Would you be enticed by an ad for a coat that is waterproof, stretchable, washable, and permanent-press, that automatically repairs small cuts, rips, and burns? How about one that’s guaranteed to last a lifetime? Sounds too good to be true, but you already have such a coat—your skin.

The skin and its derivatives (sweat and oil glands, hairs, and nails) make up a complex set of organs that serves several functions, mostly protective. Together, these organs form the **integumentary system** (in-teg’u-men’tar-e).

### The Skin

- Name the tissue types composing the epidermis and dermis. List the major layers of each and describe the functions of each layer.
- Describe the factors that normally contribute to skin color. Briefly describe how changes in skin color may be used as clinical signs of certain disease states.
The skin receives little respect from its inhabitants, but architecturally it is a marvel. It covers the entire body, has a surface area of 1.2 to 2.2 square meters, weighs 4 to 5 kilograms (4–5 kg = 9–11 lb), and accounts for about 7% of total body weight in the average adult. Also called the integument (“covering”), the skin multitasks. Its functions go well beyond serving as a bag for body contents. Pliable yet tough, it takes constant punishment from external agents. Without our skin, we would quickly fall prey to bacteria and perish from water and heat loss.

Varying in thickness from 1.5 to 4.0 millimeters (mm) or more in different parts of the body, the skin is composed of two distinct regions (Figure 5.1):

- The **epidermis** (ep”i-der’mis), composed of epithelial cells, is the outermost protective shield of the body (epi = upon).
- The underlying **dermis**, making up the bulk of the skin, is a tough, leathery layer composed mostly of fibrous connective tissue.

Only the dermis is vascularized. Nutrients reach the epidermis by diffusing through the tissue fluid from blood vessels in the dermis.

The subcutaneous tissue just deep to the skin is known as the **hypodermis** (Figure 5.1). Strictly speaking, the hypodermis is not part of the skin, but it shares some of the skin’s protective functions. The hypodermis, also called superficial fascia because it is superficial to the tough connective tissue wrapping (fascia) of the skeletal muscles, consists mostly of adipose tissue.

Besides storing fat, the hypodermis anchors the skin to the underlying structures (mostly to muscles), but loosely enough that the skin can slide relatively freely over those structures. Sliding skin protects us by ensuring that many blows just glance off our bodies. Because of its fatty composition, the hypodermis also acts as a shock absorber and an insulator that reduces heat loss. The hypodermis thickens markedly when a person gains weight. In females, this “extra” subcutaneous fat accumulates first in the thighs and breasts, but in males it first collects in the anterior abdomen (as a “beer belly”).

**Epidermis**

Structurally, the **epidermis** is a keratinized stratified squamous epithelium consisting of four distinct cell types and four or five distinct layers.
Cells of the Epidermis

The cells populating the epidermis include keratinocytes, melanocytes, dendritic cells, and tactile cells.

**Keratinocytes** The chief role of keratinocytes (kē-ratˈi-nōsēts; “keratin cells”) is to produce keratin, the fibrous protein that helps give the epidermis its protective properties (Greek kera = horn) (Figure 5.2b, orange cells). Most epidermal cells are keratinocytes.

Tightly connected to one another by desmosomes, the keratinocytes arise in the deepest part of the epidermis from a cell layer called the stratum basale. These cells undergo almost continuous mitosis in response to prompt by epidermal growth factor, a peptide produced by various cells throughout the body. As these cells are pushed upward by the production of new cells beneath them, they make the keratin that eventually dominates their cell contents. By the time the keratinocytes reach the skin surface, they are dead, scalelike structures that are little more than keratin-filled plasma membranes.

Millions of dead keratinocytes rub off every day, giving us a totally new epidermis every 25 to 45 days, but cell production and keratin formation is accelerated in body areas regularly subjected to friction, such as the hands and feet. Persistent friction (from a poorly fitting shoe, for example) causes a thickening of the epidermis called a callus.

**Melanocytes** Melanocytes (melˈə-nō-sīts), the star-shaped epithelial cells that synthesize the pigment melanin (melˈə-nin; melan = black), are found in the deepest layer of the epidermis (Figure 5.2b, gray cells). As melanin is made, it accumulates in membrane-bound granules called melanosomes that motor proteins move along actin filaments to the ends of the melanocyte's processes (the “spider arms”). From there they are transferred to a number of nearby keratinocytes (4 to 10 depending on body area). The melanin granules accumulate on the superficial, or “sunny,” side of the keratinocyte nucleus, forming a pigment shield that protects the nucleus from the damaging effects of ultraviolet (UV) radiation in sunlight.

**Dendritic Cells** The star-shaped dendritic cells arise from bone marrow and migrate to the epidermis. Also called Langhans cells (lahngˈər-ənz) after a German anatomist, they ingest foreign substances and are key activators of our immune system, as described later in this chapter. Their slender processes extend among the surrounding keratinocytes, forming a more or less continuous network (Figure 5.2b, purple cell).

**Tactile Cells** Occasional tactile (Merkel) cells are present at the epidermal-dermal junction. Shaped like a spiky hemisphere (Figure 5.2b, blue cell), each tactile cell is intimately associated with a disclike sensory nerve ending. The combination, called a tactile or Merkel disc, functions as a sensory receptor for touch.

Layers of the Epidermis

Variation in epidermal thickness determines if skin is thick or thin. In **thick skin**, which covers areas subject to abrasion—the palms, fingertips, and soles of the feet—the epidermis consists of five layers, or strata (straˈtā; “bed sheets”). From deep to superficial, these layers are stratum basale, stratum spinosum, stratum granulosum, stratum lucidum, and stratum corneum. In thin skin, which covers the rest of the body, the stratum lucidum appears to be absent and the other strata are thinner (Figure 5.2a, b).

Note that the terms “thick skin” and “thin skin” are really misnomers because they refer to the epidermis only. Indeed, the thickest skin in the body is on the upper back.

**Stratum Basale (Basal Layer)** The stratum basale (straˈtum bah-saˈle), the deepest epidermal layer, is attached to the underlying dermis along a wavy borderline that reminds one of corrugated cardboard. For the most part, it consists of a single row of stem cells—a continually renewing cell population—representing the youngest keratinocytes. The many mitotic nuclei seen in this layer reflect the rapid division of these cells and account for its alternate name, stratum germinativum (jerˈmi-nə-tiv-əm; “germinating layer”). Each time one of these basal cells divides, one daughter cell is pushed into the cell layer just above to begin its specialization into a mature keratinocyte. The other daughter cell remains in the basal layer to continue the process of producing new keratinocytes.

Some 10–25% of the cells in the stratum basale are melanocytes, and their branching processes extend among the surrounding cells, reaching well into the more superficial stratum spinosum layer. Occasional tactile cells also occur in this stratum.

**Stratum Spinosum (Prickly Layer)** The stratum spinosum (spiˈno-sum; “prickly”) is several cell layers thick. These cells contain a weibike system of intermediate filaments, mainly tension-resisting bundles of pre-keratin filaments, which span their cytosol to attach to desmosomes. Looking like tiny versions of the spiked iron balls used in medieval warfare, the keratinocytes in this layer appear to have spines, causing them to be called prickle cells. The spines do not exist in the living cells; they are artifacts that arise during tissue preparation when these cells shrink but their numerous desmosomes hold tight. Scattered among the keratinocytes are melanin granules and dendritic cells, which are most abundant in this epidermal layer.

**Stratum Granulosum (Granular Layer)** The thin stratum granulosum (granˈu-lo-ˈsum) consists of four to six cell layers in which keratinocyte appearance changes drastically, and the process of keratinization (in which the cells fill with the protein keratin) begins. These cells flatten, their nuclei and organelles begin to disintegrate, and they accumulate two types of granules. The keratohyaline granules (kerˈə-hi-ˈal-in) help to form keratin in the upper layers, as we will see.

The lamellar granules (laˈmə-lər) contain a water-resistant glycolipid that is spewed into the extracellular space and is a major factor in slowing water loss across the epidermis. The plasma membranes of these cells thicken as cytosal proteins bind to the inner membrane face and lipids released by the lamellar granules coat their external surfaces. These events produce an epidermal water barrier and make the cells more resistant to destruction. So, you might say that keratinocytes “toughen up” to make the outer strata the strongest skin region.

Like all epithelia, the epidermis relies on capillaries in the underlying connective tissue (the dermis in this case) for its nutrients. Above the stratum granulosum, the epidermal cells are
Chapter 5 The Integumentary System

5.2 The main structural features of the skin epidermis. (a) Photomicrograph of the four major epidermal layers (200×). (b) Diagram showing these four layers and the distribution of different cell types. The four cell types are keratinocytes (orange), melanocytes (gray), dendritic cells (purple), and tactile cells (blue). A sensory nerve ending (yellow), extending from the dermis (pink), is shown associated with the tactile cell forming a tactile disc (touch receptor). Notice that numerous desmosomes join the keratinocytes together. The stratum lucidum, found only in thick skin, is not illustrated here.

Stratum corneum
Most superficial layer; 20–30 layers of dead cells, essentially flat membranous sacs filled with keratin. Glycolipids in extracellular space.

Stratum granulosum
Typically five layers of flattened cells, organelles deteriorating; cytoplasm full of lamellar granules (release lipids) and keratohyaline granules.

Stratum spinosum
Several layers of keratinocytes unified by desmosomes. Cells contain thick bundles of intermediate filaments made of pre-keratin.

Stratum basale
Deepest epidermal layer; one row of actively mitotic stem cells; some newly formed cells become part of the more superficial layers. See occasional melanocytes and dendritic cells.

too far from the dermal capillaries and the glycolipids coating their external surfaces cut them off from nutrients, so they die. This is a normal sequence of events.

Stratum Lucidum (Clear Layer) Through the light microscope, the stratum lucidum (loo’sid-um; “light”), visible only in thick skin, is a thin translucent band just above the stratum granulosum. Considered by some to be a subdivision of the superficial stratum corneum, it consists of two or three rows of clear, flat, dead keratinocytes with indistinct boundaries. Here, or in the stratum corneum above, the gummy substance of the keratohyaline granules clings to the keratin filaments in the cells, causing them to aggregate in large, cable-like, parallel arrays of intermediate filaments called tonofilaments.
**Stratum Corneum (Horny Layer)** An abrupt transition occurs between the nucleated cells of the stratum granulosum and the flattened anucleate cells of the stratum corneum (kor’ne-um). This outermost epidermal layer is a broad zone 20 to 30 cell layers thick that accounts for up to three-quarters of the epidermal thickness. Keratin and the thickened plasma membranes of cells in this stratum protect the skin against abrasion and penetration, and the glycolipid between its cells nearly waterproofs this layer. For these reasons, the stratum corneum provides a durable “overcoat” for the body, protecting deeper cells from the hostile external environment (air) and from water loss, and rendering the body relatively insensitive to biological, chemical, and physical assaults. It is amazing that even dead cells can still play so many roles.

The differentiation from basal cells to those typical of the stratum corneum is a specialized form of apoptosis in which the nucleus and other organelles break down and the plasma membrane thickens. So, the terminal cells do not fragment, but instead eventually slough off the skin surface. The shingle-like cell remnants of the stratum corneum are referred to as corneifer, or horny, cells (cornu = horn). They are familiar to everyone as dandruff, shed from the scalp, and dander, the loose flakes that slough off dry skin.

The average person’s skin sheds some 50,000 dead cells every minute and 18 kg (40 lb) of these skin flakes in a lifetime, providing a lot of fodder for the dust mites that inhabit our homes and bed linens. The saying “Beauty is only skin deep” is especially interesting in light of the fact that nearly everything we see when we look at someone is dead!

1. While walking barefoot in a barn, Jeremy stepped on a rusty nail that penetrated the epidermis on the sole of his foot. Name the layers the nail pierced from the superficial skin surface to the junction with the dermis.
2. The stratum basale is also called the stratum germinativum, a name that refers to its major function. What is that function?
3. Why are the desmosomes connecting the keratinocytes so important?
4. Given that epithelia are avascular, which layer would you expect to have the best-nourished cells?

**Check Your Understanding**

The dermis has two layers, the papillary and reticular, which abut one another along an indistinct boundary (Figure 5.3).

**Papillary Layer** The thin superficial papillary layer (pap’il-er-e) is areolar connective tissue in which fine interlacing collagen and elastic fibers form a loosely woven mat that is heavily invested with small blood vessels. The looseness of this connective tissue allows phagocytes and other defensive cells to wander freely as they patrol the area for bacteria that have breached the skin.

Peglike projections from its surface, called dermal papillae (pah-pil’ e; papil = nipple), indent the overlying epidermis (see Figure 5.1). Many dermal papillae contain capillary loops. Others house free nerve endings (pain receptors) and touch receptors called tactile or Meissner’s corpuscles (mis’nerz kor’pus-lz). Note that tactile cells and tactile corpuscles are different structures. (We consider them in more detail in Chapter 13.) In thick skin, such as the palms of the hands and soles of the feet, these papillae lie atop larger mounds called dermal ridges, which in turn cause the overlying epidermis to form epidermal ridges (Figure 5.4a).

Collectively, these skin ridges, referred to as friction ridges, are assumed to enhance the gripping ability of the fingers and feet like tire treads help grip the road. Recent studies indicate that they also contribute to our sense of touch by amplifying vibrations detected by the large lamellar corpuscles (receptors) in the dermis.
The Integumentary System

Friction ridge patterns are genetically determined and unique to each of us. Because sweat pores open along their crests, our fingertips leave identifying films of sweat called *fingerprints* on almost anything we touch.

**Reticular Layer**  The deeper reticular layer, accounting for about 80% of the thickness of the dermis, is coarse, irregularly arranged, dense fibrous connective tissue (Figure 5.3). The network of blood vessels that nourishes this layer, the cutaneous plexus, lies between this layer and the hypodermis. Its extracellular matrix contains pockets of adipose cells and thick bundles of interlacing collagen fibers. The collagen fibers run in various planes, but most run parallel to the skin surface. Separations, or less dense regions, between these bundles form **cleavage (tension) lines** in the skin. These externally invisible lines tend to run longitudinally in the skin of the head and limbs and in circular patterns around the neck and trunk (Figure 5.4b).

Cleavage lines are important to surgeons because when an incision is made parallel to these lines, the skin gapes less and heals more readily. The collagen fibers of the dermis give skin strength and resiliency that prevent most jabs and scrapes from penetrating the dermis. In addition, collagen binds water, helping to keep skin hydrated. Elastic fibers provide the stretch-recoil properties of skin.

**Flexure lines**, a third type of skin marking, are dermal folds that occur at or near joints, where the dermis is tightly secured to deeper structures. (Notice the deep creases on your palms.) Since the skin cannot slide easily to accommodate joint movement in such regions, the dermis folds and deep skin creases form (Figure 5.4c). Flexure lines are also visible on the wrists, fingers, soles, and toes.

**Homeostatic Imbalance 5.1**

Extreme stretching of the skin, such as during pregnancy, can tear the dermis, leaving silvery white scars called *striae* (stri’ë; “streaks”), commonly called “stretch marks.” Short-term but acute trauma (as from a burn or wielding a hoe) can cause a *blister*, a fluid-filled pocket that separates the epidermal and dermal layers.
Check Your Understanding

5. Which layer of the dermis is responsible for producing fingerprint patterns?
6. Which tissue of the hypodermis makes it a good shock absorber?
7. You have just gotten a paper cut. It is very painful, but it doesn't bleed. Has the cut penetrated into the dermis or just the epidermis?

For answers, see Appendix H.

Skin Color

Three pigments contribute to skin color: melanin, carotene, and hemoglobin. Of these, only melanin is made in the skin.

Melanin

Melanin is a polymer made of tyrosine amino acids. Its two forms range in color from reddish yellow to brownish black. Its synthesis depends on an enzyme in melanocytes called tyrosinase (ti-ro'si-nās) and, as noted earlier, it passes from melanocytes to the basal keratinocytes. Eventually, lysosomes break down the melanosomes, so melanin pigment is found only in the deeper layers of the epidermis.

Human skin comes in different colors. However, distribution of those colors is not random—populations of darker-skinned people tend to be found nearer the equator (where greater protection from the sun is needed), and those with the lightest skin are found closer to the poles. Since all humans have the same relative number of melanocytes, differences in skin coloring reflect the kind and amount of melanin made and retained. Melanocytes of black- and brown-skinned people produce many more and darker melanosomes than those of fair-skinned individuals, and their keratinocytes retain it longer. Freckles and pigmented nevi (moles) are local accumulations of melanin.

When we expose our skin to sunlight, keratinocytes secrete chemicals that stimulate melanocytes. Prolonged sun exposure causes a substantial melanin buildup, which helps protect the DNA of viable skin cells from UV radiation by absorbing the rays and dissipating the energy as heat. Indeed, the initial signal for speeding up melanin synthesis seems to be a faster repair rate of DNA that has suffered photodamage (photo = light). In all but the darkest people, this defensive response causes skin to darken visibly (tanning occurs).

One condition sun exposure does not cause, regardless of its name, is sunspots, small circular white spots on the skin that stand out like a sore thumb in tanned skin. Actually a fungus infection, this condition—more accurately called tinea versicolor (vers = change)—occurs mainly on the arms, legs, upper chest, and back. Largely a cosmetic problem, its growth is encouraged by oily, sweaty skin. Antifungal medication works, but it takes a long time for the white patches to become pigmented again.

Many chemicals induce photosensitivity; that is, they heighten the skin's sensitivity to UV radiation and can cause an unsightly skin rash. Such substances include some antibiotic and antihistamine drugs, and many chemicals in perfumes and detergents. Small, itchy blisters erupt all over the body. Then the peeling begins—in sheets!

Carotene

Carotene (kar’o-ten) is a yellow to orange pigment found in certain plant products such as carrots. It tends to accumulate in the stratum corneum and in fatty tissue of the hypodermis. Its color is most obvious in the palms and soles, where the stratum corneum is thickest, and most intense when large amounts of carotene-rich foods are eaten. However, the yellowish tinge of the skin of some Asian peoples is due to variations in melanin, as well as to carotene. In the body, carotene can be converted to vitamin A, a vitamin that is essential for normal vision, as well as for epidermal health.

The pinkish hue of fair skin reflects the crimson color of the oxygenated pigment hemoglobin (he’mon-glo’bin) in the red blood cells circulating through the dermal capillaries. Because Caucasian skin contains only small amounts of melanin, the epidermis is nearly transparent and allows hemoglobin's color to show through.

Homeostatic Imbalance 5.3

When hemoglobin is poorly oxygenated, both the blood and the skin of Caucasians appear blue, a condition called cyanosis (si’ah-no’sis; cyan = dark blue). Skin often becomes cyanotic during heart failure and severe respiratory disorders. In dark-skinned individuals, the skin does not appear cyanotic because of the masking effects of melanin, but cyanosis is apparent in their mucous membranes and nail beds (the same sites where the red cast of oxygenated blood is normally visible).

Many alterations in skin color signal certain disease states or even emotional states:

- Redness, or erythema (er’ti-the’mah): Reddened skin may indicate embarrassment (blushing), fever, hypertension, inflammation, or allergy.
- Pallor, or blanching: During fear, anger, and certain other types of emotional stress, some people become pale. Pale skin may also signify anemia or low blood pressure.
- Jaundice (jawndis), or yellow cast: An abnormal yellow skin tone usually signifies a liver disorder, in which yellow bile pigments accumulate in the blood and are deposited in body tissues. [Normally, the liver cells secrete the bile pigments (bilirubin) as a component of bile.]
Bronzing: A bronze, almost metallic appearance of the skin is a sign of Addison's disease, in which the adrenal cortex produces inadequate amounts of its steroid hormones; or a sign of pituitary gland tumors that inappropriately secrete melanocyte-stimulating hormone (MSH).

Black-and-blue marks, or bruises: Black-and-blue marks reveal where blood escaped from the circulation and clotted beneath the skin. Such clotted blood masses are called hematomas (he‘mah-to‘mah; "blood swelling").

Check Your Understanding
1. Black-and-blue marks are bruises, hematomas, or what?
2. Name a few skin appendages.
3. List the parts of a hair follicle.
4. List the cutaneous appendages of the skin.
5. Name a few skin appendages.
6. Define melanin.
7. List the parts of a hair follicle.

Appendages of the Skin

List the parts of a hair follicle and explain the function of each part. Also describe the functional relationship of arrector pili muscles to the hair follicles.

Name the regions of a hair and explain the basis of hair color. Describe the distribution, growth, replacement, and changing nature of hair during the life span.

Describe the structure of nails.

Appendages of the Skin

Anchoring the skin itself, the integumentary system includes several derivatives of the epidermis. These skin appendages include hair and hair follicles, nails, sweat glands, and sebaceous (oil) glands. Each plays a unique role in maintaining body homeostasis.

A key step in beginning to form any of the skin’s appendages is formation of an epithelial bud. This process is stimulated by a reduced production of cell adhesion factor (cadherin). Once the cell-to-cell attractions are broken, the cells can move about and rearrange themselves, allowing an epithelial bud to form.

Hairs and Hair Follicles

Hair is an important part of our body image—consider, for example, the spiky hair style of punk rockers and the flowing, glossy manes of some high-fashion models. Millions of hairs are distributed over our entire skin surface except our palms, soles, lips, nipples, and parts of the external genitalia (the head of the penis, for instance). Although hair helps to keep other mammals warm, our sparse body hair is far less luxurious and useful. Its main function in humans is to sense insects on the skin before they bite or sting us. Hair on the scalp guards the head against physical trauma, heat loss, and sunlight. (Pity the bald man.) Eyelashes shield the eyes, and nose hairs filter large particles like lint and insects from the air we inhale.

Structure of a Hair

Hairs, or pilis (pi‘li), are flexible strands produced by hair follicles and consist largely of dead, keratinized cells. The hard keratin that dominates hairs and nails has two advantages over the soft keratin found in typical epidermal cells: (1) It is tougher and more durable, and (2) its individual cells do not flake off.

The chief regions of a hair are the shaft, the portion in which keratinization is complete, and the root, where keratinization is still ongoing. The shaft, which projects from the skin, extends about halfway down the portion of the hair embedded in the skin (Figure 5.5). The root is the remainder of the hair deep within the follicle. If the shaft is flat and ribbonlike in cross section, the hair is kinky; if it is oval, the hair is silky and wavy; if it is perfectly round, the hair is straight and tends to be coarse.

A hair has three concentric layers of keratinized cells: the medulla, cortex, and cuticle (Figure 5.5a, b).

- The medulla (mé-dül‘ah; “middle”), its central core, consists of large cells and air spaces. The medulla, which is the only part of the hair that contains soft keratin, is absent in fine hairs.
- The cortex, a bulky layer surrounding the medulla, consists of several layers of flattened cells.
- The outermost cuticle is formed from a single layer of cells overlapping one another like shingles on a roof. This arrangement helps separate neighboring hairs so the hair does not mat. (Hair conditioners smooth out the rough surface of the cuticle and make our hair look shiny.) The most heavily keratinized part of the hair, the cuticle provides strength and helps keep the inner layers tightly compacted.

Because it is subjected to the most abrasion, the cuticle tends to wear away at the tip of the hair shaft, allowing keratin fibrils in the cortex and medulla to frizz out, creating “split ends.”

Hair pigment is made by melanocytes at the base of the hair follicle and transferred to the cortical cells. Various proportions of melanins of different colors (yellow, rust, brown, and black) combine to produce hair color from blond to pitch black. Additionally, red hair is colored by the iron-containing pigment trichosiderin. When melanin production decreases (mediated by delayed-action genes) and air bubbles replace melanin in the hair shaft, hair turns gray or white.

Structure of a Hair Follicle

Hair follicles (folli = bag) fold down from the epidermal surface into the dermis. In the scalp, they may even extend into the hypodermis. The deep end of the follicle, located about 4 mm (1/6 in.) below the skin surface, expands to form a hair bulb (Figure 5.5c, d). A knot of sensory nerve endings called a hair follicle receptor, or root hair plexus, wraps around each hair bulb (see Figure 5.1). Bending the hair stimulates these endings. Consequently, our hairs act as sensitive touch receptors.

Feel the tickle as you run your hand over the hairs on your forearm.
The wall of a hair follicle is composed of an outer peripheral connective tissue sheath (or fibrous sheath), derived from the dermis; a thickened basal lamina called the glassy membrane; and an inner epithelial root sheath, derived mainly from an invagination of the epidermis (Figure 5.5). The epithelial root
sheath, which has external and internal parts, thins as it approaches the hair bulb, so that only a single layer of epithelial cells covers the papilla.

The cells that compose the hair matrix, or actively dividing area of the hair bulb that produces the hair, originate in a region called the hair bulge located a fraction of a millimeter above the hair bulb. When chemical signals diffusing from the hair papilla reach the hair bulge, some of its cells migrate toward the papilla, where they divide to produce the hair cells. As the matrix produces new hair cells, the older part of the hair is pushed upward, and its fused cells become increasingly keratinized and die.

Associated with each hair follicle is a bundle of smooth muscle cells called an arrector pili (ah-rek ‘tor pi ’li; “ raiser of hair”) muscle. As you can see in Figure 5.1, most hair follicles approach the skin surface at a slight angle. The arrector pili muscle is attached in such a way that its contraction pulls the hair follicle upright and dimples the skin surface to produce goose bumps in response to cold temperatures or fear. This “hair-raising” response is not very useful to humans, with our short sparse hairs, but it is an important way for other animals to retain heat and protect themselves. Furry animals stay warmer by trapping a layer of insulating air in their fur; and a scared animal with its hair on end looks larger and more formidable to its enemy. The more important role of the arrector pili in humans is that its contractions force sebum out of hair follicles to the skin surface where it acts as a skin lubricant.

Types and Growth of Hair

Hairs come in various sizes and shapes, but as a rule they can be classified as vellus or terminal. The body hair of children and adult females is pale, fine vellus hair (vel’us; vell = wool, fleece). The coarser, longer hair of the eyebrows and scalp is terminal hair, which may also be darker.

At puberty, terminal hairs appear in the axillary and pubic regions of both sexes and on the face and chest (and typically the arms and legs) of males. These terminal hairs grow in response to the stimulating effects of androgens (of which testosterone is the most important), and when male hormones are present in large amounts, terminal hair growth is luxuriant.

Many factors influence hair growth and density, especially nutrition and hormones. Poor nutrition means poor hair growth, whereas conditions that increase local dermal blood flow (such as chronic physical irritation or inflammation) may enhance local hair growth. Many old-time bricklayers who carried their hod on one shoulder all the time developed one hairy shoulder. Undesirable hair growth (such as on a woman’s upper lip) may be arrested by electrolysis or laser treatments, which use electricity or light energy, respectively, to destroy the hair roots.

Homeostatic Imbalance 5.4

In women, both the ovaries and the adrenal glands normally produce small amounts of androgens. Excessive hairiness, or hirsutism (her’soot-izm; hirsut = hairy), as well as other signs of masculinization, may result from an adrenal gland or ovarian tumor that secretes abnormally large amounts of androgens. Since few women want a beard or hairy chest, such tumors are surgically removed as soon as possible.

The rate of hair growth varies from one body region to another and with sex and age, but it averages 2.5 mm per week. Each follicle goes through growth cycles. In each cycle, an active growth phase, ranging from weeks to years, is followed by a regressive phase. During the regressive phase, the hair matrix cells die and the follicle base and hair bulb shrivels somewhat, dragging the hair papilla upward to abut the region of the follicle that does not regress. The follicle then enters a resting phase for one to three months. After the resting phase, the cycling part of the follicle regenerates and activated bulge cells migrate toward the papilla. As a result, the matrix proliferates again and forms a new hair to replace the old one that has fallen out or will be pushed out by the new hair.

The life span of hairs varies and appears to be controlled by a slew of proteins. The follicles of the scalp remain active for six to ten years before becoming inactive for a few months. Because only a small percentage of the hair is shed at any one time, we lose an average of 90 scalp hairs daily. The follicles of the eyebrow hairs remain active for only three to four months, which explains why your eyebrows are never as long as the hairs on your head.

Hair Thinning and Baldness

A follicle has only a limited number of cycles in it. Given ideal conditions, hair grows fastest from the teen years to the 40s, and then its growth slows. The fact that hairs are shed faster than they are replaced leads to hair thinning and some degree of baldness, or alopecia (al’o-pe’she-ah), in both sexes. By age 35, noticeable hair loss occurs in 40% of men, and by age 60 that number jumps to 85%. Much less dramatic in women, the process usually begins at the anterior hairline and progresses posteriorly. Coarse terminal hairs are replaced by vellus hairs, and the hair becomes increasingly wispy.

True, or frank, baldness is a different story entirely. The most common type, male pattern baldness, is a genetically determined, sex-influenced condition. It is thought to be caused by a delayed-action gene that “switches on” in adulthood and changes the response of the hair follicles to DHT (dihydrotestosterone), a metabolite of testosterone. As a result, the follicular growth cycles become so short that many hairs never even emerge from their follicles before shedding, and those that do are fine vellus hairs that look like peach fuzz in the “bald” area.

Until recently, the only cure for male pattern baldness was drugs that inhibit testosterone production, but they also cause loss of sex drive—a trade-off few men would choose. Quite by accident, it was discovered that minoxidil (Rogaine), a drug used to reduce high blood pressure, has the interesting side effect in some bald men (and balding women) of stimulating hair regrowth. Although its results are variable, minoxidil is available over the counter in dropper bottles or spray form for application to the scalp. Finasteride (better known as Propecia), according to some the most promising cure for male pattern baldness, has had moderate success. Available only by prescription in once-a-day pill form, it must be taken for the rest of a person’s life. Once the patient stops taking it, all of the new growth falls out.
Hair thinning can be induced by a number of factors that upset the normal balance between hair loss and replacement. Outstanding examples are acutely high fever, surgery, severe emotional trauma, and certain drugs (excessive vitamin A, some antidepressants and blood thinners, anabolic steroids, and most chemotherapy drugs). Protein-deficient diets and lactation lead to hair thinning because new hair growth stops when protein needed for keratin synthesis is not available or is being used for milk production. In all of these cases, hair regrows if the cause is removed or corrected.

In the rare condition alopecia areata, the immune system attacks the follicles and the hair falls out in patches. But again, the follicles survive. Hair loss due to severe burns, excessive radiation, or other factors that eliminate the follicles is permanent. ✚

A nail is a scalelike modification of the epidermis that forms a clear protective covering on the dorsal surface of the distal part of a finger or toe (Figure 5.6). Nails, which attach to the hooves or claws of other animals, are useful as "tools" to help pick up small objects or scratch an itch.

In contrast to soft keratin of the epidermis, nails (like hairs) contain hard keratin. Each nail has a free edge, a nail plate or body (visible attached portion), and a proximal root (embedded in the skin). The deeper layers of the epidermis extend beneath the nail as the nail bed, and the nail itself corresponds to the superficial keratinized layers. The thickened proximal portion of the nail bed, called the nail matrix, is responsible for nail growth. As the nail cells produced by the matrix become heavily keratinized, the nail body slides distally over the nail bed.

Nails normally appear pink because of the rich bed of capillaries in the underlying dermis. However, the region that lies over the thick nail matrix appears as a white crescent called the lunule (lu’nool; "little moon"). The proximal and lateral borders of the nail are overlapped by skin folds, called nail folds. The proximal nail fold projects onto the nail body as the cuticle or eponychium (ep’o-nik’e-um; "on the nail"). The thickened region beneath the free edge of the nail where dirt and debris tend to accumulate is the hyponychium ("below nail"), informally called the quick. It secures the free edge of the nail plate at the fingertip.

Changes in nail appearance can help diagnose certain conditions. For example, yellow-tinged nails may indicate a respiratory or thyroid gland disorder, while thickened yellow nails may signal a fungus infection. An outward concavity of the nail (spoon nail) may signal an iron deficiency, and horizontal lines (Beau’s lines) across the nails may hint of malnutrition.

Nails normally appear pink because of the rich bed of capillaries in the underlying dermis. However, the region that lies over the thick nail matrix appears as a white crescent called the lunule (lu’nool; “little moon”). The proximal and lateral borders of the nail are overlapped by skin folds, called nail folds. The proximal nail fold projects onto the nail body as the cuticle or eponychium (ep’o-nik’-e-um; “on the nail”). The thickened region beneath the free edge of the nail where dirt and debris tend to accumulate is the hyponychium ("below nail"), informally called the quick. It secures the free edge of the nail plate at the fingertip.

Changes in nail appearance can help diagnose certain conditions. For example, yellow-tinged nails may indicate a respiratory or thyroid gland disorder, while thickened yellow nails may signal a fungus infection. An outward concavity of the nail (spoon nail) may signal an iron deficiency, and horizontal lines (Beau’s lines) across the nails may hint of malnutrition.
Apocrine secretion contains the same basic components as true sweat, plus fatty substances and proteins. Consequently, it is viscous and sometimes has a milky or yellowish color. The secretion is odorless, but when bacteria on the skin decompose its organic molecules, it takes on a musky and generally unpleasant odor, the basis of body odor.

Apocrine glands begin functioning at puberty under the influence of the male sex hormones (androgens) and play little role in maintaining a constant body temperature. Their precise function is not yet known, but they are activated by sympathetic nerve fibers during pain and stress. Because sexual foreplay increases their activity, and they enlarge and recede with the phases of a woman’s menstrual cycle, they may be the human equivalent of other animals’ sexual scent glands.

Ceruminous glands (sĕ-roo’mĭ-nus; ceră wax) are modified apocrine glands found in the lining of the external ear canal. Their secretion mixes with sebum produced by nearby sebaceous glands to form a sticky, bitter substance called cerumen, or earwax, that is thought to deter insects and block entry of foreign material.

Apocrine glands begin functioning at puberty under the influence of the male sex hormones (androgens) and play little role in maintaining a constant body temperature. Their precise function is not yet known, but they are activated by sympathetic nerve fibers during pain and stress. Because sexual foreplay increases their activity, and they enlarge and recede with the phases of a woman’s menstrual cycle, they may be the human equivalent of other animals’ sexual scent glands.

Ceruminous glands (sĕ-roo’mĭ-nus; ceră wax) are modified apocrine glands found in the lining of the external ear canal. Their secretion mixes with sebum produced by nearby sebaceous glands to form a sticky, bitter substance called cerumen, or earwax, that is thought to deter insects and block entry of foreign material.

Apocrine glands begin functioning at puberty under the influence of the male sex hormones (androgens) and play little role in maintaining a constant body temperature. Their precise function is not yet known, but they are activated by sympathetic nerve fibers during pain and stress. Because sexual foreplay increases their activity, and they enlarge and recede with the phases of a woman’s menstrual cycle, they may be the human equivalent of other animals’ sexual scent glands.

Ceruminous glands (sĕ-roo’mĭ-nus; ceră wax) are modified apocrine glands found in the lining of the external ear canal. Their secretion mixes with sebum produced by nearby sebaceous glands to form a sticky, bitter substance called cerumen, or earwax, that is thought to deter insects and block entry of foreign material.

Sebaceous (Oil) Glands

The sebaceous glands (se-ba’shūs; “greasy”), or oil glands (Figure 5.7a), are simple branched alveolar glands that are...
found all over the body except in the thick skin of the palms and soles. They are small on the body trunk and limbs, but quite large on the face, neck, and upper chest. These glands secrete an oily substance called sebum (se’ bum). The central cells of the alveoli accumulate oily lipids until they become so engorged that they burst, so functionally these glands are holocrine glands (see p. 125). The accumulated lipids and cell fragments constitute sebum.

Most, but not all, sebaceous glands develop as outgrowths of hair follicles and secrete sebum into a hair follicle, or occasionally to a pore on the skin surface. Arrector pili contractions force sebum out of the hair follicles to the skin surface. Sebum softens and lubricates the hair and skin, prevents hair from becoming brittle, and slows water loss from the skin when external humidity is low. Perhaps even more important is its bactericidal (bacterium-killing) action.

Hormones, especially androgens, stimulate the secretion of sebum. Sebaceous glands are relatively inactive during childhood but are activated in both sexes during puberty, when androgen production begins to rise.

**Homeostatic Imbalance 5.6**

If accumulated sebum blocks a sebaceous gland duct, a whitehead appears on the skin surface. If the material oxidizes and dries, it darkens to form a blackhead. Acne is an active inflammation of the sebaceous glands accompanied by “pimples” (pustules or cysts) on the skin. It is usually caused by bacterial infection, particularly by staphylococcus, and can range from mild to severe, leading to permanent scarring.

Overactive sebaceous glands can cause seborrhea (seb’ o-re’ ah; “fast-flowing sebum”), known as “cradle cap” in infants. Seborrhea begins on the scalp as pink, raised lesions that gradually become yellow to brown and begin to slough off oily scales.

**Check Your Understanding**

17. Which cutaneous glands are associated with hair follicles?
18. When Anthony returned home from a run in 85°F weather, his face was dripping with sweat. Why?
19. What is the difference between heat-induced sweating and a “cold sweat,” and which variety of sweat gland is involved?
20. Sebaceous glands are not found in thick skin. Why is their absence in those body regions desirable?

For answers, see Appendix H.

**Functions of the Integumentary System**

Describe how the skin accomplishes at least five different functions.

First and foremost, our skin is a barrier. Like the skin of a grape, it keeps its contents juicy and whole. The skin is also a master at self (wound) repair, and interacts immediately with other body systems by making potent molecules, all the while protecting deeper tissues from damaging external agents. Even this short list hints that the skin and its derivatives perform a variety of functions, including: protection, body temperature regulation, cutaneous sensation, metabolic functions, blood reservoir, and excretion.

**Protection**

Given its superficial location, the skin is our most vulnerable organ system, exposed to microorganisms, abrasion, temperature extremes, and harmful chemicals. The skin constitutes at least three types of barriers: chemical, physical, and biological.

**Chemical Barriers**

Chemical barriers include skin secretions and melanin. Although the skin’s surface teems with bacteria, the low pH of skin secretions—the so-called acid mantle—retards their multiplication. In addition, dermcidin in sweat and bactericidal substances in sebum kill many bacteria outright. Skin cells also secrete natural antibiotics called defensins that literally punch holes in bacteria, making them look like sieves. Wounded skin releases large quantities of protective peptides called catherelcidins that are particularly effective in preventing infection by group A streptococcus bacteria.

As discussed earlier, melanin provides a chemical pigment shield to prevent UV damage to skin cells.

**Physical Barriers**

The continuity of skin and the hardness of its keratinized cells provide physical barriers. As a physical barrier, the skin is a remarkable compromise. A thicker epidermis would be more impenetrable, but we would pay the price in loss of suppleness and agility.

The outstanding barrier capacity of the skin arises from the structure of its stratum corneum, which has been compared to bricks and mortar. Multiple layers of dead flat cells are the bricks and the glycolipids surrounding them are the mortar. Epidermal continuity works hand in hand with the acid mantle and certain chemicals in skin secretions to ward off bacterial invasion. The water-resistant glycolipids of the epidermis block most diffusion of water and water-soluble substances between cells, preventing both their loss from and entry into the body through the skin. However, there is a continual small loss of water through the epidermis, and if immersed in water (other than salt water), the skin will take in some water and swell slightly.

Substances that do penetrate the skin in limited amounts include:

- Lipid-soluble substances, such as oxygen, carbon dioxide, fat-soluble vitamins (A, D, E, and K), and steroids (estrogens)
- Oleoresins (o’le-o-rez’inz) of certain plants, such as poison ivy and poison oak
- Organic solvents, such as acetone, dry-cleaning fluid, and paint thinner, which dissolve the cell lipids
- Salts of heavy metals, such as lead and mercury
- Selected drugs (nitroglycerine, nicotine, seasickness medications)
Drug agents called penetration enhancers that help ferry other drugs into the body. Alcoholic drinks dramatically enhance skin permeability for at least 24 hours after their ingestion.

**Homeostatic Imbalance 5.7**

Organic solvents and heavy metals are devastating to the body and can be lethal. Passage of organic solvents through the skin into the blood can shut down the kidneys and also cause brain damage. Absorption of lead results in anemia and neurological defects. These substances should never be handled with bare hands.

**Biological Barriers**

Biological barriers include the dendritic cells of the epidermis, macrophages in the dermis, and DNA itself.

Dendritic cells are active elements of the immune system. To activate the immune response, the foreign substances, or antigens, must be presented to specialized white blood cells called lymphocytes. In the epidermis, the dendritic cells play this role.

Dermal macrophages constitute a second line of defense to dispose of viruses and bacteria that manage to penetrate the epidermis. They, too, act as antigen “presenters.”

Although melanin provides a fairly good chemical sunscreen, DNA itself is a remarkably effective biologically based sunscreen. Electrons in DNA molecules absorb UV radiation and transfer it to the atomic nuclei, which heat up and vibrate vigorously. Since the heat dissipates to surrounding water molecules instantaneously, the DNA converts potentially destructive radiation into harmless heat.

**Body Temperature Regulation**

The body works best when its temperature remains within homeostatic limits. Like car engines, we need to get rid of the heat generated by our internal reactions. As long as the external temperature is lower than body temperature, the skin surface loses heat to the air and to cooler objects in its environment, just as a car radiator loses heat to the air and other nearby parts.

Under normal resting conditions, and as long as the environmental temperature is below 31–32°C (88–90°F), sweat glands secrete about 500 ml (0.5 L) of sweat per day. This routine and unnoticeable sweating is called insensible perspiration. When body temperature rises, the nervous system stimulates dermal blood vessels to dilate and the sweat glands into vigorous secretory activity. Indeed, on a hot day, sweat becomes noticeable and can account for the loss of up to 12 L (about 3 gallons) of body water in one day. This visible output of sweat is called sensible perspiration. Evaporation of sweat from the skin surface dissipates body heat and efficiently cools the body, preventing overheating.

When the external environment is cold, dermal blood vessels constrict. Their constriction causes the warm blood to bypass the skin temporarily and allows skin temperature to drop to that of the external environment. This slows passive heat loss from the body, conserving body heat. Chapter 24 discusses body temperature regulation.

**Cutaneous Sensation**

The skin is richly supplied with cutaneous sensory receptors, which are actually part of the nervous system. The cutaneous receptors are classified as exteroceptors (ek’ster-o-sep’torz) because they respond to stimuli arising outside the body. For example, tactile (Meissner’s) corpuscles (in the dermal papillae) and tactile discs allow us to become aware of a caress or the feel of our clothing against our skin, whereas lamellar (also called Pacinian) corpuscles (in the deeper dermis or hypodermis) alert us to bumps or contacts involving deep pressure. Hair follicle receptors report on wind blowing through our hair and a playful tug on a pigtail. Free nerve endings that meander throughout the skin sense painful stimuli (irritating chemicals, extreme heat or cold, and others). We defer detailed discussion of these cutaneous receptors to Chapter 13.

Figure 5.1 illustrates all the cutaneous receptors mentioned above except for tactile corpuscles, which are found only in skin that lacks hairs, and tactile cells, shown in Figure 5.2b.

**Metabolic Functions**

The skin is a chemical factory, fueled in part by the sun’s rays. When sunlight bombards the skin, modified cholesterol molecules are converted to a vitamin D precursor, which is transported via the blood to other body areas to be ultimately converted to vitamin D, which plays various roles in calcium metabolism. For example, calcium cannot be absorbed from the digestive tract without vitamin D.

Among its other metabolic functions, the epidermis makes chemical conversions that supplement those of the liver. For example, keratinocyte enzymes can

- “Disarm” many cancer-causing chemicals that penetrate the epidermis
- Convert some harmless chemicals into carcinogens
- Activate some steroid hormones—for instance, they can transform cortisone applied to irritated skin into hydrocortisone, a potent anti-inflammatory drug
- Skin cells also make several biologically important proteins, including collagenase, an enzyme that aids the natural turnover of collagen (and deters wrinkles).

**Blood Reservoir**

The dermal vascular supply is extensive and can hold about 5% of the body’s entire blood volume. When other body organs, such as vigorously working muscles, need a greater blood supply, the nervous system constricts the dermal blood vessels. This constriction shunts more blood into the general circulation, making it available to the muscles and other body organs.

**Excretion**

The body eliminates limited amounts of nitrogen-containing wastes (ammonia, urea, and uric acid) in sweat, although most such wastes are excreted in urine. Profuse sweating is an important avenue for water and salt (sodium chloride) loss.
Interestingly, sunburned skin accelerates its production of Fas, a protein that causes genetically damaged skin cells to commit suicide, reducing the risk of mutations that will cause sun-linked skin cancer. The death of these gene-damaged cells causes the skin to peel after a sunburn.

There is no such thing as a “healthy tan,” but the good news for sun worshippers is the newly developed skin lotions that can fix damaged DNA before the involved cells become cancerous. These lotions contain tiny oily vesicles (liposomes) filled with enzymes that initiate repair of the DNA mutations most commonly caused by sunlight. The liposomes penetrate the epidermis and enter the keratinocytes, ultimately making their way into the nuclei to bind to specific sites where two DNA bases have fused. There, by selectively cutting the DNA strands, they begin a DNA repair process that is completed by cellular enzymes.

The three major forms of skin cancer are basal cell carcinoma, squamous cell carcinoma, and melanoma.

**Basal Cell Carcinoma**

Basal cell carcinoma (kar’si-no’mah), the least malignant and most common, accounts for nearly 80% of cases. Stratum basale cells proliferate, invading the dermis and hypodermis. The cancer lesions occur most often on sun-exposed areas of the face and appear as shiny, dome-shaped nodules that later develop a central ulcer with a pearly, beaded edge (Figure 5.8a). Basal cell carcinoma is relatively slow-growing, and metastasis seldom occurs before it is noticed. Full cure by surgical excision is the rule in 99% of cases.

**Squamous Cell Carcinoma**

Squamous cell carcinoma, the second most common skin cancer, arises from the keratinocytes of the stratum spinosum. The lesion appears as a scaly reddened papule (small, rounded elevation) that arises most often on the head (scalp, ears, and lower lip), and hands (Figure 5.8b). It tends to grow rapidly and metastasize if not removed. If it is caught early and removed surgically or by radiation therapy, the chance of complete cure is good.

**Melanoma**

Melanoma (mel’uh-no’mah), cancer of melanocytes, is the most dangerous skin cancer because it is highly metastatic and resistant to chemotherapy. It accounts for only 2–3% of skin cancers.
cancers, but its incidence is increasing rapidly (by 3–8% per year in the United States). Melanoma can begin wherever there is pigment. Most such cancers appear spontaneously, and about one-third develop from preexisting moles. It usually appears as a spreading brown to black patch (Figure 5.8c) that metastasizes rapidly to surrounding lymph and blood vessels.

The key to surviving melanoma is early detection. The chance of survival is poor if the lesion is over 4 mm thick. The usual therapy for melanoma is wide surgical excision accompanied by immunotherapy (immunizing the body against its cancer cells).

The American Cancer Society suggests that we regularly examine our skin for new moles or pigmented spots. Apply the **ABCD rule** for recognizing melanoma:

- **Asymmetry**: The two sides of the pigmented spot or mole do not match.
- **Border irregularity**: The borders of the lesion exhibit indentations.
- **Color**: The pigmented spot contains several colors (blacks, browns, tans, and sometimes blues and reds).
- **Diameter**: The spot is larger than 6 mm in diameter (the size of a pencil eraser).

Some experts add an **E**, for elevation above the skin surface.

### Burns

Burns are a devastating threat to the body primarily because of their effects on the skin. A **burn** is tissue damage inflicted by intense heat, electricity, radiation, or certain chemicals, all of which denature cell proteins and kill cells in the affected areas.

The immediate threat to life resulting from severe burns is a catastrophic loss of body fluids containing proteins and electrolytes. This leads to dehydration and electrolyte imbalance, and then renal failure (kidney shutdown) and circulatory shock (inadequate blood circulation due to reduced blood volume). To save the patient, the lost fluids must be replaced immediately via the intravenous (IV) route.

#### Evaluating Burns

In adults, the volume of fluid lost can be estimated by computing the percentage of body surface burned using the **rule of nines**. This method divides the body into 11 areas, each accounting for 9% of total body area, plus an additional area surrounding the genitals accounting for 1% of body surface area (Figure 5.9). The rule of nines is only approximate, so special tables are used when greater accuracy is desired.

Burn patients also need thousands of extra food calories daily to replace lost proteins and allow tissue repair. No one can eat enough food to provide these calories, so burn patients are given supplementary nutrients through gastric tubes and IV lines. After the initial crisis has passed, infection becomes the main threat and sepsis (widespread bacterial infection) is the leading cause of death in burn victims. Burned skin is sterile for about 24 hours. Thereafter, bacteria, fungi, and other pathogens easily invade areas where the skin barrier is destroyed, and they multiply rapidly in the nutrient-rich environment of dead tissues. Adding to this problem is the fact that the immune system becomes deficient within one to two days after severe burn injury.

Burns are classified according to their severity (depth) as first-, second-, or third-degree burns. In **first-degree burns**, only the epidermis is damaged. Symptoms include localized redness, swelling, and pain. First-degree burns tend to heal in two to three days without special attention. Sunburn is usually a first-degree burn.
Second-degree burns injure the epidermis and the upper region of the dermis. Symptoms mimic those of first-degree burns, but blisters also appear. The burned area is red and painful, but skin regeneration occurs with little or no scarring within three to four weeks if care is taken to prevent infection. First- and second-degree burns are referred to as partial-thickness burns (Figure 5.10a).

Third-degree burns are full-thickness burns, involving the entire thickness of the skin (Figure 5.10b). The burned area appears gray-white, cherry red, or blackened, and initially there is little or no edema. Since the nerve endings have been destroyed, the burned area is not painful. Although skin might eventually regenerate by proliferating epithelial cells at the edges of the burn or stem cells in hair follicles, it is usually impossible to wait that long because of fluid loss and infection. Skin grafting is advised.

In general, burns are considered critical if any of the following conditions exists:
- Over 25% of the body has second-degree burns
- Over 10% of the body has third-degree burns
- There are third-degree burns of the face, hands, or feet

Facial burns introduce the possibility of burned respiratory passageways, which can swell and cause suffocation. Burns at joints are also troublesome because scar tissue can severely limit joint mobility.

Treat ing Burns

To prepare a burned area for a skin graft, the eschar (es’kar), or burned skin, must first be debrided (removed). To prevent infection and fluid loss, the area is then flooded with antibiotics and covered temporarily with a synthetic membrane, animal (pig) skin, cadaver skin, or “living bandage” made from the thin amniotic sac membrane that surrounds a fetus. Then healthy skin is transplanted to the burned site. Unless the graft is taken from the patient (an autograft), however, there is a good chance that the patient’s immune system will reject it (see p. 790 in Chapter 21). Even if the graft “takes,” extensive scar tissue often forms in the burned areas.

An exciting technique eliminates many of the traditional problems of skin grafting and rejection. Synthetic skin—a silicone “epidermis” bound to a spongy “dermal” layer composed of collagen and ground cartilage—is applied to the debrided area. In time, the patient’s own dermal tissue absorbs and replaces the artificial one. Then the silicone sheet is peeled off and replaced with a network of epidermal cells cultured from the patient’s own skin. The body does not reject this artificial skin, which saves lives and results in minimal scarring. However, it is more likely to become infected than is an autograft.

✓ Check Your Understanding

25. Which type of skin cancer develops from the youngest epidermal cells?
26. What name is given to the rule for recognizing the signs of melanoma?
The Integumentary System

27. The healing of burns and epidermal regeneration is usually uneventful unless the burn is a third-degree burn. What accounts for this difference?

28. Although the anterior head and face represent only a small percentage of the body surface, burns to this area are often more serious than those to the body trunk. Why?

---

For answers, see Appendix H.

Developmental Aspects of the Integumentary System

✓ Describe and attempt to explain the causes of changes that occur in the skin from birth to old age.

The epidermis develops from the embryonic ectoderm, and the dermis and hypodermis develop from mesoderm. By the end of the fourth month of development, the skin is fairly well formed. The epidermis has all its strata, dermal papillae are obvious, fingerprints have developed, and rudimentary epidermal derivatives have formed by downward projections of cells from the basal layer. During the fifth and sixth months, the fetus is covered with a downy coat of delicate colorless hairs called the lanugo coat (lah-nu’go; “wool”). This hairy cloak is shed by the seventh month, and vellus hairs appear.

From Infancy to Adulthood

When a baby is born, its skin is covered with vernix caseosa (ver’niks kā-se-o’sah; “varnish of cheese”), a white, cheesy-looking substance produced by the sebaceous glands that protects the fetus's skin within the water-filled amnion. The newborn’s skin is very thin and often has accumulations in the sebaceous glands on the forehead and nose that appear as small white spots called milia (mil’e-ah). These normally disappear by the third week after birth.

During infancy and childhood, the skin thickens, and more subcutaneous fat is deposited. Although we all have approximately the same number of sweat glands, the number that function increases in the first two years after birth and is determined by climate. For this reason, people who grow up in hot climates have more active sweat glands than those raised in cooler areas of the world.

During adolescence, the skin and hair become oilier as sebaceous glands are activated, and acne may appear. Acne generally subsides in early adulthood, and skin reaches its optimal appearance when we reach our 20s and 30s. Thereafter, the skin starts to show the effects of cumulative environmental assaults (abrasion, wind, sun, chemicals). Scaling and various kinds of skin inflammation, or dermatitis (der”mah-ti’tis), become more common.

Aging Skin

As old age approaches, the rate of epidermal cell replacement slows, the skin thins, and its susceptibility to bruises and other injuries increases. The lubricating substances produced by the skin glands that make young skin so soft become deficient. Skin becomes dry and itchy, although people with naturally oily skin seem to postpone this dryness until later in life. Elastic fibers clump, and collagen fibers become fewer and stiffer. The subcutaneous fat layer diminishes, leading to the intolerance to cold so common in elderly people. Additionally, declining levels of sex hormones result in similar fat distribution in elderly men and women.

The decreasing elasticity of the skin, along with the loss of subcutaneous tissue, inevitably leads to wrinkling. Decreasing numbers of melanocytes and dendritic cells enhance the risk and incidence of skin cancer in this age group. As a rule, redheads and fair-skinned individuals, who have less melanin to begin with, show age-related changes more rapidly than do those with darker skin and hair.

By age 50, the number of active hair follicles has declined by two-thirds and continues to fall, resulting in hair thinning. Hair loses its luster in old age, and the delayed-action genes responsible for graying and male pattern baldness become active.

Although there is no known way to avoid skin aging, one of the best ways to slow the process is to shield your skin from both the UVA (aging rays) and UVB (rays that burn) of the sun. Aged skin that has been protected from the sun, while it grows thinner and loses some elasticity, still remains unwrinkled and unmarked. Wear protective clothing and apply sunscreens or sunblocks with a sun protection factor (SPF) of 15 or higher. Remember, the same sunlight that produces that fashionable tan also causes the sagging, blotchy, wrinkled skin of old age complete with pigmented “liver spots.” Much of this havoc is due to UVA activation of enzymes called matrix metalloproteinases, which degrade collagen and other dermal components. A drug called tretinoin, related to vitamin A, inhibits these enzymes and is used in some skin creams to slow photo-aging.

Good nutrition, plenty of fluids, and cleanliness may also delay the process.

✓ Check Your Understanding

29. What is the source of vernix caseosa that covers the skin of the newborn baby?
30. What change in the skin leads to cold intolerance in the elderly?
31. How does UV radiation contribute to skin wrinkles?

For answers, see Appendix H.

The skin is only about as thick as a paper towel—not too impressive as organ systems go. Yet, when it is severely damaged, nearly every body system reacts. Metabolism accelerates or may be impaired, immune system changes occur, bones may soften, the cardiovascular system may fail—the list goes on and on. On the other hand, when the skin is intact and performing its functions, the body as a whole benefits. System Connections on p. 168 summarizes homeostatic interrelationships between the integumentary system and other organ systems.
Homeostatic Interrelationships Between the Integumentary System and Other Body Systems

- Nervous System  Chapter 6
  - Skin protects nervous system organs; cutaneous sensory receptors for touch, pressure, pain, and temperature located in skin (see Figure 5.1)
- Endocrine System  Chapter 16
  - Skin protects endocrine organs; converts some hormones to their active forms; synthesizes a vitamin D precursor
  - Androgens produced by the endocrine system activate sebaceous glands and are involved in regulating hair growth
- Cardiovascular System  Chapters 17–19
  - Skin protects cardiovascular organs; prevents fluid loss from body; serves as blood reservoir
  - Cardiovascular system transports oxygen and nutrients to skin and removes wastes from skin; provides substances needed by skin glands to make their secretions
- Lymphatic System/Immunity  Chapters 20–21
  - Skin protects lymphatic organs; prevents pathogen invasion; dendritic cells and macrophages help activate the immune system
  - Lymphatic system prevents edema by picking up excessive leaked fluid; immune system protects skin cells
- Respiratory System  Chapter 22
  - Skin protects respiratory organs; hairs in nose help filter out dust from inhaled air
  - Respiratory system furnishes oxygen to skin cells and removes carbon dioxide via gas exchange with blood
- Digestive System  Chapter 23
  - Skin protects digestive organs; provides vitamin D needed for calcium absorption; performs some of the same chemical conversions as liver cells
  - Digestive system provides needed nutrients to the skin
- Urinary System  Chapters 25–26
  - Skin protects urinary organs; excretes salts and some nitrogenous wastes in sweat
  - Urinary system activates vitamin D precursor made by keratinocytes; disposes of nitrogenous wastes of skin metabolism
- Reproductive System  Chapter 27
  - Skin protects reproductive organs; cutaneous receptors respond to erotic stimuli; highly modified sweat glands (mammary glands) produce milk
  - During pregnancy, skin stretches to accommodate growing fetus; changes in skin pigmentation may occur

**Skeletal System  Chapters 6–8**
- Skin protects bones; skin synthesizes a vitamin D precursor needed for normal calcium absorption and deposit of bone (calcium) salts, which make bones hard
- Skeletal system provides support for skin

**Muscular System  Chapters 9–10**
- Skin protects muscles
- Active muscles generate large amounts of heat, which increases blood flow to the skin and may activate sweat glands in skin

**Nervous System  Chapters 11–15**
- Skin protects nervous system organs; cutaneous sensory receptors for touch, pressure, pain, and temperature located in skin (see Figure 5.1)
Chapter Summary

For more chapter study tools, go to the Study Area of MasteringA&P at www.masteringaandp.com. There you will find:
- Interactive Physiology
- A&P Flix
- Practice Anatomy Lab
- PhysioEx
- Videos, Practice Quizzes and Tests, MP3 Tutor Sessions, Case Studies, and much more!

The Skin (pp. 150–157)
1. The skin, or integument, is composed of two discrete tissue layers, an outer epidermis and a deeper dermis, resting on subcutaneous tissue, the hypodermis.

Epidermis (pp. 151–154)
2. The epidermis is an avascular, keratinized sheet of stratified squamous epithelium. Most epidermal cells are keratinocytes. Scattered among the keratinocytes in the deepest epidermal layers are melanocytes, dendritic cells, and tactile cells.
3. From deep to superficial, the strata, or layers of the epidermis, are the basale, spinosum, granulosum, lucidum, and corneum. The stratum lucidum is absent in thin skin. The mitotically active stratum basale is the source of new cells for epidermal growth. The most superficial layers are increasingly keratinized and less viable.

Dermis (pp. 154–156)
4. The dermis, composed mainly of dense, irregular connective tissue, is well supplied with blood vessels, lymphatic vessels, and nerves. Cutaneous receptors, glands, and hair follicles reside within the dermis.
5. The more superficial papillary layer exhibits dermal papillae that protrude into the epidermis above, as well as dermal ridges. Dermal ridges and epidermal ridges together form the friction ridges that produce fingerprints.
6. In the deeper, thicker reticular layer, the connective tissue fibers are much more densely interwoven. Less dense regions between the collagen bundles produce cleavage, or tension, lines in the skin. Points of tight dermal attachment to the hypodermis produce dermal folds, or flexure lines.

Skin Color (pp. 156–157)
7. Skin color reflects the amount of pigments (melanin and carotene) in the skin and the oxygenation level of hemoglobin in blood.
8. Melanin production is stimulated by exposure to ultraviolet radiation in sunlight. Melanin, produced by melanocytes and transferred to keratinocytes, protects the keratinocyte nuclei from the damaging effects of UV radiation.
9. Skin color is affected by emotional state. Alterations in normal skin color (jaundice, bronzing, erythema, and others) may indicate certain disease states.

Appendages of the Skin (pp. 157–162)
1. Skin appendages, which derive from the epidermis, include hairs and hair follicles, nails, and glands (sweat and sebaceous).

Hairs and Hair Follicles (pp. 157–160)
2. A hair, produced by a hair follicle, consists of heavily keratinized cells. A typical hair has a central medulla, a cortex, and an outer cuticle and root and shaft portions. Hair color reflects the amount and kind of melanin present.
3. A hair follicle consists of an inner epithelial root sheath and an outer peripheral connective tissue sheath derived from the dermis. The base of the hair follicle is a hair bulb with a matrix that produces the hair. A hair follicle is richly vascularized and well supplied with nerve fibers. Arrector pili muscles pull the follicles into an upright position, producing goose bumps, and propel sebum to the skin surface when they contract.
4. Except for hairs of the scalp and around the eyes, hairs formed initially are fine vellus hairs; at puberty, under the influence of androgens, coarser, darker terminal hairs appear in the axillae and the genital region.
5. The rate of hair growth varies in different body regions and with sex and age. Differences in life span of hairs account for differences in length on different body regions. Hair thinning reflects factors that lengthen follicular resting periods, age-related atrophy of hair follicles, and a delayed-action gene.

Nails (p. 160)
6. A nail is a scalelike modification of the epidermis that covers the dorsum of a finger (or toe) tip. The actively growing region is the nail matrix.

Sweat (Sudoriferous) Glands (pp. 160–161)
7. Eccrine (merocrine) sweat glands, with a few exceptions, are distributed over the entire body surface. Their primary function is thermoregulation. They are simple coiled tubular glands that secrete a salt solution containing small amounts of other solutes. Their ducts usually empty to the skin surface via pores.
8. Apocrine sweat glands, which may function as scent glands, are found primarily in the axillary and anogenital areas. Their secretion is similar to eccrine secretion, but it also contains proteins and fatty substances on which bacteria thrive.

Sebaceous (Oil) Glands (pp. 161–162)
9. Sebaceous glands occur all over the body surface except for the palms and soles. They are simple alveolar glands; their oily holocrine secretion is called sebum. Sebaceous gland ducts usually empty into hair follicles.
10. Sebum lubricates the skin and hair, prevents water loss from the skin, and acts as a bactericidal agent. Sebaceous glands are activated (at puberty) and controlled by androgens.

Functions of the Integumentary System (pp. 162–164)
1. Protection. The skin protects by chemical barriers (the antibacterial nature of sebum, defensins, cathelicidins, the acid mantle, and the UV shield of melanin), physical barriers (the hardened keratinized and lipid-rich surface), and biological barriers (dendritic cells, macrophages, and DNA).
2. Body temperature regulation. The skin vasculature and sweat glands, regulated by the nervous system, play an important role in maintaining body temperature homeostasis.
3. Cutaneous sensation. Cutaneous sensory receptors respond to temperature, touch, pressure, and pain stimuli.
4. Metabolic functions. A vitamin D precursor is synthesized from cholesterol by epidermal cells. Skin cells also play a role in some chemical conversions.
5. Blood reservoir. The extensive vascular supply of the dermis allows the skin to act as a blood reservoir.

**Homeostatic Imbalances of Skin** (pp. 164–167)
1. The most common skin disorders result from infections.

**Skin Cancer** (pp. 164–165)
2. The most common cause of skin cancer is exposure to ultraviolet radiation.
3. Basal cell carcinoma and squamous cell carcinoma are cured if they are removed before metastasis. Melanoma, a cancer of melanocytes, is less common but more dangerous.

**Burns** (pp. 165–166)
4. In severe burns, the initial threat is loss of protein- and electrolyte-rich body fluids, which may lead to circulatory collapse. The second threat is overwhelming bacterial infection.
5. The extent of a burn may be evaluated by using the rule of nines. The severity of burns is indicated by the terms first degree, second degree, and third degree. Third-degree burns are full-thickness burns that require grafting for successful recovery.

**Developmental Aspects of the Integumentary System** (p. 167)
1. The epidermis develops from embryonic ectoderm; the dermis (and hypodermis) develops from mesoderm.
2. The fetus exhibits a downy lanugo coat. Fetal sebaceous glands produce vernix caseosa, which helps protect the fetus’s skin from its watery environment.

**From Infancy to Adulthood** (p. 167)
3. A newborn’s skin is thin. During childhood the skin thickens and more subcutaneous fat is deposited. At puberty, sebaceous glands are activated and terminal hairs appear in greater numbers.

**Aging Skin** (p. 167)
4. In old age, the rate of epidermal cell replacement declines and the skin and hair thin. Skin glands become less active. Loss of collagen and elastic fibers and subcutaneous fat leads to wrinkling; delayed-action genes cause graying and balding. Photodamage is a major cause of skin aging.

**Multiple Choice/Matching**
(Some questions have more than one correct answer. Select the best answer or answers from the choices given.)

1. Which epidermal cell type is most numerous? (a) keratinocyte, (b) melanocyte, (c) dendritic cell, (d) tactile cell.
2. Which cell functions as part of the immune system? (a) keratinocyte, (b) melanocyte, (c) dendritic cell, (d) tactile cell.
3. Basal cell carcinoma and squamous cell carcinoma are cured if they are removed before metastasis. Melanoma, a cancer of melanocytes, is less common but more dangerous.
4. Skin color is determined by (a) the amount of blood, (b) pigments, (c) oxygenation level of the blood, (d) all of these.
5. The sensations of touch and pressure are picked up by receptors located in (a) the stratum spinosum, (b) the dermis, (c) the hypodermis, (d) the stratum corneum.
6. Which is not a true statement about the papillary layer of the dermis? (a) It is largely areolar connective tissue, (b) It is most responsible for the toughness of the skin, (c) It contains nerve endings that respond to stimuli, (d) It is highly vascular.
7. Skin surface markings that reflect points of tight dermal attachment to underlying tissues are called (a) tension lines, (b) papillary ridges, (c) flexure lines, (d) dermal papillae.
8. Which of the following is not an epidermal derivative? (a) Hair, (b) sweat gland, (c) sensory receptor, (d) sebaceous gland.
9. An arrector pili muscle (a) is associated with each sweat gland, (b) can cause a hair to stand up straight, (c) enables each hair to be stretched when wet, (d) provides new cells for continued growth of its associated hair.
10. The product of this type of sweat gland includes protein and lipid substances that become odoriferous as a result of bacterial action: (a) apocrine gland, (b) eccrine gland, (c) sebaceous gland, (d) pancreatic gland.
11. Sebum (a) lubricates the surface of the skin and hair, (b) consists of cell fragments and fatty substances, (c) in excess may cause seborrhea, (d) all of these.
12. The rule of nines is helpful clinically in (a) diagnosing skin cancer, (b) estimating the extent of a burn, (c) estimating how serious a cancer is, (d) preventing acne.

**Short Answer Essay Questions**
13. Which epidermal cells are also called prickle cells? Which contain keratohyaline and lamellar granules?
15. You go to the beach to swim on an extremely hot, sunny summer afternoon. Describe two ways in which your integumentary system acts to preserve homeostasis during your outing.
17. Describe the process of hair formation, and list several factors that may influence (a) growth cycles and (b) hair texture.
18. What color does carotene impart to the skin?
19. Why does skin wrinkle and what factors accelerate the wrinkling process?
20. Explain each of these familiar phenomena in terms of what you learned in this chapter: (a) pimples, (b) dandruff, (c) greasy hair and “shiny nose,” (d) stretch marks from gaining weight, (e) freckles.
21. Count Dracula, the most famous vampire, rumored to have killed at least 200,000 people, was based on a real person who lived in eastern Europe about 600 years ago. He was indeed a “monster,” although he was not a real vampire. The historical Count Dracula may have suffered from which of the following? (Hint: See Related Clinical Terms.) (a) porphyria, (b) EB, (c) halitosis, (d) vitiligo. Explain your answer.
22. Why are there no skin cancers that originate from stratum corneum cells?

23. A man got his finger caught in a machine at the factory. The damage was less serious than expected, but the entire nail was torn off his right index finger. The parts lost were the body, root, bed, matrix, and eponychium of the nail. First, define each of these parts. Then, tell if this nail is likely to grow back.

24. On an outline diagram of the human body, mark off various regions according to the rule of nines. What percentage of the total body surface is affected if the skin over the following body parts is burned? (a) the entire posterior trunk and buttocks, (b) an entire lower limb, (c) the entire front of the left upper limb.

25. A common belief is that having your hair cut makes it become thicker. Explain why this belief is not true.

---

### Critical Thinking and Clinical Application Questions

1. Dean, a 40-year-old aging beach boy, is complaining to you that although his suntan made him popular when he was young, now his face is all wrinkled, and he has several darkly pigmented moles that are growing rapidly and are as big as large coins. He shows you the moles, and immediately you think “ABCD.” What does that mean and why should he be concerned?

---

### AT THE CLINIC

#### Related Clinical Terms

**Albinism** (al-bi-nizm; *alb* = white) Inherited condition in which melanocytes do not synthesize melanin owing to a lack of tyrosinase. An albino’s skin is pink, the hair pale or white, and the irises of the eyes unpigmented or poorly so.

**Boils and carbuncles** (kar-bung-klz; “little glowing embers”) Inflammation of hair follicles and sebaceous glands in which an infection has spread to the underlying hypodermis; common on the dorsal neck. Carbuncles are composite boils. A common cause is bacterial infection.

**Cold sores (fever blisters)** Small fluid-filled blisters that itch and smart; usually occur around the lips and in the mucosa of the mouth; caused by a herpes simplex infection. The virus localizes in a cutaneous nerve, where it remains dormant until activated by emotional upset, fever, or UV radiation.

**Contact dermatitis** Itching, redness, and swelling, progressing to blister formation; caused by exposure of the skin to chemicals (e.g., poison ivy oleoresin) that provoke an allergic response in sensitive individuals.

**Decubitus ulcer** (de-ku’bi-tus) Localized breakdown and ulceration of skin due to interference with its blood supply. Usually occurs over a bony prominence, such as the hip or heel, that is subjected to continuous pressure; also called a bedsore.

**Dermatology** The branch of medicine that studies and treats disorders of the skin.

**Eczema** (ek’ze-mah) A skin rash characterized by itching, blistering, oozing, and scaling of the skin. A common allergic reaction in children, but also occurs (typically in a more severe form) in adults. Frequent causes include allergic reactions to certain foods (fish, eggs, and others) or to inhaled dust or pollen. Treated by methods used for other allergic disorders.

**Epidermolysis bullosa (EB)** A group of hereditary disorders characterized by inadequate or faulty synthesis of keratin, collagen, and/or basement membrane “cement” that results in lack of cohesion between layers of the skin and mucosa. A simple touch causes layers to separate and blister. For this reason, EB victims are called “touch-me-nots.” In severe cases fatal blistering occurs in major vital organs. Because the blisters rupture easily, victims suffer frequent infections. Treatments are aimed at relieving the symptoms and preventing infection.

**Impetigo** (im’pé-ti’go; *impet* = an attack) Pink, fluid-filled, raised lesions (common around the mouth and nose) that develop a yellow crust and eventually rupture. Caused by staphylococcus infection, it is contagious, and common in school-age children.

**Porphyria** (por-fer’e-ah; “purple”) An inherited condition in which certain enzymes needed to form the heme of hemoglobin of blood are lacking. Without these enzymes, metabolic intermediates of the heme pathway called porphyrins build up, spill into the circulation, and eventually cause lesions throughout the body, especially when exposed to sunlight. The skin becomes lesioned and scarred; fingers, toes, and nose are disfigured; gums degenerate and teeth become prominent. Believed to be the basis of folklore about vampires.

**Psoriasis** (so-ri’ah-sis) A chronic autoimmune condition characterized by raised, redened epidermal patches covered with silvery scales that itch or burn, crack, and sometimes bleed or become infected. When severe, it may be disfiguring and
debilitating. Trauma, infection, hormonal changes, or stress often trigger the autoimmune attacks. Cortisone-containing topicals (medications applied to the skin surface) may control mild cases. For more severe cases, self-injected drugs called biologicals and/or phototherapy with UV light in conjunction with chemotherapeutic drugs provides some relief.

**Rosacea** (ro-za’she-ah) A chronic skin eruption produced by dilated small blood vessels of the face, particularly the nose and cheeks. Papules and acne-like pustules may or may not occur. More common in women, but tends to be more severe when it occurs in men. Cause is unknown, but stress, some endocrine disorders, and anything that produces flushing (hot beverages, alcohol, sunlight, etc.) can aggravate this condition.

**Vitiligo** (vit’il’-li’go; viti = a vine, winding) The most prevalent skin pigmentation disorder, characterized by a loss of melanocytes and uneven dispersal of melanin, so that unpigmented skin regions (light spots) are surrounded by normally pigmented areas. An autoimmune disorder.

**Scleroderma** (scler = hard) An autoimmune disorder characterized by stiff, hardened skin due to abnormal amounts of collagen in the dermis that severely limit joint movements and facial expressions. A classic sign of the disorder is Raynaud disease in which the fingers and toes become white and painful because of poor blood flow to those areas. The fibrosis that occurs in systemic cases may affect a variety of organs including the lungs, eventually leading to suffocation, and the kidneys, leading to renal hypertension because of blood vessel constriction and occlusion. Environmental factors including organic solvents, asbestos, and even silicone breast implants have all been suspect scleroderma triggers.

---

**Case Study Integumentary System**

A terrible collision between a trailer truck and a bus has occurred on Route 91. Several of the passengers are rushed to area hospitals for treatment. We will follow a few of these people in clinical case studies that will continue through the book from one organ system to the next.

Examination of Mrs. DeStephano, a 45-year-old woman, reveals several impairments of homeostasis. Relative to her integumentary system, the following comments are noted on her chart:

- Epidermal abrasions of the right arm and shoulder
- Severe lacerations of the right cheek and temple
- Cyanosis apparent

The lacerated areas are cleaned, sutured (stitched), and bandaged by the emergency room (ER) personnel, and Mrs. DeStephano is admitted for further tests.

Relative to her signs:

1. What protective mechanisms are impaired or deficient in the abraded areas?
2. Assuming that bacteria are penetrating the dermis in these areas, what remaining skin defenses might act to prevent further bacterial invasion?
3. What benefit is conferred by suturing the lacerations? (Hint: See Chapter 4, p. 149, Related Clinical Terms, healing by first intention.)
4. Mrs. DeStephano's cyanotic skin may hint at what additional problem (and impairment of what body systems or functions)?

(Answers in Appendix H)