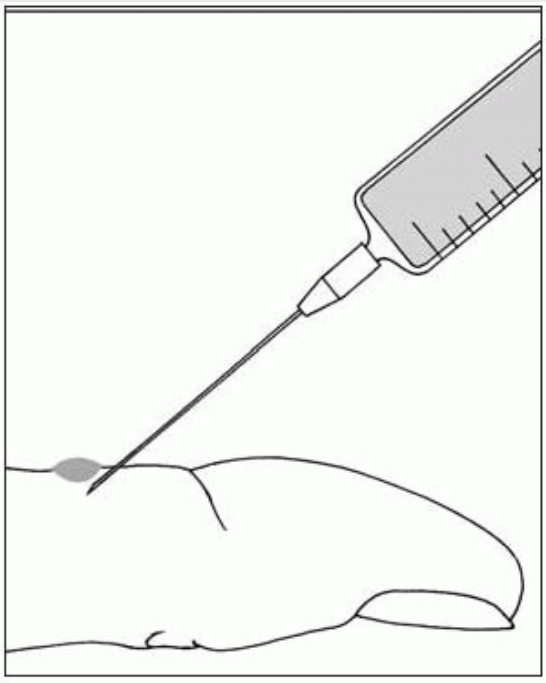
Occlusion may be beneficial to persistent warts.

Special considerations

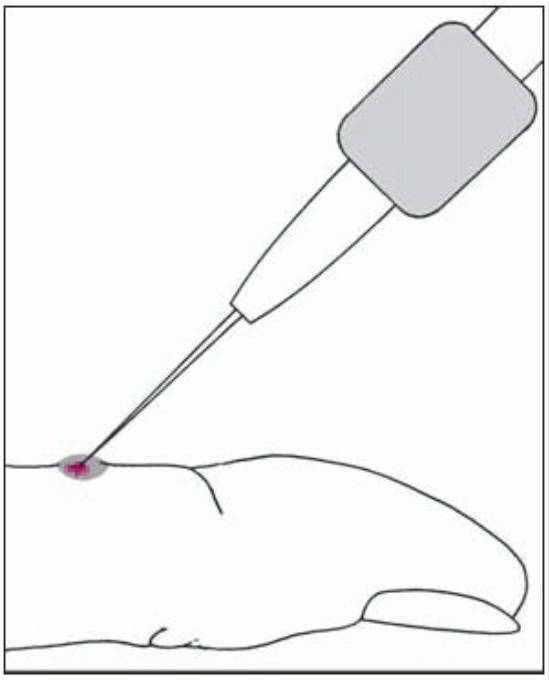
Conscientious adherence to prescribed therapy is essential. The patient's sexual partner may also require treatment. Encourage the patient to seek counseling if applicable.

REMOVING WARTS BY ELECTROSURGERY

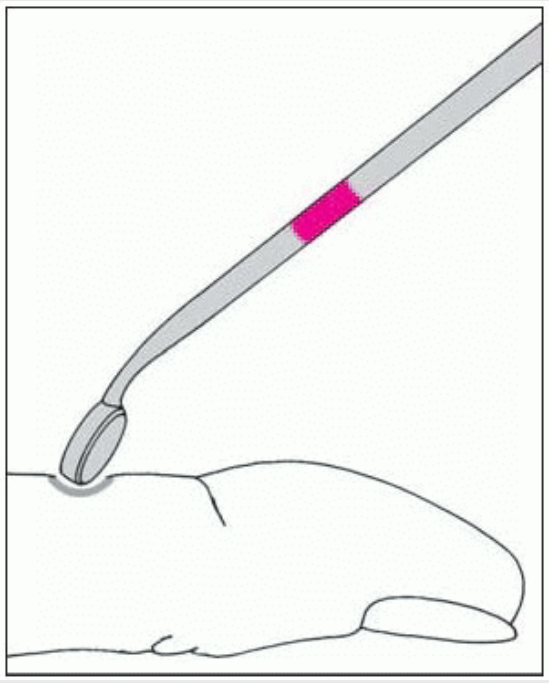
1. Injection of 1% to 2% lidocaine under and around the wart, avoiding the wart itself.



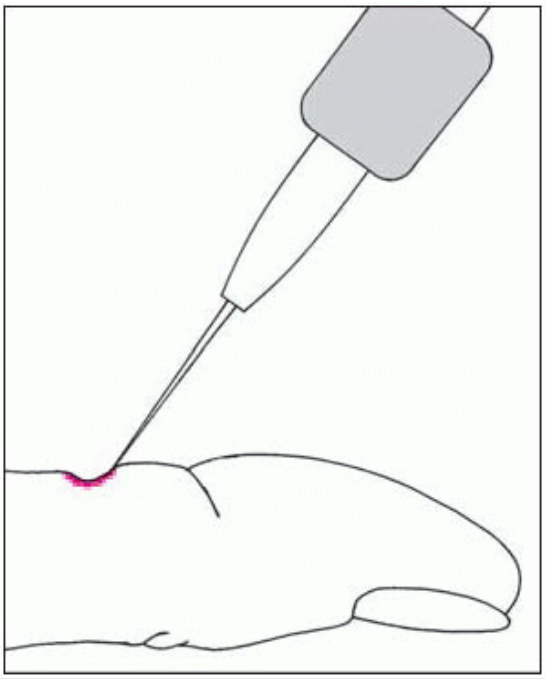
2. Electrodesiccation of the wart.



3. Removal of the wart tissue with a curette and small, curved scissors.



4. Light desiccation of the area to control bleeding and prevent recurrence.



Psoriasis

Psoriasis is a chronic, recurrent disease marked by epidermal proliferation. Its lesions, which appear as erythematous papules and plaques covered with silvery scales, vary widely in severity and distribution. Psoriasis is characterized by recurring partial remissions and exacerbations. Flare-ups are usually related to specific systemic and environmental factors but may be unpredictable; they can usually be controlled with therapy.

Causes and incidence

The tendency to develop psoriasis is 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. Onset of the disease is also influenced by environmental factors. Trauma can trigger the isomorphic effect or Koebner's phenomenon, in which lesions develop at sites of injury. Infections, especially those resulting from beta-hemolytic streptococci, may cause a flare of guttate (drop-shaped) lesions. Other contributing

factors include pregnancy, endocrine changes, climate (cold weather tends to exacerbate psoriasis), and emotional stress.

Generally, a skin cell takes 14 days to move from the basal layer to the stratum corneum, where, after 14 days of normal wear and tear, it's sloughed off. The life cycle of a normal skin cell is 28 days, compared with only 4 days for a psoriatic skin cell. This markedly shortened cycle doesn't allow time for the cell to mature. Consequently, the stratum corneum becomes thick and flaky, producing the cardinal manifestations of psoriasis.

Psoriasis affects approximately 2% of the population in the United States, and incidence is higher in whites than other races. Although this disorder is most common in young adults, it may strike at any age, including infancy.

Complications

- Depression
- Infection

PSORIATIC PLAQUES

In this patient with psoriasis, plaques consisting of silver scales cover a large area of the face.

Signs and symptoms

The most common complaint of the patient with psoriasis is itching and, occasionally, pain from dry, cracked, encrusted lesions. Psoriatic lesions are erythematous and usually form well-defined plaques, sometimes covering large areas of the body. (See *Psoriatic plaques*.) Such lesions most commonly appear on the scalp, chest, elbows, knees, shins, back, and buttocks. The plaques consist of characteristic silver scales that either flake off easily or can thicken, covering the lesion. Removal of psoriatic scales frequently produces fine bleeding points (Auspitz sign). Occasionally, small guttate lesions appear, either alone or with plaques; these lesions are typically thin and erythematous, with few scales.

Widespread shedding of scales is common in exfoliative or erythrodermic psoriasis and may also develop in chronic psoriasis.

Rarely, psoriasis becomes pustular, taking one of two forms. In localized pustular (Barber's) psoriasis, pustules appear on the palms and soles and remain sterile until opened. In generalized pustular (*von Zumbusch's)* psoriasis, which often occurs with

fever, leukocytosis, and malaise, groups of pustules coalesce to form lakes of pus on red skin. These pustules also remain sterile until opened and commonly involve the tongue and oral mucosa.

In about 30% of patients, 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.

Some patients with psoriasis develop arthritic symptoms (psoriatic arthritis), usually in one or more joints of the fingers or toes, or sometimes in the sacroiliac joints, which may progress to spondylitis. Such patients may complain of morning stiffness. Joint symptoms show no consistent linkage to the course of the cutaneous manifestations of psoriasis; they demonstrate remissions and exacerbations similar to those of rheumatoid arthritis.

Diagnosis

Diagnosis depends on patient history, appearance of the lesions and, if needed, the results of skin biopsy. Typically, serum uric acid level is elevated as a result of accelerated nucleic acid degradation, but indications of gout are absent. HLA-Cw6, B-13, and Bw-57 may be present in early-onset psoriasis. Sudden onset of psoriasis may be associated with human immunodeficiency virus.

Treatment

Treatment depends on the type of psoriasis, the extent of the disease and the patient's response to it, and what effect the disease has on the patient's lifestyle. No permanent cure exists, and all methods of treatment are merely palliative. Ideally, all patients should see a dermatologist at least once.

Removal of psoriatic scales necessitates application of occlusive ointment bases, such as petroleum jelly, salicylic acid preparations, or preparations containing urea. Baker P & S liquid (phenol, sodium chloride, and liquid paraffin), applied to the scalp at bedtime, or liquor carbonis detergens in Nivea oil applied for 6 to 8 hours, is also effective. Shampoo or tar-based preparations are also used. These medications soften the scales, which can then be removed by scrubbing them carefully with a soft brush while bathing. Some preparations, such as tar-based preparations, can be used in whirlpools for extensively involved areas.

Methods to retard rapid cell production include exposure to ultraviolet light (UVB or natural sunlight) to the point of minimal erythema. Tar preparations or crude coal tar itself may be applied to affected areas about 15 minutes before exposure or may be left on overnight and wiped off the next morning. A thin layer of petroleum jelly may be applied before UVB exposure (the most common treatment for generalized psoriasis). Exposure time can increase gradually. Outpatient or day treatment with UVB prevents long hospitalizations and prolongs remission.

Steroid creams and ointments are useful to control psoriasis. A potent fluorinated steroid works well, except on the face and intertriginous areas. These creams require application twice daily, preferably after bathing to facilitate absorption, and overnight use of occlusive dressings, such as plastic wrap, plastic gloves or booties, or a vinyl exercise suit (under direct medical or nursing supervision). Small, stubborn plaques may require intralesional steroid injections. Anthralin, combined with a

paste mixture, may be used for well-defined plaques but must not be applied to unaffected areas because it causes injury and stains normal skin. Apply petroleum jelly around the affected skin before applying anthralin. Commonly used concurrently with steroids, anthralin is applied at night and steroids during the day.

In a patient with severe chronic psoriasis, the Goeckerman regimen—which combines tar baths and UVB treatments—may help achieve the longest remission and clear the skin in 3 to 5 weeks. The Ingram technique is a variation of this treatment, using anthralin instead of tar. A therapy called PUVA combines administration of psoralens with exposure to high-intensity UVA. As a last resort, a cytotoxin, usually methotrexate or cyclosporine, an immunosuppressant, may help severe, refractory psoriasis.

Etretinate, a retinoid compound, is effective in treating extensive cases of psoriasis. However, because this drug is a strong teratogen, it's unsafe for use in women of childbearing age. It also has numerous adverse effects that many patients find intolerable. Tacarotene, a newer topical retinoid, combined with a medium-strength topical corticosteroid is also effective.

Low-dose antihistamines, oatmeal baths, emollients, and open wet dressings may help relieve pruritus. Aspirin and local heat help alleviate the pain of psoriatic arthritis; severe cases may require nonsteroidal anti-inflammatory drugs.

Therapy for psoriasis of the scalp consists of a tar shampoo followed by application of a steroid lotion; ketoconazole and anthralin may also be effective. No effective treatment exists for psoriasis of the nails.

Special considerations

Design your patient's care plan to include patient teaching and careful monitoring for adverse effects of therapy.

- Make sure the patient understands his prescribed therapy; provide written instructions to avoid confusion. Teach correct application of prescribed ointments, creams, and lotions. A steroid cream, for example, should be applied in a thin film and rubbed gently into the skin until the cream disappears. All topical medications, especially

those containing anthralin and tar, should be applied with a downward motion to avoid rubbing them into the follicles. Gloves must be worn because anthralin stains and injures the skin. After application, the patient may dust himself with powder to prevent anthralin from rubbing off on his clothes. Warn the patient never to put an occlusive dressing over anthralin. Suggest use of mineral oil, then soap and water, to remove anthralin. Caution the patient to avoid scrubbing his skin vigorously, to prevent Koebner's phenomenon. If a medication has been applied to the scales to soften them, suggest the patient use a soft brush to remove them.

- Watch for adverse effects, especially allergic reactions to anthralin, atrophy and acne from steroids, and burning, itching, nausea, and squamous cell epitheliomas from PUVA. Initially, evaluate the patient on methotrexate weekly, then monthly for red blood cell, white blood cell, and platelet counts because cytotoxins may cause hepatic or bone marrow toxicity. Liver biopsy may be done to assess the effects of methotrexate.
- Caution the patient receiving PUVA therapy to stay out of the sun on the day of treatment, and to protect his eyes with sunglasses that screen UVA for 24 hours after treatment. Tell him to wear goggles during exposure to this light.
- Be aware that psoriasis can cause psychological problems. Assure the patient that psoriasis isn't contagious and, although exacerbations and remissions occur, they're controllable with treatment. However, be sure he understands there's no cure. Also, because stressful situations tend to exacerbate psoriasis, help the patient learn to cope with these situations. Explain the relationship between psoriasis and arthritis, but point out that psoriasis causes no other systemic disturbances. Refer all patients to the National Psoriasis Foundation, which provides information and directs patients to local chapters.

Lichen planus

A benign but pruritic skin eruption, lichen planus is a relatively rare disorder that usually produces scaling, purple papules marked by white lines or spots. The features of these lesions are called the “4 Ps”—purple, polygonal, pruritic, and papule.

In most patients, lichen planus resolves spontaneously in 6 to 18 months. In a few, chronic lichen planus may persist for several years.

Causes and incidence

The cause of lichen planus is unknown. Eruptions similar to lichen planus have been induced by arsenic, bismuth, gold, quinidine, propranolol, statins, and naproxen. Exposure to developers used in color photography may likewise cause an eruption that's indistinguishable from lichen planus.

Lichen planus is found in all geographic areas, with equal distribution among races.

Eruptions of lesions with features characteristic of lichen planus occur most often in middle-age people and are uncommon in young and elderly people.

Signs and symptoms

Lichen planus may develop suddenly or insidiously. Initial lesions commonly appear on the arms or legs (generally on the wrist and medial sides of the thighs) and evolve into the generalized eruption of flat, glistening, purple papules marked with white lines or spots (Wickham's striae). These lesions may be linear from scratching or may coalesce into plaques. Lesions often affect the mucous membranes (especially the buccal mucosa), male genitalia and, less often, the nails. These lesions are painful, especially when ulcers develop. Mild to severe pruritus is common.

Diagnosis

CONFIRMING DIAGNOSIS

Although characteristic skin lesions usually establish the diagnosis of lichen planus, confirmation may require a skin biopsy.

Treatment

Treatment is essentially symptomatic. The goal of therapy is to relieve itching with topical fluorinated steroids and occlusive dressings,

intralesional injections of steroids, oatmeal baths, and antihistamines. Erosive oral lesions should be treated with triamcinolone acetonide in Orabase twice daily. Generalized severely pruritic skin lesions may be treated with systemic corticosteroids. An initial dosage of oral prednisone may be prescribed; thereafter, the dosage is decreased by approximately one-third each week. If the patient experiences a recurrence of itching after the drug is discontinued, he'll be given a low dose every other morning. If a drug is suspected as the cause, it should be discontinued.

Special considerations

- Administer medications as indicated, and inform the patient of possible adverse effects, especially drowsiness produced by antihistamines.
- Provide emotional support and reassure the patient that lichen planus, although annoying, is usually a benign, self-limiting condition, although lesions may persist for months or years.

Corns and calluses

Usually located on areas of repeated trauma (especially the feet), corns and calluses are acquired skin conditions marked by hyperkeratosis of the stratum corneum. The prognosis is good with proper foot care.

Causes and incidence

A corn (also known as a *clavus*) is a hyperkeratotic area that usually results from external pressure, such as that from ill-fitting shoes or, less commonly, from internal pressure, such as that caused by a protruding underlying bone (due to arthritis for example). A callus is an area of thickened skin, generally found on the foot or hand, produced by external pressure or friction. Persons whose activities produce repeated trauma (for example, manual laborers or guitarists) commonly develop calluses.

The severity of a corn or callus depends on the degree and duration of trauma.

Complication

- Secondary infection

Signs and symptoms

Both corns and calluses cause pain through pressure placed on underlying tissue by localized thickened skin. Corns contain a central keratinous core, are smaller and more clearly defined than calluses, and are usually more painful. The pain they cause may be dull and constant or sharp when pressure is applied. “Soft” corns are caused by the pressure of a bony prominence. They appear as whitish thickenings and are commonly found between the toes, most often in the fourth interdigital web. “Hard” corns are sharply delineated and conical, and appear most frequently over the dorsolateral aspect of the fifth toe.

Calluses have indefinite borders and may be quite large. They usually produce dull pain on pressure, rather than constant pain. Although calluses commonly appear over plantar warts, they're distinguished from these warts by normal skin markings.

Diagnosis

Diagnosis depends on careful physical examination of the affected area and on patient history revealing chronic trauma.

Treatment

Surgical debridement may be performed to remove the nucleus of a corn, usually under a local anesthetic. In intermittent debridement, keratolytics—usually 40% salicylic acid plasters—are applied to affected areas. Injections of corticosteroids beneath the corn may be necessary to relieve pain. However, the simplest and best treatment is essentially preventive—avoidance of trauma. Corns and calluses disappear after the source of trauma has been removed. Metatarsal pads may redistribute the weight-bearing areas of the foot; corn pads may prevent painful pressure. (See *Aids for relieving painful pressure*.)

Patients with persistent corns or calluses require referral to a podiatrist or dermatologist; those with corns or calluses caused by a bony malformation, as in arthritis, require orthopedic consultation.

Special considerations

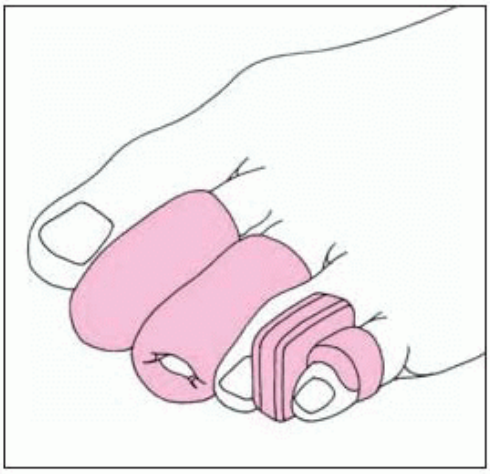
- Teach the patient how to apply salicylic acid plasters. Make sure the plaster is large enough to cover the affected area. Place the sticky side down on the foot; then cover the plaster with adhesive tape. Plasters are usually taken off after an overnight application but may be left in place for as long as 7 days.
- After removing the plaster, the patient should soak the area in water and abrade the soft, macerated skin with a towel or pumice stone. He should then reapply the plaster and repeat the entire procedure until he has removed all the hyperkeratotic skin.
- Warn the patient against removing corns or calluses with a sharp instrument such as a razor blade.

PREVENTION

- *Advise the patient to wear properly fitted shoes. Suggest the use of metatarsal or corn pads to relieve pressure. Refer to a podiatrist, dermatologist, or orthopedist, if necessary.*
- *Assure the patient that good foot care can correct this condition.*
- *Wear padded gloves when using hand tools.*
- *Keep hands and feet soft by applying moisturizer.*

AIDS FOR RELIEVING PAINFUL PRESSURE

Both metatarsal and corn pads can help relieve painful pressure. Commercial products available include, from left to right, foam toe cap, foam toe sleeve, soft corn shield, and hard corn (fifth toe) shield.



Pityriasis rosea

An acute, self-limiting, inflammatory skin disease, pityriasis rosea usually produces a “herald” patch—which usually goes undetected—followed by a generalized eruption of papulosquamous lesions.

Causes and incidence

The cause of pityriasis rosea is unknown, but the brief course of the disease and the virtual absence of recurrence suggest a viral agent (herpes virus 7 is suspected) or an autoimmune disorder.

Although this noncontagious disorder may develop at any age, it's most apt to occur in adolescents and young adults. Incidence rises in the spring and fall. In the United States, less than 3% of people have this disease.

Complication

- Secondary infection

Signs and symptoms

Pityriasis typically begins with an erythematous “herald” patch, which may appear anywhere on the body, although it occurs most commonly on the trunk. Although this slightly raised, oval lesion is about 2 to 6 cm in diameter, approximately 25% of patients don't notice it. A few days to

several weeks later, yellow-tan or erythematous patches with scaly edges (about 0.5 to 1 cm in diameter) erupt on the trunk and extremities—and, rarely, on the face, hands, and feet in adolescents. Eruption continues for 7 to 10 days, and the patches persist for 2 to 6 weeks. Occasionally, these patches are macular, vesicular, or urticarial. A characteristic of this disease is the arrangement of lesions, which produces a pattern similar to that of a pine tree. Accompanying pruritus, if present, is usually mild but may be severe.

Diagnosis

Characteristic skin lesions support the diagnosis. Differential diagnosis must also rule out secondary syphilis (through serologic testing), dermatophytosis, and drug reaction.

Treatment

Treatment focuses on relief of pruritus, with emollients, oatmeal baths, antihistamines, topical steroids, and occasionally exposure to ultraviolet light or sunlight. Rarely, if inflammation is severe, systemic corticosteroids may be required.

Special considerations

- Reassure the patient that pityriasis rosea isn't contagious, spontaneous remission usually occurs in 4 to 12 weeks, and lesions generally don't recur.
- Urge the patient not to scratch. Advise him to avoid hot baths because they may intensify itching. Encourage the use of antipruritics.

Hyperhidrosis

Primary hyperhidrosis is the excessive secretion of sweat from the eccrine glands. It usually occurs in the axillae (typically after puberty) and on the palms and soles (often starting during infancy or childhood). Abnormal and excessive heat loss can occur, causing most patients to have body temperatures less than 98.6° F (37°C). In addition, secondary hyperhidrosis commonly occurs as a clinical manifestation of an underlying disorder.

Causes and incidence

Genetic factors may contribute to the development of primary hyperhidrosis and, in susceptible individuals, emotional stress appears to be the most prominent cause, although most patients aren't anxious. Increased central nervous system (CNS) impulses may provoke excessive release of acetylcholine, producing a heightened sweat response. Exercise and a hotclimate can cause profuse sweating in these patients. Certain drugs (such as antipyretics, emetics, meperidine, and anticholinesterases) and certain foods (such as tomato sauce, chocolate, coffee, and spicy foods) have been known to increase sweating.

Secondary hyperhidrosis may be a result of infections and chronic diseases, such as tuberculosis, malaria, or lymphoma, may cause excessive nighttime sweating. A person with diabetes commonly demonstrates hyperhidrosis during a hypoglycemic crisis. Other predisposing conditions include hyperthyroidism, pheochromocytomas; cardiovascular disorders, such as shock or heart failure; CNS disturbances (generally lesions of the hypothalamus); withdrawal from drugs or alcohol; menopause; and Graves' disease.

Primary hyperhidrosis occurs in 2% to 3% of people in the U.S.

Complication

- Fungal infections

Signs and symptoms

Axillary hyperhidrosis frequently produces such extreme sweating that patients often

ruin their clothes in 1 day and develop contact dermatitis from clothing dyes; similarly, hyperhidrosis of the soles can easily damage a pair of shoes. Profuse sweating from both the soles and palms hinders the patient's ability to work and interact socially. Patients with this condition often report increased emotional strain.

Diagnosis

CONFIRMING DIAGNOSIS

Clinical observations and patient history confirm hyperhidrosis.

Treatment

The treatment of choice is application of 20% aluminum chloride in absolute ethanol. (Most antiperspirants contain a 5% solution.) Formaldehyde may also be used but may lead to allergic contact sensitization. Glutaraldehyde produces less contact sensitivity than formaldehyde but stains the skin; it's used more often on the feet than on the hands, as a soak or applied directly several times a week and then weekly as needed. Botulinum toxin type A (Botox) is helpful in temporarily blocking the nerves that stimulate sweating. It's injected in small doses in the axillae. It may cause side effects, such as pain at the injection site or flulike symptoms.

Iontophoresis (low-level electric current applied locally to skin surfaces) reduces sweat secretion at the site. Repeated treatments will be necessary for sustained relief.

Therapy sometimes includes anticholinergics, except in patients with glaucoma or prostatic hypertrophy. Severe hyperhidrosis unresponsive to conservative therapy may require local axillary removal of sweat glands or, as a last resort, an endoscopic thoracic sympathectomy, which stops the signal for excessive sweating. The procedure is performed under general anesthesia.

Special considerations

- Provide support and reassurance because hyperhidrosis may be socially embarrassing.
- Tell the patient to apply aluminum chloride in absolute ethanol nightly to dry axillae, soles, or palms. The area should be covered with plastic wrap for 6 to 8 hours, preferably overnight, then washed with soap and water. Tell him to repeat this procedure for several nights, until profuse daytime sweating subsides. Frequency of treatments can then be reduced.

- Advise the patient with hyperhidrosis of the soles to wear leather sandals and white or colorfast cotton socks.

Pressure ulcers

Pressure ulcers, commonly called *pressure sores* or *bedsores,* are localized areas of cellular necrosis that occur most often in the skin and subcutaneous tissue over bony prominences. These ulcers may be superficial, caused by local skin irritation with subsequent surface maceration, or deep, originating in underlying tissue. Deep lesions typically go undetected until they penetrate the skin; but, by then, they've usually caused subcutaneous damage.

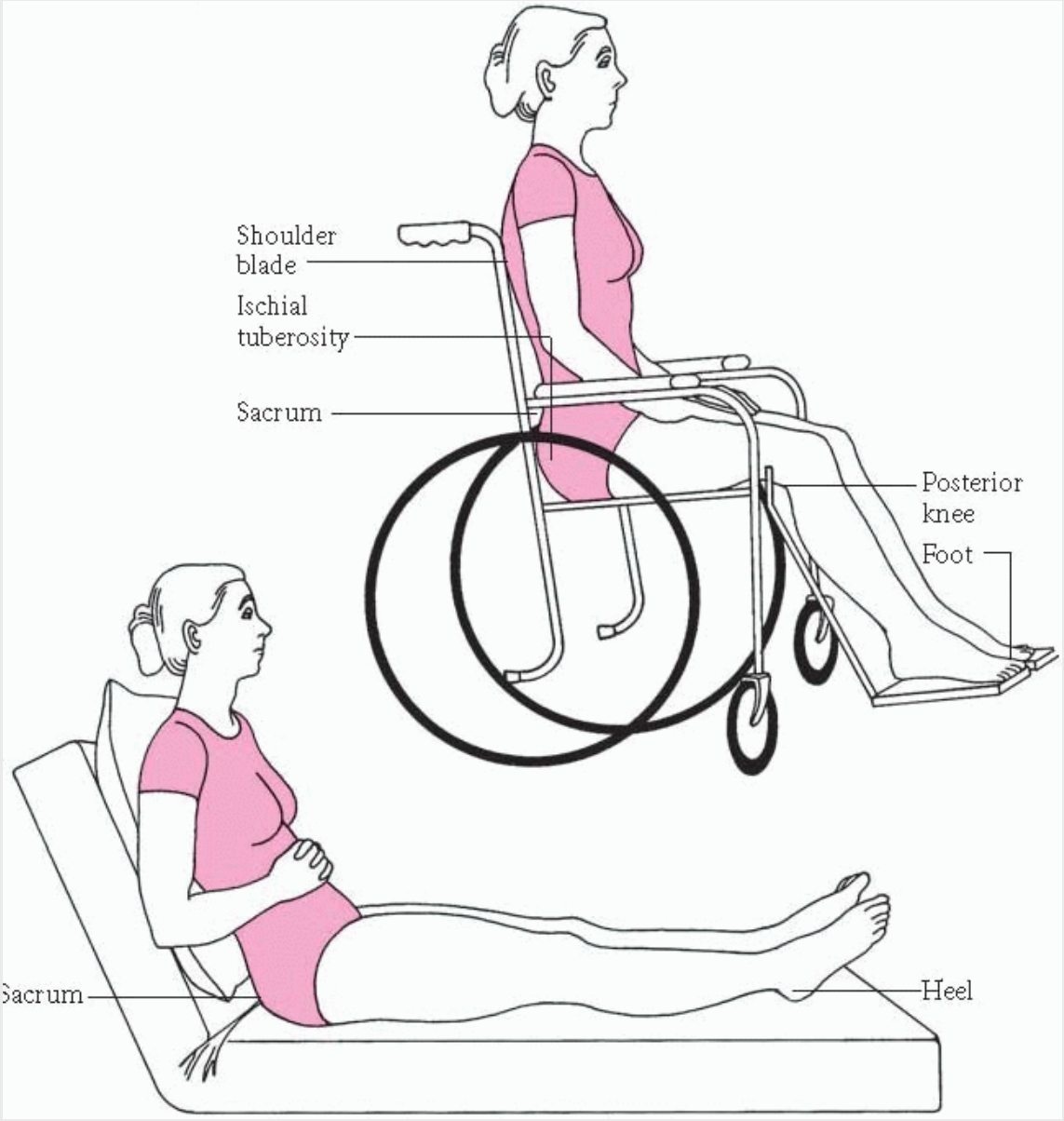
Causes and incidence

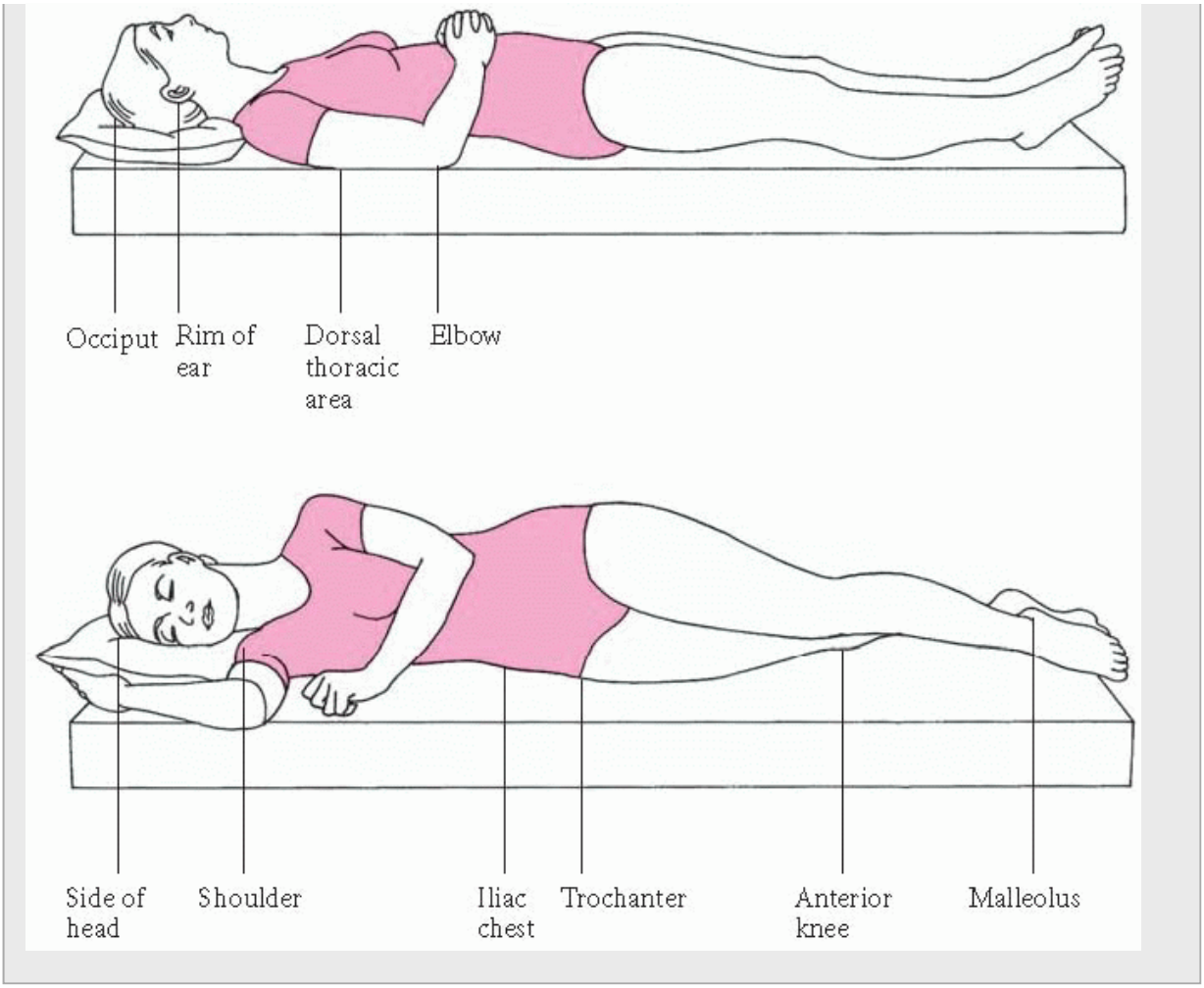
Most pressure ulcers are caused by pressure, particularly over bony prominences, that interrupts normal circulatory function, leading to ischemia of the underlying structures of skin, fat, and muscles. (See *Pressure points: Common sites of pressure ulcers*, pages 776 and 777.) The intensity and duration of such pressure govern the severity of the ulcer; pressure exerted over an area for a moderate period (1 to 2 hours) produces tissue ischemia and increased capillary pressure, leading to edema and multiple small-vessel thromboses. An inflammatory reaction gives way to ulceration and necrosis of ischemic cells. In turn, necrotic tissue predisposes to bacterial invasion and subsequent infection.

The patient's position determines the pressure exerted on the tissues. For example, if the head of the bed is elevated, or the patient assumes a slumped position, gravity pulls his weight downward and forward. This shearing force causes deep ulcers due to ischemic changes in the muscles and subcutaneous tissues, and occurs most often over the sacrum and ischial tuberosities.

PRESSURE POINTS: COMMON SITES OF PRESSURE ULCERS

Pressure ulcers may develop in any of these 16 pressure points. To prevent sores, reposition the patient every 1 to 2 hours, and carefully check for any change in the patient's skin tone.


Shoulder blade
Ischial tuberosity
Sacrum
Posterior knee
Foot
Sacrum
Heel



Predisposing conditions for pressure ulcers include altered mobility, inadequate nutrition (leading to weight loss, subsequent reduction of subcutaneous tissue and muscle bulk and, possibly, a poorly functioning immune system), and a breakdown in skin or subcutaneous tissue (as a result of edema, incontinence, fever, pathologic conditions, or obesity).

Pressure ulcers occur in 10% to 17% of all hospitalized patients and 20% to 40% of all nursing home patients. Patients living at home aren't free from risk, either: 20% of all pressure ulcers occur in the home. In the United States, there are approximately 2 million new cases of pressure ulcers diagnosed every year.

Complications

- Bacteremia
- Fluid and electrolyte loss

- Septicemia

Signs and symptoms

Pressure ulcers commonly develop over bony prominences. Early features of superficial lesions are shiny, erythematous changes over the compressed area, caused by localized vasodilation when pressure is relieved. Superficial erythema progresses to small blisters or erosions and, ultimately, to necrosis and ulceration.

An inflamed area on the skin's surface may be the first sign of underlying damage when pressure is exerted between deep tissue and bone. Bacteria in a compressed site cause inflammation and, eventually, infection, which leads to further necrosis. A foul-smelling, purulent discharge may seep from a lesion that penetrates the skin from beneath. Infected, necrotic tissue prevents healthy granulation of scar tissue; a black eschar may develop around and over the lesion. (See *Pressure ulcer staging,* pages 778 and 779.)

PRESSURE ULCER STAGING

The National Pressure Ulcer Advisory Panel has updated the staging of pressure ulcers to include the original four stages but also has added two other stages called *suspected deep tissue injury* and *unstageable*.

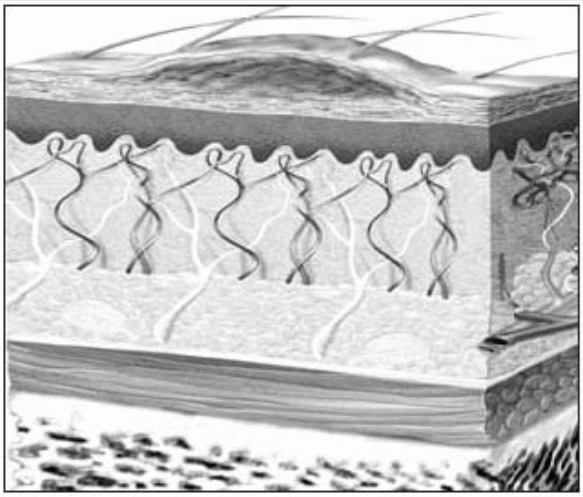
Suspected deep tissue injury

Suspected deep tissue injury involves maroon or purple intact skin or a blood-filled blister due to damage from shearing or pressure on the underlying soft tissue. Before the discoloration occurs, the area may be painful, mushy or firm or boggy, warmer or cooler as compared to other tissue.

Stage I

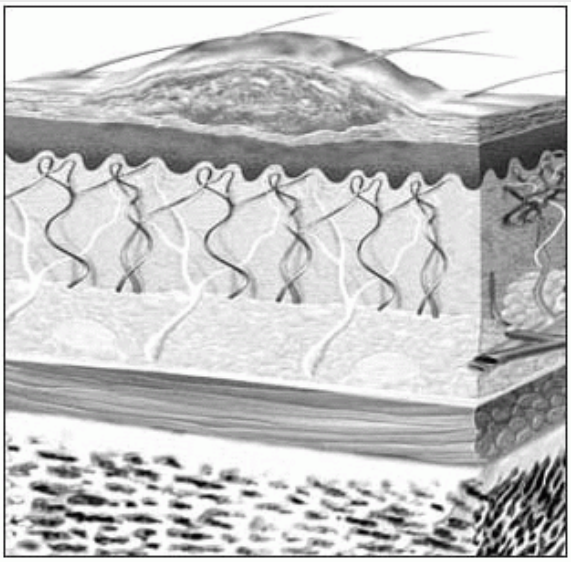
A stage I pressure ulcer is an area of intact skin that does not blanch and is usually over a bony prominence. Skin that is darkly pigmented may not show blanching but its

color may differ from surrounding area. The area may be painful, firm or soft, or warmer or cooler when compared to the surrounding tissue.



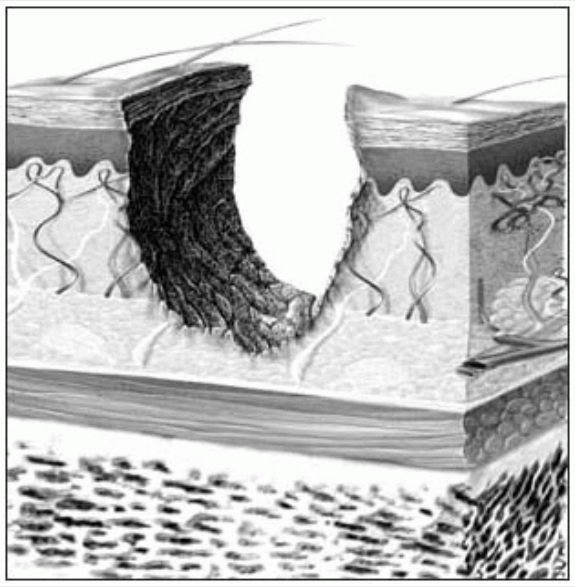
Stage II

A stage II pressure ulcer is a superficial partial-thickness wound that presents clinically a shallow and open ulcer without slough and with a red and pink wound bed. This term shouldn't be used to describe perineal dermatitis, maceration, tape burns, skin tears or excoriation, only an abrasion, a blister, or a shallow crater that involves the epidermis and dermis.



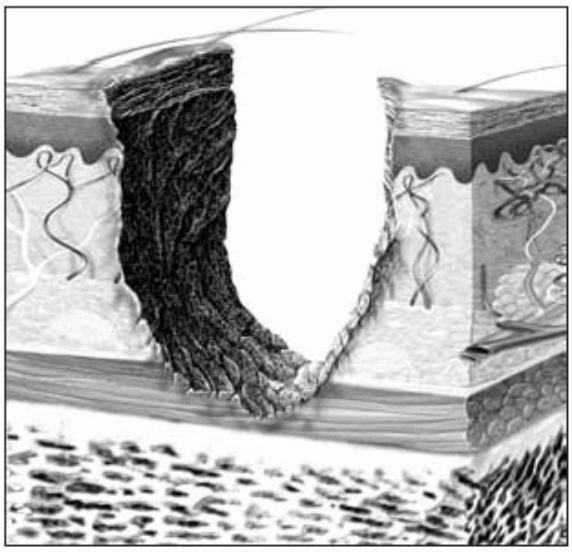
Stage III

A stage III pressure ulcer is a full-thickness wound with tissue loss. The subcutaneous tissue may be visible but muscle, tendon or bone is not exposed. Slough may be present but it does not hide the depth of the tissue loss. Undermining and tunneling may be present.



Stage IV

A stage IV pressure ulcer involves full-thickness skin loss with exposed muscle, bone, and tendon. Eschar and sloughing may be present as well as undermining and tunneling.



Unstageable
An unstageable pressure ulcer involves full thickness tissue loss. The base of the ulcer is covered by yellow, tan, gray, green, or brown slough or tan, brown or black eschar. Some may have both slough and eschar. The pressure cannot be staged until enough eschar or slough is removed to expose the base of the wound.

Diagnosis

Pressure ulcers are obvious on physical examination. Wound culture and sensitivity testing of the exudate in the ulcer identify infecting organisms and antibiotics that may be needed. If severe hypoproteinemia is suspected, total serum protein values and serum albumin studies may be appropriate.

Treatment

Successful treatment must relieve pressure on the affected area, keep the area clean and dry, and promote healing. (See *Preventing pressure ulcers*, page 780.)

Special considerations

- During each shift, check the skin of bedridden patients for possible changes in color, turgor, temperature, and sensation. Examine an existing ulcer for any change

  in size or degree of damage. When using pressure relief aids or topical agents, explain their function to the patient.
- Clean open lesions with normal saline solution. Dressings, if needed, should be porous and lightly taped to healthy skin. Debridement of necrotic tissue may be necessary to allow healing. One method is to apply open wet dressings and allow them to dry on the ulcer. Removal of the dressings mechanically débrides exudate and necrotic tissue. Other methods include surgical debridement with a fine scalpel blade and chemical debridement using proteolytic enzyme agents.
- Encourage adequate intake of food and fluids to maintain body weight and promote healing. Consult with the dietary department to provide a diet that promotes granulation of new tissue. Encourage the debilitated patient to eat frequent, small meals that are rich in protein, iron, calories,

  and vitamin C to promote healing. Assist weakened patients with their meals.

PREVENTION

PREVENTING PRESSURE ULCERS

Prevent pressure ulcers by repositioning the bedridden patient at least every 2 hours around the clock. To minimize the effects of a shearing force, use a footboard and raise the head of the bed to an angle not exceeding 60°. Also, use a draw or pull sheet to turn the patient or to pull him up. Keep the patient's knees slightly flexed for short periods. Perform passive range-of-motion exercises, or encourage the patient to do active exercises if possible.

Pressure relief aids

To prevent pressure ulcers in immobilized patients, use pressure relief aids on their beds.

- *Gel flotation pads* disperse pressure over a greater skin surface area; convenient and adaptable for home and wheelchair use.
- *Alternating pressure mattress* contains tubelike sections, running lengthwise, that deflate and reinflate, changing areas of pressure. Use mattress with a single untucked sheet because layers of line decrease its effectiveness.
- *Convoluted foam mattress* minimizes area of skin pressure with its alternating areas of depression and elevation: soft, elevate foam areas cushion skin; depressed areas relieve pressure. This mattress should be used with a single, loosely tucked sheet and is adaptable for home and wheelchair use. If the patient is incontinent, cover the mattress with the provided plastic sleeve.
- *Spanco* mattress has polyester fibers with silicon tubes to decrease pressure without limiting the patient's position. It has no weight limitations.
- *Sheepskin* is soft, dry, absorbent, and easy to clean. It should be in direct contact with the patient's skin. It's available in sizes to fit elbows and heels and is adaptable for home use.
- *Air-fluidized bed* supports the patient at a subcapillary pressure point and provides a warm, relaxing, therapeutic airflow. It eliminates friction and maceration.
- *Low air-loss beds*, such as Flexicare and Accucare, slow the drying of any saline soaks, and elderly patients often experience less disorientation than with high air-loss beds. The head of the bed can be elevated so there's less chance of aspiration, especially in patients who require tube feeding. Patients can get out of bed more easily on low air-loss surfaces.

Skin care

Provide meticulous skin care. Keep the skin clean and dry without the use of harsh soaps. Gently massaging the skin around the affected area—not on it— promotes healing. Thoroughly rub moisturizing lotions into the skin to prevent maceration of the skin surface. Change bed linens frequently for patients who are diaphoretic or incontinent. Use a fecal incontinence bag for incontinent patients.

Skin-damaging agents to avoid include:

- harsh alkaline soaps
- alcohol-based products (can cause vasoconstriction)
- tincture of benzoin (may cause painful erosions)
- hexachlorophene (may irritate the central nervous system)
- petroleum gauze.

Topical dressings

Types of topical dressings that aid in prevention and treatment of pressure ulcers include:

- transparent films
- hydrocolloid dressings
- hydrogel dressings
- foam dressings
- calcium alginate dressings
- gauze dressings.

Selected references

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