SECTION 4

Protection and survival

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CHAPTER 14

The skin

ANIMATION

The skin

Learning outcomes

After studying this section, you should be able to:

- describe the structure of the skin
- explain the principal functions of the skin
- compare and contrast the processes of primary and secondary wound healing.

The first part of this chapter explores the structure and functions of the skin, which is also known as the integumentary system. The effects of ageing on the skin are discussed in the following section. The chapter concludes with a review of common skin conditions.

The skin completely covers the body and is continuous with the membranes lining the body orifices. It:

- protects the underlying structures from injury and from invasion by microbes
- contains sensory nerve endings that enable discrimination of pain, temperature and touch
- is involved in the regulation of body temperature.

Structure of the skin

The skin is the largest organ in the body and has a surface area of about 1.5–2 m^2 in adults. In certain areas, it contains accessory structures: glands, hair and nails. There are two main layers; the *epidermis*, which covers the *dermis*. Between the skin and underlying structures is a subcutaneous layer composed of areolar tissue and adipose (fat) tissue.

Epidermis

This is the most superficial layer and is composed of *stratified keratinised squamous epithelium* (see Fig. 3.13, p. 39). It varies in thickness, being thickest on the palms of the hands and soles of the feet. There are no blood vessels or nerve endings in the epidermis, but its deeper layers are bathed in interstitial fluid from the dermis, which provides oxygen and nutrients, and drains away as lymph.

There are several layers (strata) of cells in the epidermis which extend from the deepest *germinative layer* to the most superficial *stratum corneum* (a thick horny layer) (Fig. 14.1). Epidermal cells originate in the germinative layer and undergo gradual change as they progress towards the skin surface. The cells on the surface are flat, thin, non-nucleated, dead cells, or *squames*, in which the cytoplasm has been replaced by the fibrous protein *keratin*. The surface cells are constantly rubbed off and replaced by those beneath. Complete replacement of the epidermis takes about a month.

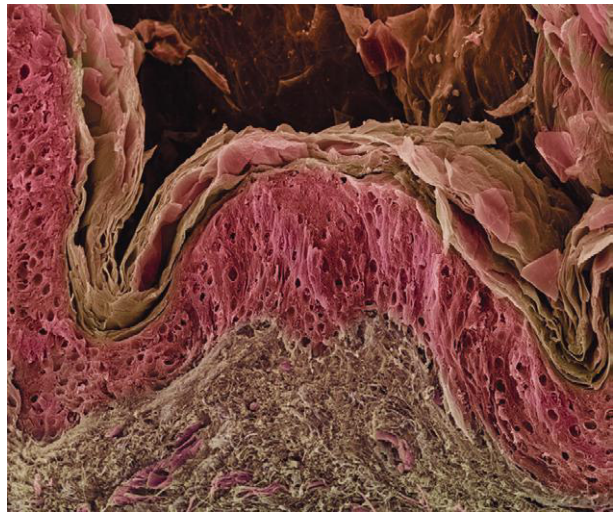

Figure 14.1 Coloured scanning electron micrograph of the skin showing the superficial stratum corneum (pale brown), above the lower layers of the epidermis (pink) and the dermis (grey brown).

Healthy epidermis depends upon three processes being synchronised:

- desquamation (shedding) of the keratinised cells from the surface
- effective keratinisation of cells approaching the surface
- continual cell division in the deeper layers with newly formed cells being pushed upwards to the surface.

Hairs, secretions from sebaceous glands and ducts of sweat glands pass through the epidermis to reach the surface.

Upward projections of the dermal layer, the *dermal papillae* (Fig. 14.2), anchor this securely to the more superficial epidermis and allow passage and exchange of nutrients and wastes to the lower part of the epidermis. This arrangement stabilises the two layers preventing damage due to shearing forces. *Blisters* develop when trauma causes separation of the dermis and epidermis, and serous fluid collects between the two layers.

In areas where the skin is subject to greater wear and tear, e.g. the palms and fingers of the hands and soles of the feet, the epidermis is thicker and hairs are absent. In these areas the dermal papillae are arranged in parallel lines giving the skin surface a ridged appearance. The pattern of ridges on the fingertips is unique to every individual and the impression made by them is the 'fingerprint'.

Skin colour is affected by various factors.

- Melanin, a dark pigment derived from the amino acid tyrosine and secreted by *melanocytes* in the deep germinative layer, is absorbed by surrounding epithelial cells. The amount is genetically determined and varies between different parts of the body,

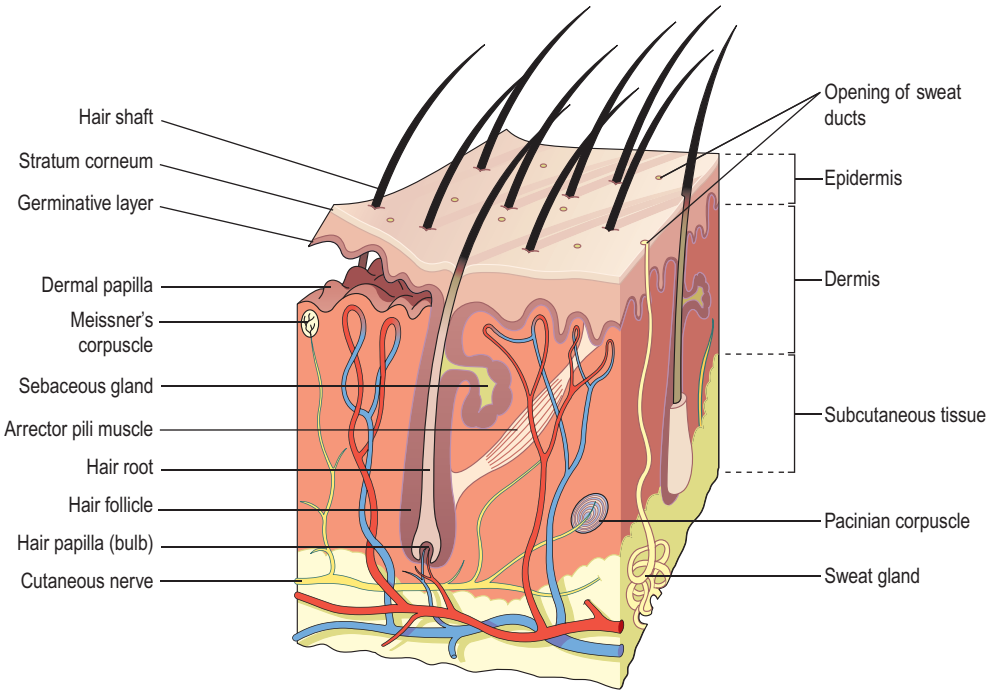

Figure 14.2 The skin showing the main structures in the dermis.

between people of the same ethnic origin and between ethnic groups. The number of melanocytes is fairly constant so the differences in colour depend on the amount of melanin secreted. It protects the skin from the harmful effects of ultraviolet rays in sunlight. Exposure to sunlight promotes synthesis of melanin.

- Normal saturation of haemoglobin (p. 66) and the amount of blood circulating in the dermis give white skin its pink colour. When oxygen saturation is very low, the skin may appear bluish (*cyanosis*).
- Excessive levels of bile pigments in blood and carotenes in subcutaneous fat give the skin a yellowish colour.

Dermis (Fig. 14.2)

The dermis is tough and elastic. It is formed from connective tissue and the matrix contains *collagen fibres* (see Fig. 3.16, p. 40) interlaced with *elastic fibres*. Rupture of elastic fibres occurs when the skin is overstretched, resulting in permanent *striae*, or stretch marks, that may be found in pregnancy and obesity. Collagen fibres bind water and give the skin its tensile strength, but as this ability declines with age, wrinkles develop. Fibroblasts (see Fig. 3.5, p. 34), macrophages (Fig. 3.17, p. 40) and mast cells are the main cells found in the dermis. Underlying its deepest layer is the subcutaneous layer containing areolar tissue and varying amounts of adipose (fat) tissue. The structures in the dermis are:

- blood and lymph vessels
- sensory nerve endings
- sweat glands and their ducts
- hairs, arrector pili muscles and sebaceous glands.

Blood and lymph vessels. Arterioles form a fine network with capillary branches supplying sweat glands, sebaceous glands, hair follicles and the dermis. Lymph vessels form a network throughout the dermis.

Sensory nerve endings. Sensory receptors (specialised nerve endings) sensitive to *touch, temperature, pressure* and *pain* are widely distributed in the dermis. Incoming stimuli activate different types of sensory receptors (Fig. 14.2, Box 14.1); for example, the Pacinian corpuscle is sensitive to deep pressure (Fig. 14.3). The skin is an important sensory organ through which individuals receive information about their environment. Nerve impulses, generated in the sensory receptors in the dermis, are transmitted to the spinal cord by sensory nerves (Fig. 14.4). From there impulses are conducted to the sensory area of the cerebrum where the sensations are perceived (see Fig. 7.22B, p. 158).

Box 14.1 Sensory receptors in the skin

Sensory receptor	Stimulus
Meissner's corpuscle	Light pressure
Pacinian corpuscle	Deep pressure
Free nerve ending	Pain

Figure 14.3 Pacinian corpuscle.

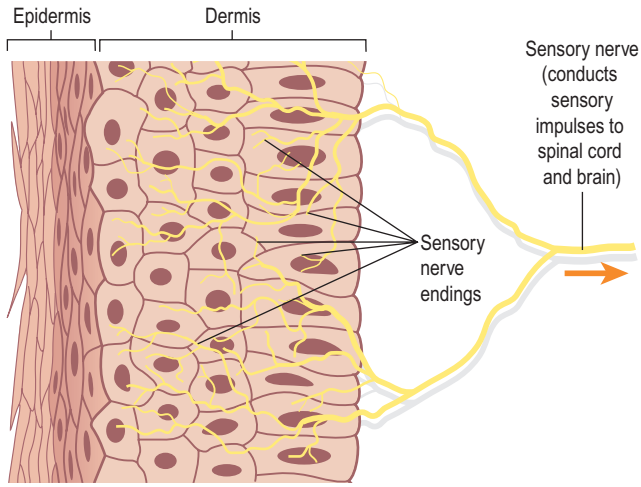

Figure 14.4 Sensory nerves in the dermis.

Sweat glands

These are widely distributed throughout the skin and are most numerous in the palms of the hands, soles of the feet, axillae and groins. They are formed from epithelial cells. The bodies of the glands lie coiled in the subcutaneous tissue. There are two types of sweat gland. Eccrine sweat glands are the more common type and open onto the skin surface through tiny pores, and the sweat produced here is a clear, watery fluid important in regulating body temperature. Apocrine glands open into hair follicles and become active at puberty. They may play a role in sexual arousal. These glands are found, for example, in the axilla. Bacterial decomposition of their secretions causes an unpleasant odour. A specialised example of this type of gland is the ceruminous gland of the outer ear, which secretes earwax (Ch. 8).

The most important function of sweat is in the regulation of body temperature (p. 367). Excessive sweating may lead to dehydration and serious depletion of sodium chloride unless intake of water and salt is appropriately increased. After 7–10 days' exposure to high environmental temperatures the amount of salt lost is substantially reduced but water loss remains high.

Figure 14.5 Coloured scanning electron micrograph of hair shafts growing through the skin.

Hairs

These grow from *hair follicles*, downgrowths of epidermal cells into the dermis or subcutaneous tissue. At the base of the follicle is a cluster of cells called the *hair papilla* or *bulb*. The hair is formed by multiplication of cells of the bulb and as they are pushed upwards, away from their source of nutrition, the cells die and become keratinised. The part of the hair above the skin is the *shaft* and the remainder, the *root* (Fig. 14.2). Figure 14.5 shows hair growing through the skin and also desquamation, which roughens the skin surface; the roughened surface may harbour microbial growth although many are removed by the constant rubbing off of the topmost layers.

Hair colour is genetically determined and depends on the amount and type of melanin present. White hair is the result of the replacement of melanin by tiny air bubbles.

Arrector pili (Fig. 14.2)**.** These are little bundles of smooth muscle fibres attached to the hair follicles. Contraction makes the hair stand erect and raises the skin around the hair, causing 'goose flesh'. The muscles are stimulated by sympathetic nerve fibres in response to fear and cold. Erect hairs trap air, which acts as an insulating layer. This is an efficient warming mechanism, especially when accompanied by shivering, i.e. involuntary contraction of skeletal muscles.

Sebaceous glands (Fig. 14.2). These consist of secretory epithelial cells derived from the same tissue as the hair follicles. They secrete an oily antimicrobial substance, *sebum*, into the hair follicles and are present in the skin of all parts of the body except the palms of the hands and the soles of the feet. They are most numerous in the scalp, face, axillae and groins. In regions of transition from one type of superficial epithelium to another, such as lips, eyelids, nipple, labia minora and glans penis, there are sebaceous glands that are independent of hair follicles, secreting sebum directly onto the surface.

Sebum keeps the hair soft and pliable and gives it a shiny appearance. On the skin it provides some waterproofing and acts as a bactericidal and fungicidal agent, preventing infection. It also prevents drying and cracking of skin, especially on exposure to heat and sunlight. The activity of these glands increases at puberty and is less at the extremes of age, rendering the skin of infants and older adults prone to the effects of excessive moisture (*maceration*).

Nails (Fig. 14.6)

Human nails are equivalent to the claws, horns and hooves of animals. Derived from the same cells as epidermis and hair these are hard, horny keratin plates that protect the tips of the fingers and toes.

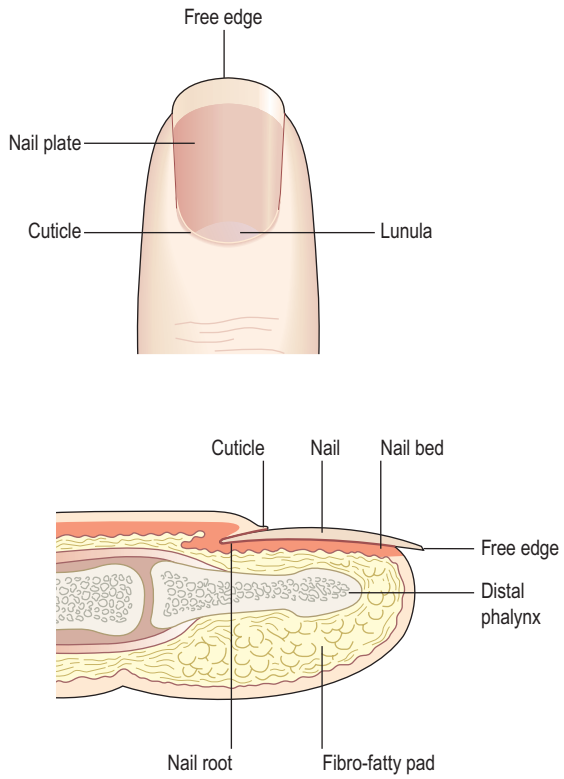

Figure 14.6 The nail and related skin.

The *root* of the nail is embedded in the skin and covered by the cuticle, which forms the hemispherical pale area called the *lunula*.

The *nail plate* is the exposed part that has grown out from the *nail bed*, the germinative zone of the epidermis.

Finger nails grow more quickly than toe nails and growth is faster when the environmental temperature is high.

Functions of the skin

Protection

The skin forms a relatively waterproof layer, provided mainly by its keratinised epithelium, which protects the deeper, more delicate structures. As an important non-specific defence mechanism it acts as a barrier against:

- invasion by micro-organisms
- chemicals
- physical agents, e.g. mild trauma, ultraviolet light
- dehydration.

The epidermis contains specialised immune cells called dendritic (Langerhans) cells, which are a type of macrophage. They phagocytose intruding antigens and travel to lymphoid tissue, where they present antigen to T-lymphocytes, thus stimulating an immune response (Ch. 15).

Abundant sensory nerve endings in the dermis enable perception, discrimination and location of internal and external stimuli. This allows responses to changes in the environment, e.g. by reflex action (withdrawal) to unpleasant or painful stimuli, protecting it from further injury (p. 164).

The pigment melanin protects against harmful ultraviolet rays in sunlight.

Regulation of body temperature

Body temperature remains fairly constant around 36.8°C across a wide range of environmental temperatures ensuring that the optimal range for enzyme activity required for metabolism is maintained. In health, variations are usually limited to between 0.5 and 0.75°C, although it rises slightly in the evening, during exercise and in women just after ovulation. To maintain this constant temperature, a negative feedback system regulates the balance between heat produced in the body and heat lost to the environment.

Heat production

When metabolic rate increases, body temperature rises, and when it decreases body temperature falls. Some of the energy released during metabolic activity is in the form of heat; the most active organs produce most heat. The principal organs involved are:

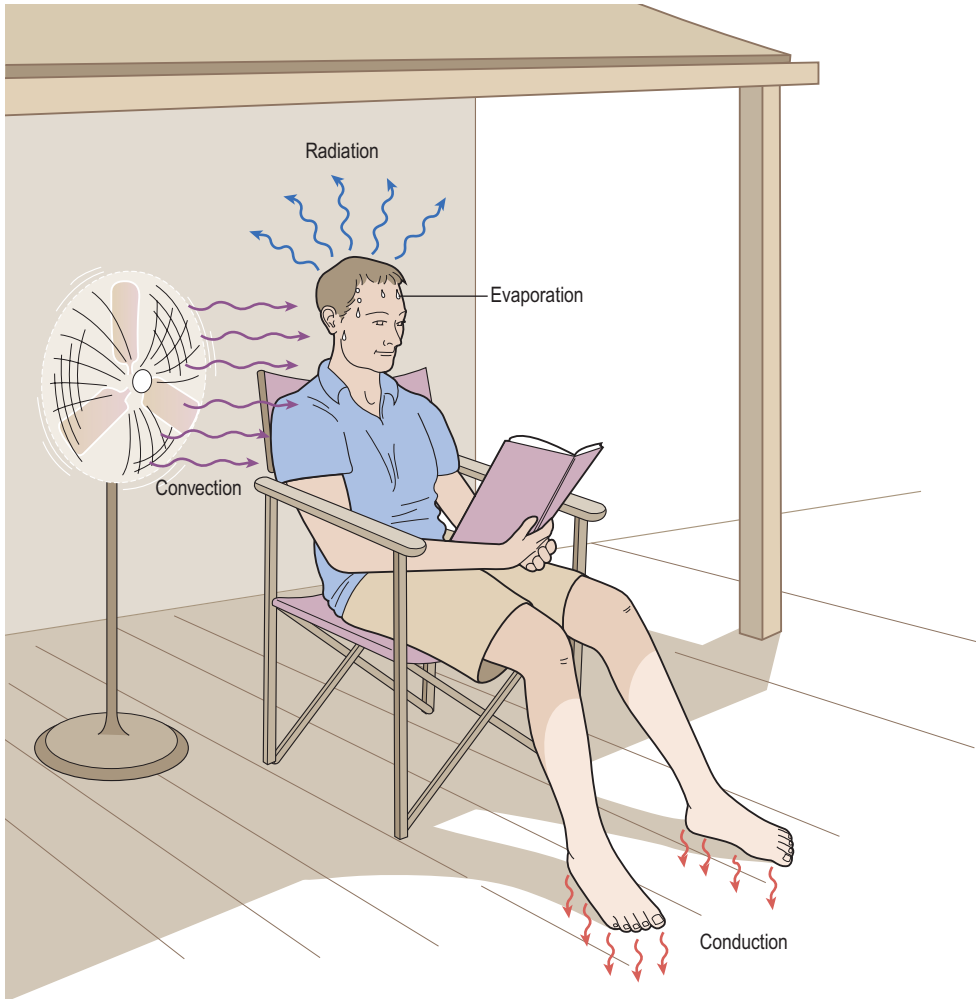

Figure 14.7 Mechanisms of heat loss.

- *skeletal muscles* – contraction of skeletal muscles produces a large amount of heat and the more strenuous the muscular exercise, the greater the heat produced. Shivering also involves skeletal muscle contraction, which increases heat production when there is the risk of body temperature falling below normal.
- *the liver* is very metabolically active, which produces heat as a by-product. Metabolic rate and heat production are increased after eating.
- *the digestive organs* that generate heat during peristalsis and the chemical reactions involved in digestion.

Heat loss

Most heat loss from the body occurs through the skin. Small amounts are lost in expired air, urine and faeces. Only heat loss through the skin can be regulated; heat lost by the other routes cannot be controlled.

Heat loss through the skin is affected by the difference between body and environmental temperatures, the amount of the body surface exposed and the type of clothes worn. Air insulates against heat loss when trapped in layers of clothing and between the skin and clothing. For this reason several layers of lightweight clothes provide more effective insulation against low environmental temperatures than one heavy garment.

Mechanisms of heat loss (Fig. 14.7)**.** In *radiation*, the main mechanism, exposed parts of the body radiate heat away from the body. In *evaporation*, the body is cooled as body heat converts the water in sweat to water vapour. In *conduction*, clothes and other objects in direct contact with the skin take up heat. In *convection*, air passing over the exposed parts of the body is heated and rises, cool air replaces it and convection currents are set up. Convection also cools the body when clothes are worn, except when they are windproof.

Control of body temperature

The *temperature regulating centre* in the hypothalamus is sensitive to the temperature of circulating blood. This

centre responds to decreasing temperature by sending nerve impulses to:

- arterioles in the dermis, which constrict decreasing blood flow to the skin
- skeletal muscles stimulating shivering.

As heat is conserved, body temperature rises and when it returns to the normal range again the negative feedback mechanism is switched off (see Fig. 1.5, p. 7).

Conversely when body temperature rises, heat loss is increased by dilation of arterioles in the dermis, increasing blood flow to the skin, and stimulation of the sweat glands causing sweating, until it falls into the normal range again when the negative feedback mechanism is switched off.

Activity of the sweat glands. When body temperature is increased by 0.25 to 0.5°C the sweat glands secrete sweat onto the skin surface. Evaporation of sweat cools the body, but is slower in humid conditions.

Loss of heat from the body by evaporation of water through the skin and expired air still occurs even when the environmental temperature is low. This is called *insensible water loss* (around 500 mL per day) and is accompanied by insensible heat loss.

Regulation of blood flow through the skin. The amount of heat lost from the skin depends largely on blood flow through dermal capillaries. As body temperature rises, the arterioles dilate and more blood enters the capillary network in the skin. The skin is warm and pink in colour. In addition to increasing the amount of sweat produced, the temperature of the skin rises and more heat is lost by radiation, conduction and convection.

If the environmental temperature is low or if heat production is decreased, the arterioles in the dermis are constricted. This reduces blood flow to the body surface, conserving heat. The skin appears paler and feels cool.

Fever

This is often the result of infection and is caused by release of chemicals (*pyrogens*) from inflammatory cells and invading bacteria. Pyrogens, e.g. *interleukin 1* (p. 379), act on the hypothalamus, which releases prostaglandins that reset the hypothalamic thermostat to a higher temperature. The body responds by activating heat-promoting mechanisms, e.g. shivering and vasoconstriction, until the new higher temperature is reached. When the thermostat is reset to the normal level, heat-loss mechanisms are activated. There is profuse sweating and vasodilation accompanied by warm, pink (flushed) skin until body temperature falls to the normal range again.

Hypothermia

This means a core (e.g. rectal) temperature below 35°C. At a core temperature below 32°C, compensatory mechanisms that restore body temperature normally fail, e.g. shivering is replaced by muscle rigidity and cramps, vasoconstriction fails and blood pressure, pulse and respiration rates fall. Confusion and disorientation occur. Death usually occurs when the temperature falls below 25°C.

Individuals at the extremes of age are prone to hypothermia as temperature regulation is less effective in the young and older adults.

Formation of vitamin D

7-Dehydrocholesterol is a lipid-based substance in the skin and is converted to vitamin D by sunlight. This vitamin is used with calcium and phosphate in the formation and maintenance of bone.

Cutaneous sensation

Sensory receptors are nerve endings in the dermis that are sensitive to touch, pressure, temperature or pain. Stimulation generates nerve impulses in sensory nerves that are transmitted to the cerebral cortex (see Fig. 7.22, p. 158). Some areas have more sensory receptors than others causing them to be especially sensitive, e.g. the lips and fingertips.

Absorption

This property is limited but substances that can be absorbed include:

- some drugs, in transdermal patches, e.g. hormone replacement therapy during the menopause, nicotine as an aid to smoking cessation
- some toxic chemicals, e.g. mercury.

Excretion

The skin is a minor excretory organ for some substances including:

- sodium chloride in sweat; excess sweating may lead to low blood sodium levels (hyponatraemia)
- urea, especially when kidney function is impaired
- aromatic substances, e.g. garlic and other spices.

Wound healing

Conditions required for wound healing

Systemic factors. These include good nutritional status and general health. Infection, impaired immunity, poor blood supply and systemic conditions, e.g. diabetes mellitus and cancer, reduce the rate of wound healing.

Local factors. Local factors that facilitate wound healing include a good blood supply to provide oxygen and nutrients and remove waste products, and freedom from contamination by, e.g., microbes, foreign bodies or toxic chemicals.

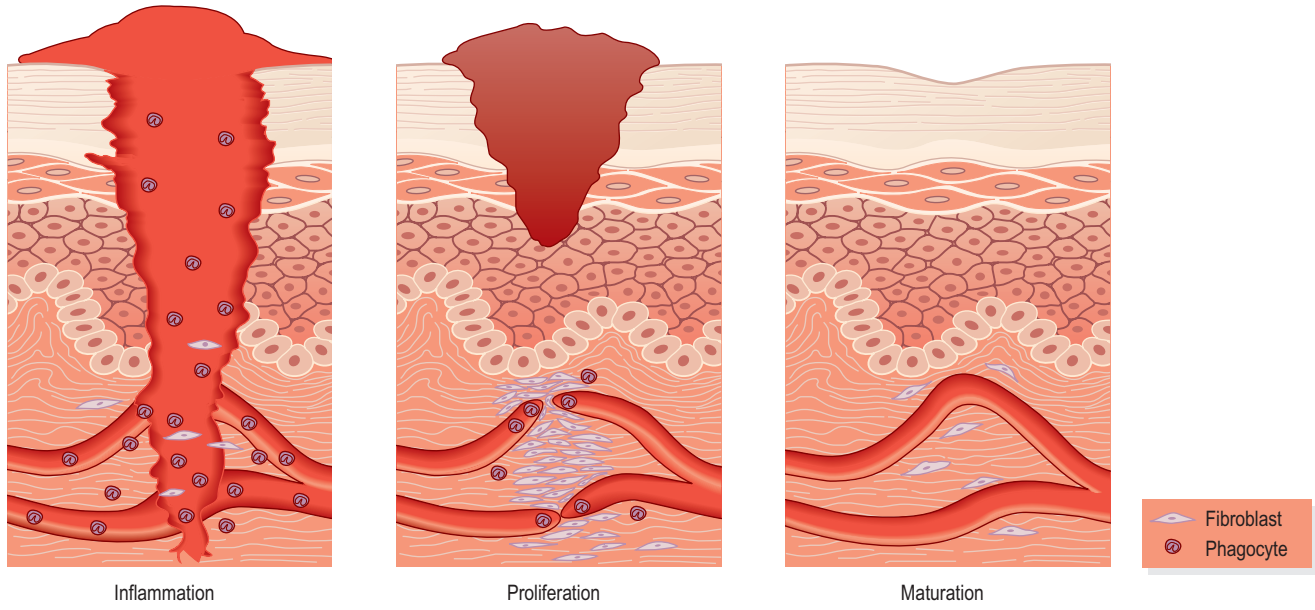

Figure 14.8 Stages in primary wound healing.

Primary healing (healing by first intention)

This type of healing follows minimal destruction of tissue when the damaged edges of a wound are in close apposition, e.g. a surgical incision (Fig. 14.8). There are several overlapping stages in the repair process.

Inflammation. In the first few hours the cut surfaces become inflamed and blood clot (mainly fibrin, Fig. 4.15, p. 71) and cell debris fill the gap between them. Phagocytes, including macrophages, and fibroblasts migrate into the blood clot:

- phagocytes begin to remove the clot and cell debris, stimulating fibroblast activity
- fibroblasts secrete collagen fibres which begin to bind the wound margins together.

Proliferation. Epithelial cells proliferate across the wound, through the clot. The epidermis meets and grows upwards until full thickness is restored. The clot above the new tissue becomes the scab, which separates after 3–10 days. *Granulation tissue*, consisting of new capillary buds, phagocytes and fibroblasts, develops, invading the clot and restoring blood supply to the wound. Fibroblasts continue to secrete collagen fibres as the clot and any bacteria are removed by phagocytosis.

Maturation. The granulation tissue is replaced by fibrous scar tissue. Rearrangement of collagen fibres occurs and the strength of the wound increases. In time the scar becomes less vascular, appearing after a few months as a fine line.

The channels left when stitches are removed heal by the same process.

Secondary healing (healing by second intention)

This type of healing follows extensive tissue destruction or when the edges of a wound cannot be brought into apposition, e.g. varicose ulcers and pressure (decubitus) ulcers. The stages of secondary healing (Fig. 14.9) are the same as in primary healing (see above); healing time depends on effective removal of the cause and the size of the wound.

Inflammation. This develops on the surface of the healthy tissue and separation of necrotic tissue (*slough*) begins, due mainly to the action of phagocytes in the inflammatory exudate. The inflammatory process is described on page 377.

Proliferation. This begins as granulation tissue; consisting of capillary buds, phagocytes and fibroblasts; develops at the base of the cavity. Granulation tissue grows towards the surface, probably stimulated by macrophages and a range of locally released chemicals. Phagocytes in the plentiful blood supply reduce or prevent infection of the wound by ingesting bacteria after separation of the slough. Some fibroblasts in the wound develop a limited ability to contract, reducing the size of the wound and healing time. When granulation tissue reaches the level of the dermis, epithelial cells at the edges proliferate and grow towards the centre.

Maturation. This occurs by *fibrosis* (see below), in which scar tissue replaces granulation tissue, usually over several months until the full thickness of the skin is restored. Scar tissue is shiny and does not contain sweat glands, hair follicles or sebaceous glands.

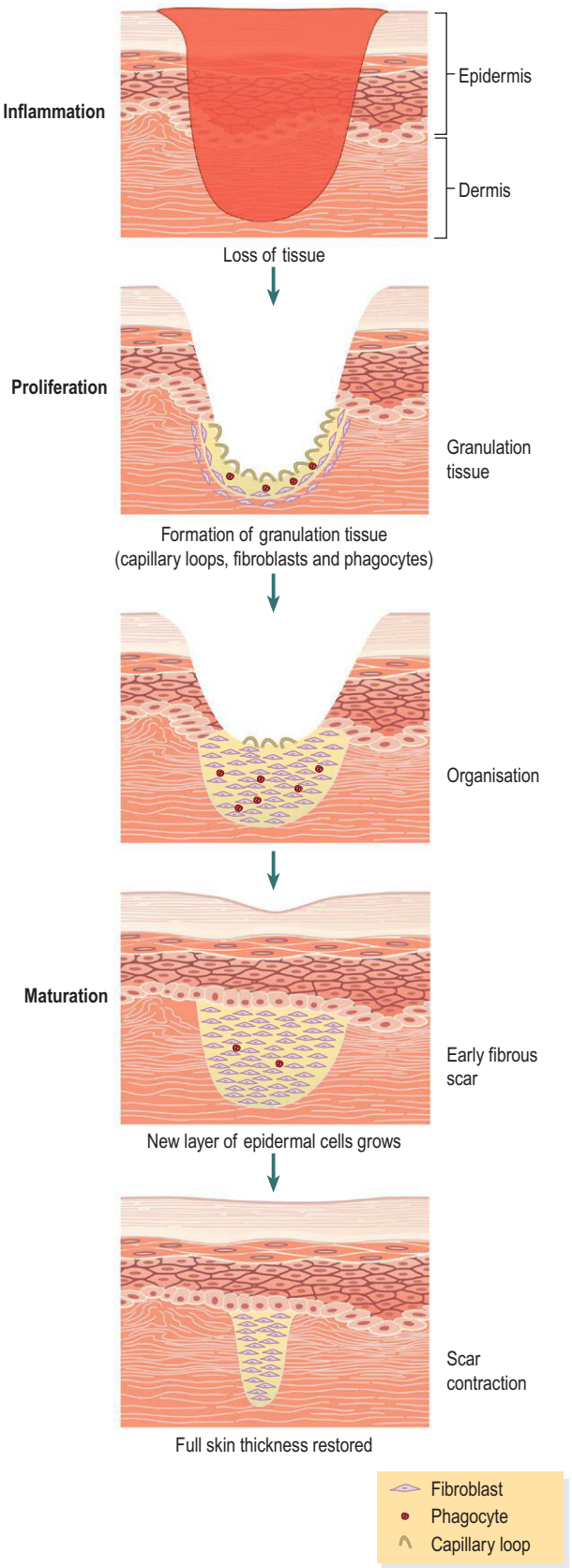

Figure 14.9 Stages in secondary wound healing.

Fibrosis (scar formation)

Fibrous tissue is formed during healing by secondary intention, e.g. following chronic inflammation, persistent ischaemia, suppuration or extensive trauma. The process begins with formation of granulation tissue, then, over time, the inflammatory material is removed leaving only the collagen fibres secreted by the fibroblasts. Fibrous tissue may have long-lasting damaging effects.

Adhesions. These consist of fibrous tissue, which causes adjacent structures to stick together and may limit movement, e.g. between the layers of pleura, preventing inflation of the lungs or between loops of bowel, interfering with peristalsis.

Fibrosis of infarcts. Blockage of a vessel by a thrombus or an embolus causes an infarction. Fibrosis of one large infarct or of numerous small infarcts may follow, leading to varying degrees of organ dysfunction, e.g. in heart, brain, kidneys, liver.

Tissue shrinkage. This occurs as fibrous tissue ages. The effects depend on the site and extent of the fibrosis, e.g.:

- small tubes, such as blood vessels, air passages, ureters, the urethra and ducts of glands may become narrow or obstructed and lose their elasticity
- contractures (bands of shrunken fibrous tissue) may extend across joints, e.g. in a limb or digit there may be limitation of movement.

Complications of wound healing

In addition to the effects of adhesions, fibrosis of infarcts and tissue shrinkage described above, other complications are outlined below.

Infection. This arises from microbial contamination, usually by bacteria, and results in formation of pus (*suppuration*).

Pus consists of dead phagocytes, dead cells, cell debris, fibrin, inflammatory exudate and living and dead microbes. The most common pyogenic (pus-forming) pathogens are *Staphylococcus aureus* and *Streptococcus pyogenes*. Small amounts of pus form *boils* and larger amounts form *abscesses*. *S. aureus* produces the enzyme coagulase, which converts fibrinogen to fibrin, localising the pus. *S. pyogenes* produces toxins that break down tissue, spreading the infection. Healing, following pus formation, is by secondary intention (see above).

Superficial abscesses tend to rupture and discharge pus through the skin. Healing is usually complete unless tissue damage is extensive.

Deep abscesses have a variety of outcomes. There may be:

- early rupture with complete discharge of pus on to the surface, followed by healing

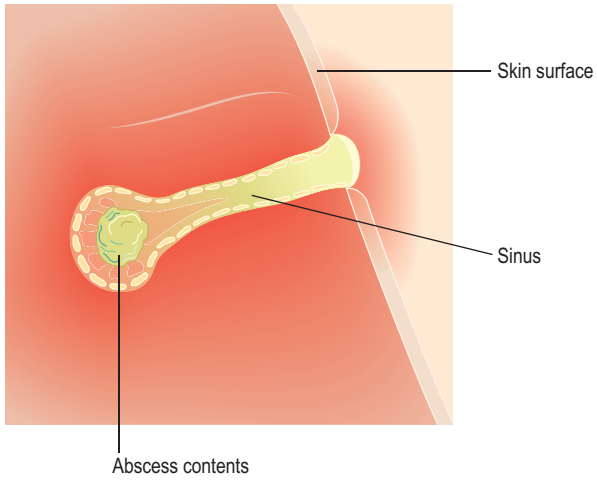

Figure 14.10 Sinus between an abscess and the body surface.

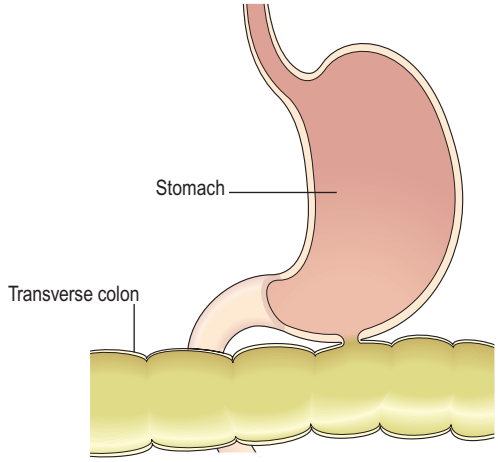

Figure 14.11 Fistula between the stomach and the colon.

- rupture and limited discharge of pus on to the surface, followed by the development of a chronic abscess with an infected open channel or *sinus* (Fig. 14.10)
- rupture and discharge of pus into an adjacent organ or cavity, forming an infected channel open at both ends, a *fistula* (Fig. 14.11)
- eventual removal of pus by phagocytes, followed by healing
- enclosure of pus by fibrous tissue that may become calcified, harbouring live organisms which may become a source of future infection, e.g. tuberculosis
- formation of adhesions (see above) between adjacent membranes, e.g. pleura, peritoneum
- shrinkage of fibrous tissue as it ages, which may reduce the lumen or obstruct a tube, e.g. oesophagus, bowel, blood vessel.

Effects of ageing on the skin

Learning outcome

After studying this section, you should be able to:

- describe the effects of ageing on the structure and function of the skin.

From the third decade, there are gradual changes in the structure and functioning of the skin which become much more prominent in old age. As the germinative layer becomes less active, the epidermis thins. The dermis also thins and there are fewer elastic and collagen fibres, which causes wrinkling and sagging. These changes may be accelerated by chronic exposure to strong sunlight, which is also associated with the development of malignant melanoma.

Sweat gland activity and temperature regulation become less efficient, putting older adults at greater risk in extreme temperatures making them increasingly prone to heatstroke and hypothermia. Less sebum is secreted making the skin dry and susceptible to continual exposure to moisture (maceration).

Production of vitamin D decreases predisposing older adults to deficiency and reduction in bone strength, especially when exposure to sunlight is limited.

Melanocytes become less active causing older adults to be more sensitive to sunlight and more prone to sunburn. In hair, when the pigment melanin is replaced by air bubbles, greying occurs. There are fewer active hair follicles and therefore hair thins although in some areas this is not the case; notably the eyebrows, nose and ears in males, and the face and upper lip in females.

Disorders of the skin

Learning outcomes

After studying this section, you should be able to:

- list the causes of diseases in this section
- explain the pathological features and effects of common skin conditions.

Infections

Viral infections

Human papilloma virus (HPV)

This causes *warts* or *veruccas* that are spread by direct contact, e.g. from another lesion or another infected individual. There is proliferation of the epidermis and development of a small firm growth, which is nearly always benign. Common sites are the hands, the face and soles of the feet.

Herpes viruses

Rashes seen in chickenpox and shingles (p. 184) are caused by the herpes zoster virus. Other herpes viruses cause *cold sores* (HSV1, p. 320) and *genital herpes* (HSV2, p. 466).

Bacterial infections

Impetigo

This is a highly infectious condition commonly caused by *Staphylococcus aureus*. Superficial pustules develop, usually round the nose and mouth. It is spread by direct contact and affects mainly children and immunosuppressed individuals. When caused by *Streptococcus pyogenes* (group A β-haemolytic streptococcus) the infection may be complicated by an immune reaction causing glomerulonephritis (p. 350) a few weeks later.

Cellulitis

This is a spreading infection caused by some anaerobic bacteria including *Streptococcus pyogenes* and *Clostridium perfringens* that enter through a break in the skin. Their spread is facilitated by the formation of enzymes that break down the connective tissue that normally isolates an area of inflammation. If untreated, the bacteria may enter the blood causing septicaemia.

In severe cases *necrotising fasciitis* may occur. There is rapid and progressive necrosis of subcutaneous tissue that usually includes the fascia in the affected area. Multiple organ failure is common and mortality is high.

Fungal infections (mycoses)

Ringworm and tinea pedis

These are superficial skin infections. In ringworm there is an outward spreading ring of inflammation. It most commonly affects the scalp, feet and groin and is easily spread to others. Tinea pedis (athlete's foot) affects the skin between the toes. Both infections are spread by direct contact.

Non-infective inflammatory conditions

Dermatitis (eczema)

Dermatitis is a common inflammatory skin condition that may be either acute or chronic. In acute dermatitis there is redness, swelling and exudation of serous fluid usually accompanied by *pruritus* (itching). This is often followed by crusting and scaling. If the condition becomes chronic, the skin thickens and may become leathery due to long-term scratching, which may cause infection.

Atopic dermatitis is associated with allergy and commonly affects atopic individuals, i.e. those prone to hypersensitivity disorders (p. 385). Children, who may also suffer from hay fever or asthma (pp. 262 and 264), are often affected.

Contact dermatitis may be caused by direct contact with irritants, e.g. cosmetics, soap, detergent, strong acids or alkalis, industrial chemicals or a hypersensitivity reaction (see Fig. 15.9, p. 386) to, e.g., latex, nickel, dyes and other chemicals.

Psoriasis

This common condition is genetically determined and characterised by exacerbations and periods of remission of varying duration. Proliferating cells of the basal layers of the epidermis progress more rapidly upwards through the epidermis resulting in incomplete maturation of the upper layer. Psoriasis is characterised by red, scaly plaques with a silvery surface (Fig. 14.12). Bleeding may occur when scales are scratched or rubbed off. The elbows, knees and scalp are common sites but other parts can also be affected. Trigger factors that lead to exacerbation of the condition include trauma, infection and sunburn. Sometimes psoriasis is associated with rheumatoid arthritis (p. 432).

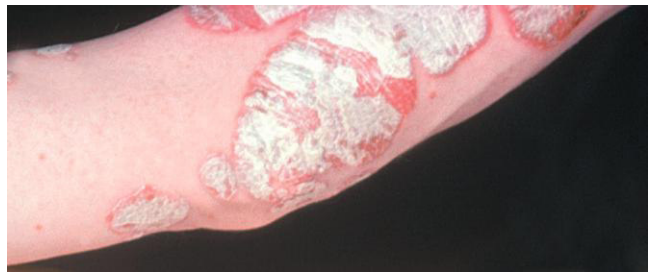

Figure 14.12 Psoriasis.

Acne vulgaris

This is commonest in adolescent males and thought to be caused by increased levels of testosterone after puberty. Sebaceous glands (in hair follicles) become blocked and then infected, leading to inflammation and pustule formation. In severe cases permanent scarring may result. The most common sites are the face, chest and upper back.

Pressure ulcers

Also known as *decubitus ulcers* or bedsores, these occur over 'pressure points', areas where the skin may be compressed for long periods between a bony prominence and a hard surface, e.g. a bed or chair. When this occurs, blood flow to the affected area is impaired and ischaemia develops. Initially the skin reddens, and later as ischaemia and necrosis occur, the skin sloughs and an ulcer forms that may then enlarge into a cavity. If infection occurs, this can result in septicaemia. Healing takes place by secondary intention (p. 368).

Predisposing factors

These may be:

- extrinsic, e.g. pressure, shearing forces, trauma, immobility, moisture, infection
- intrinsic, e.g. poor nutritional status, emaciation, incontinence, infection, concurrent illness, sensory impairment, poor circulation.

Burns

These may be caused by many types of trauma including: heat, cold, electricity, ionising radiation and corrosive chemicals, including strong acids or alkalis (bases). Local damage occurs disrupting the structure and functions of the skin.

Burns are classified according to their depth:

- *first degree* when only the epidermis is involved, the surface is moist and there are signs of inflammation including redness, swelling and pain. There are no blisters and tissue damage is minimal.
- *second degree* when the epidermis and upper dermis are affected. In addition to the signs and symptoms above, blistering is usually present.
- *third degree* (deep or full thickness) when the epidermis and dermis are destroyed. These burns are usually relatively painless as the sensory nerve endings in the dermis are destroyed. After a few days the destroyed tissue coagulates and forms an *eschar*, or thick scab, which sloughs off after 2 to 3 weeks. In *circumferential burns*, which encircle any area of the body, complications may arise from constriction of the part by eschar, e.g. respiratory impairment may follow circumferential burns of the chest, or the circulation to the distal part of an affected limb may be seriously impaired. Skin grafting is required except for small injuries. Healing, which is prolonged, occurs by secondary intention (p. 368) and there is no regeneration of sweat glands, hair follicles or sebaceous glands. Resultant scar tissue often limits movement of affected joints. **14.1**

The extent of burns in adults is roughly estimated using the 'rule of nines' (Fig. 14.13). In adults, hypovolaemic shock usually develops when 15% of the surface area is affected. Fatality is likely in adults with third degree burns if the surface area affected is added to the patient's age and the total is greater than 80.

Complications of burns

Although burns affect the skin, when extensive, their systemic consequences can also be life-threatening or fatal.

Dehydration and hypovolaemia. These may occur in extensive burns when there is excessive leakage of water and plasma proteins from the damaged skin surface.

Shock. This may accompany severe hypovolaemia.

Hypothermia. This develops when excessive heat is lost in leakage from burns.

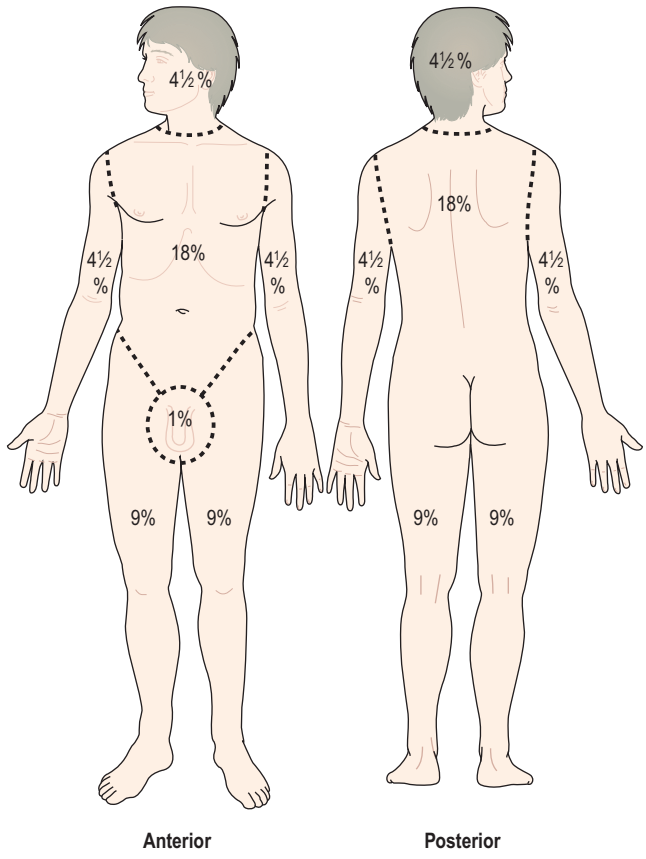

Figure 14.13 The 'rule of nines' for estimating the extent of burns in adults.

Infection. This occurs easily when subcutaneous tissue is exposed to the environment and may result in septicaemia.

Renal failure. This occurs when the kidney tubules cannot deal with the large amount of waste from haemolysed erythrocytes and damaged tissue.

Contractures. These may develop later as fibrous scar tissue contracts distorting joints, e.g. the hands, restricting their range of motion.

Malignant tumours

Basal cell carcinoma

This is the least malignant and most common type of skin cancer. It is associated with long-term exposure to sunlight and is therefore most likely to occur on sun-exposed sites, usually the head or neck. It appears as a shiny nodule and later this breaks down, becoming an ulcer with irregular edges, commonly called a *rodent ulcer*. Although this is locally invasive it seldom metastasises.

Malignant melanoma

This is malignant proliferation of melanocytes, usually originating in a mole that enlarges and may have an irregular outline (Fig. 14.14). It may ulcerate and bleed and most commonly affects young and middle-aged adults. Predisposing factors are a fair skin and recurrent episodes of intensive exposure to sunlight including repeated episodes of sunburn, especially in childhood. Sites for this tumour show a gender bias, with the lower leg being the commonest site in females and the upper back in males. Metastases usually develop early and are associated with a poor prognosis. Initial spread is usually to nearby lymph nodes and is followed by blood-spread metastases in the liver, brain, lungs, bowel and bone marrow.

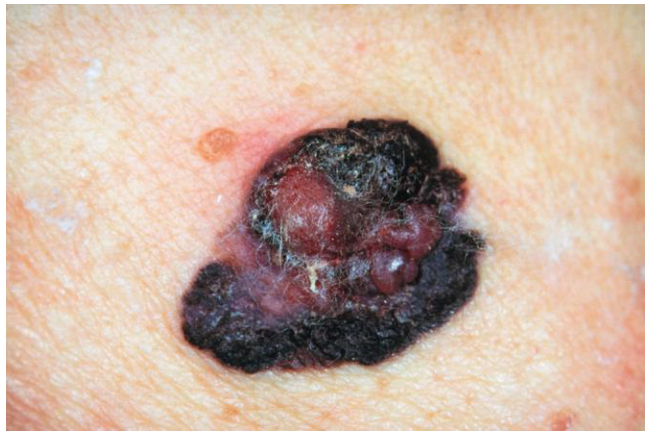

Figure 14.14 Malignant melanoma.

Kaposi's sarcoma

In this condition, which is usually AIDS-related, a malignant tumour arises in the walls of lymphatic vessels. A small red-blue patch or nodule develops, usually on the lower limbs but the mouth, oesophagus, stomach and intestines can also be affected. Without treatment skin lesions enlarge and become more numerous.

For a range of self-assessment exercises on the topics in this chapter, visit Evolve online resources: https://evolve.elsevier.com/Waugh/anatomy/