16

The skeleton

BONE

Learning outcomes

After studying this section you should be able to:

- list five types of bones and give an example of each
- outline the general structure of a long bone
- describe the structure of compact and cancellous bone tissue
- describe the development of bone
- outline hormonal regulation of growth of bone
- state the functions of bones.

Bone is a strong and durable type of connective tissue. It consists of:

- water (25%)
- organic constituents including *osteoid* (the carbon-containing part of the matrix) and bone cells (25%)
- inorganic constituents, mainly calcium phosphate (50%).

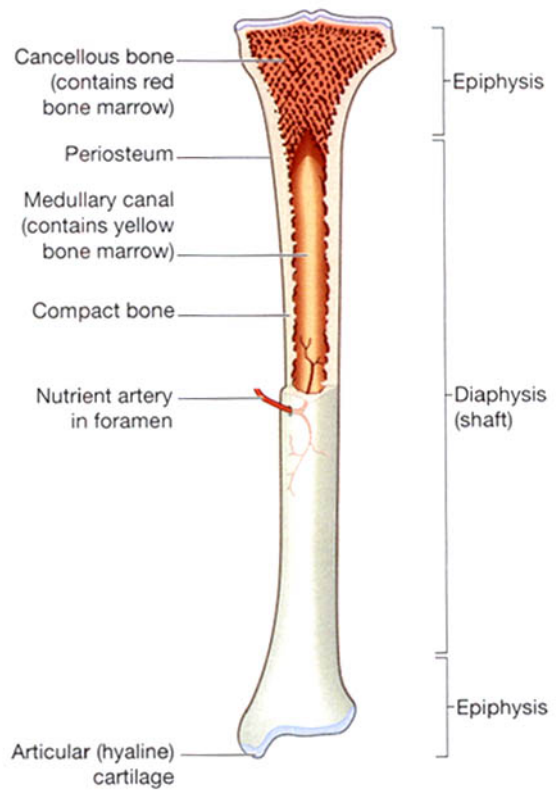


Figure 16.1 A mature long bone – partially sectioned.

Although bones are often thought to be static or permanent they are highly vascular living structures that are continuously being remodelled.

Types of bones

Bones are classified as long, short, irregular, flat and sesamoid.

Long bones. These consist of a shaft and two extremities. As the name suggests the length is much greater than the width. Examples include the femur, tibia and fibula.

Short, irregular, flat and sesamoid bones. These have no shafts or extremities and are diverse in shape and size. Examples include:

- short bones – carpals (wrist)
- irregular bones – vertebrae and some skull bones
- flat bones – sternum, ribs and most skull bones
- sesamoid bones – patella (knee cap).

Bone structure

General structure of a long bone (Fig. 16.1)

These have a *diaphysis* or shaft and two *epiphyses* or extremities. The diaphysis is composed of *compact bone* with a central medullary canal, containing fatty *yellow bone marrow*. The epiphyses consist of an outer covering of compact bone with *cancellous bone* inside. The diaphysis and epiphyses are separated by *epiphyseal cartilages*, which ossify when growth is complete. Thickening of a bone occurs by the deposition of new bone tissue under the periosteum.

Long bones are almost completely covered by a vascular membrane, the *periosteum*. The outer layer is fibrous and the inner layer is osteogenic containing *osteoblasts* (bone-forming cells) and *osteoclasts* (bone-destroying cells), which are involved in maintenance and remodelling of bones; it gives attachment to muscles and tendons and protects bones from injury. *Hyaline cartilage* replaces periosteum on the articular surfaces of bones forming synovial joints.

Structure of short, irregular, flat and sesamoid bones

These have a relatively thin outer layer of compact bone with cancellous bone inside containing *red bone marrow* (Fig. 16.2). They are enclosed by periosteum except the inner layer of the cranial bones where it is replaced by dura mater.

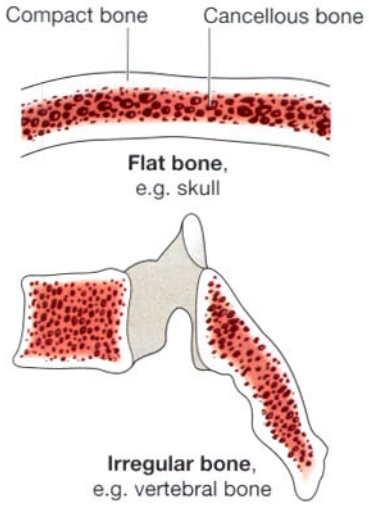


Figure 16.2 Sections of flat and irregular bones.

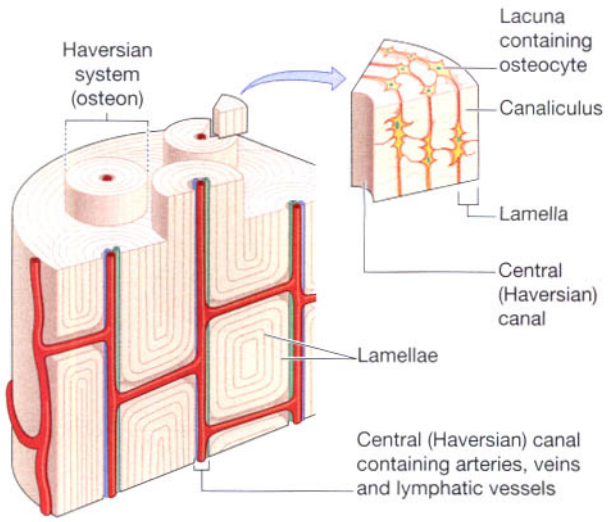


Figure 16.3 Microscopic structure of compact bone.

Microscopic structure of bone

Compact (cortical) bone

To the naked eye, compact bone appears solid but on microscopic examination large numbers of *Haversian systems* or *osteons* are seen (Fig. 16.3). These consist of a central Haversian canal, containing blood and lymph vessels and nerves, surrounded by concentric rings or plates of bone (*lamellae*). Between these are *lacunae*, tiny spaces, containing tissue fluid and spider-shaped *osteocytes* (mature bone cells). *Canaliculi* link the lacunae with each other and with the central Haversian canal. The tissue fluid nourishes the bone cells. The areas between Haversian systems contain *interstitial lamellae*, remains of older systems partially broken down during remodelling or growth of bone. The 'tubular' arrangement of lamellae gives bone greater strength than a solid structure of the same size.

Cancellous (trabecular, spongy) bone

To the naked eye, cancellous bone looks like a honeycomb. Microscopic examination reveals a framework formed from *trabeculae* (meaning 'little beams'), which consist of a few lamellae and osteocytes interconnected by canaliculi (Fig. 16.4). The spaces between the trabeculae contain *red bone marrow* that nourishes the osteocytes.

Bone cells

The cells responsible for bone formation are *osteoblasts* (these later mature into *osteocytes*). Osteoblasts and *chondrocytes* (cartilage-forming cells) develop from the same parent fibrous tissue cells. Differentiation into *osteogenic cells*, rather than *chondroblasts*, is believed to depend upon an adequate oxygen supply. This may be a factor affecting healing of fractures, i.e. if the oxygen supply is deficient there may be a preponderance of chondroblasts, resulting in a cartilaginous union of the fracture.

Osteoblasts

These are the bone-forming cells that secrete collagen and other constituents of bone tissue. They are present:

- in the deeper layers of periosteum
- in the centres of ossification of immature bone
- at the ends of the diaphysis adjacent to the epiphyseal cartilages of long bones
- at the site of a fracture.

Osteocytes

As bone develops, osteoblasts become trapped and remain isolated in lacunae. They stop forming new bone at this stage and are called *osteocytes*. Osteocytes are nourished by tissue fluid in the canaliculi that radiate from the Haversian canals. Their functions are not clear but they may be associated with the movement of calcium between the bones and the blood.

Osteoclasts

Their function is resorption of bone to maintain the optimum shape. This takes place at bone surfaces:

- under the periosteum, to maintain the shape of bones during growth and to remove excess callus formed during healing of fractures
- round the walls of the medullary canal during growth and to canalise callus during healing.

A fine balance of osteoblast and osteoclast activity maintains normal bone structure and functions.

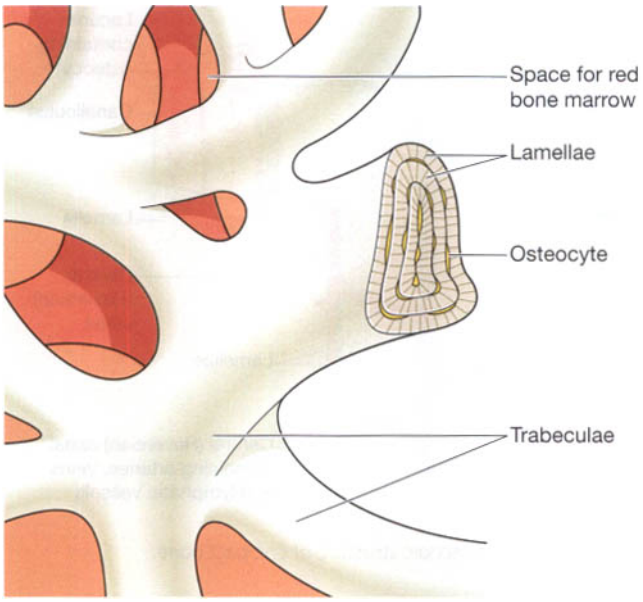


Figure 16.4 Microscopic structure of cancellous bone.

Development of bone tissue (osteogenesis or ossification)

This begins before birth and is not complete until about the 21st year of life (Fig. 16.5). Long, short and irregular bones develop from rods of cartilage, *cartilage models*. Flat bones develop from *membrane models* and sesamoid bones from *tendon models*. Bone development consists of two processes:

- secretion by osteoblasts of *osteoid*, i.e. collagen fibres in a mucopolysaccharide matrix which gradually replaces the original cartilage and membrane models
- calcification of osteoid immediately after its deposition.

There are two types of arrangement of collagen in osteoid.

Woven (non-lamellar) bone. Collagen fibres are deposited in irregular bundles, then ossified. This primitive bone structure is part of normal fetal development occurring during ossification of bones that originate as membrane models, e.g. skull bones. In adults it is also present in bone tumours and healing fractures (p. 407).

Lamellar bone. The collagen fibres are deposited as in woven bone, organised into characteristic lamellae found in compact and cancellous bone then ossified. This occurs when cartilage models are replaced by bone and in healing of fractures.

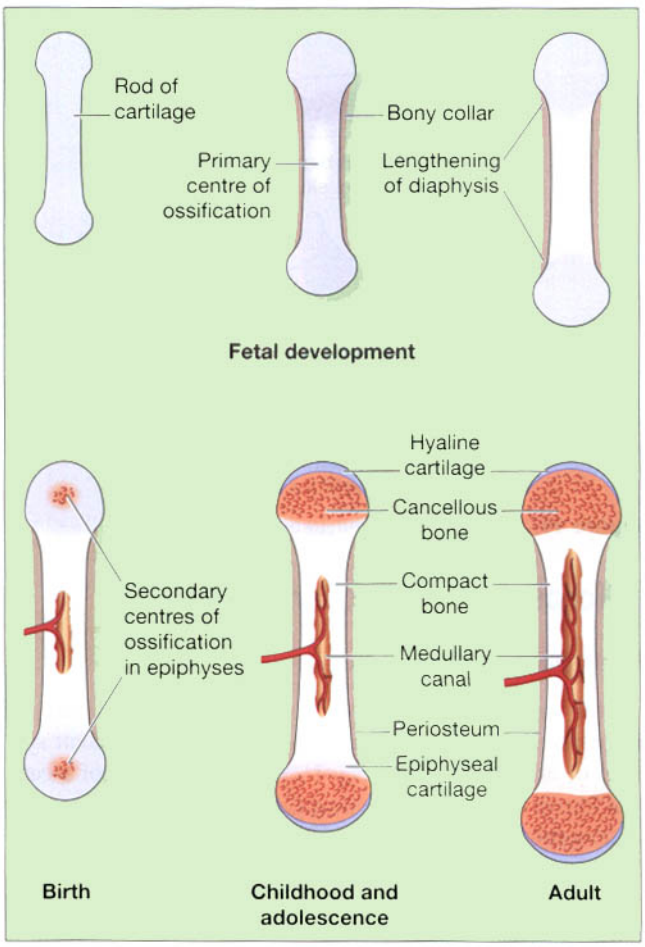


Figure 16.5 The stages of development of a long bone.

Development of long bones

In long bones the focal points from which ossification begins are small areas of osteogenic cells, or *centres of ossification* in the cartilage model. This is accompanied by development of a bone collar at about 8 weeks of gestation. Later the blood supply develops and bone tissue replaces cartilage as osteoblasts secrete osteoid components in the shaft. The bone lengthens as ossification continues and spreads to the epiphyses. Around birth, secondary centres of ossification develop in the epiphyses and the medullary canal forms when osteoclasts break down the central bone tissue in the middle of the shaft. After birth, the bone grows in length by ossification of the diaphyseal surface of the epiphyseal cartilages and growth is complete when the cartilages become completely ossified (Fig. 16.5).

Hormonal regulation of bone growth

Hormones that regulate the growth and consistency of size and shape of bones include the following.

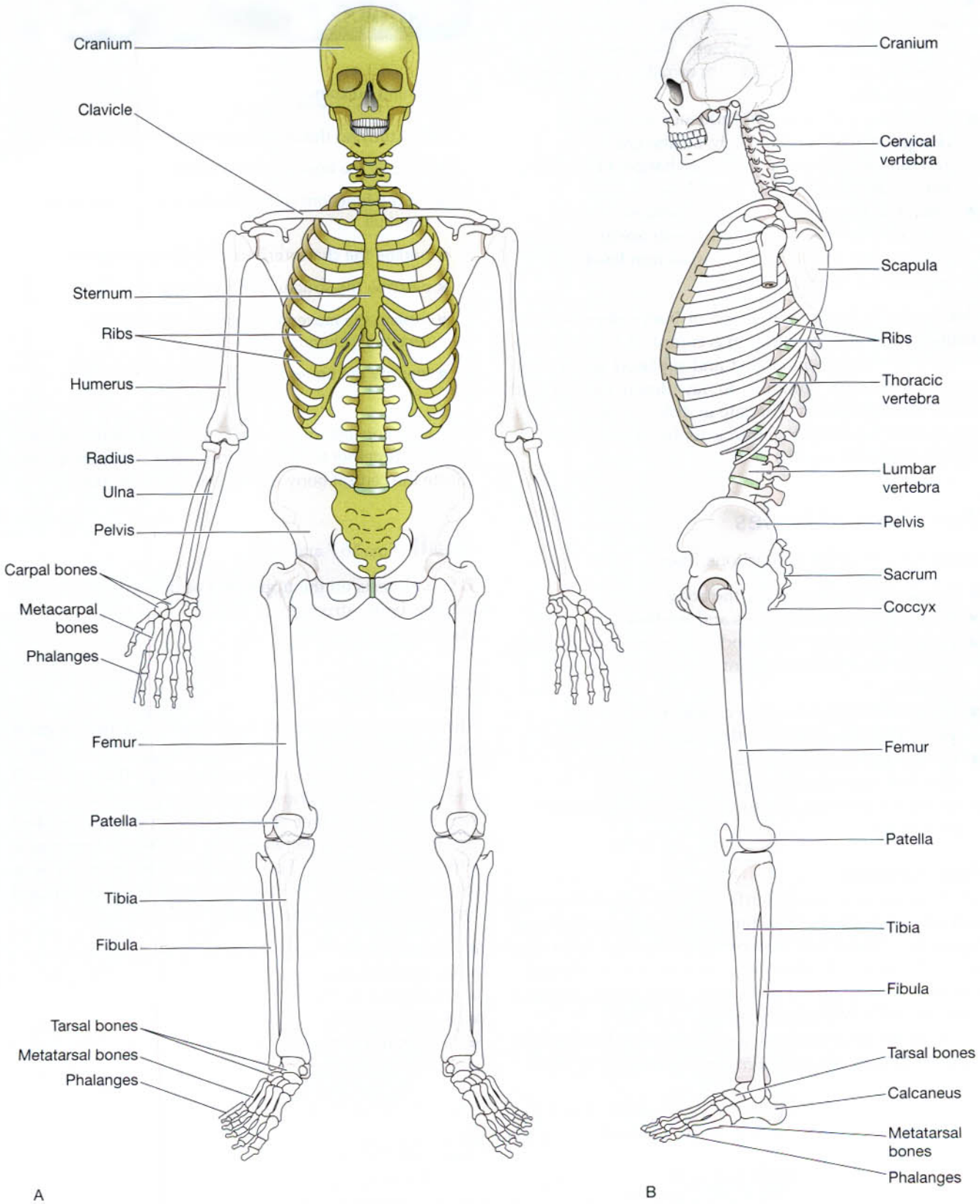


Figure 16.6 The skeleton. A. Anterior view: axial skeleton – gold; appendicular skeleton – brown. B. Lateral view.

- *Growth hormone* and the thyroid hormones, *thyroxine* and *triiodothyronine*, are especially important during infancy and childhood; deficient or excessive secretion of these results in abnormal development of the skeleton.
- *Testosterone* and *oestrogens* influence the physical changes that occur at puberty, i.e. the growth spurt and masculinising or feminising changes of specific parts of the skeleton, e.g. the pelvis.
- *Calcitonin* from the thyroid gland and *parathyroid hormone* from the parathyroid glands are involved in homeostasis of blood and bone calcium levels required for bone development.

Although the length and shape of bones does not normally change after ossification is complete, bone tissue is continually being remodelled and replaced when damaged. Osteoblasts continue to lay down osteoid and osteoclasts reabsorb it. The rate in different bones varies, e.g. the distal part of the femur is replaced gradually over a period of 5 to 6 months.

Functions of bones

Bones have a variety of functions. They:

- provide the framework of the body
- give attachment to muscles and tendons
- permit movement of the body as a whole and of parts of the body, by forming joints that are moved by muscles
- form the boundaries of the cranial, thoracic and pelvic cavities, protecting the organs they contain
- contain red bone marrow in which blood cells develop: haematopoiesis (see Fig. 4.2, p. 62)
- provide a reservoir of minerals, especially calcium phosphate.

Bone markings

Most bones have rough surfaces, raised protuberances and ridges which give attachment to muscle tendons and ligaments. These are not included in the following descriptions of individual bones unless they are of particular note, but many are marked on illustrations. Related terminology is defined on page 45.

The bones of the skeleton are divided into two groups: the *axial skeleton* and the *appendicular skeleton* (Fig. 16.6).

AXIAL SKELETON

Learning outcomes

After studying this section you should be able to:

- identify the bones of the skull (face and cranium)
- list the functions of the sinuses and fontanelles of the skull
- outline the characteristics of a typical vertebra
- describe the structure of the vertebral column
- explain the movements and functions of the vertebral column
- identify the bones that form the thoracic cage.

This part consists of the *skull, vertebral column, ribs* and *sternum*. Together the bones forming these structures constitute the central bony core of the body, the axis.

Skull (Figs 16.7 and 16.8)

The skull rests on the upper end of the vertebral column and its bony structure is divided into two parts: the cranium and the face.

Cranium

The cranium is formed by a number of flat and irregular bones that provide a bony protection for the brain. It has a *base* upon which the brain rests and a *vault* that surrounds and covers it. The periosteum inside the skull bones consists of the outer layer of dura mater. In the mature skull the joints (*sutures*) between the bones are immovable (fibrous). The bones have numerous perforations (e.g. foramina, fissures) through which nerves, blood and lymph vessels pass. The bones of the cranium are:

- 1 frontal bone
- 2 parietal bones
- 2 temporal bones
- 1 occipital bone
- 1 sphenoid bone
- 1 ethmoid bone.

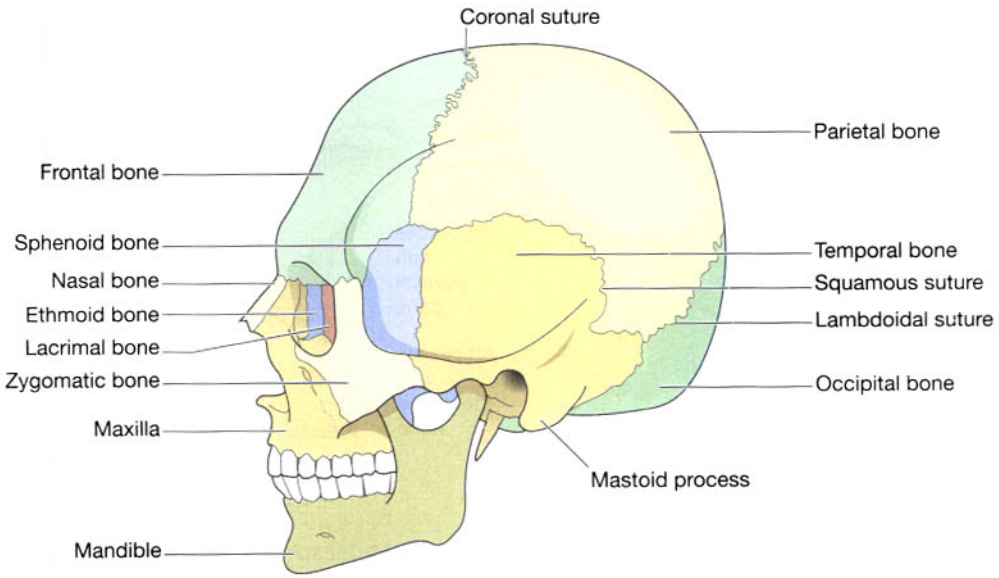


Figure 16.7 The bones of the skull and their sutures (joints).

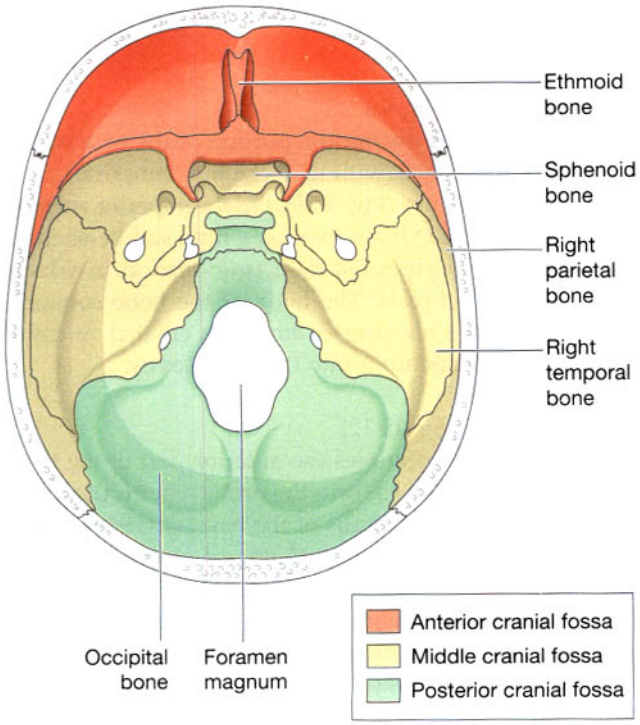


Figure 16.8 The bones forming the base of the skull and the cranial fossae. Viewed from above.

Frontal bone

This is the bone of the forehead. It forms part of the *orbital cavities* (eye sockets) and the prominent ridges above the eyes, the *supraorbital margins*. Just above the supraorbital margins, within the bone, there are two air-filled cavities or *sinuses* lined with ciliated mucous membrane which have openings into the nasal cavity.

The *coronal suture* joins the frontal and parietal bones and other fibrous joints are formed with the sphenoid, zygomatic, lacrimal, nasal and ethmoid bones. The bone originates in two parts joined in the midline by the *frontal suture* (Fig. 16.15).

Parietal bones

These bones form the sides and roof of the skull. They articulate with each other at the *sagittal suture*, with the frontal bone at the coronal suture, with the occipital bone at the *lambdoidal suture* and with the temporal bones at the *squamous sutures*. The inner surface is concave and is grooved by the brain and blood vessels.

Temporal bones (Fig. 16.9)

These bones lie one on each side of the head and form immovable joints with the parietal, occipital, sphenoid and zygomatic bones. Each temporal bone has several important features.

The *squamous part* is the thin fan-shaped part that articulates with the parietal bone. *The zygomatic process* articulates with the zygomatic bone to form the zygomatic arch (cheekbone).

The *mastoid part* contains the *mastoid process*, a thickened region behind the ear. It contains a large number of very small air sinuses which communicate with the middle ear and are lined with squamous epithelium.

The *petrous portion* forms part of the base of the skull and contains the organs of hearing (the spiral organ) and balance.

The temporal bone articulates with the mandible at the *temporomandibular joint*, the only movable joint of the skull. Immediately behind this articulating surface is the

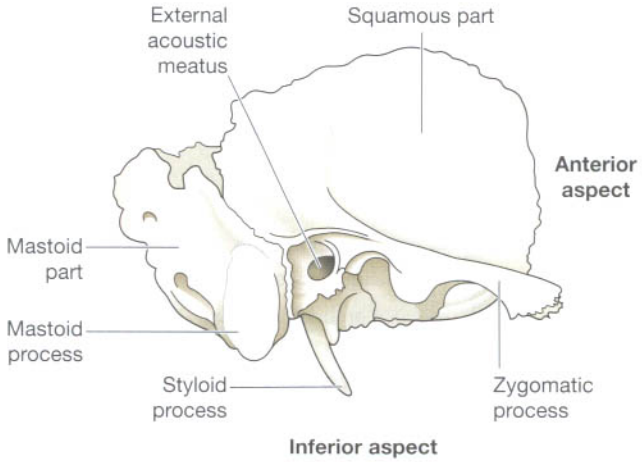


Figure 16.9 The right temporal bone. Lateral view.

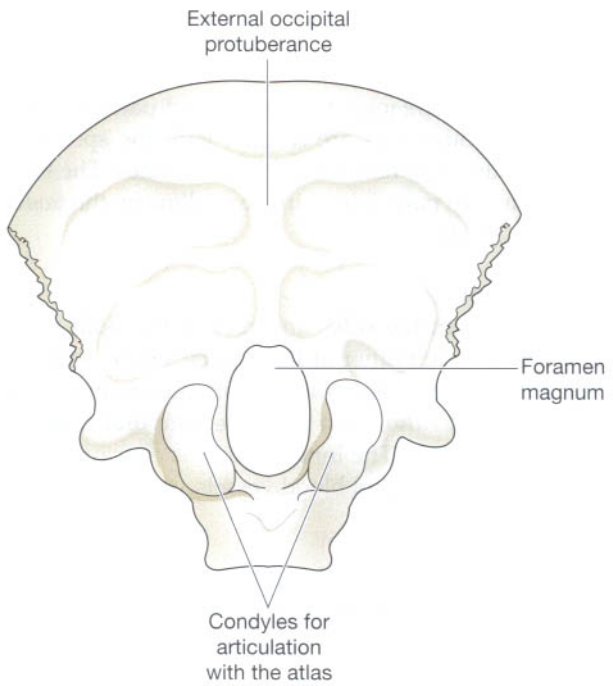


Figure 16.10 The occipital bone viewed from below.

external auditory meatus (auditory canal), which passes inwards towards the petrous portion of the bone.

Occipital bone (Fig. 16.10)

This bone forms the back of the head and part of the base of the skull. It has immovable joints with the parietal, temporal and sphenoid bones. Its inner surface is deeply concave and the concavity is occupied by the occipital lobes of the cerebrum and by the cerebellum. The occiput has two articular condyles that form hinge joints with the first bone of the vertebral column, the *atlas*. Between the condyles there is the *foramen magnum* (meaning 'large hole') through which the spinal cord passes into the cranial cavity.

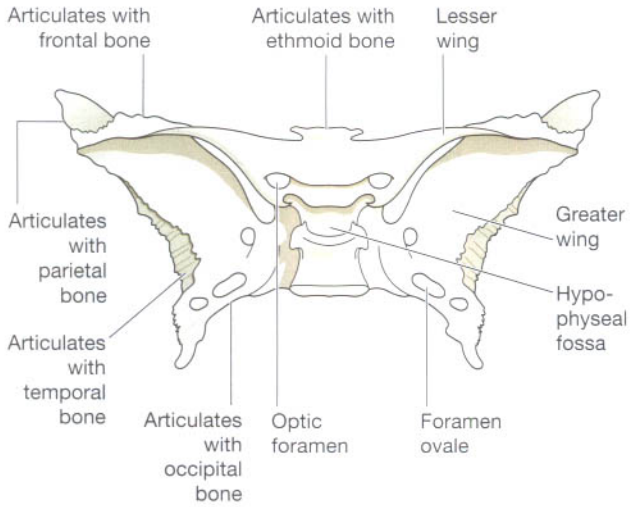


Figure 16.11 The sphenoid bone viewed from above.

Sphenoid bone (Fig. 16.11)

This bone occupies the middle portion of the base of the skull and it articulates with the occipital, temporal, parietal and frontal bones (Fig. 16.8). On the superior surface in the middle of the bone there is a little saddle-shaped depression, the *hypophyseal fossa* (*sella turcica*) in which the *pituitary gland* rests. The body of the bone contains some fairly large air sinuses lined by ciliated mucous membrane with openings into the nasal cavity.

Ethmoid bone (Fig. 16.12)

The ethmoid bone occupies the anterior part of the base of the skull and helps to form the orbital cavity, the nasal septum and the lateral walls of the nasal cavity. On each side are two projections into the nasal cavity, the *upper* and *middle conchae* or *turbinated processes*. It is a very delicate bone containing many air sinuses lined with ciliated epithelium and with openings into the nasal cavity. The horizontal flattened part, the *cribriform plate*, forms the roof of the nasal cavity and has numerous small foramina through which nerve fibres of the *olfactory nerve* (sense of smell) pass upwards from the nasal cavity to the brain. There is also a very fine *perpendicular plate* of bone that forms the upper part of the *nasal septum*.

Face

The skeleton of the face is formed by 13 bones in addition to the frontal bone, already described. Figure 16.13 shows the relationships between the bones:

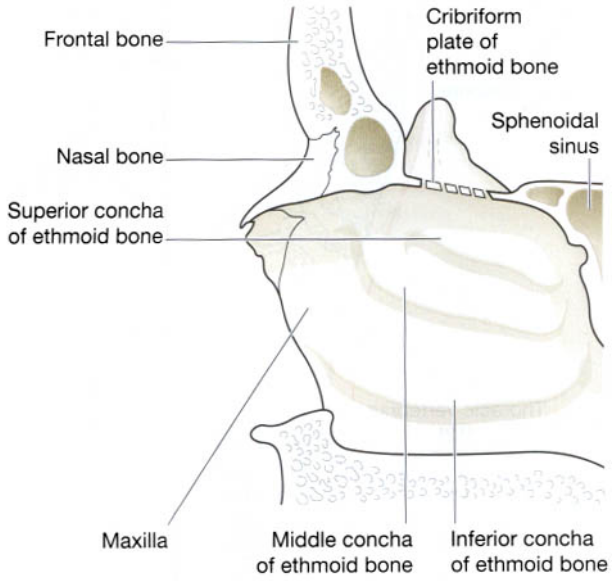


Figure 16.12 The right ethmoid bone and its related structures.

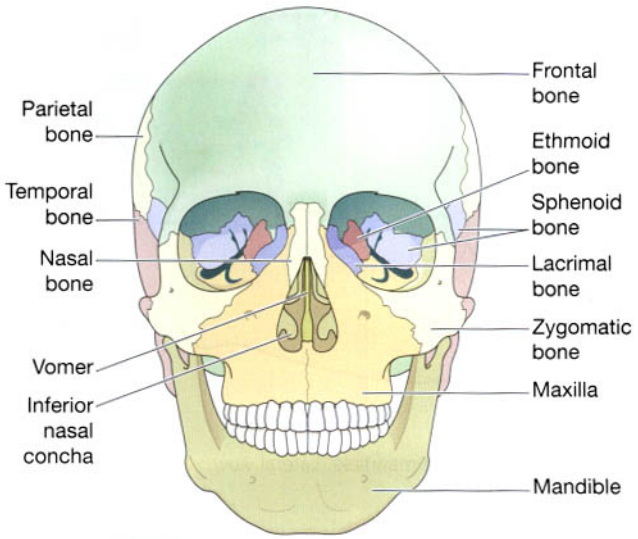


Figure 16.13 The bones of the face. Anterior view.

- 2 zygomatic or cheek bones
- 1 maxilla (originated as 2)
- 2 nasal bones
- 2 lacrimal bones
- 1 vomer
- 2 palatine bones
- 2 inferior conchae
- 1 mandible (originated as 2).

Zygomatic or cheek bones

The zygomatic bones form the prominences of the cheeks and part of the floor and lateral walls of the orbital cavities.

Maxilla or upper jaw bone

This originates as two bones but fusion takes place before birth. The maxilla forms the upper jaw, the anterior part of the roof of the mouth, the lateral walls of the nasal cavity and part of the floor of the orbital cavities. The *alveolar ridge*, or *process*, projects downwards and carries the upper teeth. On each side there is a large air sinus, the *maxillary sinus*, lined with ciliated mucous membrane and with openings into the nasal cavity.

Nasal bones

These are two small flat bones which form the greater part of the lateral and superior surfaces of the bridge of the nose.

Lacrimal bones

These two small bones are posterior and lateral to the nasal bones and form part of the medial walls of the orbital cavities. Each is pierced by a foramen for the passage of the *nasolacrimal duct* which carries the tears from the medial canthus of the eye to the nasal cavity.

Vomer

The vomer is a thin flat bone which extends upwards from the middle of the hard palate to form the main part of the nasal septum. Superiorly it articulates with the perpendicular plate of the ethmoid bone.

Palatine bones

These are two L-shaped bones. The horizontal parts unite to form the posterior part of the hard palate and the perpendicular parts project upwards to form part of the lateral walls of the nasal cavity. At their upper extremities they form part of the orbital cavities.

Inferior conchae

Each concha is a scroll-shaped bone which forms part of the lateral wall of the nasal cavity and projects into it below the middle concha. The superior and middle conchae are parts of the ethmoid bone.

Mandible (Fig. 16.14)

This is the only movable bone of the skull. It originates as two parts which unite at the midline. Each half consists of two main parts: a *curved body* with the *alveolar ridge* containing the lower teeth and a *ramus* which projects upwards almost at right angles to the posterior end of the body.

At the upper end the ramus divides into the *condylar process* which articulates with the temporal bone to form the *temporomandibular* joint and the *coronoid process* that gives attachment to muscles and ligaments. The point where the ramus joins the body is the *angle* of the jaw.

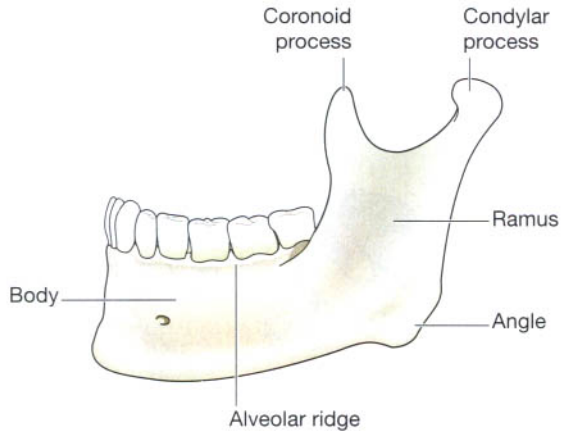


Figure 16.14 The left mandible. Lateral view.

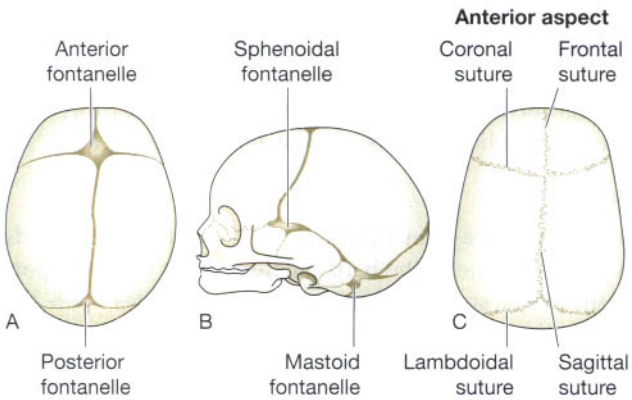


Figure 16.15 The skull showing the fontanelles and sutures. A. Fontanelles viewed from above. B. Fontanelles viewed from the side. C. Main sutures viewed from above when ossification is complete.

Hyoid bone

This is an isolated horse-shoe-shaped bone lying in the soft tissues of the neck just above the *larynx* and below the *mandible* (see Fig. 10.4, p. 242). It does not articulate with any other bone but is attached to the styloid process of the temporal bone by ligaments. It gives attachment to the base of the tongue.

Sinuses

Sinuses containing air are present in the sphenoid, ethmoid, maxillary and frontal bones. They all communicate with the nasal cavity and are lined with ciliated mucous membrane. Their functions are:

- to give resonance to the voice
- to lighten the bones of the face and cranium, making it easier for the head to balance on top of the vertebral column.

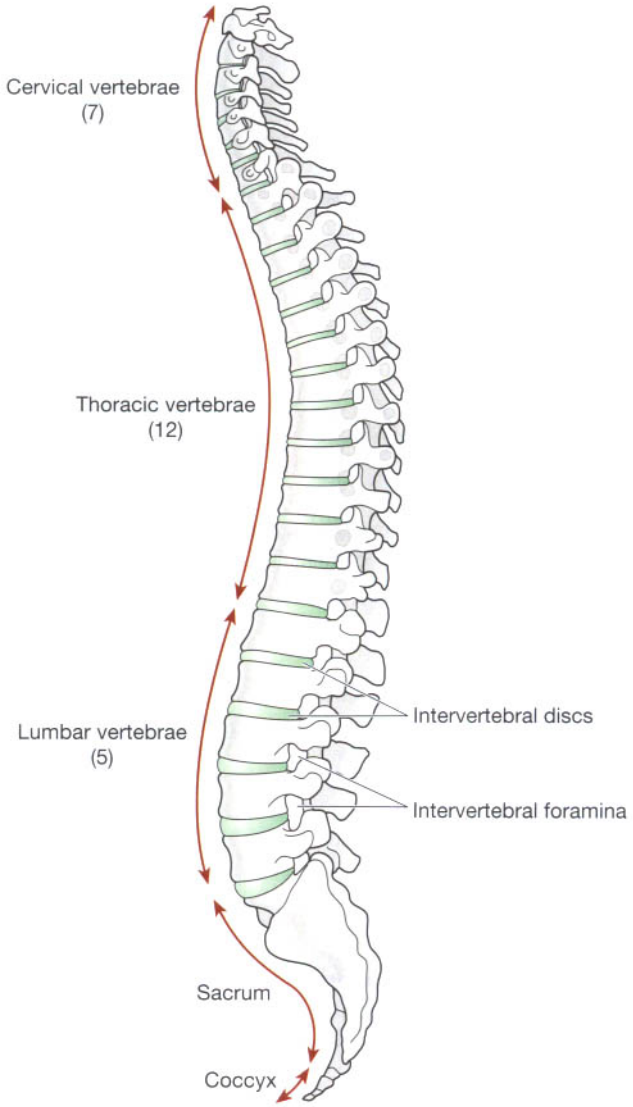


Figure 16.16 The vertebral column. Lateral view.

Fontanelles of the skull (Fig. 16.15)

At birth, ossification of the cranial sutures is incomplete. Where three or more bones meet there are distinct membranous areas, or *fontanelles*. The two largest are the *anterior fontanelle*, not fully ossified until the child is 12 to 18 months old, and the *posterior fontanelle*, usually ossified 2 to 3 months after birth. The skull bones do not fuse before birth to allow for moulding of the baby's head during its passage through the birth canal.

Vertebral column (Fig. 16.16)

The vertebral column consists of 24 separate movable, irregular bones, the *sacrum* (five fused bones) and the

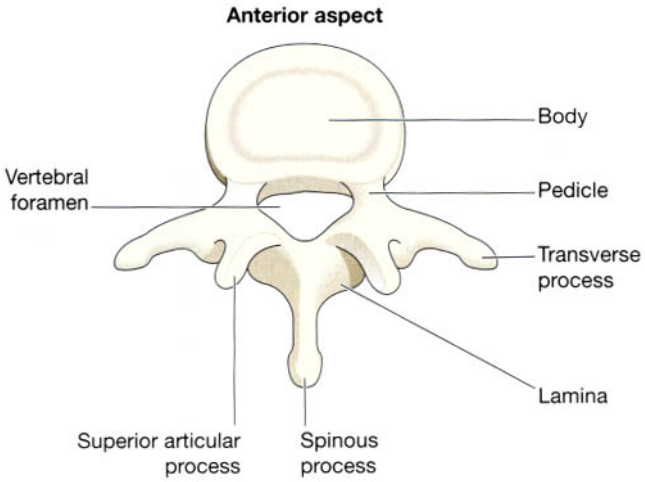


Figure 16.17 A lumbar vertebra showing the features of a typical vertebra – viewed from above.

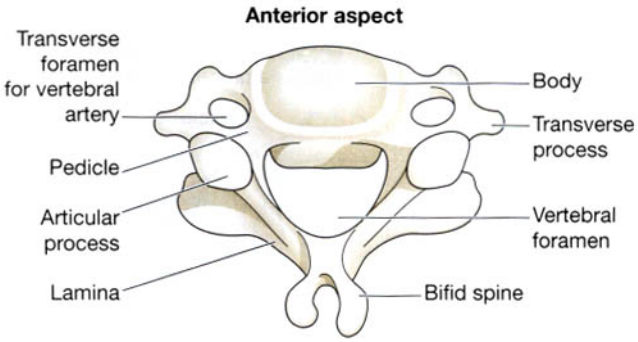


Figure 16.18 A cervical vertebra showing typical features viewed from above.

coccyx (four fused bones). The 24 separate bones are in three groups: 7 cervical, 12 thoracic and 5 lumbar.

The movable vertebrae have many characteristics in common but some groups have distinguishing features.

Characteristics of a typical vertebra (Fig. 16.17)

The body. The body of each vertebra is situated anteriorly. The size varies with the site. They are smallest in the cervical region and become larger towards the lumbar region.

The vertebral (neural) arch encloses a large *vertebral foramen*. The ring of bone consists of two *pedicles* that project backwards from the body and two *laminae*. Where the pedicles and laminae unite, *transverse processes* project laterally and where the two laminae meet in the midline posteriorly they form a *spinous process*. The neural arch has four articular surfaces: two articulate with the vertebra above and two with the one below. The vertebral foramina form the vertebral (neural) canal that contains the spinal cord.

Special features of vertebrae in different parts of the vertebral column

Cervical vertebrae (Fig. 16.18)

The transverse processes have a foramen through which a vertebral artery passes upwards to the brain. The first two cervical vertebrae are atypical.

The *atlas* (Fig. 16.19A) is the 1st cervical vertebra and it consists simply of a ring of bone with two short transverse processes. The anterior part of the large vertebral foramen is occupied by the *odontoid process* of the axis, which is held in position by a *transverse ligament* (Fig. 16.19C).

Thus the odontoid process forms the body of the atlas. The posterior part is the true vertebral foramen and is occupied by the spinal cord. On its superior surface the bone has two articular facets which form joints with the condyles of the occipital bone of the skull. The nodding movement of the head takes place at these joints.

The *axis* (Fig. 16.19B) is the 2nd cervical vertebra. The body is small and has the upward projecting *odontoid process* or *dens* that articulates with the first cervical vertebra, the atlas. The movement at this joint is turning the head from side to side.

Thoracic vertebrae (Fig. 16.20)

The bodies and transverse processes have facets for articulation with the ribs.

Lumbar vertebrae (Fig. 16.17)

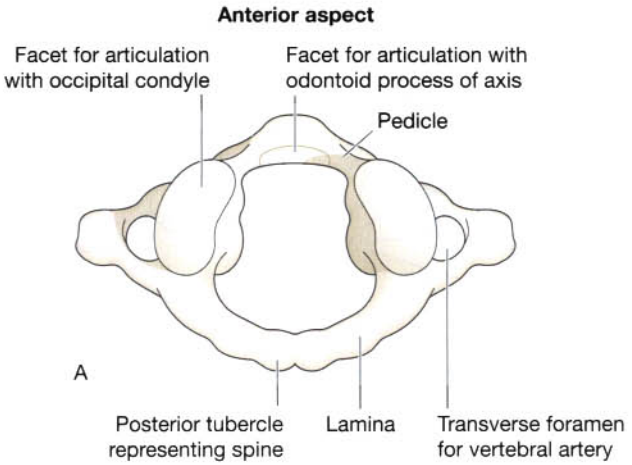
These have no special features.

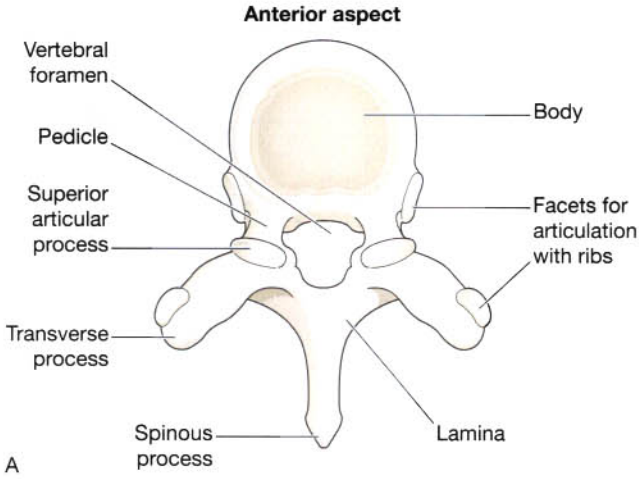
Sacrum (Fig. 16.21)

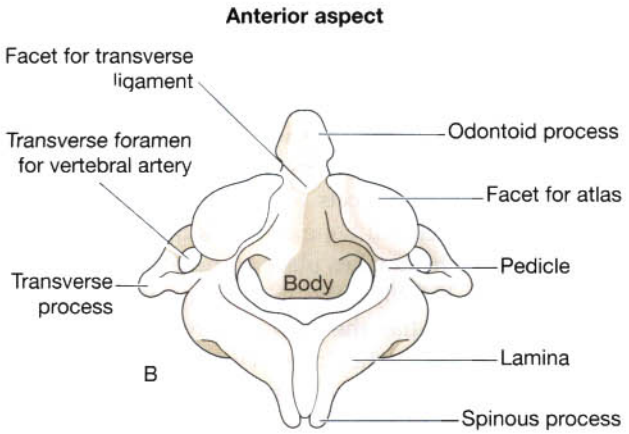
This consists of five rudimentary vertebrae fused to form a triangular or wedge-shaped bone with a concave anterior surface. The upper part, or base, articulates with the 5th lumbar vertebra. On each side it articulates with the ilium to form a *sacroiliac joint*, and at its inferior tip it articulates with the *coccyx*. The anterior edge of the base, the *promontory*, protrudes into the pelvic cavity. The vertebral foramina are present, and on each side of the bone there is a series of foramina for the passage of nerves.

Coccyx (Fig. 16.21)

This consists of the four terminal vertebrae fused to form a very small triangular bone, the broad base of which articulates with the tip of the sacrum.







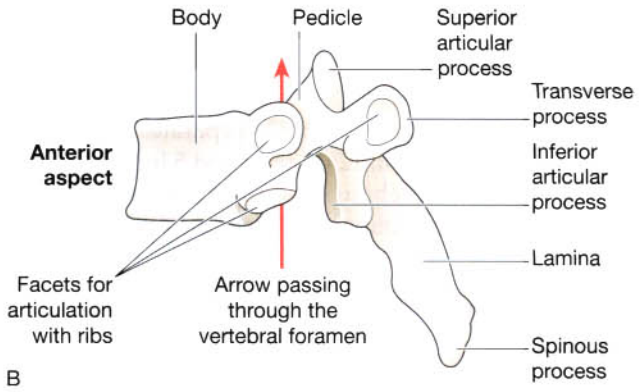


Figure 16.20 A thoracic vertebra: A. Viewed from above. B. Viewed from the side.

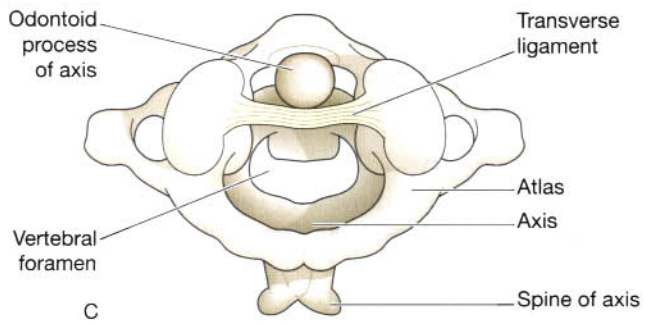


Figure 16.19 The upper cervical vertebrae viewed from above: A. The atlas. B. The axis. C. The atlas and axis in position showing the transverse ligament.

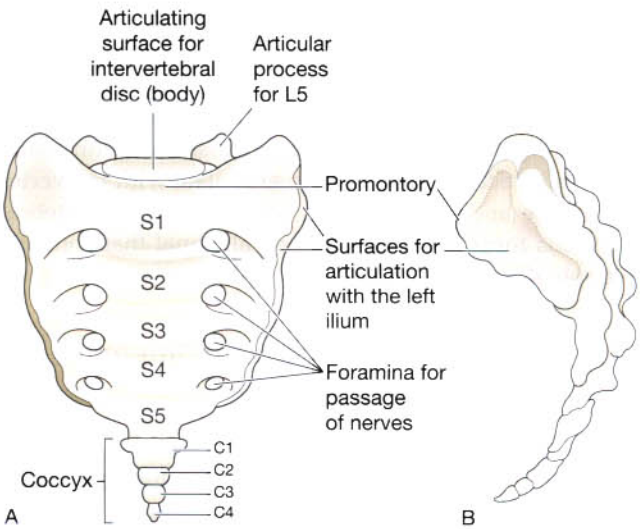


Figure 16.21 The sacrum and coccyx: A. Anterior view. B. Lateral view.

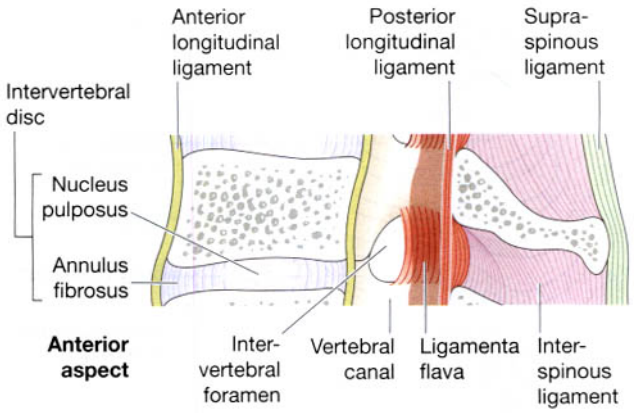


Figure 16.22 Section of the vertebral column showing the ligaments, intervertebral discs and intervertebral foramina.

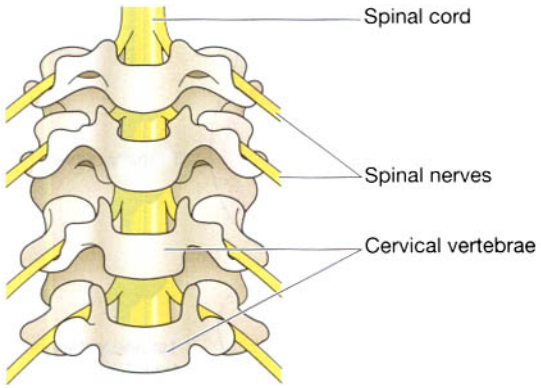


Figure 16.23 Lower cervical vertebrae separated to show the spinal cord and spinal nerves emerging through the intervertebral foramina. Anterior view.

Features of the vertebral column

Intervertebral discs

The bodies of adjacent vertebrae are separated by *intervertebral discs*, consisting of an outer rim of fibrocartilage (*annulus fibrosus*) and a central core of soft gelatinous material (*nucleus pulposus*) (Fig. 16.22). They are thinnest in the cervical region and become progressively thicker towards the lumbar region. The posterior longitudinal ligament in the vertebral canal helps to keep them in place. They have a shock-absorbing function and the cartilaginous joints they form contribute to the flexibility of the vertebral column as a whole.

Intervertebral foramina

When two adjacent vertebrae are viewed from the side, a foramen can be seen. Half of the wall is formed by the vertebra above and half by the one below (Fig. 16.23).

Throughout the length of the column there is an intervertebral foramen on each side between every pair of vertebrae, through which the spinal nerves, blood vessels and lymph vessels pass.

Ligaments of the vertebral column (Fig. 16.22)

These ligaments hold the vertebrae together and help to maintain the intervertebral discs in position.

The *transverse ligament* maintains the odontoid process of the axis in the correct position in relation to the atlas (Fig. 16.19C).

The *anterior longitudinal ligament* extends the whole length of the column and lies in front of the vertebral bodies.

The *posterior longitudinal ligament* lies inside the vertebral canal and extends the whole length of the vertebral column in close contact with the posterior surface of the bodies of the bones.

The *ligamenta flava* connect the laminae of adjacent vertebrae.

The *ligamentum nuchae* and the *supraspinous ligament* connect the spinous processes, extending from the occiput to the sacrum.

Curves of the vertebral column (Fig. 16.24)

When viewed from the side the vertebral column presents four curves, two *primary* and two *secondary*.

The fetus in the uterus lies curled up so that the head and the knees are more or less touching. This position shows the *primary curvature*. The secondary *cervical curve* develops when the child can hold up his head (after about 3 months) and the secondary *lumbar curve* develops when he stands upright (after 12 to 15 months). The thoracic and sacral primary curves are retained.

Movements of the vertebral column

The movements between the individual bones of the vertebral column are very limited. However, the movements of the column as a whole are quite extensive and include *flexion* (bending forward), *extension* (bending backward), *lateral flexion* (bending to the side) and *rotation*. There is more movement in the cervical and lumbar regions than elsewhere.

Functions of the vertebral column

These include the following.

- Collectively the vertebral foramina form the vertebral canal which provides a strong bony protection for the delicate spinal cord lying within it.
- The pedicles of adjacent vertebrae form intervertebral foramina, one on each side, providing access to the spinal cord for spinal nerves, blood vessels and lymph vessels.

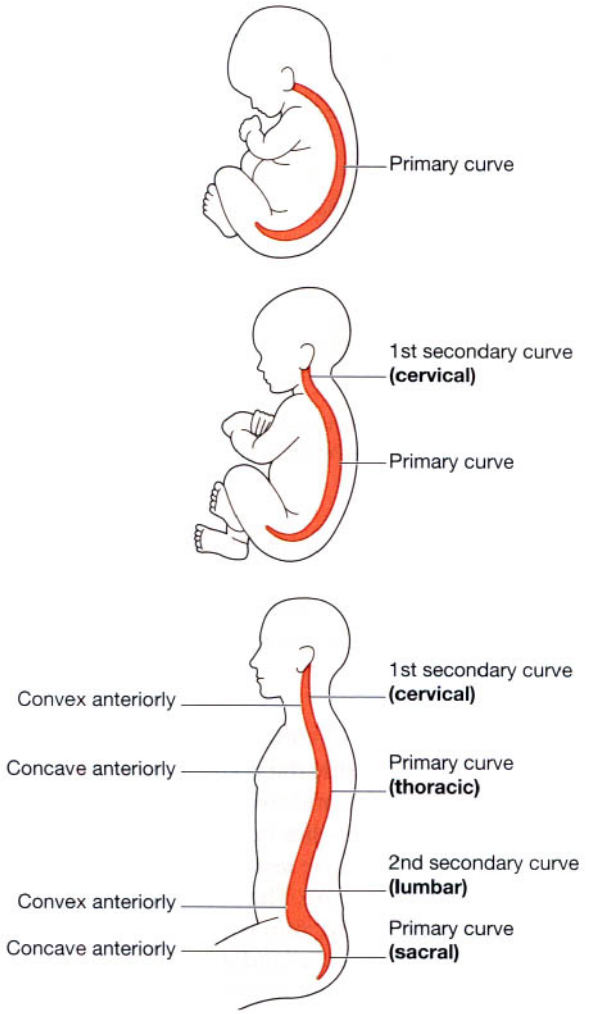


Figure 16.24 The order of development of the curves of the spine.

- The numerous individual bones enable a certain amount of movement.
- It supports the skull.
- The intervertebral discs act as shock absorbers, protecting the brain.
- It forms the axis of the trunk, giving attachment to the ribs, shoulder girdle and upper limbs, and the pelvic girdle and lower limbs.

Thoracic cage (Fig. 16.25)

The bones of the thorax or thoracic cage are:

- 1 sternum
- 12 pairs of ribs
- 12 thoracic vertebrae.

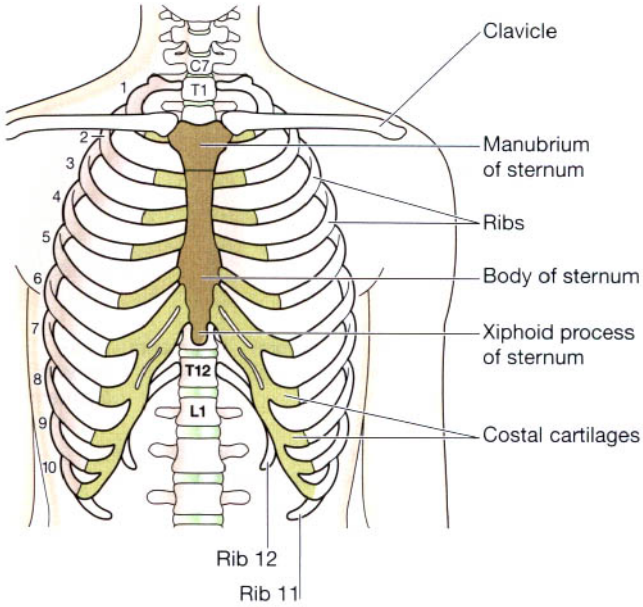


Figure 16.25 The thoracic cage. Anterior view.

Sternum or breast bone (Fig. 16.26)

This *flat bone* can be felt just under the skin in the middle of the front of the chest.

The *manubrium* is the uppermost section and articulates with the clavicles at the *sternoclavicular joints* and with the first two pairs of ribs.

The *body* or *middle portion* gives attachment to the ribs.

The *xiphoid process* is the tip of the bone. It gives attachment to the diaphragm, muscles of the anterior abdominal wall and the *linea alba*.

Ribs

There are 12 pairs of ribs which form the bony lateral walls of the thoracic cage and articulate posteriorly with the thoracic vertebrae. The first 10 pairs are attached anteriorly to the sternum by *costal cartilages*, some directly and some indirectly (Fig. 16.25).

The last two pairs (*floating ribs*) have no anterior attachment.

Characteristics of a rib (Fig. 16.27). The head articulates posteriorly with the bodies of two adjacent thoracic vertebrae and on the tubercle there is a facet that articulates with the transverse process of one. The sternal end is attached to the sternum by a costal cartilage, i.e. a band of hyaline cartilage. The superior border is rounded and smooth while the inferior border has a marked groove occupied by the intercostal blood vessels and nerves.

The first rib does not move during respiration. The spaces between the ribs are occupied by the intercostal muscles. During inspiration, when these muscles contract,

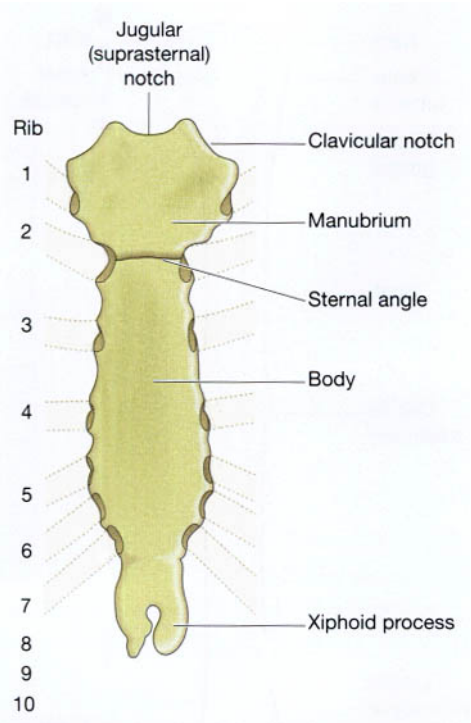


Figure 16.26 The sternum and its attachments.

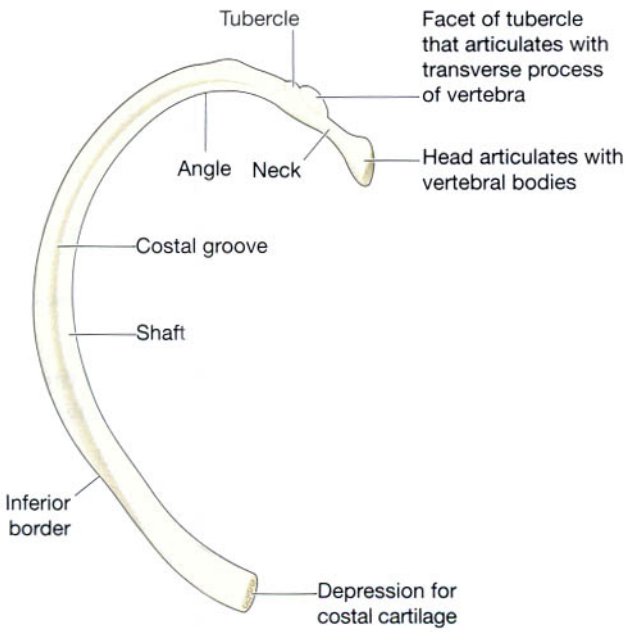


Figure 16.27 A typical rib viewed from below.

the ribs and sternum are lifted upwards and outwards, increasing the capacity of the thoracic cavity (see Fig. 10.21, p. 254).

Thoracic vertebrae

The 12 thoracic vertebrae are described on page 397.

APPENDICULAR SKELETON

Learning outcomes

After studying this section you should be able to:

- identify the bones that form the appendicular skeleton
- state the characteristics of the bones forming the appendicular skeleton
- outline the differences in structure between the male and female pelves.

The appendicular skeleton consists of the shoulder girdle with the upper limbs and the pelvic girdle with the lower limbs (Fig. 16.6).

Shoulder girdle and upper limb

Each shoulder girdle consists of:

- 1 clavicle
- 1 scapula.

Each upper limb consists of the following bones:

- 1 humerus
- 1 radius
- 1 ulna
- 8 carpal bones
- 5 metacarpal bones
- 14 phalanges.

Clavicle or collar bone (Fig. 16.28)

The clavicle is a long bone which has a double curve. It articulates with the manubrium of the sternum at the *sternoclavicular joint* and forms the *acromioclavicular joint* with the *acromion process* of the scapula. The clavicle provides the only bony link between the upper limb and the axial skeleton.

Scapula or shoulder blade (Fig. 16.29)

The scapula is a flat triangular-shaped bone, lying on the posterior chest wall superficial to the ribs and separated from them by muscles.

At the lateral angle there is a shallow articular surface, the *glenoid cavity* which, with the *head of the humerus*, forms the *shoulder joint*.

On the posterior surface there is a *spinous process* that projects beyond the lateral angle of the bone that overhangs the shoulder joint, called the *acromion process*. It articulates with the clavicle at the *acromioclavicular joint*. The *coracoid process*, a projection from the upper border of the bone, gives attachment to muscles that move the shoulder joint.

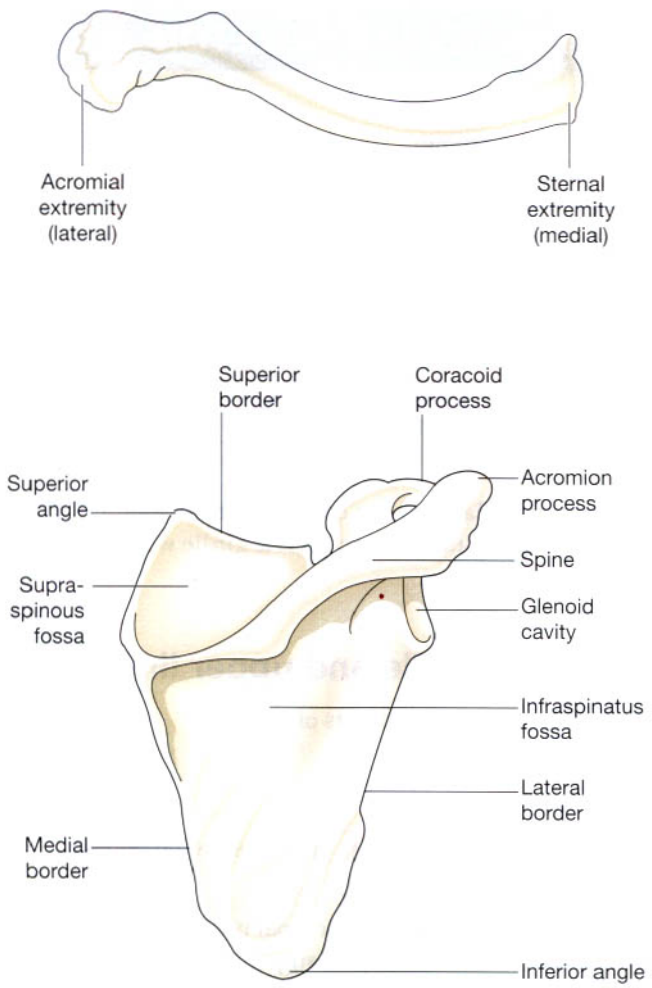


Figure 16.29 The right scapula. Posterior view.

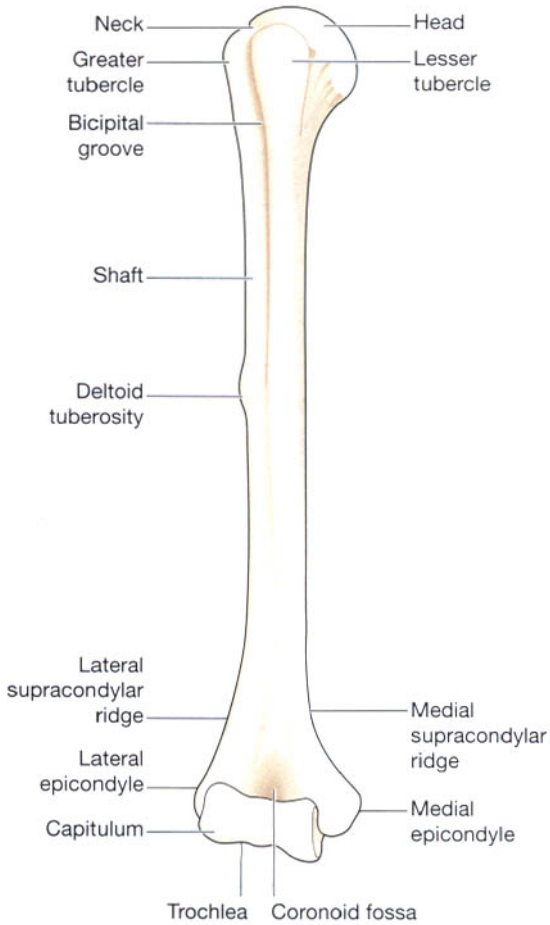


Figure 16.30 The right humerus. Anterior view.

Humerus (Fig. 16.30)

This is the bone of the upper arm. The head articulates with the glenoid cavity of the scapula, forming the shoulder joint. Distal to the head there are two roughened projections of bone, the *greater* and *lesser tubercles,* and between them there is a deep groove, the *bicipital groove* or *intertubercular sulcus*, occupied by one of the tendons of the biceps muscle.

The distal end of the bone presents two surfaces that articulate with the radius and ulna to form the elbow joint.

Ulna and radius (Fig. 16.31)

These are the two bones of the forearm. The ulna is longer than and medial to the radius and when the arm is in the anatomical position, i.e. with the palm of the hand facing forward, the two bones are parallel. They articulate with the humerus at the *elbow joint*, the carpal bones at the *wrist joint* and with each other at the *proximal* and *distal radioulnar* joints.

Carpal or wrist bones (Fig. 16.32)

There are eight carpal bones arranged in two rows of four. From outside inwards they are:

- *proximal row*: scaphoid, lunate, triquetral, pisiform
- *distal row*: trapezium, trapezoid, capitate, hamate.

These bones are closely fitted together and held in position by ligaments which allow a certain amount of movement between them. The bones of the proximal row are associated with the wrist joint and those of the distal row form joints with the metacarpal bones. Tendons of muscles lying in the forearm cross the wrist and are held close to the bones by strong fibrous bands, called retinacula (see Fig. 17.8, p. 420).

Metacarpal bones or the bones of the hand

These five bones form the palm of the hand. They are numbered from the thumb side inwards. The proximal ends articulate with the carpal bones and the distal ends with the phalanges.

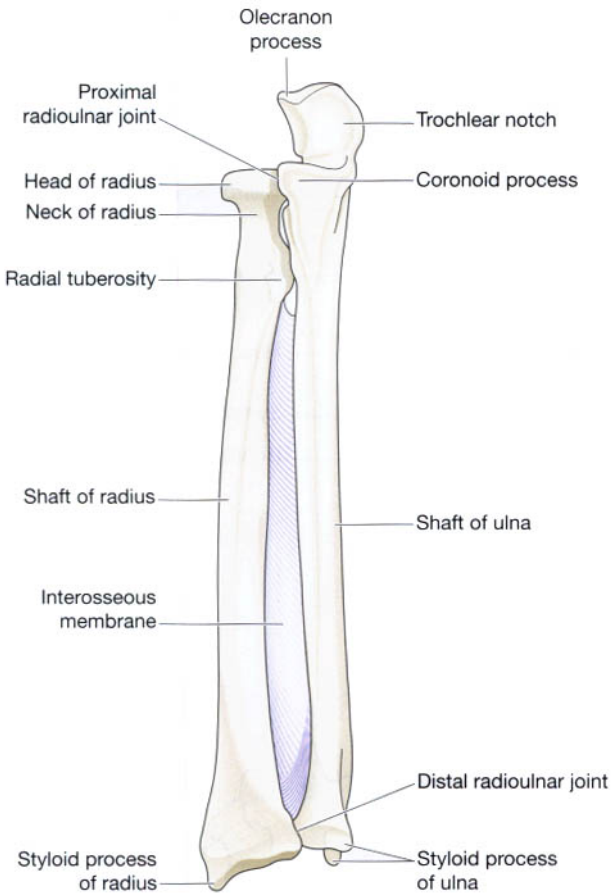


Figure 16.31 The right radius and ulna with the interosseous membrane. Anterior view.

Phalanges or finger bones

There are 14 phalanges, three in each finger and two in the thumb. They articulate with the metacarpal bones and with each other.

Pelvic girdle and lower limb

The bones of the pelvic girdle are:

- 2 innominate bones
- 1 sacrum.

The bones of the lower limb are:

- 1 femur
- 1 tibia
- 1 fibula
- 1 patella
- 7 tarsal bones
- 5 metatarsal bones
- 14 phalanges.

Innominate or hip bones (Fig. 16.33)

Each hip bone consists of three fused bones, the *ilium*, *ischium* and *pubis*. On its outer surface there is a deep depression, the *acetabulum*, which forms the hip joint with the almost-spherical head of femur.

The *ilium* is the upper flattened part of the bone and it presents the *iliac crest*, the anterior point of which is called the *anterior superior iliac spine*.

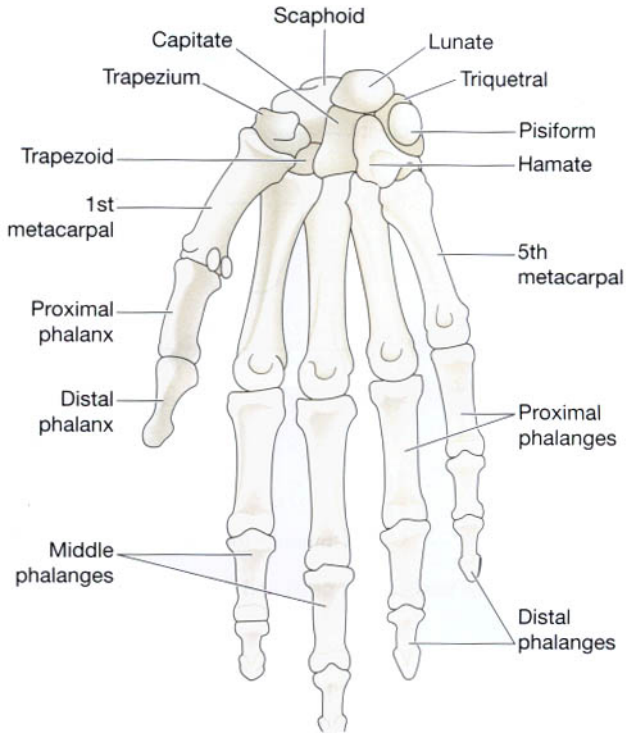


Figure 16.32 The bones of the wrist, hand and fingers. Anterior view.

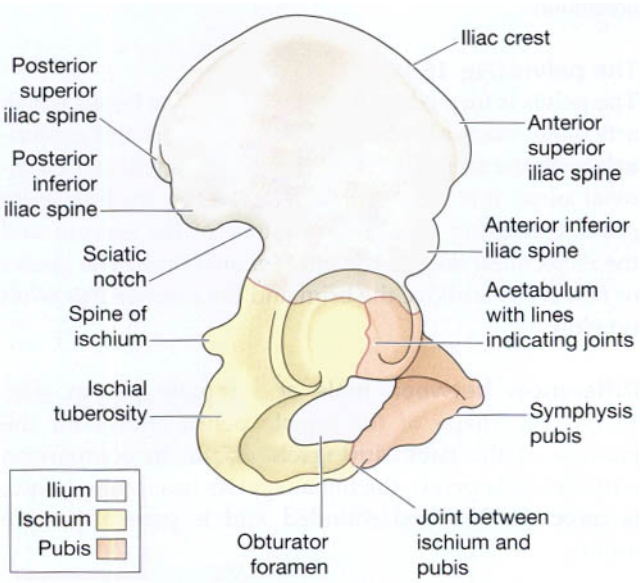


Figure 16.33 The right innominate bone. Lateral view.

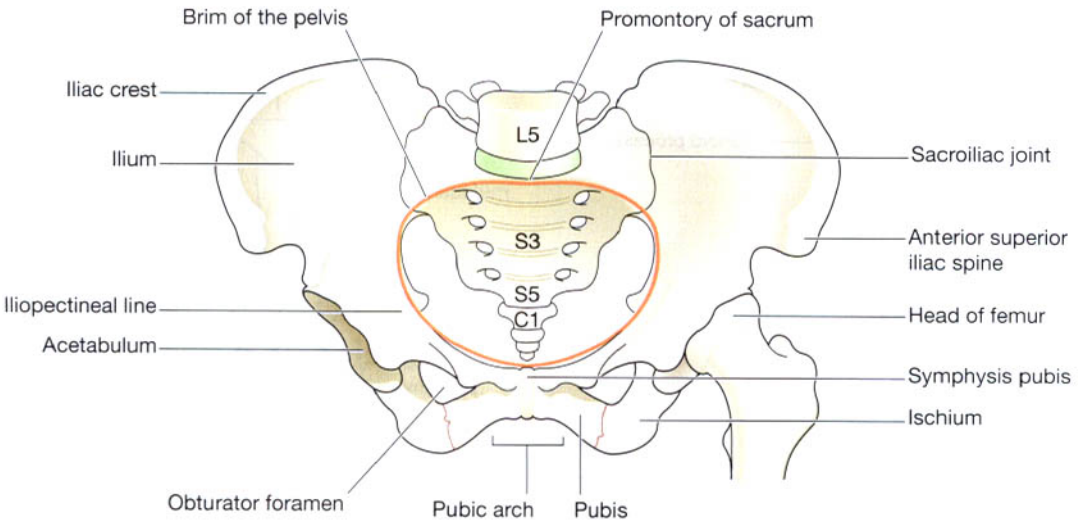


Figure 16.34 The bones of the pelvis and the upper part of the left femur.

The *pubis* is the anterior part of the bone and it articulates with the pubis of the other hip bone at a cartilaginous joint, the *symphysis pubis*.

The *ischium* is the inferior and posterior part.

The union of the three parts takes place in the *acetabulum*.

The pelvis (Fig. 16.34)

The pelvis is formed by the two innominate bones which articulate anteriorly at the symphysis pubis and posteriorly with the sacrum at the *sacroiliac joints* which are synovial joints. It is divided into two parts by the *brim of the pelvis*, consisting of the promontory of the sacrum and the *iliopectineal lines* of the innominate bones. The *greater* or *false pelvis* is above the brim and the *lesser* or *true pelvis* is below.

Differences between male and female pelves (Fig. 16.35)

The shape of the female pelvis allows for the passage of the baby during childbirth. In comparison with the male pelvis, the female pelvis has lighter bones, is more shallow and rounded and is generally more roomy.

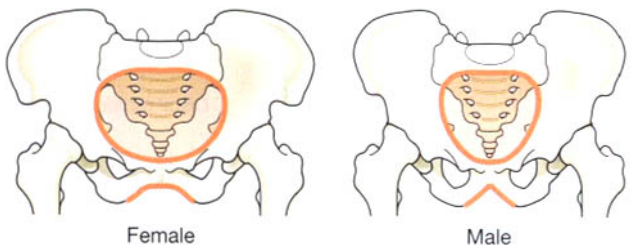


Figure 16.35 The difference in shape of the male and female pelves.

Femur or thigh bone (Fig. 16.36)

The femur is the longest and strongest bone of the body. The head is almost spherical and fits into the *acetabulum* of the hip bone to form the *hip joint*. In the centre of the head there is a small depression for the attachment of the *ligament of the head of the femur*. This extends from the acetabulum to the femur and contains a blood vessel that supplies blood to an area of the head of the bone. The neck extends outwards and slightly downwards from the head to the shaft and most of it is within the capsule of the hip joint.

The posterior surface of the lower third forms a flat triangular area called the *popliteal surface*. The distal extremity has two articular *condyles* which, with the tibia and patella, form the knee joint.

Tibia or shin bone (Fig. 16.37)

The tibia is the medial of the two bones of the lower leg. The proximal extremity is broad and flat and presents two *condyles* for articulation with the femur at the *knee joint*. The head of the fibula articulates with the inferior aspect of the lateral condyle, forming the *proximal tibiofibular* joint.

The distal extremity of the tibia forms the *ankle joint*. with the *talus* and the fibula. The *medial malleolus* is a downward projection of bone medial to the ankle joint.

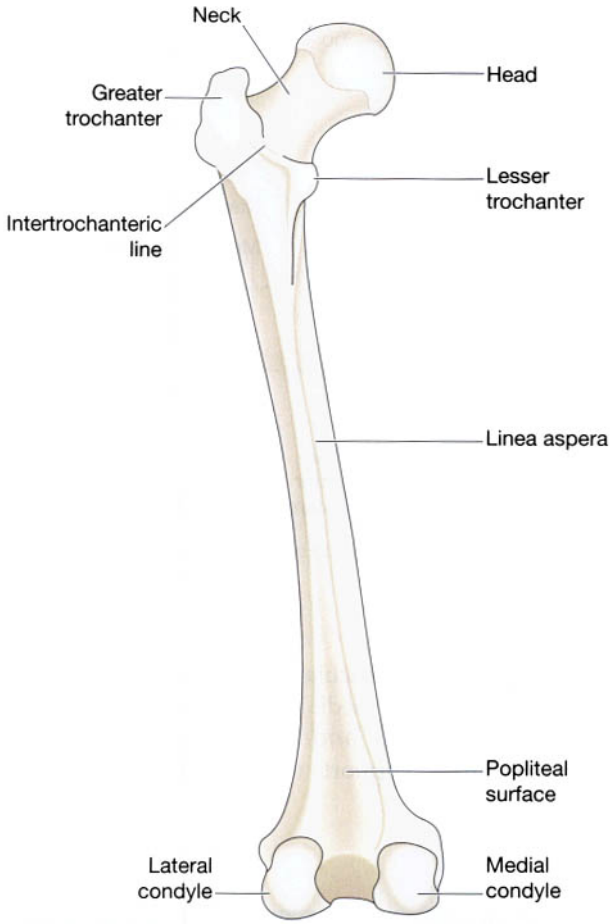


Figure 16.36 The left femur. Posterior view.

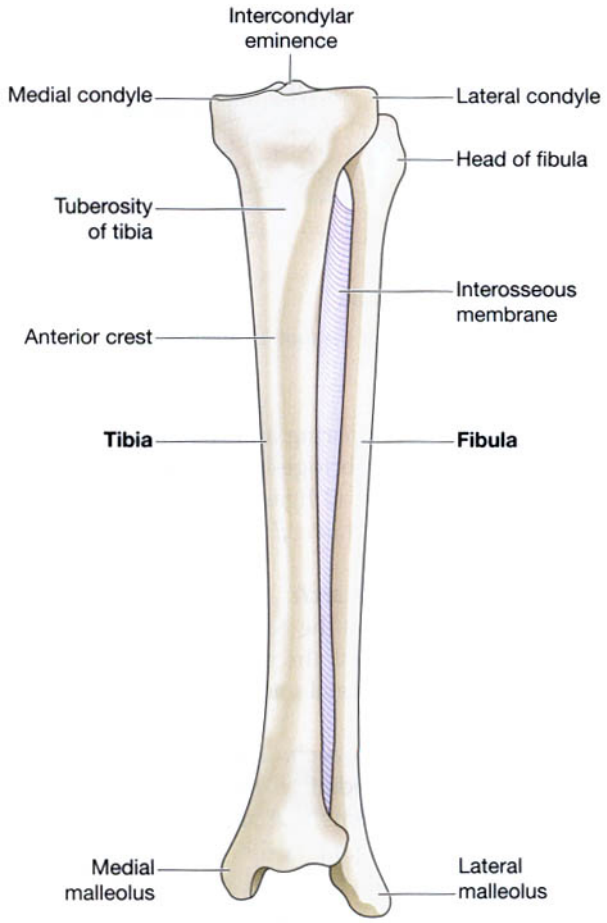


Figure 16.37 The left tibia and fibula with the interosseous membrane. Anterior view.

Fibula (Fig. 16.37)

The fibula is the long slender lateral bone in the leg. The head or upper extremity articulates with the lateral condyle of the tibia forming the proximal tibiofibular joint and the lower extremity articulates with the tibia then projects beyond it to form the *lateral malleolus*.

Patella or knee cap

This is a roughly triangular-shaped *sesamoid* bone associated with the knee joint. Its posterior surface articulates with the patellar surface of the femur in the knee joint and its anterior surface is in the *patellar tendon*, i.e. the tendon of the quadriceps femoris muscle.

Tarsal or ankle bones (Fig. 16.38)

There are seven tarsal bones which form the posterior part of the foot. They are:

- 1 talus
- 1 calcaneus
- 1 navicular
- 3 cuneiform
- 1 cuboid.

The *talus* articulates with the tibia and fibula at the ankle joint. The *calcaneus* forms the heel of the foot. The other bones articulate with each other and with the metatarsal bones.

Metatarsal bones of the foot (Fig. 16.38)

These are five bones, numbered from within outwards, which form the greater part of the dorsum of the foot. At their proximal ends they articulate with the tarsal bones and at their distal ends, with the phalanges. The enlarged distal head of the 1st metatarsal bone forms the 'ball' of the foot.

Phalanges of the toes (Fig. 16.38)

There are 14 phalanges arranged in a similar manner to those in the fingers, i.e. two in the great toe (the *halux*) and three in each of the other toes.

Arches of the foot. The arrangement of the bones of the foot is such that it is not a rigid structure. This point is

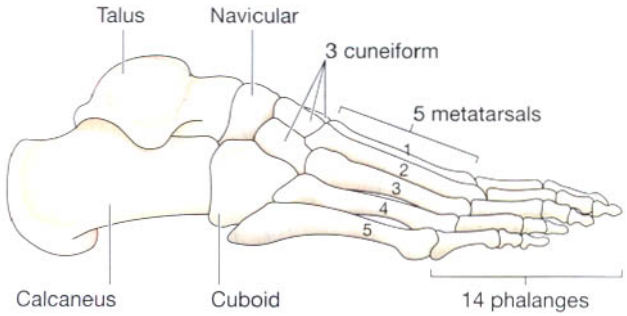


Figure 16.38 The bones of the foot. Lateral view.

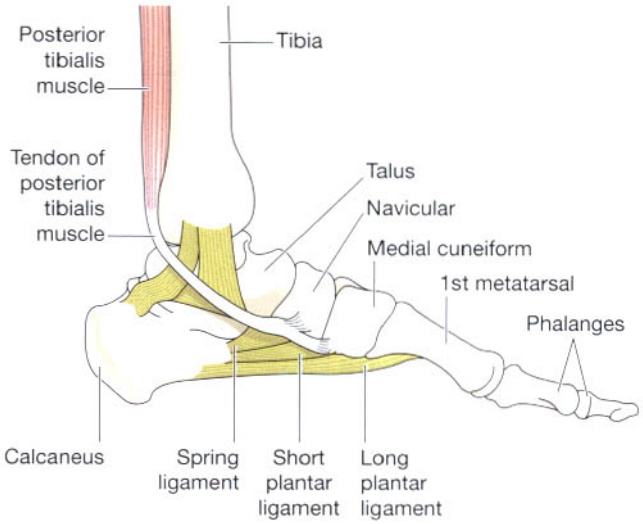


Figure 16.39 The tendons and ligaments supporting the arches of the left foot. Medial view.

well illustrated by comparing a normal foot with a 'flat' foot. The bones have a bridge-like arrangement and are supported by muscles and ligaments so that four arches are formed, a *medial* and *lateral longitudinal arch* and two *transverse arches*.

Medial longitudinal arch. This is the highest of the arches and is formed by the calcaneus, talus, navicular, three cuneiform and first three metatarsal bones. Only the calcaneus and the distal end of the metatarsal bones should touch the ground.

Lateral longitudinal arch. The lateral arch is much less marked than its medial counterpart. The bony components are the calcaneus, cuboid and the two lateral metatarsal bones. Again only the calcaneus and metatarsal bones should touch the ground.

Transverse arches. These run across the foot and can be more easily seen by examining the skeleton than the live model. They are most marked at the level of the three cuneiform and cuboid bones.

Muscles and ligaments which support the arches of the foot (Fig. 16.39). As there are movable joints between all the bones of the foot, very strong muscles and ligaments are necessary to maintain the strength, resilience and stability of the foot during walking, running and jumping.

Posterior tibialis muscle. This is the most important muscular support of the medial longitudinal arch. It lies on the posterior aspect of the lower leg, originates from the middle third of the tibia and fibula and its tendon passes behind the medial malleolus to be inserted into the navicular, cuneiform, cuboid and metatarsal bones. It acts as a sling or 'suspension apparatus' for the arch.

Short muscles of the foot. This group of muscles is mainly concerned with the maintenance of the lateral longitudinal and transverse arches. They make up the fleshy part of the sole of the foot.

Plantar calcaneonavicular ligament or 'spring' ligament. This is a very strong thick ligament stretching from the calcaneus to the navicular bone. It plays an important part in supporting the medial longitudinal arch.

Plantar ligaments and interosseous membranes. These structures support the lateral and transverse arches.

HEALING OF BONES

Learning outcomes

After studying this section you should be able to:

- state three types of fractures
- outline the process of bone healing
- list the factors that delay healing of fractures
- describe two complications of fractures.

Bone fractures are classified as:

- *simple*: the bone ends do not protrude through the skin
- *compound*: the bone ends protrude through the skin
- *pathological*: fracture of a bone weakened by disease.

Following a fracture, the broken ends of bone are joined by the deposition of new bone. This occurs in several stages (Fig. 16.40).

- A haematoma forms between the ends of bone and in surrounding soft tissues.
- There follows development of acute inflammation and accumulation of macrophages which phagocytose the haematoma, inflammatory exudate and small fragments of bone without blood supply (this takes about 5 days). Fibroblasts migrate to the site; granulation tissue and new capillaries develop.
- New bone forms as large numbers of osteoblasts secrete woven (non-lamellar) bone (p. 390), which is then quickly organised into lamellar bone and calcified, forming a *callus* (after about a week).
- Osteoblasts and osteoclasts remain active and the callus matures, reuniting the bone ends (after about 3 weeks).
- Reshaping of the bone continues and gradually the medullary canal is reopened through the callus (in weeks or months).
- In time the bone heals completely regaining its original features. Osteoblasts and osteoclasts are no longer present.

Factors that delay healing of fractures

Tissue fragments between the ends of bone. Splinters of dead bone (*sequestrae*) and soft tissue fragments not removed by phagocytosis delay healing.

Deficient blood supply. This delays growth of granulation tissue and new blood vessels. Hypoxia also reduces the number of osteoblasts and increases the number of chondrocytes that develop from their common parent cells. This may lead to cartilagenous union of the fracture, which results in a weaker repair. The most vulnerable sites, because of their normally poor blood supply, are the neck of femur, the scaphoid and the shaft of tibia.

Poor alignment of bone ends. This may result in the formation of a large and irregular callus which heals slowly and often results in permanent disability.

Continued mobility of bone ends. Continuous movement results in fibrosis of the granulation tissue followed by fibrous union of the fracture.

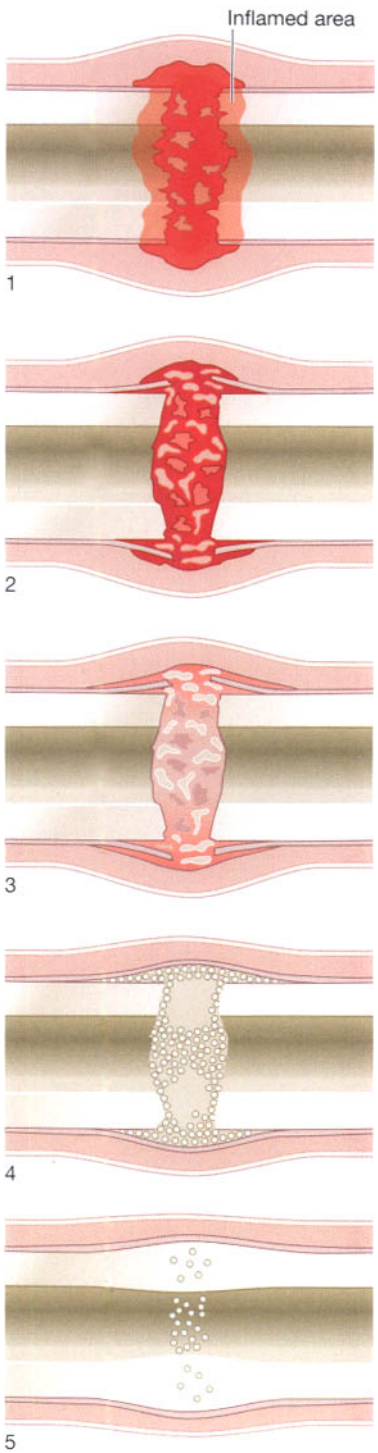


Figure 16.40 Stages in bone healing.

Miscellaneous. These include:

- infection (see below)
- systemic illness
- malnutrition
- drugs, e.g. corticosteroids
- ageing.

Complications of fractures

Infection (osteomyelitis). Microbes gain access through broken skin, although they may occasionally be blood-borne (p. 410). Healing will not occur until the infection resolves.

Fat embolism. Emboli consisting of fat from the marrow in the medullary canal may enter the circulation through torn veins. They are most likely to lodge in the lungs.

DISEASES OF BONES

Learning outcomes

After studying this section you should be able to:

- explain the pathological features of osteoporosis, Paget's disease, rickets and osteomalacia
- outline the causes and effects of osteomyelitis
- describe abnormalities of bone development
- explain the effects of bone tumours.

Osteoporosis

In this condition bone mass (the amount of bone tissue) is reduced because its deposition does not keep pace with resorption. Peak bone mass occurs around 35 years and then gradually declines in both sexes. Lowered oestrogen levels after the menopause are associated with a period of accelerated bone loss in women. Thereafter bone density in women is less than in men for any given age. Bone is progressively weakened with cancellous bone affected first by thinning and loss of trabeculae. In the post-menopausal period an imbalance of hormones probably causes bone weakening, i.e. between anabolic steroids (oestrogen and androgens) and antianabolic steroids (glucocorticoids). A range of environmental factors and diseases are associated with decreased bone mass and are implicated in development of osteoporosis (Box 16.1). Some can be influenced by changes in lifestyle. Exercise and calcium intake during childhood and adolescence are thought to be important in determining eventual bone mass of an individual. As bone mass decreases, susceptibility to fractures increases. Immobility causes reversible osteoporosis, the extent of which corresponds to the area of impaired movement, e.g.:

- localised – following immobilisation of a fractured limb or around a joint affected by rheumatoid arthritis
- generalised – in e.g. prolonged unconsciousness.

Common features of osteoporosis are:

- skeletal deformity – gradual loss of height with age, which is caused by compression of vertebrae
- bone pain
- fractures – especially of the hip (neck of femur), wrist (Colles fracture) and vertebrae.

Box 16.1 Causes of decreased bone mass

Risk factors	Drugs
Female gender	Corticosteroids
Increasing age	
White ethnic origin	**Diseases**
Family history	Cushing's syndrome
Lack of exercise/ immobility	Hyperparathyroidism
Diet (low calcium)	Type I diabetes mellitus
Smoking	Rheumatoid arthritis
Excess alcohol intake	Chronic renal failure
Early menopause/ oophorectomy	Chronic liver disease
Thin build (small bones)	Anorexia nervosa
	Neoplasia

Paget's disease

This disease can affect one bone, part of a bone or many bones. Osteocytes reabsorb excess bone, softening the tissue, and then overactive osteoblasts deposit abnormal new bone that is thickened or enlarged and structurally weak. This predisposes to deformities and fractures, commonly of the pelvis, femur, tibia and skull. Most cases occur after 40 years and the incidence increases with age. The cause is unknown and it often goes undetected until complications arise. These include:

- bone pain
- bony deformities, e.g. bowing of the tibia and femur
- fractures that are pathological (spontaneous) or follow minor trauma
- osteoarthritis due to bony deformities, especially in the hip joint
- osteosarcoma, which often occurs in the elderly and is associated with a poor prognosis (p. 411)
- compression of nerves in the diminished cranial foramina due to thickening of the bones, e.g. compression of the vestibulocochlear nerve causing deafness.

Rickets and osteomalacia

Rickets occurs in children and osteomalacia in adults after ossification is complete. They are caused by deficiency of vitamin D which promotes calcification of bone and absorption of calcium in the small intestine (see p. 274 and Table 11.1, p. 276). Deficiency may be due to:

- dietary deficiency of vitamin D
- malabsorption, e.g. coeliac disease or following gastrointestinal surgery

- lack of exposure to sunlight, e.g. pigmented skin, housebound people
- excessive loss of vitamin D or its precursors, e.g. in chronic renal failure, haemodialysis
- drugs that result in breakdown of vitamin D, e.g. anticonvulsants, including phenytoin.

In *rickets*, osteoid is deposited but calcification is incomplete. Although growth of the epiphyseal cartilage continues, growth is stunted. The bones remain soft and those of the lower limbs become bowed by the weight of the body.

In *osteomalacia* there is increased and abnormal turnover of bone. As in rickets, osteoid is not calcified and the bones become soft, bowed and prone to fractures.

Infection of bones

Osteomyelitis

Microbes gain access to bones:

- through the skin in compound fractures
- by spread from a local focus of infection, e.g. from an infected prosthesis or tooth abscess
- via the blood—commonly from a boil or paronychia (infection of the nail bed)
- during a surgical procedure.

The most common infecting organism is *Staphylococcus aureus*, which typically affects the growth regions of long bones in children. Infection of soft tissues of the feet, common in elderly diabetics, may spread to the bones. Infection of the bone causes inflammation that may completely resolve. In more severe cases healing may be delayed by the presence of *sequestra* (pieces of dead bone) in the wound. Complications include bone necrosis, suppuration (pus formation), local spread to the periosteum and then to surrounding soft tissues and joints. This may be followed by formation of a *subperiosteal abscess* that ruptures forming a sinus discharging pus to the skin, which in chronic cases can continue for several years.

Developmental abnormalities of bone

Achondroplasia

This is caused by a genetic abnormality. There is abnormal growth of cartilage, especially the epiphyseal cartilage of long bones, leading to characteristic dwarfism and under-development of the bones of the base of the skull (Fig. 16.41).

Figure 16.41 Achondroplasia.

Osteogenesis imperfecta ('brittle bone syndrome

This is a group of conditions in which there is a congen tal defect of osteoblasts, resulting in failure of ossific tion. The bones are brittle and fracture easily, eith spontaneously or following very slight trauma.

Tumours of bone

Benign tumours

Single or multiple tumours may develop for unknow reasons in bone and cartilage. They may cause patholo cal fractures or pressure damage to soft tissues, e.g. benign vertebral tumour may damage the spinal co or a spinal nerve. Benign tumours of cartilage have tendency to undergo malignant change.

Malignant tumours

Metastatic tumours

The most common malignancies of bone are metastas of primary carcinomas of the breast, lungs, thyroid, ki neys and prostate gland. The usual sites are those wi the best blood supply, i.e. cancellous bone, especially t bodies of the lumbar vertebrae and the epiphyses of t humerus and femur. Tumour fragments are spread blood, and possibly along the walls of the veins fro pelvic tumours to vertebrae. The effects of the malignan may be:

- destruction of bone, leading to pathological fracture
- collapse of vertebrae causing damage to the spinal cord and/or spinal nerves

- fibrosis of bone
- anaemia, leukopenia and thrombocytopenia but in most cases the link is not known.

Primary tumours

Osteosarcoma. This is a rapidly growing and often metastatic tumour believed to develop from the precursors of osteogenic cells. In young people between 10 and 25 years of age the tumour develops most commonly in the medullary canal of long bones, especially the femur. It is usually well advanced before it becomes evident. In older people, usually over 60 years of age, it is often associated with Paget's disease and the bones most commonly affected are the vertebrae, skull and pelvis.

Chondrosarcoma. These relatively slow-growing tumours are usually the result of malignant change in benign tumours of cartilage cells. They occur mainly between the ages of 40 and 70 years.

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17

The joints

A joint is the site at which any two or more bones articulate or come together. Some joints have no movement (*fibrous*), some only slight movement (*cartilaginous*) and some are freely movable (*synovial*).

TYPES OF JOINT

Learning outcomes

After studying this section you should be able to:

- state the characteristics of fixed and fibrous joints
- state the different types of synovial joints
- outline the movements possible at five types of synovial joints
- describe the structure and functions of a typical synovial joint.

Fibrous or fixed joints (Fig. 17.1)

These immovable joints have fibrous tissue between the bones, e.g. joints between the bones of the skull (sutures) and those between the teeth and the maxilla and mandible.

Cartilaginous or slightly movable joints (Fig. 17.2)

There is a pad of *fibrocartilage* between the ends of the bones that form the joint which allows for very slight movement where the pad of cartilage is compressed. Examples include the symphysis pubis and the joints between the vertebral bodies.

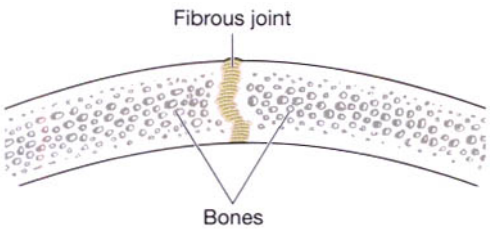

Figure 17.1 A fibrous or fixed joint, e.g. the sutures of the skull.

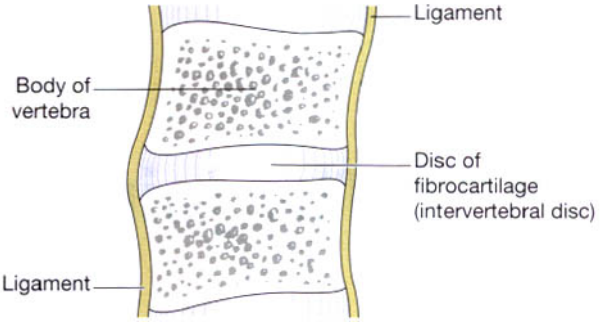

Figure 17.2 A cartilaginous or slightly movable joint, e.g. between the vertebral bodies.

Table 17.1 Movements possible at synovial joints

Movement	Definition
Flexion	Bending, usually forward but occasionally backward, e.g. knee joint
Extension	Straightening or bending backward
Abduction	Movement away from the midline of the body
Adduction	Movement towards the midline of the body
Circumduction	Combination of flexion, extension, abduction and adduction
Rotation	Movement round the long axis of a bone
Pronation	Turning the palm of the hand down
Supination	Turning the palm of the hand up
Inversion	Turning the sole of the foot inwards
Eversion	Turning the sole of the foot outwards

Synovial or freely movable joints

Synovial joints have characteristic features that enable a wide range of movements (Table 17.1). They are classified according to the range of movement possible or to the shape of the articulating parts of the bones involved.

Ball and socket. The head or ball of one bone articulates with a socket of another and the shape of the bones allows for a wide range of movement. Those possible are flexion, extension, adduction, abduction, rotation and circumduction. Examples are the shoulder and hip.

Hinge joints. These allow the movements of flexion and extension only. They are the elbow, knee, ankle, the joints between the atlas and the occipital bone, and the interphalangeal joints of the fingers and toes.

Gliding joints. The articular surfaces glide over each other, e.g. sternoclavicular joints, acromioclavicular joints and joints between the carpal bones and those between the tarsal bones.

Pivot joints. Movement is round one axis (rotation), e.g. proximal and distal radioulnar joints and the joint between the atlas and the odontoid process of the axis.

Condyloid and saddle joints. Movements take place round two axes, permitting flexion, extension, abduction, adduction and circumduction, e.g. the wrist, temporomandibular, metacarpophalangeal and metatarsophalangeal joints.

Characteristics of a synovial joint (Fig. 17.3)

All synovial joints have certain characteristics in common.

Articular or hyaline cartilage

The parts of the bones which are in contact are always covered with hyaline cartilage. It provides a smooth articular surface and is strong enough to absorb compression forces and bear the weight of the body. The cartilage lining, which is up to 7 mm thick in young people, becomes thinner and less compressible with age. This leads to increasing stress on other structures in the joint. Cartilage has no blood supply and receives its nourishment from synovial fluid.

Capsule or capsular ligament

The joint is surrounded and enclosed by a sleeve of fibrous tissue which holds the bones together. It is sufficiently loose to allow freedom of movement but strong enough to protect it from injury.

Synovial membrane

This is composed of epithelial cells and is found:

- lining the capsule
- covering those parts of the bones within the joint not covered by articular cartilage
- covering all intracapsular structures that do not bear weight.

Synovial fluid. This is a thick sticky fluid, of egg-white consistency, secreted by synovial membranes into the *synovial cavity*, and it:

- provides nutrients for the structures within the joint cavity

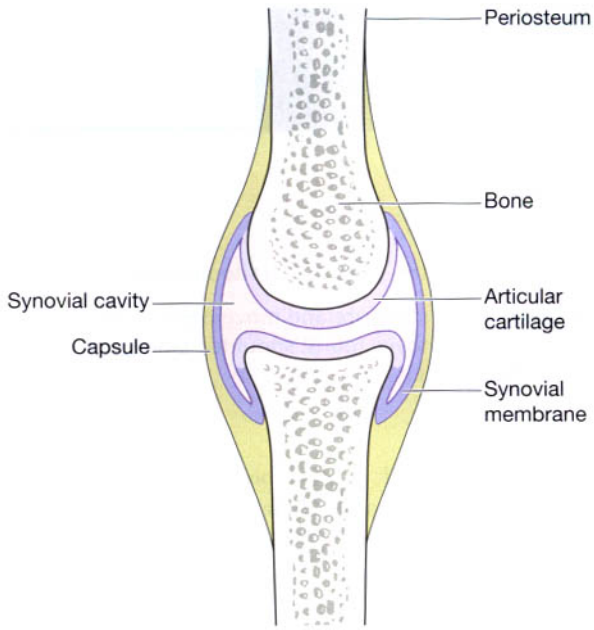

Figure 17.3 Diagram of the basic structure of a synovial joint.

- contains phagocytes, which remove microbes and cellular debris
- acts as a lubricant
- maintains joint stability
- prevents the ends of the bones from being separated, as does a little water between two glass surfaces.

Little sacs of synovial fluid or *bursae* are present in some joints, e.g. the knee. They act as cushions to prevent friction between a bone and a ligament or tendon, or skin where a bone in a joint is near the surface.

Other intracapsular structures

Some joints have structures within the capsule, but outside the synovial membrane, which assist in maintenance of stability, e.g. fat pads and menisci in the knee joint. When these structures do not bear weight they are covered by synovial membrane.

Extracapsular structures

- *Ligaments* that blend with the capsule provide additional stability at most joints.
- *Muscles* or their *tendons* also provide stability and stretch across the joints they move. When the muscle contracts it shortens, pulling one bone towards the other.

Nerve and blood supply

Nerves and blood vessels crossing a joint usually supply the capsule and the muscles that move it.

MAIN SYNOVIAL JOINTS OF THE LIMBS

Learning outcome

After studying this section you should be able to:

- describe the structure and movements of the following synovial joints: shoulder, elbow, wrist, hip, knee, ankle.

Individual synovial joints have the characteristics described above so only their distinctive features are included in this section.

Shoulder joint (Fig. 17.4)

This ball and socket joint is formed by the glenoid cavity of the scapula and the head of the humerus. The capsular ligament is very loose inferiorly to allow for the free movement normally possible at this joint. The glenoid cavity is deepened by a rim of fibrocartilage, the *glenoidal labrum*, which provides additional stability without limiting movement. The tendon of the long head of the *biceps muscle*, lying in the intertubercular (bicipital) groove of the humerus, extends through the joint cavity and is attached to the upper rim of the glenoid cavity. It has an important stabilising effect on the joint.

Synovial membrane forms a sleeve round the part of the tendon of the long head of the biceps muscles within the capsular ligament and covers the glenoidal labrum.

Extracapsular structures consist of:

- the *coracohumeral ligament*, extending from the coracoid process of the scapula to the humerus
- the *glenohumeral ligaments*, which blend with and strengthen the capsule
- the *transverse humeral ligament*, retaining the biceps tendon in the intertubercular groove.

The stability of the joint may be reduced if these structures, together with the tendon of the biceps muscle, are stretched by repeated dislocations of the joint.

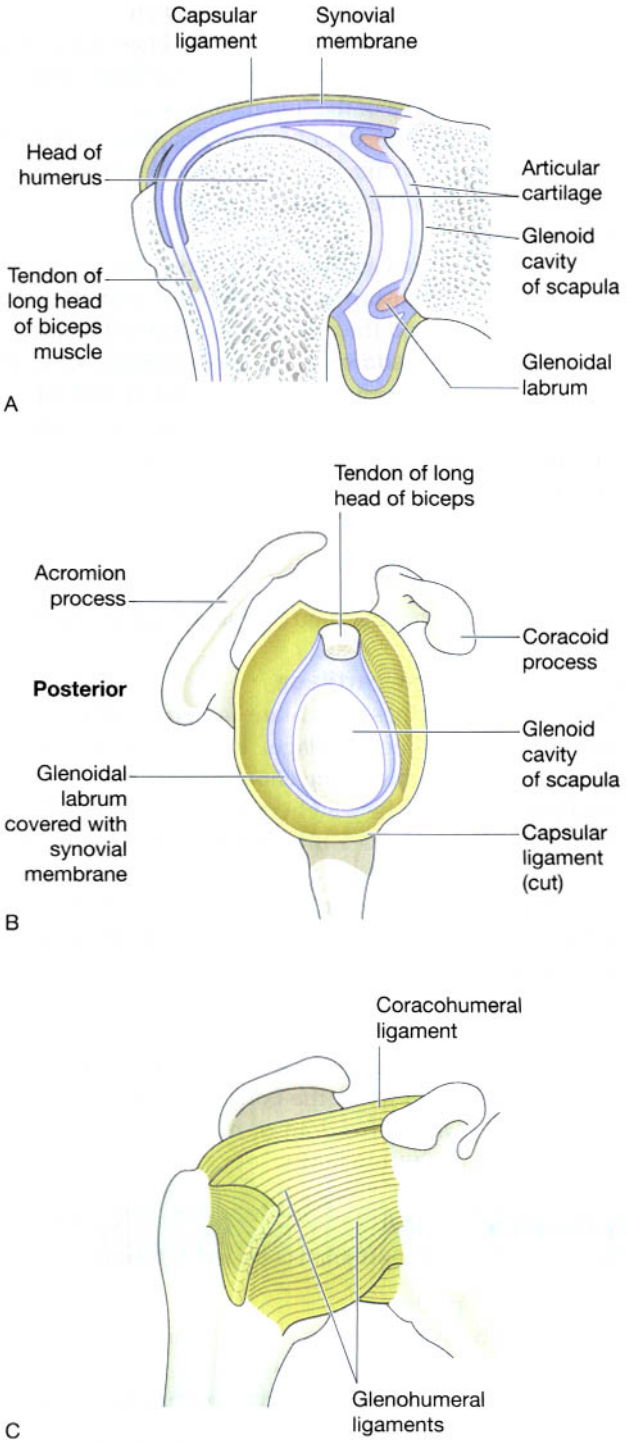

Figure 17.4 The right shoulder joint: A. Section viewed from the front. B. The position of glenoidal labrum with the humerus removed, viewed from the side. C. The supporting ligaments viewed from the front.

Muscles and movements

Muscles (Fig. 17.5)

Coracobrachialis muscle. This lies on the upper medial aspect of the arm. It arises from the coracoid process of the scapula, stretches across in front of the shoulder joint and is inserted into the middle third of the humerus. It flexes the shoulder joint.

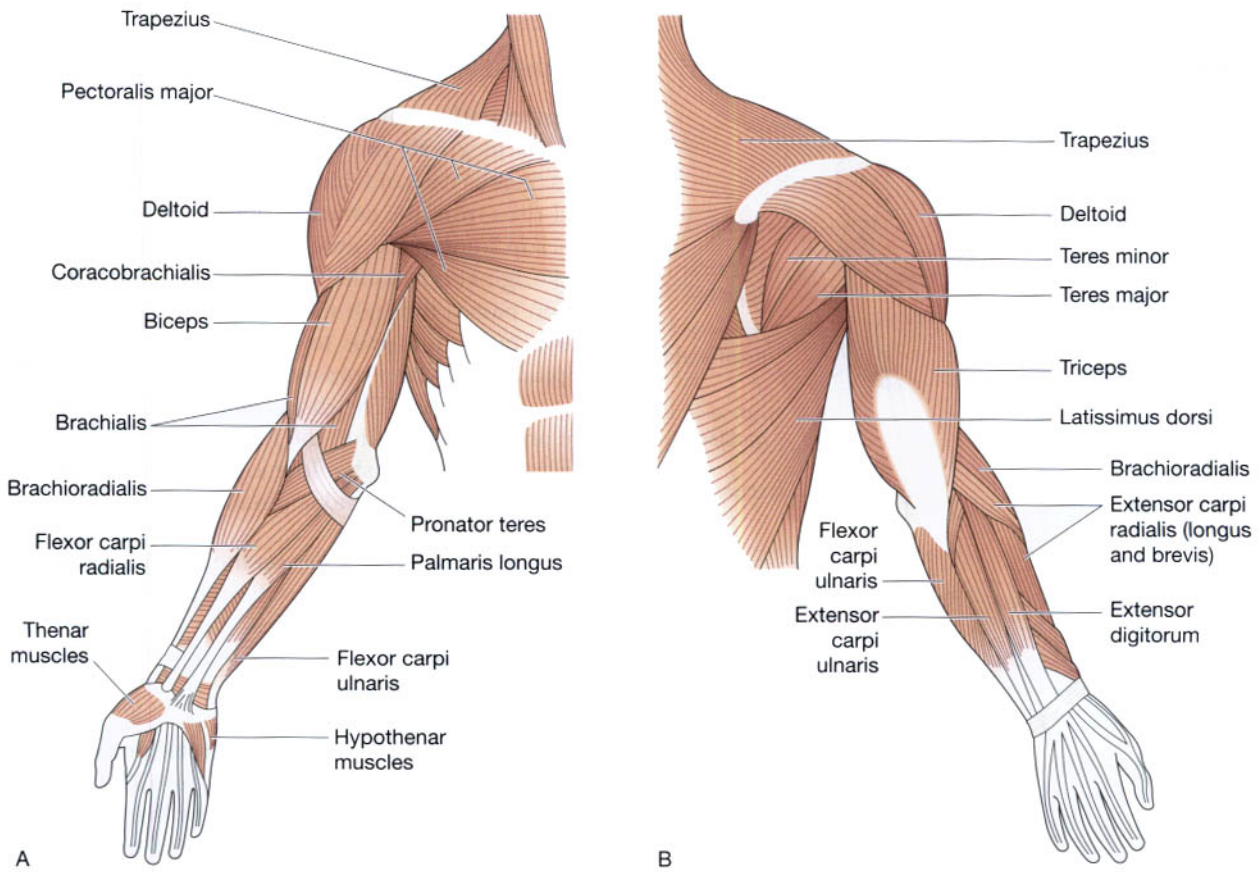

Figure 17.5 The main muscles that move the joints of the upper limb. A. Anterior view. B. Posterior view.

Deltoid muscle. These muscle fibres originate from the clavicle, acromion process and spine of scapula and radiate over the shoulder joint to be inserted into the deltoid tuberosity of the humerus. It forms the fleshy and rounded contour of the shoulder. The anterior fibres cause flexion, the middle or main part, abduction and the posterior fibres extend the shoulder joint.

Pectoralis major. This lies on the anterior thoracic wall. The fibres originate from the middle third of the clavicle and from the sternum and are inserted into the lip of the intertubercular groove of the humerus. It draws the arm forward and towards the body, i.e. flexes and adducts.

Latissimus dorsi. This arises from the posterior part of the iliac crest and the spinous processes of the lumbar and lower thoracic vertebrae. It passes upwards across the back then under the arm to be inserted into the bicipital groove of the humerus. It adducts, medially rotates and extends the arm.

Teres major. This originates from the inferior angle of the scapula and is inserted into the humerus just below the shoulder joint. It extends, adducts and medially rotates the arm.

Movements

Flexion: coracobrachialis, anterior fibres of deltoid and pectoralis major.
Extension: teres major, latissimus dorsi and posterior fibres of deltoid.
Abduction: deltoid.
Adduction: combined action of flexors and extensors.
Circumduction: flexors, extensors, abductors and adductors acting in series.
Medial rotation: pectoralis major, latissimus dorsi, teres major and anterior fibres of deltoid.
Lateral rotation: posterior fibres of deltoid.

Elbow joint (Fig. 17.6)

This *hinge* joint is formed by the trochlea and the capitulum of the humerus and the trochlear notch of the ulna and the head of the radius.

Extracapsular structures consist of anterior, posterior, medial and lateral strengthening ligaments.

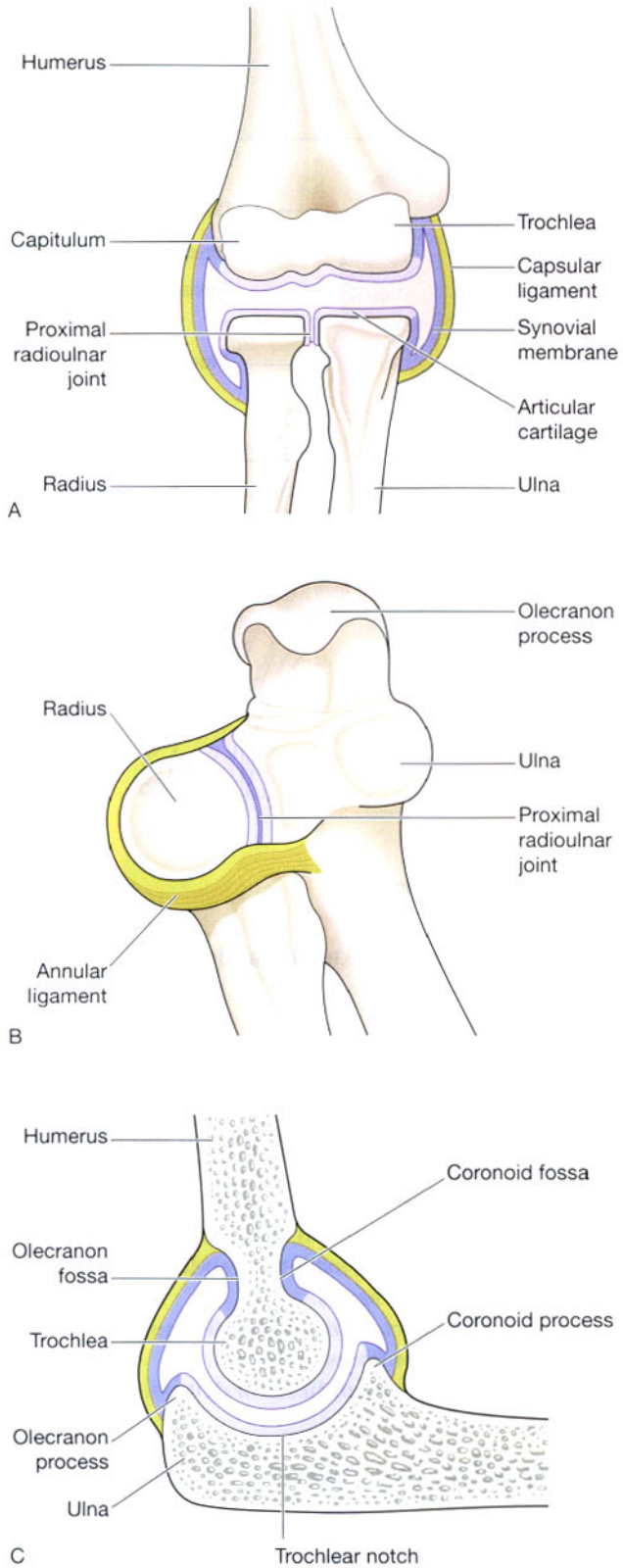

Figure 17.6 The elbow and proximal radioulnar joints. A. Section viewed from the front. B. The proximal radioulnar joint, viewed from above. C. Section of the elbow joint, partly flexed, viewed from the side.

Muscles and movements

Muscles (Fig. 17.5)

Biceps muscle. This lies on the anterior aspect of the upper arm. At its proximal end it is divided into two parts (heads) each of which has its own tendon. The short head rises from the coracoid process of the scapula and passes in front of the shoulder joint to the arm. The long head originates from the rim of the glenoid cavity and its tendon passes through the joint cavity and the bicipital groove of the humerus to the arm. It is retained in the bicipital groove by a transverse ligament which stretches across the groove. The distal tendon crosses the elbow joint and is inserted into the radial tuberosity. It helps to stabilise and flex the shoulder joint and at the elbow joint it assists with flexion and supination.

Brachialis muscle. This lies on the anterior aspect of the upper arm deep to the biceps. It originates from the shaft of the humerus, extends across the elbow joint and is inserted into the ulna just distal to the joint capsule. It is the main flexor of the elbow joint.

Triceps muscle. This lies on the posterior aspect of the humerus. It arises from three heads, one from the scapula and two from the posterior surface of the humerus. The insertion is by a common tendon to the olecranon process of the ulna. It helps to stabilise the shoulder joint, assists in adduction of the arm and extends the elbow joint.

Movements

Flexion: biceps and brachialis.
Extension: triceps.

Proximal and distal radioulnar joints

The *proximal radioulnar joint*, formed by the rim of the head of the radius rotating in the radial notch of the ulna, is in the same capsule as the elbow joint. The *annular ligament* is a strong extracapsular ligament which encircles the head of the radius and keeps it in contact with the radial notch of the ulna (Fig. 17.6B).

The distal *radioulnar joint* is a pivot joint between the distal end of the radius and the head of the ulna (Fig. 17.7).

Muscles and movements

Muscles (Fig. 17.5)

Pronator teres. This lies obliquely across the upper third of the front of the forearm. It arises from the medial epicondyle of the humerus and the coronoid process of the ulna and passes obliquely across the forearm to be

inserted into the lateral surface of the shaft of the radius. It rotates the radioulnar joints, changing the hand from the anatomical to the writing position, i.e. pronation.

Supinator muscle. This lies obliquely across the posterior and lateral aspects of the forearm. Its fibres arise from the lateral epicondyle of the humerus and the upper part of the ulna and are inserted into the lateral surface of the upper third of the radius. It rotates the radioulnar joints, changing the hand from the writing to the anatomical position, i.e. supination. It lies deep to the muscles shown in Figure 17.5.

Movements

Pronation: pronator teres.
Supination: supinator and biceps.

Wrist joint (Fig. 17.7)

This is a *condyloid* joint between the distal end of the radius and the proximal ends of the scaphoid, lunate and triquetral. A disc of white fibrocartilage separates the ulna from the joint cavity and articulates with the carpal bones. It also separates the inferior radioulnar joint from the wrist joint.

Extracapsular structures consist of medial and lateral ligaments and anterior and posterior radiocarpal ligaments.

Muscles and movements

Muscles (Fig. 17.5)

Flexor carpi radialis. This lies on the anterior surface of the forearm. It originates from the medial epicondyle of the humerus and is inserted into the second and third metacarpal bones. It flexes the wrist joint, and when acting with the extensor carpi radialis, abducts the joint.

Flexor carpi ulnaris. This lies on the medial aspect of the forearm. It originates from the medial epicondyle of the humerus and the upper parts of the ulna and is inserted into the pisiform, the hamate and the fifth metacarpal bones. It flexes the wrist, and when acting with the extensor carpi ulnaris, adducts the joint.

Extensor carpi radialis longus and brevis. These lie on the posterior aspect of the forearm. The fibres originate from the lateral epicondyle of the humerus and are inserted by a long tendon into the second and third metacarpal bones. They extend and abduct the wrist.

Extensor carpi ulnaris. This lies on the posterior surface of the forearm. It originates from the lateral epicondyle of the humerus and is inserted into the fifth metacarpal bone. It extends and adducts the wrist.

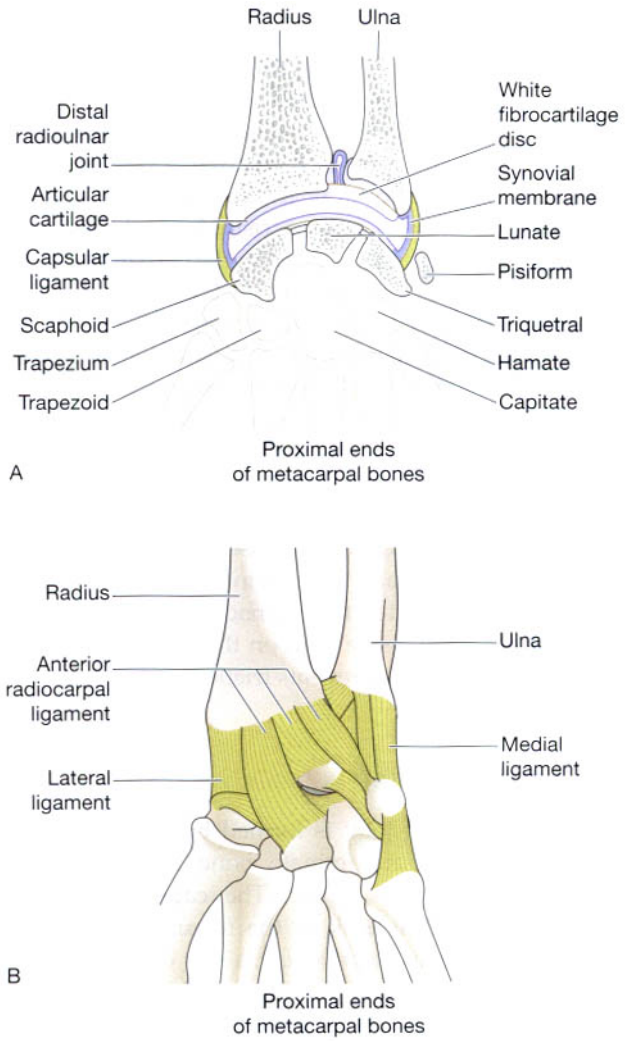

Figure 17.7 The wrist and distal radioulnar joints. Anterior view. A. Section. B. Supporting ligaments.

Movements

Flexion: flexor carpi radialis and the flexor carpi ulnaris.
Extension: extensors carpi radialis (longus and brevis) and the extensor carpi ulnaris.
Abduction: flexor and extensors carpi radialis.
Adduction: flexor and extensor carpi ulnaris.

Joints of the hands and fingers

There are synovial joints between the carpal bones, between the carpal and metacarpal bones, between the metacarpal bones and proximal phalanges and between the phalanges. The powerful movements that occur at

these joints are produced by muscles in the forearm which have tendons extending into the hand. Many of the finer movements of the fingers are produced by numerous small muscles in the hand.

The flexor retinaculum is a strong fibrous band that stretches across the front of the carpal bones, enclosing their concavity and forming the *carpal tunnel*. The tendons of flexor muscles of the wrist joint and the fingers and the median nerve pass through the carpal tunnel, the retinaculum holding them close to the bones. Synovial membrane forms sleeves around these tendons in the carpal tunnel and extends some way into the palm of the hand. Synovial sheaths also enclose the tendons on the flexor surfaces of the fingers. Synovial fluid prevents friction that might damage the tendons as they move over the bones (Fig. 17.8).

The extensor retinaculum is a strong fibrous band that extends across the back of the wrist. Tendons of muscles that extend the wrist and finger joints are encased in synovial membrane under the retinaculum. The synovial sheaths are less extensive than on the flexor aspect. The synovial fluid secreted prevents friction.

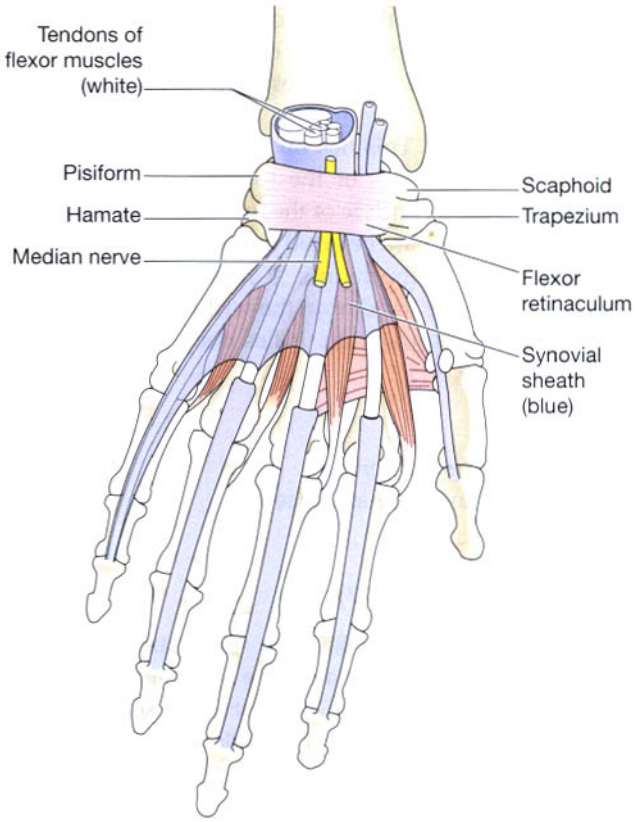

Figure 17.8 The carpal tunnel and synovial sheaths in the wrist and hand in green; tendons in white. Palmar view, left hand.

Hip joint (Fig. 17.9)

This ball and socket joint is formed by the cup-shaped acetabulum of the innominate bone and the almost spherical head of the femur. The capsular ligament includes most of the neck of the femur. The cavity is deepened by the *acetabular labrum*, a ring of fibrocartilage attached to the rim of the acetabulum. This adds stability to the joint without limiting its range of movement. The ligament of the head of the femur extends from the shallow depression in the middle of the head of the femur to the acetabulum. It conveys a blood vessel to the head of the femur. Synovial membrane covers both sides of the acetabular labrum and forms a sleeve around the ligament of the head of the femur. There are three important ligaments that surround and strengthen the capsule. They are the *iliofemoral, ischiofemoral* and *pubofemoral ligaments*.

Muscles and movements

Muscles (Figs 17.10 and 17.11)

Psoas muscle. This arises from the transverse processes and bodies of the lumbar vertebrae. It passes across the flat part of the ilium and behind the inguinal ligament to be inserted into the femur. Together with the iliacus it flexes the hip joint (Fig. 17.10).

Iliacus muscle. This lies in the iliac fossa of the innominate bone. It originates from the iliac crest, passes over the iliac fossa and joins the tendon of the psoas muscle to be inserted into the lesser trochanter of the femur. The combined action of iliacus and psoas flexes the hip joint.

Quadriceps femoris. This is a group of four muscles lying on the front and sides of the thigh. They are the *rectus femoris* and three *vasti*. The rectus femoris originates from the ilium and the three vasti from the upper end of the femur. Together they pass over the front of the knee joint to be inserted into the tibia by the patellar tendon. Only the rectus femoris flexes the hip joint. Together the group acts as a very strong extensor of the knee joint.

Gluteal muscles. These consist of the *gluteus maximus, medius* and *minimus* which together form the fleshy part of the buttock. They originate from the ilium and sacrum and are inserted into the femur. They cause extension, abduction and medial rotation at the hip joint.

Sartorius. This is the longest muscle in the body and crosses both the hip and knee joints. It originates from the

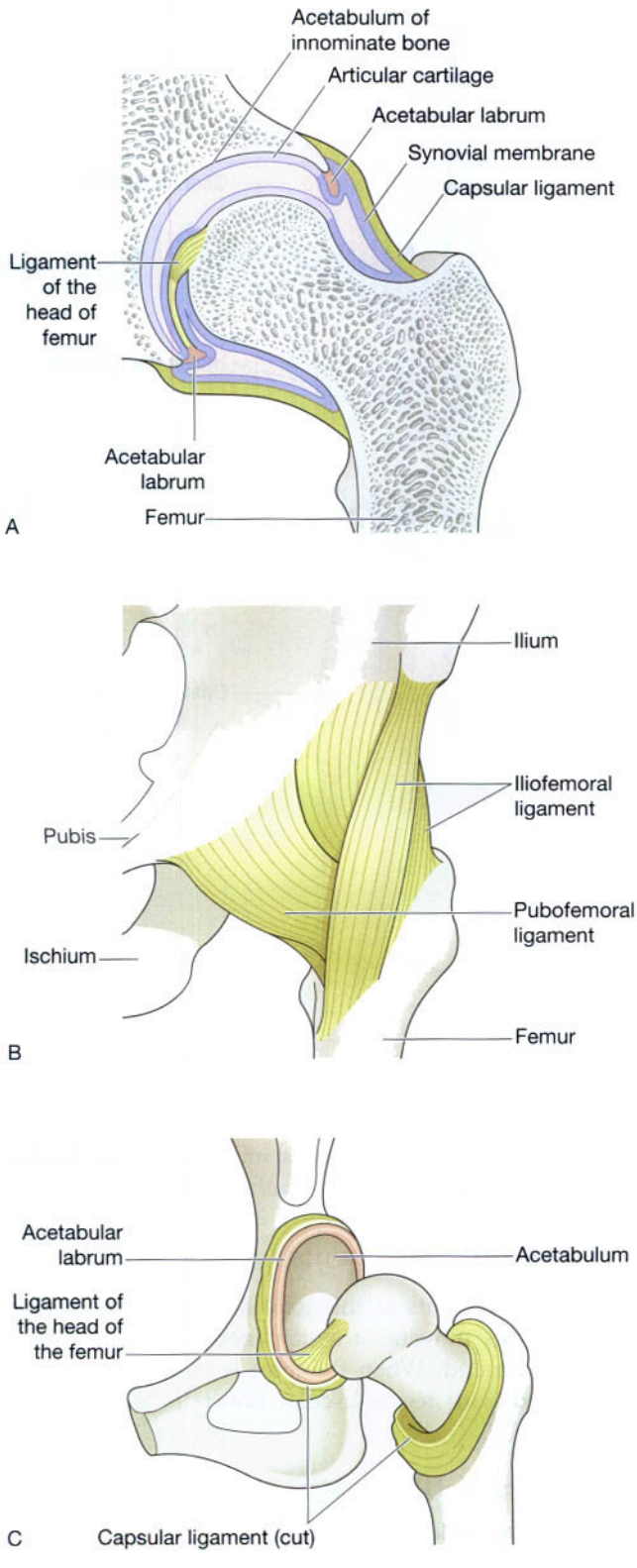

Figure 17.9 The hip joint. Anterior view. A. Section. B. Supporting ligaments. C. Head of femur and acetabulum separated to show acetabular labrum and ligament of head of femur.

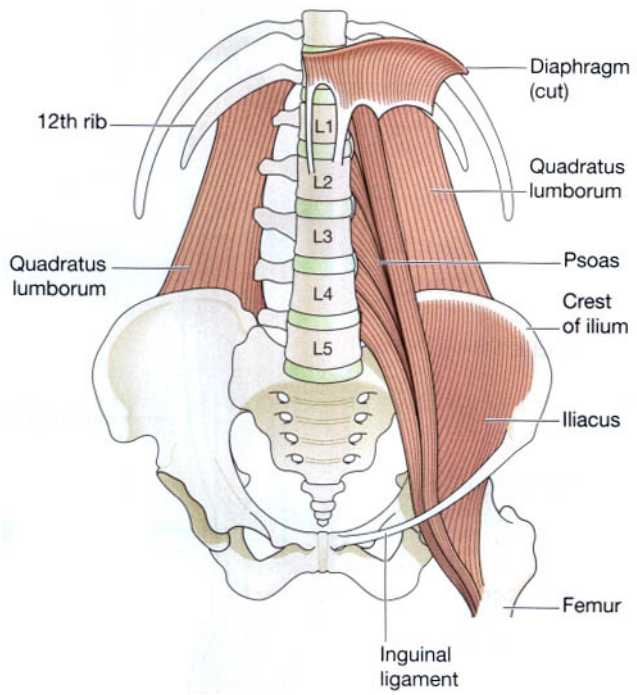

Figure 17.10 The muscles of the posterior abdominal wall and pelvis which flex the hip joint.

anterior superior iliac spine and passes obliquely across the hip joint, thigh and knee joint to be inserted into the medial surface of the upper part of the tibia. It is associated with flexion and abduction at the hip joint and flexion at the knee.

Adductor group. This lies on the medial aspect of the thigh. They originate from the pubic bone and are inserted into the linea aspera of the femur. They adduct and medially rotate the thigh.

Movements

Flexion: psoas, iliacus, rectus femoris and sartorius.
Extension: gluteus maximus and the hamstrings.
Abduction: gluteus medius and minimus, sartorius and others.
Adduction: adductor group.
Lateral rotation: mainly gluteal muscles and adductor group.
Medial rotation: gluteus medius and minimus and others.

Knee joint (Fig. 17.12)

This is the largest and most complex joint. It is a hinge joint formed by the condyles of the femur, the condyles of

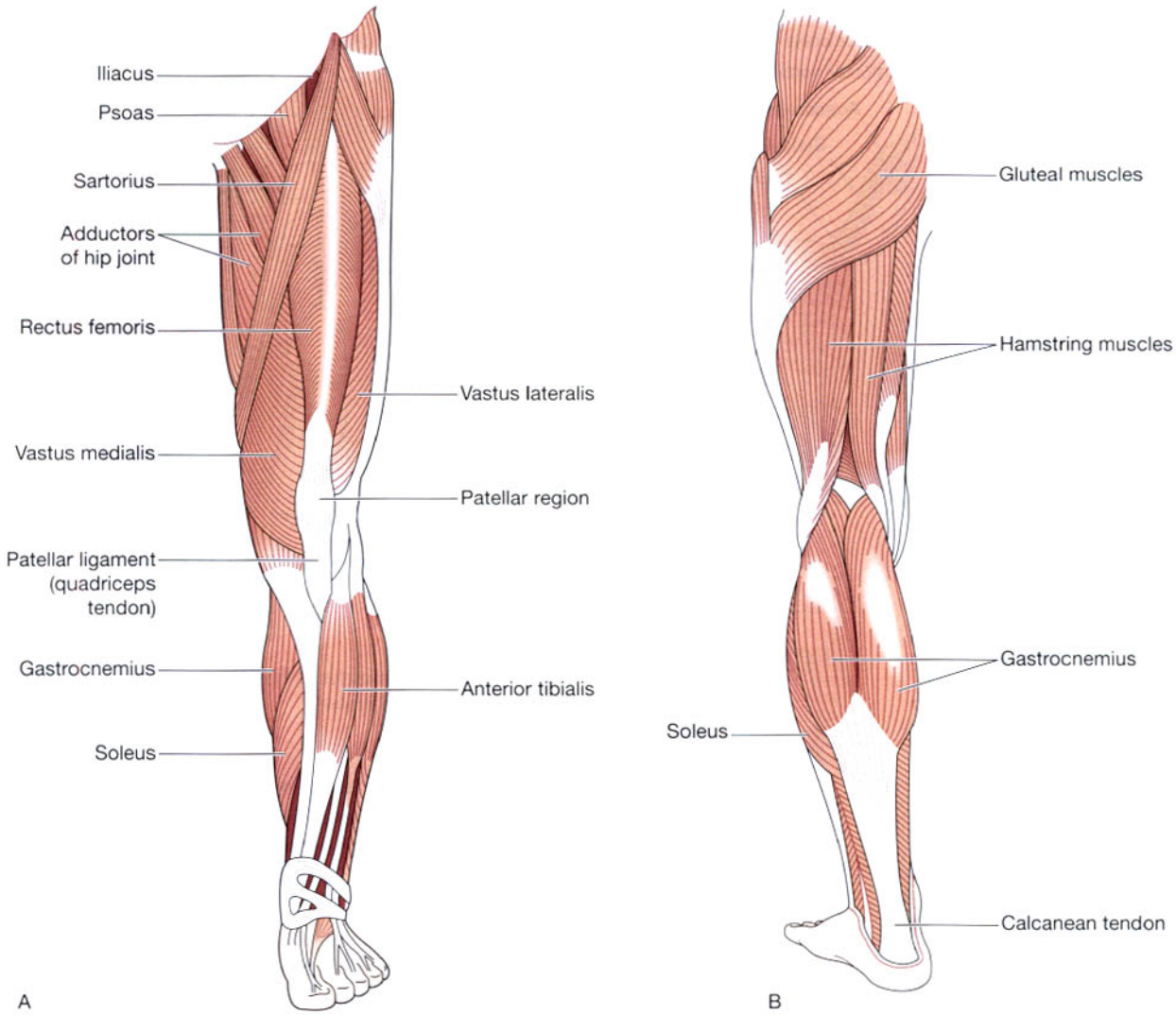

Figure 17.11 The main muscles of the lower limb. A. Anterior view. B. Posterior view.

the tibia and the posterior surface of the patella. The anterior part of the capsule consists of the tendon of the quadriceps femoris muscle which also supports the patella. Intracapsular structures include two *cruciate ligaments* that cross each other, extending from the *intercondylar notch* of the femur to the *intercondylar eminence* of the tibia. They help to stabilise the joint.

Semilunar cartilages or *menisci* are incomplete discs of white fibrocartilage lying on top of the articular condyles of the tibia. They are wedge-shaped, being thicker at their outer edges. They help to stabilise the joint by preventing lateral displacement of the bones.

Bursae and pads of fat are numerous. They prevent friction between a bone and a ligament or tendon and between the skin and the patella. Synovial membrane covers the cruciate ligaments and the pads of fat. The menisci are not covered with synovial membrane because they are weight bearing. The most important strengthening ligaments are the *medial* and *lateral ligaments*.

Muscles and movements

Possible movements at this joint are flexion, extension and a rotatory movement which 'locks' the joint when it is fully extended. When the joint is locked, balance is maintained with less muscular effort than when it is flexed.

Muscles (Fig. 17.11)

Hamstring muscles. These lie on the posterior aspect of the thigh. They originate from the ischium and are inserted into the upper end of the tibia. They are *biceps femoris, semimembranosus* and *semitendinosus muscles*. They flex the knee joint.

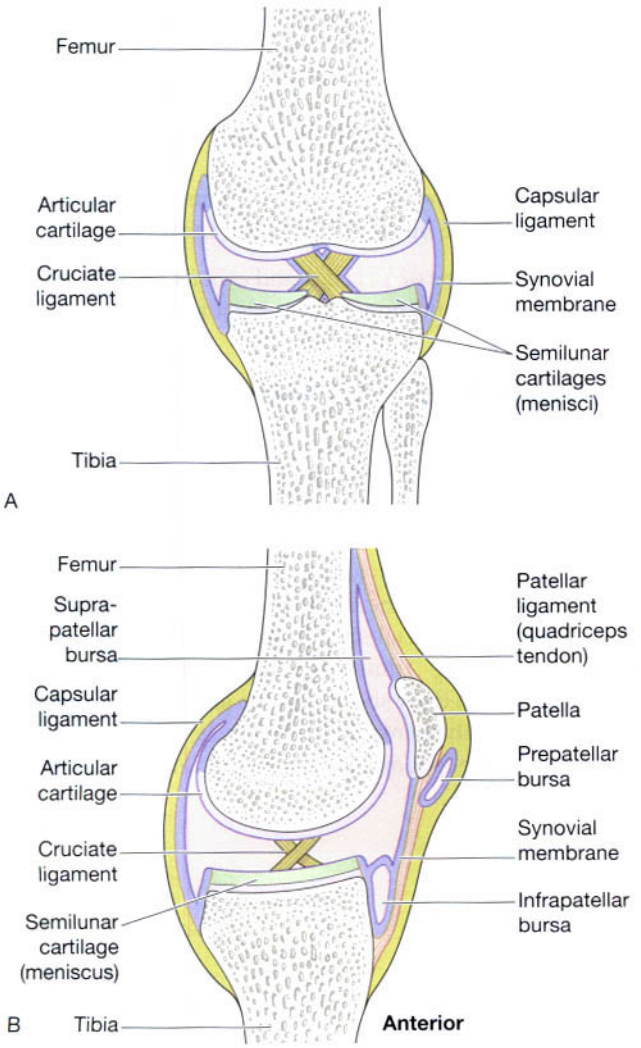

Figure 17.12 The knee joint. A. Section viewed from the front. B. Section viewed from the side. C. The superior surface of the tibia showing the semilunar cartilages and the cruciate ligaments.

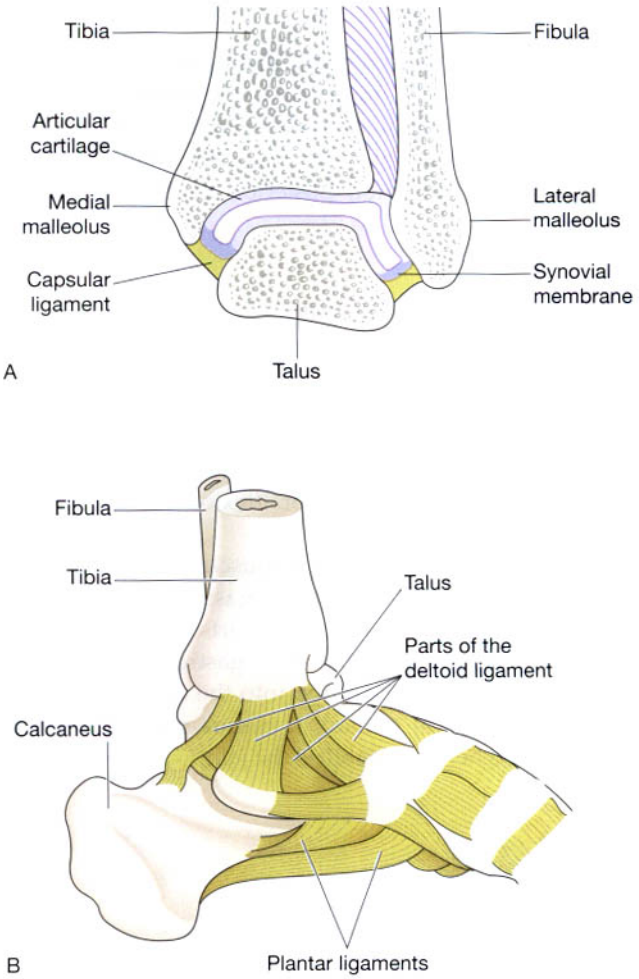

Figure 17.13 The left ankle joint. A. Section viewed from the front. B. Supporting ligaments. Medial view.

Gastrocnemius. This forms the bulk of the calf of the leg. It arises by two heads, one from each condyle of the femur, and passes down behind the tibia to be inserted into the calcaneus by the *calcanean tendon* (*Achilles tendon*). It crosses both knee and ankle joints, causing flexion at the knee and plantarflexion at the ankle.

Quadriceps femoris (described above). This extends the knee joint.

Movements

Flexion (bending backwards): gastrocnemius and hamstrings.

Extension (straightening): quadriceps femoris muscle.

Ankle joint (Fig. 17.13)

This *hinge* joint is formed by the distal end of the tibia and its malleolus (medial malleolus), the distal end of the fibula (lateral malleolus) and the talus. There are four important ligaments strengthening this joint. They are the deltoid and anterior, posterior, medial and lateral ligaments.

Muscles and movements

Muscles (Fig. 17.11)

Anterior tibialis muscle. This originates from the upper end of the tibia, lies on the anterior surface of the leg and is inserted into the middle cuneiform bone by a long tendon. It is associated with dorsiflexion of the foot.

Soleus. This is one of the main muscles of the calf of the leg, lying immediately deep to the gastrocnemius. It originates from the heads and upper parts of the fibula and the tibia. Its tendon joins that of the gastrocnemius so that they have a common insertion into the calcaneus by the calcanean (Achilles) tendon. It causes plantarflexion at the ankle and helps to stabilise the joint when standing.

Gastrocnemius. This (described above) is a powerful plantarflexor.

Movements

Flexion (*dorsiflexion*): anterior tibialis assisted by the muscles which extend the toes.
Extension (*plantarflexion*): gastrocnemius and soleus assisted by the muscles which flex the toes.

The movements of *inversion* and *eversion* occur between the tarsal bones and not at the ankle joint.

Joints of the foot and toes

There are a number of synovial joints between the tarsal bones, between the tarsal and metatarsal bones, between the metatarsals and proximal phalanges and between the phalanges. Movements are produced by muscles in the leg with long tendons which cross the ankle joint, and by muscles of the foot. The tendons crossing the ankle joint are encased in synovial sheaths and are held close to the bones by strong transverse ligaments. They move smoothly within their sheaths as the joints move. In addition to moving the joints of the foot these muscles support the arches of the foot and help to maintain body balance.

DISORDERS OF JOINTS

Learning outcomes

After studying this section you should be able to:

- relate the features of the diseases in this section to abnormal anatomy and physiology
- compare and contrast the features of rheumatoid arthritis and osteoarthritis.

The tissues involved in diseases of the synovial joints are synovial membrane, hyaline cartilage and bone.

Inflammatory diseases of joints (arthritis)

Rheumatoid arthritis (RA, rheumatoid disease)

This is a chronic progressive inflammatory autoimmune disease. It is a systemic disorder where inflammatory changes not only affect synovial joints but also many other sites including the heart, blood vessels and skin.

It is more common in females than males and can affect all ages, including children (Still's disease), although it usually develops between the ages of 35 and 55 years. The cause is not clearly understood but development of autoimmunity may be initiated by microbial infection, possibly by viruses, in genetically susceptible people. Antigen/antibody complexes (*rheumatoid factors*) are formed and are often found in the blood and synovial fluid (*seropositive RA*). Seropositive individuals tend to have a more aggressive form of RA than those without rheumatoid factors, i.e. seronegative RA. Rheumatoid factors appear early in severe cases of sudden onset, and later when the disease develops gradually. Acute exacerbations of rheumatoid arthritis are usually accompanied by fever, and are interspersed with periods of remission. The joints most commonly affected are those of the hands and feet, but in severe cases most of the synovial joints may be involved. With each febrile exacerbation there is additional and cumulative damage to the joints, leading to increasing deformity, pain and loss of function. The primary changes that may be reversible include hypertrophy and hyperplasia of synovial cells and fibrinous inflammatory effusion into the joint. If the disease progresses there are further secondary changes which may be irreversible, including:

- erosion of articular cartilage and the growth of granulation tissue (*pannus*) that separates the bones and distorts the shape of the joint
- fibrosis of pannus which causes adhesions between the bones, limiting movement
- ossification of the fibrosed pannus, further restricting joint movement
- spread of granulation tissue to tendons
- weakening and atrophy of muscles possibly due to limited exercise
- development of *rheumatoid nodules* (subcutaneous collagen nodules) outside the joints, e.g. in pressure areas such as the elbow, over the knuckles and in the lungs, pleura, heart and eyes
- enlargement of lymph nodes and spleen (lymphadenopathy and splenomegaly).

In the later stages of the disease the inflammation and fever are less marked and movement is limited by deformity of the joint, muscle weakness and pain. The extent of disability varies between slight and severe. Table 17.2 highlights differences between osteoarthritis and rheumatoid arthritis.

Table 17.2 **Features of the two main types of arthritis**

	Osteoarthritis	Rheumatoid arthritis
Type of disease	Degenerative	Inflammatory and autoimmune
Tissue affected	Articular cartilage	Synovial membrane
Age of onset	Late middle age	Any age, mainly 30 to 55 years, occasionally children
Joints affected	Weight bearing, e.g. hip, knee; often only a single joint	Small, e.g. hands, feet; often many joints

Other types of polyarthritis

This group of autoimmune inflammatory arthritic diseases has many characteristics similar to rheumatoid arthritis but the rheumatoid factor is absent. The causes are not known but genetic features may be involved. The joints affected are mainly those of the axial skeleton.

Ankylosing spondylitis. In this the sacroiliac and vertebral joints become progressively ossified.

Psoriatic arthritis. This occurs in a proportion of people who suffer from psoriasis, especially if the nails are involved.

Reiter's syndrome (polyarthritis with urethritis and conjunctivitis). This syndrome, it is believed, may be precipitated by infection with *Chlamydia trachomatis*; the affected joints are usually those of the lower limb.

Rheumatic fever. Polyarthritis is a common presenting feature often involving the wrists, elbows, knees and ankles. Unlike cardiac effects, arthritis usually resolves spontaneously without complications (p. 122).

Infective arthritis

Microbes may be carried in the blood to the joints from foci of infection elsewhere in the body. In most cases of septic arthritis the joint has been damaged by previous injury or arthritic disease. The outcome may be:

- resolution without complications
- suppuration followed by healing with the formation of fibrous tissue that may become ossified
- development of chronic infection, especially in brucellosis, gonorrhoea and tuberculosis.

Traumatic injury to joints

Sprains, strains and dislocations

These damage the soft tissues, tendons and ligaments round the joint without penetrating the joint capsule. In dislocations there may be additional damage to intracapsular structures by stretching, e.g. long head of biceps muscle in the shoulder joint, cruciate ligaments in the knee joint, ligament of head of femur in the hip joint. If repair is incomplete there may be some loss of stability which increases the risk of repeated injury.

Penetrating injuries

These may be caused by a compound fracture of one of the articulating bones, or trauma caused by, e.g., gun shot. Healing may be uneventful or it may be delayed by:

- the presence in the joint of tissue fragments or sequestra (tiny pieces of bone) too large to be removed by phagocytes
- incomplete healing of torn ligaments inside the capsule
- infection that may be blood-borne or enter through broken skin.

When healing is incomplete there is a tendency for irreversible degenerative changes to occur.

Osteoarthritis (osteoarthrosis, OA)

This is a degenerative non-inflammatory disease that results in pain and restricted movement of affected joints. Osteoarthrosis is the more appropriate name but is less commonly used. Articular cartilage gradually becomes thinner because its renewal does not keep pace with its breakdown. Eventually the bony articular surfaces come in contact and the bones begin to degenerate. There is abnormal bone repair and the articular surfaces become misshapen. Chronic inflammation develops with effusion into the joint, possibly due to irritation caused by tissue debris not removed by phagocytes. Sometimes there is abnormal outgrowth of cartilage at the edges of bones which becomes ossified, forming *osteophytes*.

Primary osteoarthritis

This is the more common type and the cause is unknown. Changes may be due to acceleration of the normal ageing process in joints that have had excessive use. It usually develops in late middle age and affects large weight-bearing joints, i.e. the hips, knees and joints of the cervical and lower lumbar spine. In many cases only one joint is involved.

Osteoarthritis of spine

This condition is relatively common in the elderly. Degenerative changes cause narrowing of intervertebral discs and osteophytes may develop round the margins of joints of the vertebral column, commonly in the cervical region (*cervical spondylosis*). They may cause damage to the nervous system, varying from compression of individual spinal nerves to spinal cord injury.

Secondary osteoarthritis

This occurs in joints in which cartilage has already been damaged due to:

- congenital deformity of bones, e.g. in congenital dislocation of the hip
- trauma, e.g. intracapsular fracture of a bone, injury to intracapsular structures
- other conditions, e.g. inflammatory diseases, haemophilia following repeated haemorrhages into the joints, peripheral nerve lesions, gout, acromegaly, diabetic neuropathy.

Gout

This condition is more prevalent in males than females and there is a familial tendency. It is caused by the deposition of sodium urate crystals in joints and tendons that provokes an acute inflammatory response. It occurs in some people whose blood uric acid is abnormally high due to either overproduction or defective excretion by the kidneys. Uric acid is a waste product of the breakdown of nucleic acids, i.e. DNA and RNA, and is produced in excess when there is large-scale cell destruction, e.g. following trauma or treatment with cytotoxic drugs and in anaemia, starvation and malignancy. Defective excretion occurs in renal failure. In many cases only one joint is involved (monoarthritis) and it is typically red, hot and painful. The sites most commonly affected are the metatarsophalangeal joint of the big toe and the ankle, knee, wrist and elbow joints. Episodes of arthritis lasting days or weeks are interspersed with periods of remission. After repeated acute attacks permanent damage may occur with chronic deformity and loss of function of the affected joints. Gout is sometimes complicated by the development of renal calculi.

Connective tissue diseases

This group of disorders has common features. They:

- affect many systems of the body, especially the joints, skin and subcutaneous tissues
- tend to occur in early adult life
- usually affect more females than males
- are chronic conditions
- are autoimmune diseases in which abnormal autoantibodies are formed that attack the individual's tissues.

These disorders include the following.

- *Systemic lupus erythematosus* (SLE) – in this the affected joints are usually the hands, knees and ankles. A characteristic red 'butterfly' rash may occur on the face. Kidney involvement is common and can result in glomerulonephritis that may be complicated by chronic renal failure.
- *Systemic sclerosis* (*scleroderma*) – this is a group of disorders in which there is progressive thickening of connective tissue. There is increased production of collagen that affects many organs. In the skin there is dermal fibrosis and tightness that impairs the functioning of joints, especially of the hands. It also affects the walls of blood vessels, intestinal tract and other organs.
- *Polyarteritis nodosa* (p. 114).
- *Rheumatoid arthritis* (p. 425).
- *Ankylosing spondylitis* (p. 426).
- *Reiter's disease* (p. 426).

Carpal tunnel syndrome

This occurs when the median nerve is compressed in the wrist as it passes through the carpal tunnel (Fig. 17.8). It is a common condition, especially in women, between the ages of 30 and 50 years. There is pain and numbness in the hand and wrist affecting the thumb, index and middle fingers, and half of the ring finger. Many cases are idiopathic or secondary to other conditions, e.g. rheumatoid arthritis, diabetes mellitus, acromegaly and hypothyroidism. Repetitive flexion and extension of the wrist joint also cause the condition, e.g. following prolonged keyboard use.

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18

The muscular system

The three types of muscle tissue, their features and the nomenclature of skeletal muscles are described on page 40.

This chapter considers the skeletal muscles not involved in the movements of the joints of the limbs:

- muscles of the face and neck
- muscles of the back
- muscles of the abdominal wall
- muscles of the pelvic floor.

Muscles of respiration are described on page 252. The muscles that move the joints are described in Chapter 17.

MUSCLES OF THE FACE AND NECK (Fig. 18.1)

Learning outcomes

After studying this section you should be able to:

- name the main muscles of the face and neck
- outline the functions of the main muscles of the face and neck.

Muscles of the face

There are many muscles involved in changing facial expression and with movement of the lower jaw during chewing and speaking. Only the main muscles are described here. Except where indicated the muscles are present in pairs, one on each side.

Occipitofrontalis (unpaired). This consists of a posterior muscular part over the occipital bone (*occipitalis*), an anterior part over the frontal bone (*frontalis*) and an extensive flat tendon or *aponeurosis* that stretches over the dome of the skull and joins the two muscular parts. It raises the eyebrows.

Levator palpebrae superioris. This muscle extends from the posterior part of the orbital cavity to the upper eyelid. It raises the eyelid.

Orbicularis oculi. This muscle surrounds the eye, eyelid and orbital cavity. It closes the eye and when strongly contracted 'screws up' the eyes.

Buccinator. This flat muscle of the cheek draws the cheeks in towards the teeth in chewing and in forcible expulsion of air from the mouth ('the trumpeter's muscle').

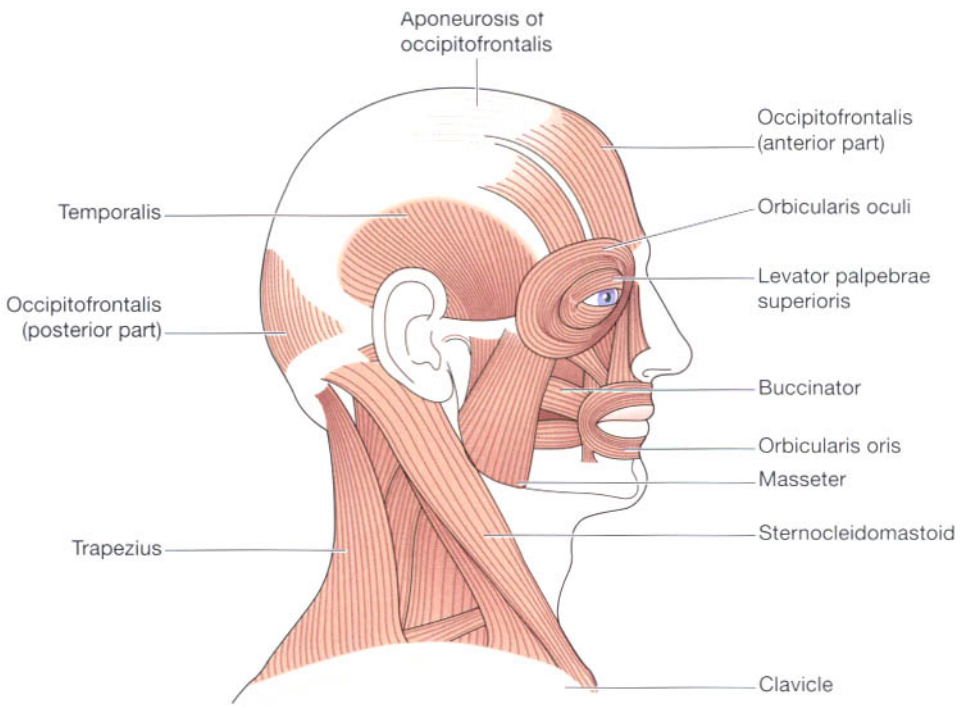


Figure 18.1 The main muscles on the right side of the face, head and neck.

Orbicularis oris (unpaired). This muscle surrounds the mouth and blends with the muscles of the cheeks. It closes the lips and, when strongly contracted, shapes the mouth for whistling.

Masseter. This is a broad muscle, extending from the zygomatic arch to the angle of the jaw. In chewing it draws the mandible up to the maxilla, closing the jaw, and exerts considerable pressure on the food.

Temporalis. This muscle covers the squamous part of the temporal bone. It passes behind the zygomatic arch to be inserted into the coronoid process of the mandible. It closes the mouth and assists with chewing.

Pterygoid. This muscle extends from the sphenoid bone to the mandible. It closes the mouth and pulls the lower jaw forward.

Muscles of the neck

There are many muscles situated in the neck but only the two largest are considered here.

Sternocleidomastoid. This muscle arises from the manubrium of the sternum and the clavicle and extends upwards to the mastoid process of the temporal bone. It assists in turning the head from side to side. When the muscle on one side contracts it draws the head towards the shoulder. When both contract at the same time they flex the cervical vertebrae or draw the sternum and clavicles upwards when the head is maintained in a fixed position, e.g. in forced respiration.

Trapezius. This muscle covers the shoulder and the back of the neck. The upper attachment is to the occipital protuberance, the medial attachment is to the transverse processes of the cervical and thoracic vertebrae and the lateral attachment is to the clavicle and to the spinous and acromion processes of the scapula. It pulls the head backwards, squares the shoulders and controls the movements of the scapula when the shoulder joint is in use.

MUSCLES OF THE BACK (Fig. 18.2)

Learning outcomes

After studying this section you should be able to:

- name the main muscles of the back
- outline the functions of the main muscles of the back.

There are six pairs of large muscles in the back in addition to those that form the posterior abdominal wall. The arrangement of these muscles is the same on each side of the vertebral column. They are:

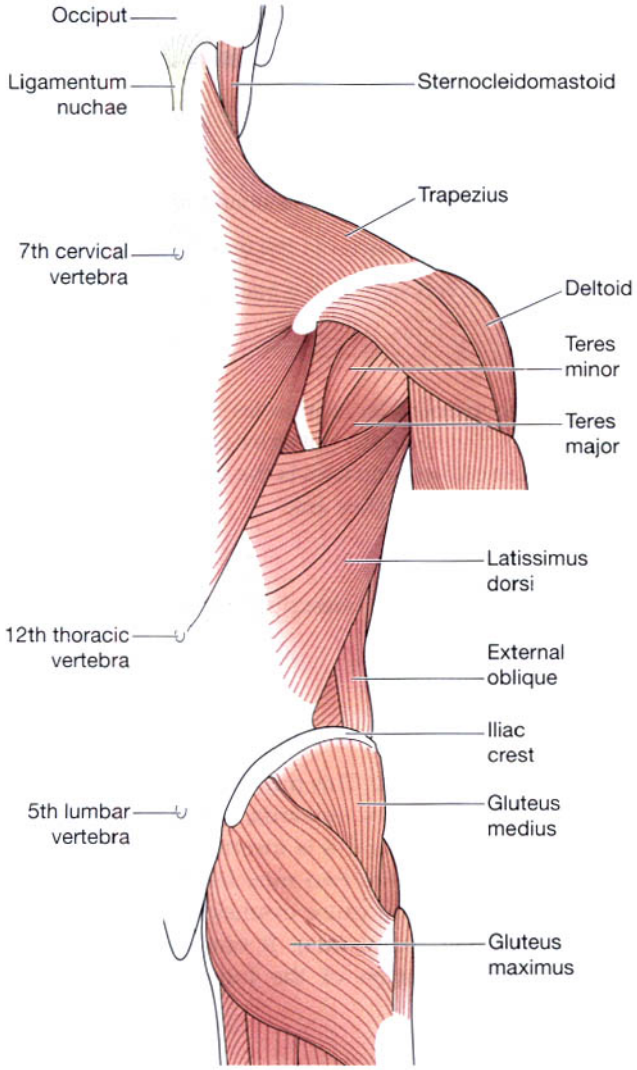
- trapezius
- teres major
- psoas
- latissimus dorsi

} described in Chapter 17

- quadratus lumborum
- sacrospinalis.

Quadratus lumborum. This muscle originates from the iliac crest then it passes upwards, parallel and close to the vertebral column and it is inserted into the 12th rib (Fig. 18.5). Together the two muscles fix the lower rib during respiration and cause extension of the vertebral column (bending backwards). If one muscle contracts it causes lateral flexion of the lumbar region of the vertebral column.

Sacrospinalis (erector spinae). This is a group of muscles lying between the spinous and transverse processes of the vertebrae (Fig. 18.6). They originate from the sacrum and are finally inserted into the occipital bone. Their contraction causes extension of the vertebral column.



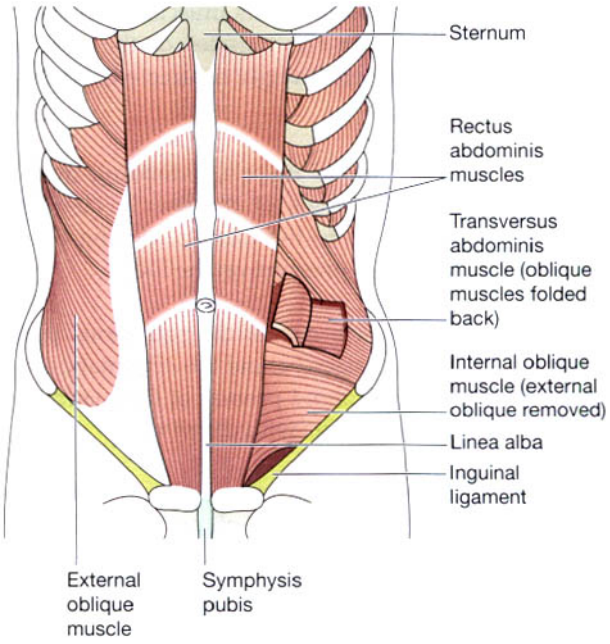


Figure 18.3 The muscles of the anterior abdominal wall.

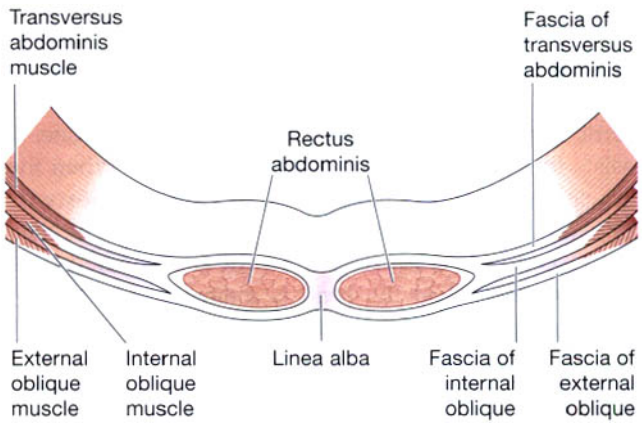


Figure 18.4 Transverse section of the muscles and fasciae of the anterior abdominal wall.

MUSCLES OF THE ABDOMINAL WALL (Figs 18.3, 18.4 and 18.5)

Learning outcomes

After studying this section you should be able to:

- name the main muscles of the abdominal wall
- outline the functions of the main muscles of the abdominal wall.

There are six pairs of muscles that form the abdominal wall. From the surface inwards they are:

- rectus abdominis
- external oblique
- internal oblique
- transversus abdominis
- quadratus lumborum
- psoas – described in Chapter 17.

The anterior abdominal wall is divided longitudinally by a very strong midline tendinous cord, the *linea alba* (meaning 'white cord') which extends from the xiphoid

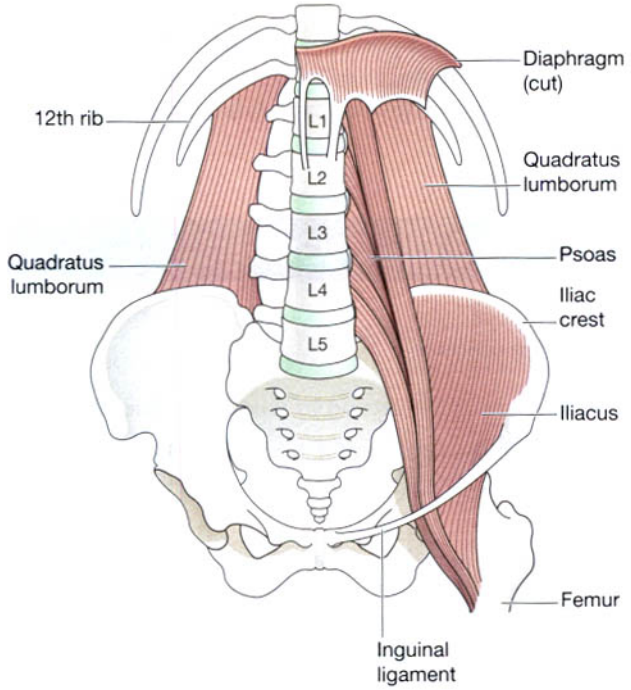


Figure 18.5 The deep muscles of the posterior abdominal wall.

process of the sternum to the symphysis pubis. The structure of the abdominal wall on each side of the linea alba is identical.

Rectus abdominis. This is the most superficial muscle. It is broad and flat, originating from the transverse part of the pubic bone then passing upwards to be inserted into the lower ribs and the xiphoid process of the sternum. Medially the two muscles are attached to the linea alba.

External oblique. This muscle extends from the lower ribs downwards and forward to be inserted into the iliac crest and, by an aponeurosis, to the linea alba.

Internal oblique. This muscle lies deep to the external oblique. Its fibres arise from the iliac crest and by a broad band of fascia from the spinous processes of the lumbar vertebrae. The fibres pass upwards towards the midline to be inserted into the lower ribs and, by an aponeurosis, into the linea alba. The fibres are at right angles to those of the external oblique.

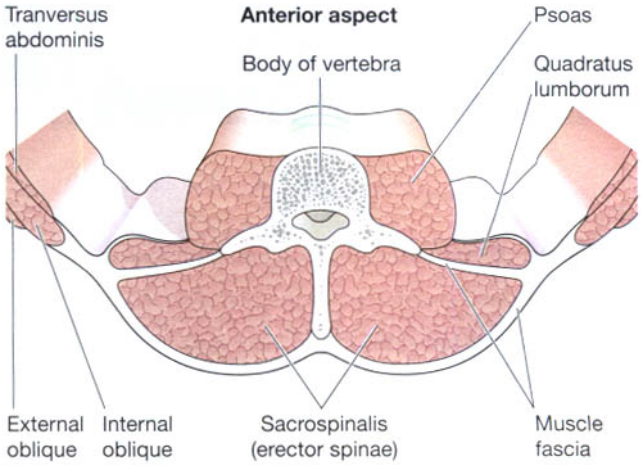


Figure 18.6 Transverse section of the posterior abdominal wall: a lumbar vertebra and its associated muscles.

Transversus abdominis. This is the deepest muscle of the abdominal wall. The fibres arise from the iliac crest and the lumbar vertebrae and pass across the abdominal wall to be inserted into the linea alba by an aponeurosis. The fibres are at right angles to those of the rectus abdominis.

Functions

The main function of the four pairs of muscles is to form the strong muscular anterior wall of the abdominal cavity. When the muscles contract together they:

- compress the abdominal organs
- flex the vertebral column in the lumbar region (Fig. 18.6).

Contraction of the muscles on one side only bends the trunk towards that side. Contraction of the oblique muscles on one side rotates the trunk.

Inguinal canal

This canal is 2.5 to 4 cm long and passes obliquely through the abdominal wall. It runs parallel to and immediately in front of the transversalis fascia and part of the inguinal ligament (Fig. 18.5). In the male it contains the *spermatic cord* and in the female, the *round ligament*. It constitutes a weak point in the otherwise strong abdominal wall through which herniation may occur (see p. 330).

MUSCLES OF THE PELVIC FLOOR
(Fig. 18.7)

Learning outcomes

After studying this section you should be able to:

- name the main muscles of the pelvic floor
- outline the functions of the main muscles of the pelvic floor.

The pelvic floor is divided into two identical halves that unite along the midline. Each half consists of fascia and muscle. The muscles are:

- levator ani
- coccygeus.

Levator ani. This is a broad flat muscle, forming the anterior part of the pelvic floor. They originate from the inner surface of the true pelvis and unite in the midline. Together they form a sling which supports the pelvic organs.

Coccygeus. This is a triangular sheet of muscle and tendinous fibres situated behind the levator ani. They originate from the medial surface of the ischium and are inserted into the sacrum and coccyx. They complete the formation of the pelvic floor which is perforated in the male by the urethra and anus, and in the female by the urethra, vagina and anus.

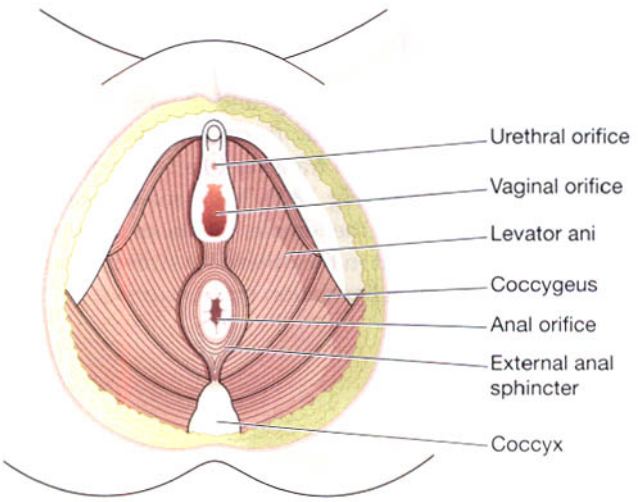


Figure 18.7 The muscles of the female pelvic floor.

Functions

The pelvic floor supports the organs of the pelvis and maintains continence, i.e. it resists raised intrapelvic pressure during micturition and defaecation.

HEALING OF MUSCLE

Learning outcome

After studying this section you should be able to:

- describe the healing of damaged muscle.

Muscle fibres may be damaged accidentally or be cut during surgery. The extent of the damage determines the mode and effectiveness of healing. In all cases, damaged tissue is removed by phagocytosis and replaced by granulation tissue.

- In *slight injury* the small gap in the muscle fibre is bridged by outgrowths from the surviving ends of the fibre, completely restoring its integrity.
- In *more extensive injury* the muscle fibre outgrowths may not be able to extend far enough into the granulation tissue to restore it completely. When this happens the remaining granulation tissue becomes fibrosed and scar tissue forms. In time this contracts and may restrict joint movement.
- In *very extensive injury* repair is by fibrosis. In *crush syndrome* (p. 436) there may also be serious systemic effects.

REPAIR OF NERVES SUPPLYING MUSCLES

Learning outcome

After studying this section you should be able to:

- describe how the nerve supply to muscles may be restored following injury.

A *motor unit* consists of a lower motor neurone (LMN) and the muscle fibres it supplies. When the nerve supply is cut the muscle cannot contract and gradually atrophies

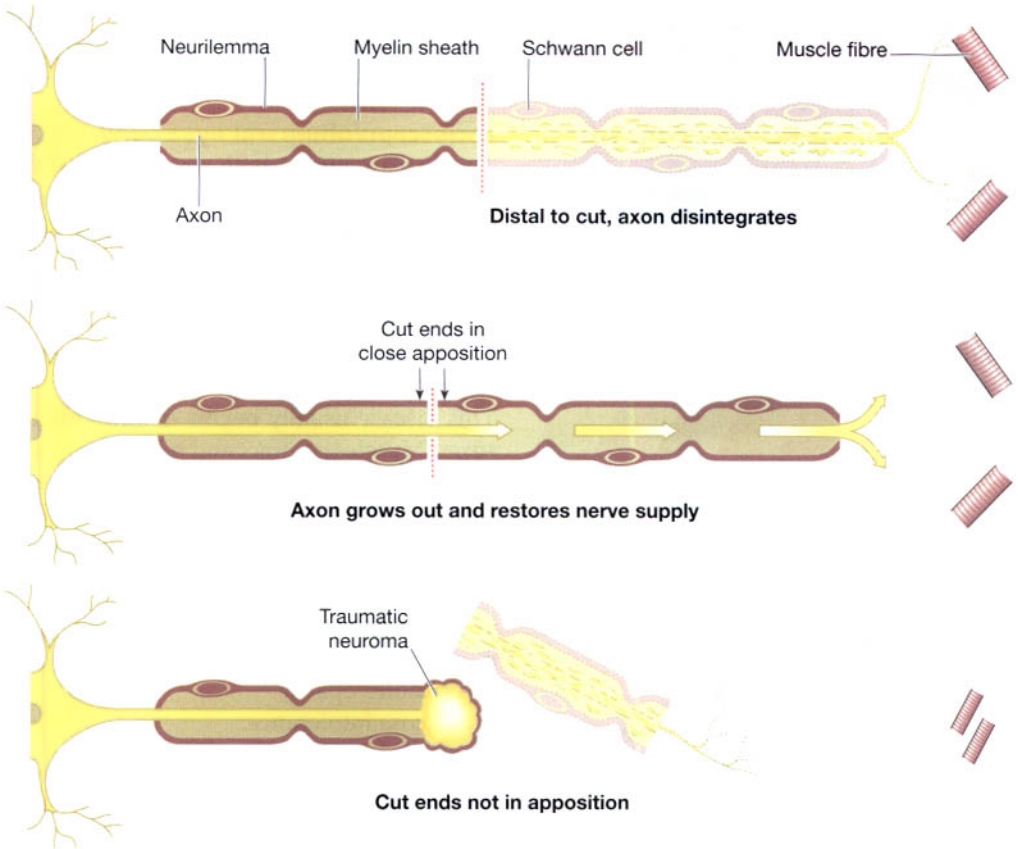


Figure 18.8 Regrowth of a peripheral nerve.

but, if the nerve regenerates, muscle fibre function is restored. The axon of the LMN in a peripheral nerve divides into numerous terminal branches, each of which supplies a muscle fibre. In a bundle of muscle fibres the nerve supply is derived from several LMNs (see Fig. 7.10, p. 147). Nerve supply to muscle fibres may be restored by:

- regeneration of the nerve if cut near the parent cell and if the cut ends are in close apposition (Fig. 18.8)
- the outgrowth of new terminal nerve fibres from the other axons supplying adjacent muscle fibres in the same bundle (Fig. 18.9).

When the neurilemma is out of position or destroyed a traumatic neuroma develops in which there is sprouting of axons and Schwann cells.

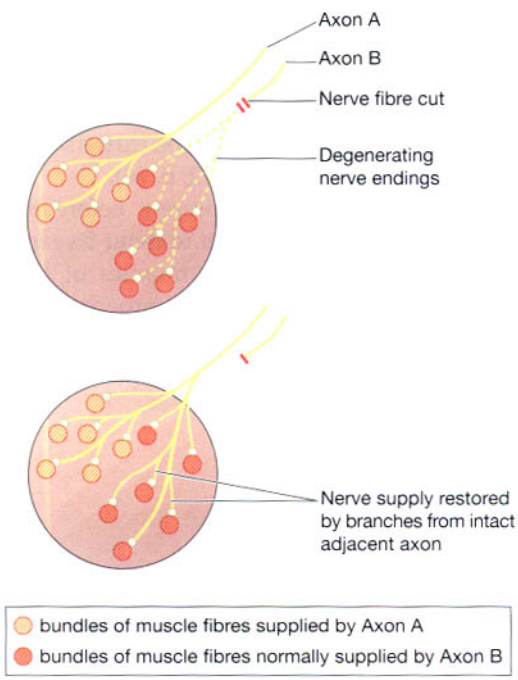


Figure 18.9 Restoration of nerve supply to muscle.

DISEASES OF MUSCLES

Learning outcomes

After studying this section you should be able to:

- list the causes of the diseases in this section
- compare and contrast the characteristics of different types of muscular dystrophy
- describe the effects of crush syndrome.

Myasthenia gravis

See page 385.

Myopathies

Muscular dystrophies

In this group of inherited diseases there is progressive degeneration of groups of muscles. The main differences in the types are:

- age of onset
- rate of progression
- groups of muscles involved.

Duchenne muscular dystrophy

Inheritance of this condition is sex linked, the affected gene being carried on the long X chromosome of female carriers. Their children may be affected by the condition if they are males (50% chance), or be carriers if they are females (50% chance) (see Fig. 4.12, p. 76).

The muscle abnormality is present before birth but may not be evident until the child is about 5 years of age. Wasting and weakness begin in muscles of the lower limbs then spread to the upper limbs, progressing rapidly without remission. Death usually occurs in adolescence, often from respiratory failure, cardiac arrhythmias or cardiomyopathy.

Facioscapulohumeral dystrophy

This disease affects both sexes. It usually begins in adolescence and the younger the age of onset the more rapidly it progresses. Muscles of the face and shoulders are affected first. This is a chronic condition that usually progresses slowly and may not cause complete disability. Life expectancy is normal.

Myotonic dystrophy

This disease usually begins in adult life and affects both sexes. Muscles contract and relax slowly, often seen as difficulty in releasing an object held in the hand. Muscles of the tongue and the face are first affected then muscles of the limbs. Systemic conditions associated with myotonic dystrophy include:

- cataracts
- atrophy of the gonads
- cardiomyopathy
- glucose intolerance.

The disease progresses without remission and with increasing disability. Death usually occurs in middle age from respiratory or cardiac failure.

Crush syndrome

Sustained pressure, on the trunk or a limb, causes ischaemia resulting in massive muscle necrosis. When pressure is relieved and the circulation is restored, myoglobin and other necrotic products are released from damaged muscle and enter the blood. This material is highly toxic to the kidneys and acute renal failure may develop. A common complication of this type of injury is infection, especially by anaerobic microbes, e.g. *Clostridium perfringens (Cl. welchii)* and other clostridia causing *gas gangrene*.

Healing of such extensive injury is by fibrosis.

19

The reproductive systems

The ability to reproduce is one of the properties which distinguishes living from non-living matter. The more primitive the animal, the simpler the process of reproduction. In human beings the process is one of sexual reproduction in which the male and female organs differ anatomically and physiologically.

Both males and females produce specialised reproductive germ cells, called *gametes*. The male gametes are called *spermatozoa* and the female gametes are called *ova*. They contain the genetic material, or *genes*, on *chromosomes*, which pass inherited characteristics on to the next generation. In other body cells there are 46 chromosomes arranged in 23 pairs but in the gametes there are only 23, one from each pair. Gametes are formed by *meiosis* (p. 33). When the ovum is fertilised by a spermatozoon the resultant *zygote* contains 23 *pairs* of chromosomes, one of each pair obtained from the father and one from the mother.

The zygote embeds itself in the wall of the uterus where it grows and develops during the 40-week *gestation period* before birth.

The functions of the female reproductive system are:

- formation of female gametes, ova
- reception of male gametes, spermatozoa
- provision of suitable environments for fertilisation of the ovum by spermatozoa and development of the resultant fetus
- parturition (childbirth)
- lactation, the production of breast milk, which provides complete nourishment for the baby in its early life.

The functions of the male reproductive system are:

- production of male gametes, spermatozoa
- transmission of spermatozoa to the female.

FEMALE REPRODUCTIVE SYSTEM

Learning outcomes

After studying this section, you should be able to:

- describe the main structures comprising the external genitalia
- explain the structure and function of the vagina
- describe the location, structure and function of the uterus and the uterine tubes
- discuss the process of ovulation and the hormones that control it
- outline the changes that occur in the female at puberty, including the physiology of menstruation
- describe the structure and function of the female breast.

The female reproductive organs, or genitalia, are divided into external and internal organs (Fig. 19.1).

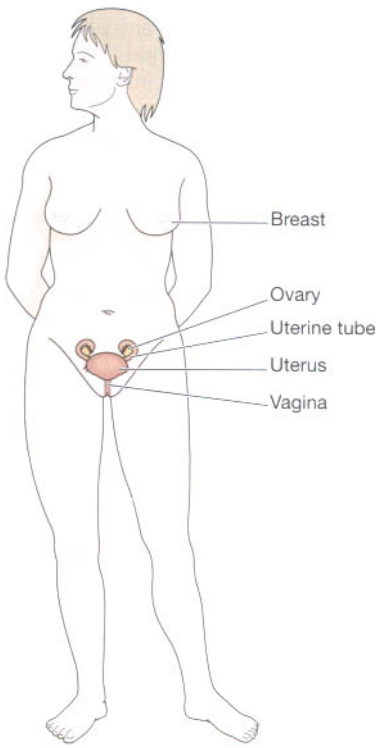
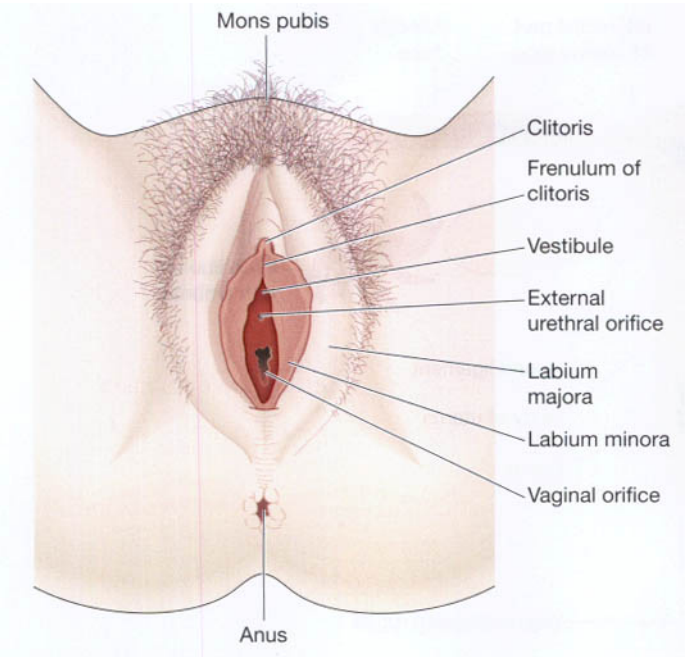


Figure 19.1 The female reproductive organs. Faint lines indicate the positions of the lower ribs and the pelvis.



External genitalia (vulva)

The external genitalia (Fig. 19.2) are known collectively as the vulva, and consist of the labia majora and labia minora, the clitoris, the vaginal orifice, the vestibule, the hymen and the vestibular glands (Bartholin's glands).

Labia majora

These are the two large folds which form the boundary of the vulva. They are composed of skin, fibrous tissue and fat and contain large numbers of sebaceous glands. Anteriorly the folds join in front of the symphysis pubis, and posteriorly they merge with the skin of the perineum. At puberty hair grows on the mons pubis and on the lateral surfaces of the labia majora.

Labia minora

These are two smaller folds of skin between the labia majora, containing numerous sebaceous glands.

The cleft between the labia minora is the *vestibule*. The vagina, urethra and ducts of the greater vestibular glands open into the vestibule.

Clitoris

The clitoris corresponds to the penis in the male and contains sensory nerve endings and erectile tissue but it has no reproductive significance.

Hymen

The hymen is a thin layer of mucous membrane which partially occludes the opening of the vagina. It is normally incomplete to allow for passage of menstrual flow.

Vestibular glands

The vestibular glands (Bartholin's glands) are situated one on each side near the vaginal opening. They are about the size of a small pea and have ducts, opening into the vestibule immediately lateral to the attachment of the hymen. They secrete mucus that keeps the vulva moist.

Blood supply, lymph drainage and nerve supply

The arterial supply. This is by branches from the *internal pudendal arteries* that branch from the internal iliac arteries and by *external pudendal arteries* that branch from the femoral arteries.

The venous drainage. This forms a large plexus which eventually drains into the internal iliac veins.

Lymph drainage. This is through the superficial inguinal nodes.

Nerve supply. This is by branches from pudendal nerves.

Perineum

The perineum is the area extending from the base of the labia minora to the anal canal. It is roughly triangular and consists of connective tissue, muscle and fat. It gives attachment to the muscles of the pelvic floor (p. 434).

Internal genitalia

The internal organs of the female reproductive system (Figs 19.3 and 19.4) lie in the pelvic cavity and consist of the vagina, uterus, two uterine tubes and two ovaries.

Vagina

The vagina is a fibromuscular tube lined with stratified squamous epithelium, connecting the external and internal organs of reproduction. It runs obliquely upwards and backwards at an angle of about 45° between the bladder in front and rectum and anus behind. In the adult the anterior wall is about 7.5 cm (3 inches) long and the posterior wall about 9 cm long. The difference is due to the angle of insertion of the cervix through the anterior wall.

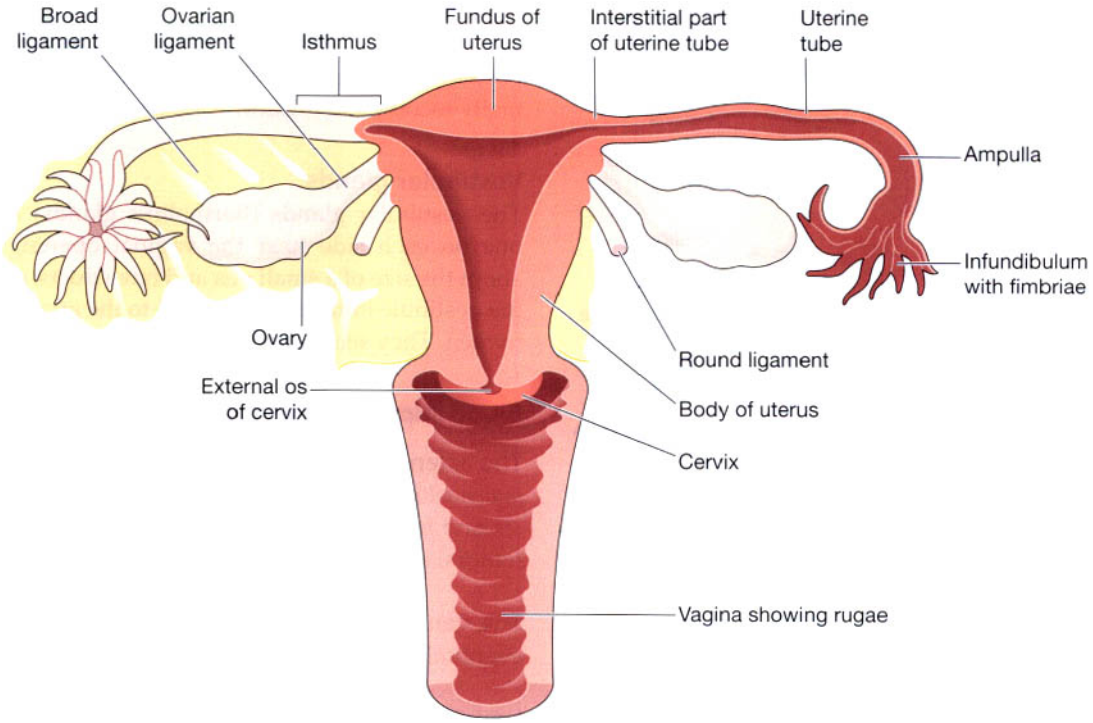


Figure 19.3 The female reproductive organs in the pelvis.

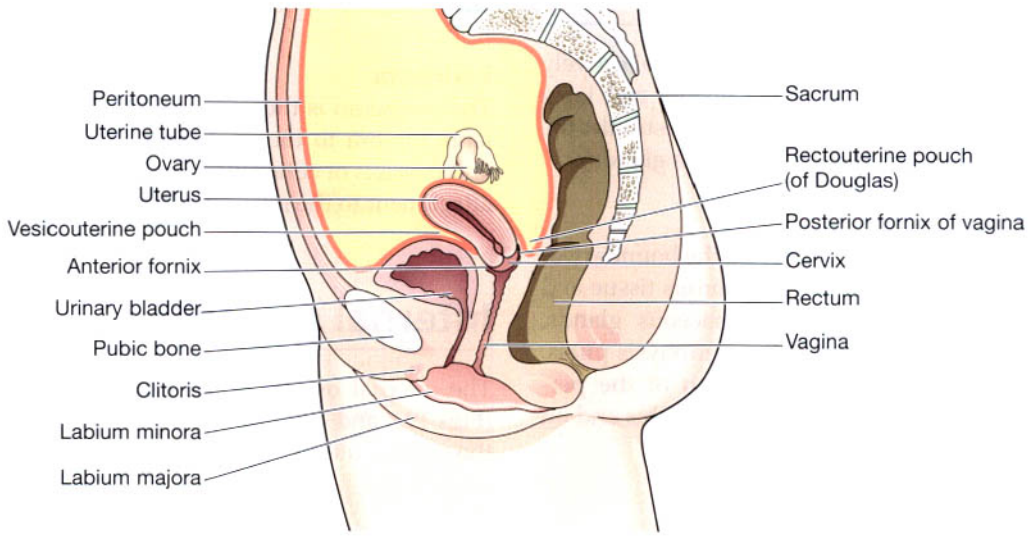


Figure 19.4 Lateral view of the female reproductive organs in the pelvis and their associated structures.

Structure of the vagina

The vagina has three layers: an outer covering of areolar tissue, a middle layer of smooth muscle and an inner lining of stratified squamous epithelium that forms ridges or *rugae*. It has no secretory glands but the surface is kept moist by cervical secretions. Between puberty and the menopause, *Lactobacillus acidophilus* bacteria are normally present, which secrete *lactic acid*, maintaining the pH between 4.9 and 3.5. The acidity inhibits the growth of most other microbes that may enter the vagina from the perineum.

Blood supply, lymph drainage and nerve supply

Arterial supply. An arterial plexus is formed round the vagina, derived from the uterine and vaginal arteries which are branches of the internal iliac arteries.

Venous drainage. A venous plexus, situated in the muscular wall, drains into the internal iliac veins.

Lymph drainage. This is through the deep and superficial iliac glands.

Nerve supply. This consists of parasympathetic fibres from the sacral outflow, sympathetic fibres from the lumbar outflow and somatic sensory fibres from the pudendal nerves.

Functions of the vagina

The vagina acts as the receptacle for the penis during coitus, and provides an elastic passageway through which the baby passes during childbirth.

Uterus

The uterus is a hollow muscular pear-shaped organ, flattened anteroposteriorly. It lies in the pelvic cavity between the urinary bladder and the rectum (Fig. 19.4).

In most women, it leans forward (*anteversion*), and is bent forward (*anteflexion*) almost at right angles to the vagina, so that its anterior wall rests partly against the bladder below, and forming the vesicouterine pouch between the two organs.

When the body is in the upright position the uterus lies in an almost horizontal position. It is about 7.5 cm long, 5 cm wide and its walls are about 2.5 cm thick. It weighs from 30 to 40 grams. The parts of the uterus are the fundus, body and cervix (Fig. 19.3).

The fundus. This is the dome-shaped part of the uterus above the openings of the uterine tubes.

The body. This is the main part. It is narrowest inferiorly at the *internal os* where it is continuous with the cervix.

The cervix ('neck' of the uterus). This protrudes through the anterior wall of the vagina, opening into it at the *external os*.

Structure of the uterus

The walls of the uterus are composed of three layers of tissue: perimetrium, myometrium and endometrium (Fig. 19.5).

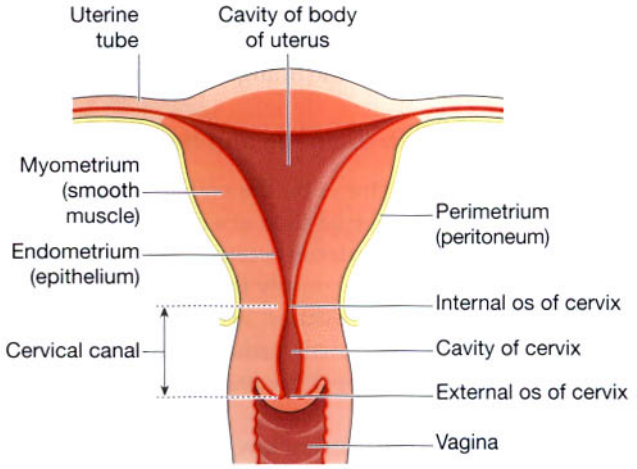


Figure 19.5 A section of the uterus.

Perimetrium

This is peritoneum, which is distributed differently on the various surfaces of the uterus (Fig. 19.4).

Anteriorly it extends over the fundus and the body where it is folded on to the upper surface of the urinary bladder. This fold of peritoneum forms the *vesicouterine pouch*.

Posteriorly the peritoneum extends over the fundus, the body and the cervix, then it continues on to the rectum to form the *rectouterine pouch* (of Douglas).

Laterally only the fundus is covered because the peritoneum forms a double fold with the uterine tubes in the upper free border. This double fold is the *broad ligament* which, at its lateral ends, attaches the uterus to the sides of the pelvis.

Myometrium

This is the thickest layer of tissue in the uterine wall. It is a mass of smooth muscle fibres interlaced with areolar tissue, blood vessels and nerves.

Endometrium

This consists of columnar epithelium containing a large number of mucus-secreting tubular glands. It is divided functionally into two layers.

- The functional layer is the upper layer and it thickens and becomes rich in blood vessels in the first half of the menstrual cycle. If the ovum is not fertilised and does not implant, this layer is shed during menstruation.
- The basal layer lies next to the myometrium, and is not lost during menstruation. It is the layer from which the fresh functional layer is regenerated during each cycle.

The upper two-thirds of the cervical canal is lined with this mucous membrane.

Further towards the vagina, however, the mucosa changes, becoming stratified squamous epithelium, which is continuous with the lining of the vagina itself.

Blood supply, lymph drainage and nerve supply

The arterial supply. This is by the *uterine arteries* which are branches of the internal iliac arteries. They pass up the lateral aspects of the uterus between the two layers of the broad ligaments. They supply the uterus and uterine tubes and join with the ovarian arteries to supply the ovaries. Branches pass downwards to anastomose with the vaginal arteries to supply the vagina.

Venous drainage. The veins follow the same route as the arteries and eventually drain into the internal iliac veins.

Lymph drainage. There are deep and superficial lymph vessels which drain lymph from the uterus and the uterine tubes to the aortic lymph nodes and groups of nodes associated with the iliac blood vessels.

Nerve supply. The nerves supplying the uterus and the uterine tubes consist of parasympathetic fibres from the sacral outflow and sympathetic fibres from the lumbar outflow.

Supports of the uterus

The uterus is supported in the pelvic cavity by surrounding organs, muscles of the pelvic floor and ligaments that suspend it from the walls of the pelvis.

Supporting structures (Fig. 19.6)

The broad ligaments. These are formed by a double fold of peritoneum, one on each side of the uterus. They hang down from the uterine tubes as though draped over them and at their lateral ends they are attached to the sides of the pelvis. The uterine tubes are enclosed in the upper free border and near the lateral ends they penetrate the posterior wall of the broad ligament and open into the peritoneal cavity. The ovaries are attached to the posterior wall, one on each side. Blood and lymph vessels and nerves pass to the uterus and uterine tubes between the layers of the broad ligaments.

The round ligaments. These are bands of fibrous tissue between the two layers of broad ligament, one on each side of the uterus. They pass to the sides of the pelvis then through the *inguinal canal* to end by fusing with the labia majora.

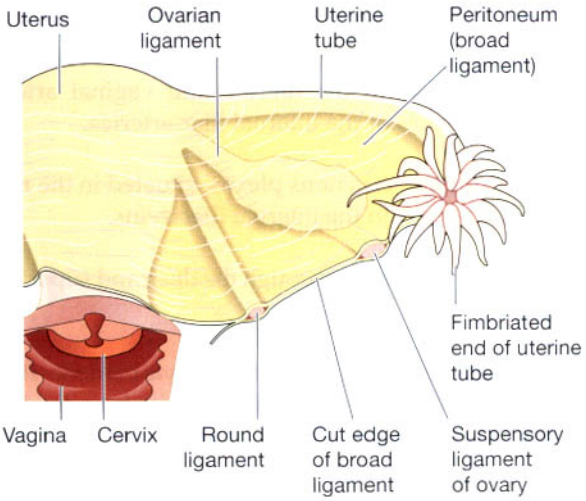


Figure 19.6 The main ligaments supporting the uterus. Only one side is shown.

The uterosacral ligaments. These originate from the posterior walls of the cervix and vagina and extend backwards, one on each side of the rectum, to the sacrum.

The transverse cervical ligaments (cardinal ligaments). These extend one from each side of the cervix and vagina to the side walls of the pelvis.

The pubocervical fascia. This extends forward from the transverse cervical ligaments on each side of the bladder and is attached to the posterior surface of the pubic bones.

Functions of the uterus

After puberty, the endometrium of the uterus goes through a regular monthly cycle of changes, the menstrual cycle, which is under the control of hypothalamic and anterior pituitary hormones (Ch. 9). The purpose of the cycle is to prepare the uterus to receive, nourish and protect a fertilised ovum. The cycle is usually regular, lasting between 26 and 30 days. If the ovum is not fertilised a new cycle begins with a short period of bleeding (menstruation).

If the ovum is fertilised the zygote embeds itself in the uterine wall. The uterine muscle grows to accommodate the developing baby, which is called an *embryo* during its first 8 weeks, and a *fetus* for the remainder of the pregnancy. Uterine secretions nourish the ovum before it implants in the endometrium, and after implantation the rapidly expanding ball of cells is nourished by the endometrial cells themselves. This is sufficient for only

the first few weeks and the *placenta* is the organ that takes over thereafter. The placenta, which is attached to the fetus by the umbilical cord, is firmly attached to the wall of the uterus, and provides the means by which the growing baby receives oxygen and nutrients, and gets rid of its wastes. During pregnancy, which normally lasts about 40 weeks, the muscular walls of the uterus are prevented from contracting and expelling the baby early by high levels of the hormone progesterone secreted by the placenta. At the end of pregnancy (at term) the hormone oestrogen, which increases uterine contractility, becomes the predominant sex hormone in the blood. Additionally, oxytocin is released from the posterior pituitary, and also stimulates the uterine muscle. Control of oxytocin release is by positive feedback (see also Fig. 9.5, p. 219). During labour, the uterus forcefully expels the baby by means of powerful rhythmical contractions.

Uterine tubes (Fallopian tubes)

The uterine tubes (Fig. 19.3) are about 10 cm long and extend from the sides of the uterus between the body and the fundus. They lie in the upper free border of the broad ligament and their trumpet-shaped lateral ends penetrate the posterior wall, opening into the peritoneal cavity close to the ovaries. The end of each tube has fingerlike projections called *fimbriae*. The longest of these is the *ovarian fimbria* which is in close association with the ovary.

Structure of the uterine tubes

The uterine tubes have an outer covering of peritoneum (broad ligament), a middle layer of smooth muscle and are lined with ciliated epithelium.

Blood supply, lymph drainage and nerve supply

These are the same as for the uterus.

Function of the uterine tubes

The uterine tubes convey the ovum from the ovary to the uterus by peristalsis and ciliary movement. The mucus secreted by the lining membrane provides ideal conditions for movement of ova and spermatozoa. Fertilisation of the ovum usually takes place in the uterine tube, and the zygote is propelled into the uterus for implantation.

Ovaries

The ovaries (Fig. 19.4) are the female gonads, or glands, and they lie in a shallow fossa on the lateral walls of the pelvis. They are 2.5 to 3.5 cm long, 2 cm wide and 1 cm thick. Each is attached to the upper part of the uterus by the *ovarian ligament* and to the back of the broad ligament by a broad band of tissue, the *mesovarium*. Blood vessels and nerves pass to the ovary through the mesovarium (Fig. 19.7).

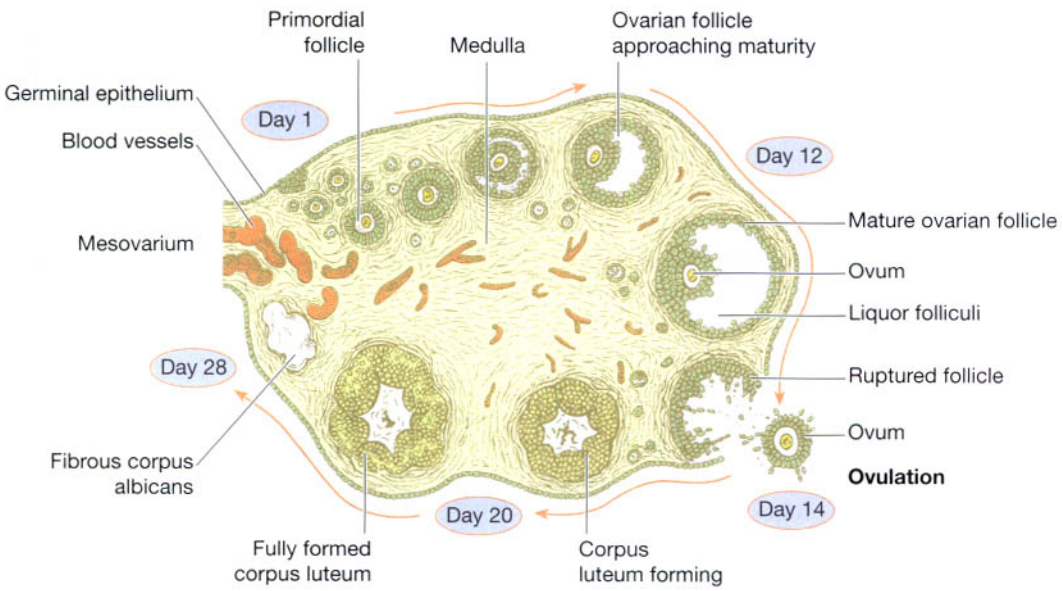


Figure 19.7 A section of an ovary showing the stages of development of one ovarian follicle.

Structure of the ovaries

The ovaries have two layers of tissue.

The medulla. This lies in the centre and consists of fibrous tissue, blood vessels and nerves.

The cortex. This surrounds the medulla. It has a framework of connective tissue, or *stroma*, covered by *germinal epithelium*. It contains *ovarian follicles* in various stages of maturity, each of which contains an ovum. Before puberty the ovaries are inactive but the stroma already contains immature (primordial) follicles, which the female has from birth. During the childbearing years, about every 28 days, one ovarian follicle (Graafian follicle) matures, ruptures and releases its ovum into the peritoneal cavity. This is called *ovulation* and it occurs during most menstrual cycles (Fig. 19.7).

Blood supply, lymph drainage and nerve supply

Arterial supply. This is by the *ovarian arteries*, which branch from the abdominal aorta just below the renal arteries.

Venous drainage. This is into a plexus of veins behind the uterus from which the ovarian veins arise. The right ovarian vein opens into the inferior vena cava and the left into the left renal vein.

Lymph drainage. This is to the lateral aortic and preaortic lymph nodes. The lymph vessels follow the same route as the arteries.

Nerve supply. The ovaries are supplied by parasympathetic nerves from the sacral outflow and sympathetic nerves from the lumbar outflow. Their precise functions are not yet fully understood.

Functions of the ovaries

Maturation of the follicle is stimulated by follicle stimulating hormone (FSH) from the anterior pituitary, and oestrogen secreted by the follicle lining cells. Ovulation is triggered by a surge of luteinising hormone (LH) from the anterior pituitary, which occurs a few hours before ovulation. After ovulation, the follicle lining cells develop into the *corpus luteum* (yellow body), under the influence of LH from the anterior pituitary. The corpus luteum produces the hormone progesterone and some oestrogen. If the ovum is fertilised it embeds itself in the wall of the uterus where it grows and develops and produces the hormone *human chorionic gonadotrophin* (hCG), which stimulates the corpus luteum to continue secreting progesterone and oestrogen for the first 3 months of the pregnancy (Figs 19.8 and 19.9), after which time this function is continued by the placenta. If the ovum is not fertilised the corpus luteum degenerates and a new cycle begins with menstruation. At the site of the degenerate corpus luteum an inactive mass of fibrous tissue forms, called the *corpus albicans*. Sometimes more than one follicle matures at a time, releasing two or more ova in the same cycle. When this happens and the ova are fertilised the result is a multiple pregnancy.

Puberty in the female

Puberty is the age at which the internal reproductive organs reach maturity. This is called the *menarche*, and marks the beginning of the childbearing period. The ovaries are stimulated by the gonadotrophins from the anterior pituitary, follicle stimulating hormone and luteinising hormone.

The age of puberty varies between 10 and 14 years and a number of physical and psychological changes take place at this time:

- the uterus, the uterine tubes and the ovaries reach maturity
- the menstrual cycle and ovulation begin (menarche)
- the breasts develop and enlarge
- pubic and axillary hair begins to grow

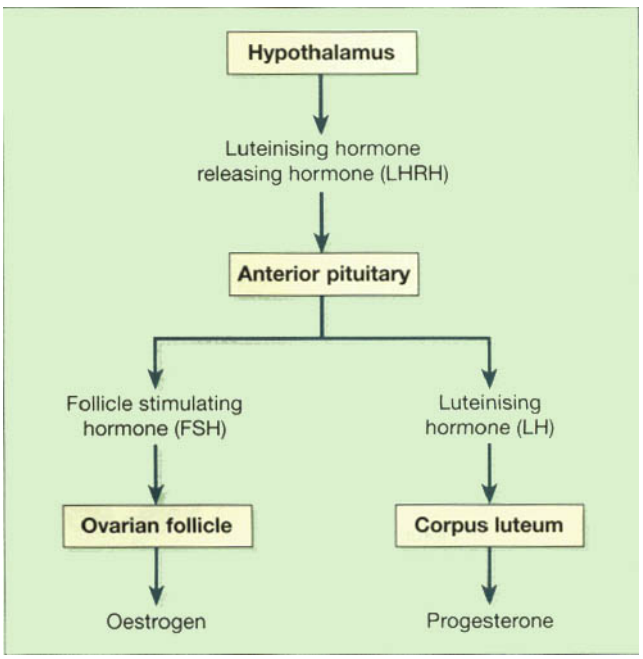


Figure 19.8 Female reproductive hormones and target tissues.

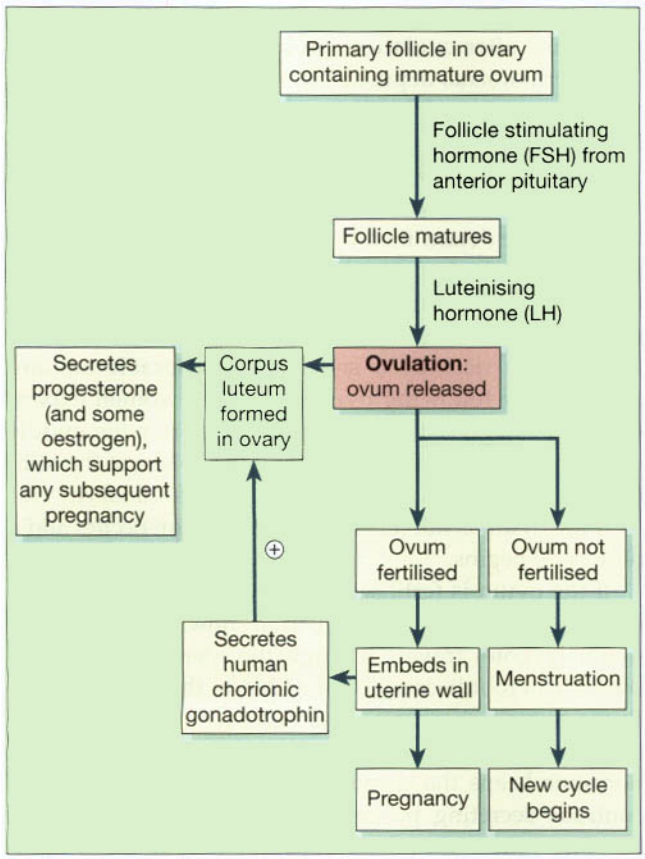


Figure 19.9 Summary of the stages of development of the ovum and the associated hormones.

- there is an increase in the rate of growth in height and widening of the pelvis
- there is an increase in the amount of fat deposited in the subcutaneous tissue, especially at the hips and breasts.

The menstrual (sexual) cycle

This is a series of events, occurring regularly in females every 26 to 30 days throughout the childbearing period of about 36 years (Fig. 19.10). The cycle consists of a series of changes that take place concurrently in the ovaries and uterine walls, stimulated by changes in the blood concentrations of hormones (Fig. 19.10B and D). Hormones secreted in the cycle are regulated by negative feedback mechanisms.

The hypothalamus secretes luteinising hormone releasing hormone (LHRH) which stimulates the anterior pituitary to secrete (see Table 9.1, p. 216):

- follicle stimulating hormone (FSH), which promotes the maturation of ovarian follicles and the secretion of oestrogen, leading to ovulation

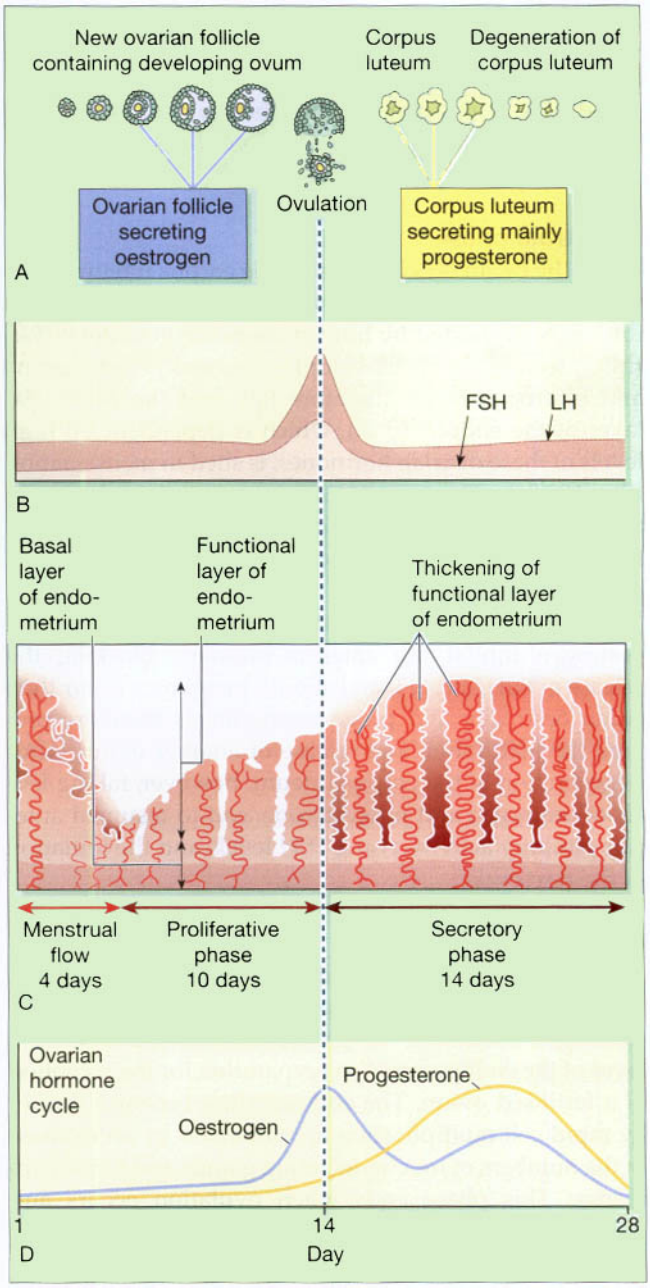


Figure 19.10 Summary of one female menstrual cycle: A. Ovarian cycle; maturation of follicle and development of corpus luteum. B. Anterior pituitary cycle; LH and FSH levels. C. Uterine cycle; menstrual, proliferative and secretory phases. D. Ovarian hormone cycle; oestrogen and progesterone levels.

- luteinising hormone (LH), which triggers ovulation, stimulates the development of the corpus luteum and the secretion of progesterone.

The hypothalamus responds to changes in the blood levels of oestrogen and progesterone. It is switched off by high levels and stimulated when they are low.

The average length of the menstrual cycle is about 28 days. By convention the days of the cycle are numbered from the beginning of the *menstrual phase* of the menstrual cycle which usually lasts about 4 days. This is followed by the *proliferative phase* (about 10 days), then by the *secretory phase* (about 14 days).

Menstrual phase

When the ovum is not fertilised, the corpus luteum starts to degenerate. (In the event of pregnancy, the corpus luteum is supported by human chorionic gonadotrophin (hCG) secreted by the developing embryo.) Progesterone and oestrogen levels therefore fall, and the functional layer of the endometrium, which is dependent on high levels of these ovarian hormones, is shed in menstruation (Fig. 19.10C). The menstrual flow consists of the secretions from endometrial glands, endometrial cells, blood from the broken down capillaries and the unfertilised ovum.

High circulating levels of ovarian progesterone and oestrogen inhibit the anterior pituitary, blocking the release of FSH and LH, and should pregnancy occur then rising oestrogen and progesterone levels therefore prevent the maturation and release of another ovum. After degeneration of the corpus luteum, however, falling levels of oestrogen and progesterone lead to resumed anterior pituitary activity, rising FSH levels and the initiation of the next cycle.

Proliferative phase

At this stage an ovarian follicle, stimulated by FSH, is growing towards maturity and is producing oestrogen. Oestrogen stimulates the proliferation of the functional layer of the endometrium in preparation for the reception of a fertilised ovum. The endometrium becomes thicker by rapid cell multiplication accompanied by an increase in the numbers of mucus-secreting glands and blood capillaries. This phase ends when ovulation occurs and oestrogen production declines.

Secretory phase

Immediately after ovulation, the lining cells of the ovarian follicle are stimulated by LH to develop the corpus luteum, which produces progesterone and some oestrogen. Under the influence of progesterone the endometrium becomes oedematous and the secretory glands produce increased amounts of watery mucus. This is believed to assist the passage of the spermatozoa through the uterus to the uterine tubes where the ovum is usually fertilised. There is a similar increase in the secretion of watery mucus by the glands of the uterine tubes and by cervical glands which lubricate the vagina.

The ovum may survive in a fertilisable form for a very short time after ovulation, probably as little as 8 hours. The spermatozoa, deposited in the vagina during coitus, may be capable of fertilising the ovum for only about 24 hours although they may survive for several days. This means that the period in each cycle during which fertilisation can occur is relatively short. The time of ovulation can be determined by observing certain changes in the woman's body around this period. Changes in cervical mucus, from thick and dry in consistency to thin, elastic and watery, are detected and, in addition, body temperature rises by a small but measurable amount immediately following ovulation. Some women experience some degree of abdominal discomfort in the middle of the cycle, thought to correspond to rupture of the follicle and release of its contents into the abdominal cavity.

If the ovum is not fertilised menstruation occurs and a new cycle begins.

If the ovum is fertilised there is no breakdown of the endometrium and no menstrual flow. The fertilised ovum (zygote) travels through the uterine tube to the uterus where it becomes embedded in the wall and produces human chorionic gonadotrophin (hCG), which is similar to anterior pituitary luteinising hormone. This hormone keeps the corpus luteum intact, enabling it to continue secreting progesterone and oestrogen for the first 3 to 4 months of the pregnancy, inhibiting the maturation of further ovarian follicles (Fig. 19.9). During that time the placenta develops and produces oestrogen, progesterone and gonadotrophins.

Menopause (climacteric)

The menopause usually occurs between the ages of 45 and 55 years, marking the end of the childbearing period. It may occur suddenly or over a period of years, sometimes as long as 10 years, and is caused by changes in sex hormone levels. The ovaries gradually become less responsive to FSH and LH, and ovulation and the menstrual cycle become irregular, eventually ceasing. Several other phenomena may occur at the same time including:

- short-term unpredictable vasodilatation with flushing, sweating and palpitations, causing discomfort and disturbance of the normal sleep pattern
- shrinkage of the breasts
- axillary and pubic hair become sparse
- atrophy of the sex organs
- episodes of uncharacteristic behaviour sometimes occur, e.g. irritability, mood changes
- gradual thinning of the skin

- loss of bone mass that predisposes to osteoporosis (p. 409)
- slow increase in blood cholesterol levels that predisposes postmenopausal women to cardiovascular disorders.

Similar changes occur after bilateral irradiation or surgical removal of the ovaries.

Breasts or mammary glands

The breasts or mammary glands are accessory glands of the female reproductive system. They exist also in the male but in only a rudimentary form.

In the female the breasts are small and immature until puberty. Thereafter they grow and develop to their mature size under the influence of oestrogen and progesterone. During pregnancy these hormones stimulate further growth. After the baby is born the hormone *prolactin* from the anterior pituitary stimulates the production of milk, and *oxytocin* from the posterior pituitary stimulates the release of milk in response to the stimulation of the nipple by the sucking baby, by a positive feedback mechanism.

Structure of the breast

The mammary glands (Fig. 19.11) consist of glandular tissue, fibrous tissue and fatty tissue.

Each breast consists of about 20 lobes of glandular tissue, each lobe being made up of a number of lobules that radiate around the nipple. The lobules consist of a cluster of alveoli which open into small ducts and these unite to form large excretory ducts, called *lactiferous ducts*. The lactiferous ducts converge towards the centre of the breast where they form dilatations or reservoirs for milk. Leading from each dilatation, or *lactiferous sinus*, is a narrow duct which opens on to the surface at the nipple. Fibrous tissue supports the glandular tissue and ducts, and fat covers the surface of the gland and is found between the lobes.

The nipple. This is a small conical eminence at the centre of the breast surrounded by a pigmented area, the *areola*. On the surface of the areola are numerous sebaceous glands (Montgomery's tubercles) which lubricate the nipple during lactation.

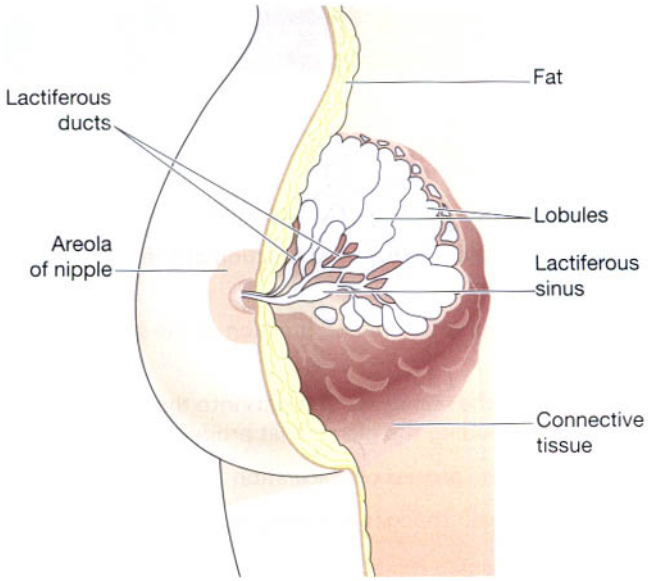


Figure 19.11 Structure of the breast.

Blood supply, lymph drainage and nerve supply

Arterial blood supply. The breasts are supplied with blood from the thoracic branches of the axillary arteries and from the internal mammary and intercostal arteries.

Venous drainage. This describes an anastomotic circle round the base of the nipple from which branches carry the venous blood to the circumference and end in the axillary and mammary veins.

Lymph drainage (see Fig. 6.1, p. 130). This is mainly into the axillary lymph vessels and nodes. Lymph may drain through the internal mammary nodes if the superficial route is obstructed.

Nerve supply. The breasts are supplied by branches from the 4th, 5th and 6th thoracic nerves which contain sympathetic fibres. There are numerous somatic *sensory nerve endings* in the breast especially around the nipple. When these *touch receptors* are stimulated by sucking, impulses pass to the hypothalamus and the flow of the hormone oxytocin is increased, promoting the release of milk.

Function of the breast

The mammary glands are only active during late pregnancy and after the birth of a baby when they produce milk (lactation). Lactation is stimulated by the hormone prolactin (p. 217).

MALE REPRODUCTIVE SYSTEM

Learning outcomes

After studying this section, you should be able to:

- describe the structure and function of the testes in the scrotum
- outline the structure and function of the spermatic cords
- describe the secretions that pass into the spermatic fluid, including the glands that produce them
- explain the process of ejaculation
- list the main changes occurring at puberty in the male.

The male reproductive system consists of the following organs (Fig. 19.12):

- 2 testes, 2 epididymides } in the scrotum
- 2 deferent ducts (vas deferens)
- 2 spermatic cords
- 2 seminal vesicles
- 2 ejaculatory ducts
- 1 prostate gland
- 1 penis.

Scrotum

The scrotum is a pouch of deeply pigmented skin, fibrous and connective tissue and smooth muscle. It is divided into two compartments each of which contains one testis, one epididymis and the testicular end of a spermatic cord. It lies below the symphysis pubis, in front of the upper parts of the thighs and behind the penis.

Testes

The testes (Fig. 19.13A and B) are the reproductive glands of the male and are the equivalent of the ovaries in the female. They are about 4.5 cm long, 2.5 cm wide and 3 cm thick and are suspended in the scrotum by the spermatic cords. They are surrounded by three layers of tissue.

The tunica vaginalis. This is a double membrane, forming the outer covering of the testes, and is a downgrowth of the abdominal and pelvic peritoneum. During early fetal life the testes develop in the lumbar region of the abdominal cavity just below the kidneys. They then descend into the scrotum taking with them coverings of peritoneum, blood and lymph vessels, nerves and the deferent duct. The peritoneum eventually surrounds the testes in the scrotum, and becomes detached from the abdominal peritoneum. Descent of the testes into the scrotum should be complete by the 8th month of fetal life.

The tunica albuginea. This is a fibrous covering beneath the tunica vaginalis that surrounds the testes. Ingrowths form septa dividing the glandular structure of the testes into *lobules*.

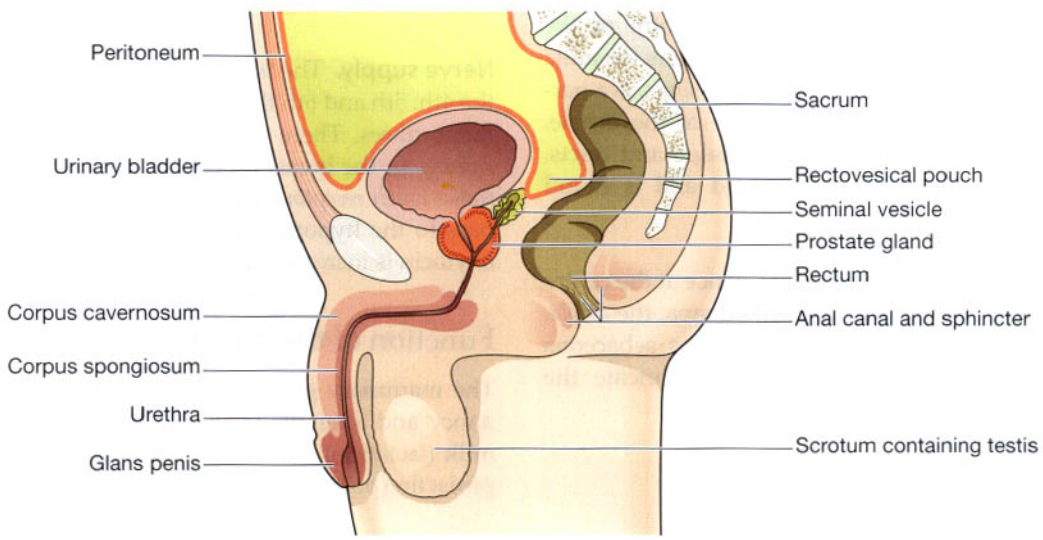


Figure 19.12 The male reproductive organs and their associated structures.

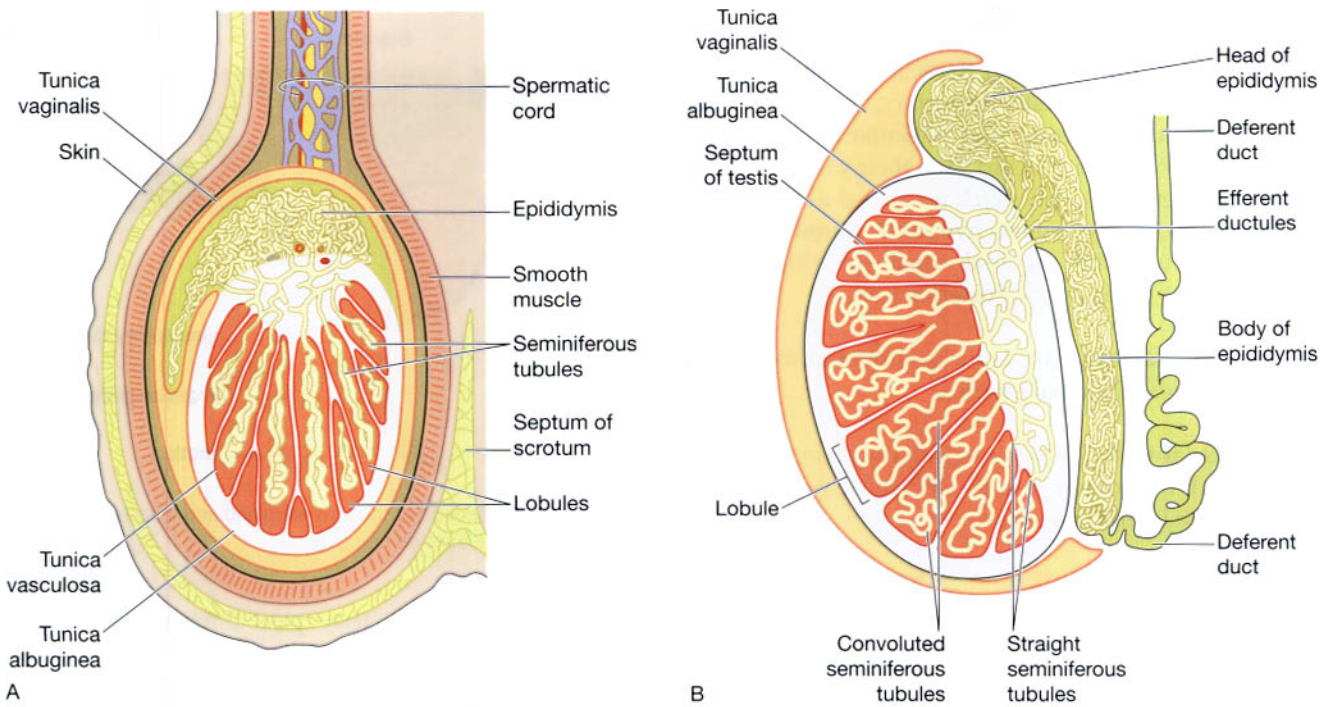


Figure 19.13 The testis: A. A section of the testis and its coverings. B. A longitudinal section of a testis and deferent duct.

The tunica vasculosa. This consists of a network of capillaries supported by delicate connective tissue.

Structure of the testes

In each testis are 200 to 300 lobules and within each lobule are 1 to 4 convoluted loops composed of *germinal epithelial cells*, called *seminiferous tubules*. Between the tubules there are groups of *interstitial cells (of Leydig)* that secrete the hormone testosterone after puberty. At the upper pole of the testis the tubules combine to form a single tubule. This tubule, about 6 m in its full length, is repeatedly folded and tightly packed into a mass called the epididymis. It leaves the scrotum as the *deferent duct* (vas deferens) in the *spermatic cord*. Blood and lymph vessels pass to the testes in the spermatic cords.

Functions of the testes

Spermatozoa (sperm) are produced in the seminiferous tubules of the testes, and mature as they pass through the long and convoluted epididymis, where they are stored. The hormone controlling sperm production is FSH from the anterior pituitary (p. 218). A mature sperm (Fig. 19.14) has a head, a body, and a long whip-like tail that is used for motility. The head is almost completely filled by the nucleus, containing its DNA. It also contains the enzymes required to penetrate the outer layers of the ovum to reach, and fuse with, its nucleus. The body of the sperm is packed with mitochondria, which fuel the propelling action of the tail that powers the sperm on its journey into the female reproductive tract.

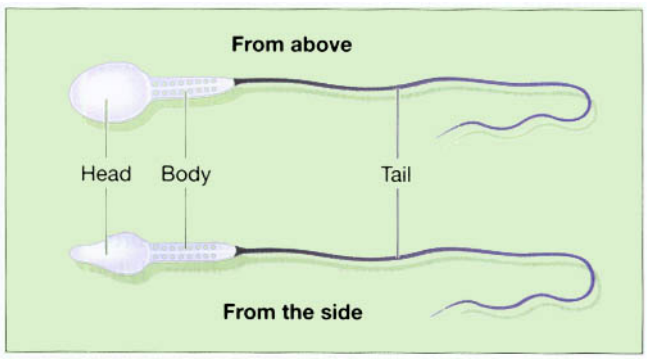


Figure 19.14 A spermatozoon.

Successful spermatogenesis takes place at a temperature about 3°C below normal body temperature. The testes are cooled by their position outside the abdominal cavity, and the thin outer covering of the scrotum has very little insulating fat.

The spermatic cords

The spermatic cords suspend the testes in the scrotum. Each cord contains a testicular artery, testicular veins, lymphatics, a deferent duct and testicular nerves, which come together to form the cord from their various origins in the abdomen. The cord, which is covered in a sheath of smooth muscle and connective and fibrous tissues, extends through the inguinal canal (p. 433) and is attached to the testis on the posterior wall.

The testicular artery. This branches from the abdominal aorta, just below the renal arteries.

The testicular vein. This passes into the abdominal cavity. The left vein opens into the left renal vein and the right into the inferior vena cava.

Lymph drainage. This is through lymph nodes around the aorta.

The deferent duct. This is some 45 cm long. It passes upwards from the testis through the inguinal canal and ascends medially towards the posterior wall of the bladder where it is joined by the duct from the *seminal vesicle* to form the *ejaculatory duct* (Fig. 19.15).

The nerve supply. This is provided by branches from the 10th and 11th thoracic nerves.

Seminal vesicles

The seminal vesicles are two small fibromuscular pouches lined with columnar epithelium, lying on the posterior aspect of the bladder (Fig. 19.15).

At its lower end each seminal vesicle opens into a short duct which joins with the corresponding deferent duct to form an ejaculatory duct.

Functions of the seminal vesicles

The seminal vesicles contract and expel their stored contents, seminal fluid, during ejaculation. Seminal fluid, which forms 60% of the bulk of the fluid ejaculated at male orgasm, contains nutrients to support the sperm during their journey through the female reproductive tract.

Ejaculatory ducts

The ejaculatory ducts are two tubes about 2 cm long, each formed by the union of the duct from a seminal vesicle and a deferent duct. They pass through the prostate gland and join the prostatic urethra, carrying seminal fluid and spermatozoa to the urethra (Fig. 19.15).

The ejaculatory ducts are composed of the same layers of tissue as the seminal vesicles.

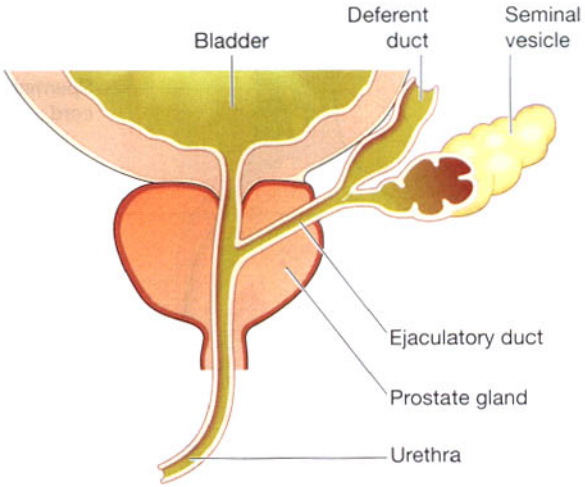


Figure 19.15 Section of the prostate gland and associated reproductive structures on one side.

Prostate gland

The prostate gland (Fig. 19.15) lies in the pelvic cavity in front of the rectum and behind the symphysis pubis, surrounding the first part of the urethra. It consists of an outer fibrous covering, a layer of smooth muscle and glandular substance composed of columnar epithelial cells.

Functions of the prostate gland

The prostate gland secretes a thin, milky fluid that makes up about 30% of *semen*, and gives it its milky appearance. It is slightly alkaline, which provides a protective local environment for sperm arriving in the acidic vagina. It also contains a clotting enzyme, which thickens the semen in the vagina, increasing the likelihood of semen being retained in the vicinity of the cervix.

Urethra and penis

Urethra

The male urethra provides a common pathway for the flow of urine and semen, the combined secretions of the male reproductive organs. It is about 19 to 20 cm long and consists of three parts. The *prostatic urethra* originates

at the urethral orifice of the bladder and passes through the prostate gland. The *membranous urethra* is the shortest and narrowest part and extends from the prostate gland to the bulb of the penis, after passing through the perineal membrane. The *spongiose* or *penile urethra* lies within the corpus spongiosum of the penis and terminates at the external urethral orifice in the *glans penis*.

There are two urethral sphincters (Fig. 19.16). The *internal sphincter* consists of smooth muscle fibres at the neck of the bladder above the prostate gland. The *external sphincter* consists of skeletal muscle fibres surrounding the membranous part.

Penis

The penis (Fig. 19.16) has a *root* and a *body*. The root lies in the perineum and the body surrounds the urethra. It is formed by three cylindrical masses of *erectile tissue* and involuntary muscle. The erectile tissue is supported by fibrous tissue and covered with skin and has a rich blood supply.

The two lateral columns are called the *corpora cavernosa* and the column between them, containing the urethra, is the *corpus spongiosum*. At its tip it is expanded into a triangular structure known as the *glans penis*. Just above the glans the skin is folded upon itself and forms a movable double layer, the *foreskin* or *prepuce*. Arterial blood is supplied by deep, dorsal and bulbar arteries of the penis which are branches from the internal pudendal arteries. A series of veins drain blood to the internal pudendal and internal iliac veins. The penis is supplied by autonomic and somatic nerves. Parasympathetic stimulation leads to filling of the spongy erectile tissue with blood, caused by arteriolar dilatation and venoconstriction, which increases blood flow into the penis and obstructs outflow. The penis therefore becomes engorged and erect, an essential prerequisite for coitus to occur.

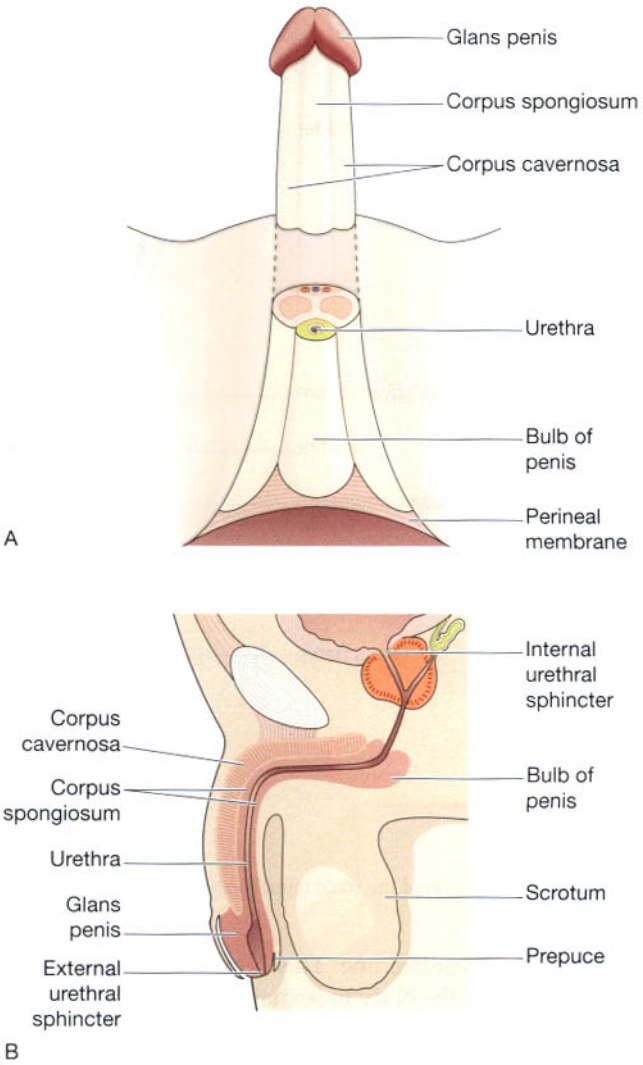


Figure 19.16 The penis: A. Viewed from below. B. Viewed from the side.

Ejaculation

During ejaculation, which occurs at the point of male orgasm, spermatozoa are expelled from the epididymis and pass through the deferent duct, the ejaculatory duct and the urethra. The semen is propelled by powerful rhythmical contraction of the smooth muscle in the walls of the deferent duct; the muscular contractions are sympathetically mediated. Muscle in the walls of the seminal vesicles and prostate gland also contracts, adding their contents to the fluid passing through the genital ducts. The force generated by these combined processes leads to emission of the semen through the external urethral sphincter (Fig. 19.17).

Sperm comprise only 10% of the final ejaculate, the remainder being made up of seminal and prostatic fluids, which are added to the sperm during male orgasm, as well as mucus produced in the urethra. Between 2 and 5 ml of semen are produced in a normal ejaculate, and contain between 40 and 100 million spermatozoa per ml. If not ejaculated, sperm gradually lose their fertility after several months and are reabsorbed by the epididymis.

Puberty in the male

This occurs between the ages of 10 and 14. Luteinising hormone from the anterior lobe of the pituitary gland stimulates the interstitial cells of the testes to increase the

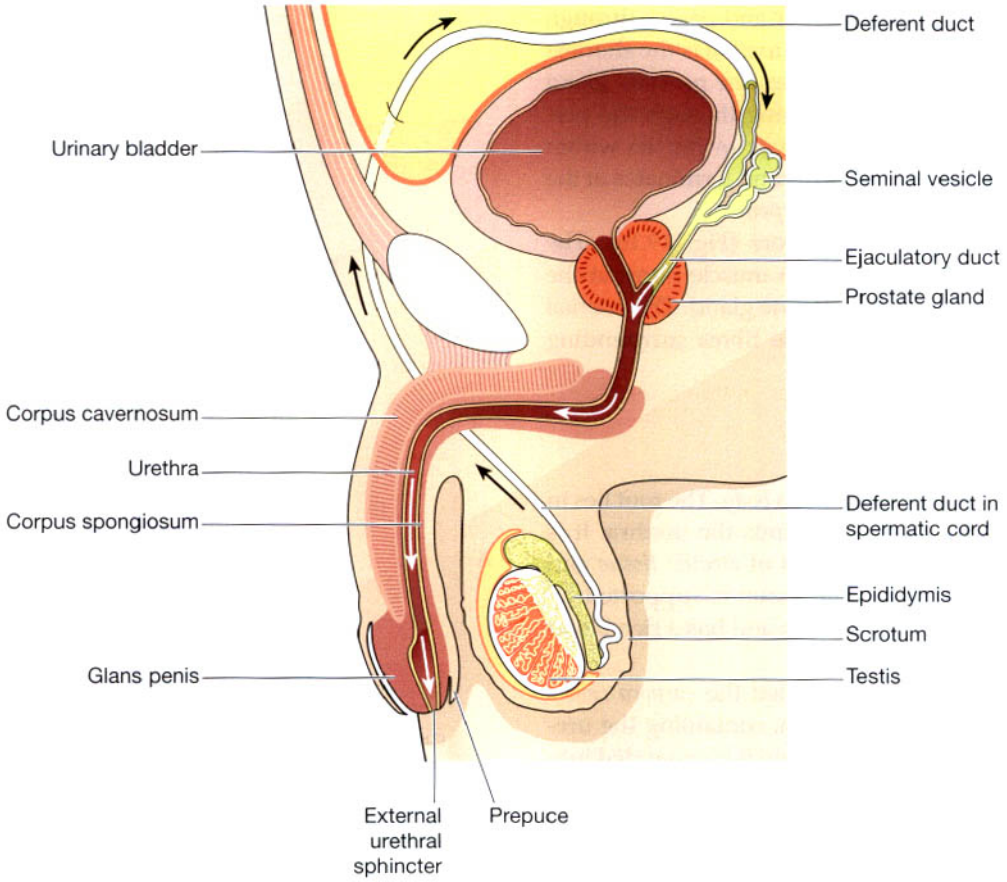


Figure 19.17 Section of the male reproductive organs. Arrows show the route taken by spermatozoa during ejaculation.

production of testosterone. This hormone influences the development of the body to sexual maturity. The changes which occur at puberty are:

- growth of muscle and bone and a marked increase in height and weight
- enlargement of the larynx and deepening of the voice—it 'breaks'
- growth of hair on the face, axillae, chest, abdomen and pubis
- enlargement of the penis, scrotum and prostate gland
- maturation of the seminiferous tubules and production of spermatozoa
- the skin thickens and becomes more oily.

In the male, fertility and sexual ability tend to decline gradually with ageing. The secretion of testosterone gradually declines, usually beginning at about 50 years of age. There is no period comparable to the female menopause.

SEXUALLY TRANSMITTED DISEASE (VENEREAL DISEASE)

Learning outcomes

After studying this section, you should be able to:

- list the principal causes of sexually transmitted diseases
- explain the effects of sexually transmitted diseases.

Infection of the reproductive system may be classified as:

- *non-specific*, usually caused by a mixture of microbes, e.g. staphylococci, streptococci, coliform bacteria, *Clostridium perfringens (Cl. welchii)*
- *specific*, caused by sexually transmitted microbes, the most common of which being *Neisseria gonorrhoeae, Trichomonas vaginalis*, chlamydia, herpes viruses, human immunodeficiency virus (HIV) and hepatitis B.

In general, the microbes that cause sexually transmitted diseases:

- are unable to survive outside the body for long periods
- have no intermediate host
- produce lesions in the genital area which discharge the infecting microbes.

Chlamydia

The microbe *Chlamydia trachomatis* causes inflammation of the female cervix, which may ascend through the reproductive tract and cause pelvic inflammatory disease, and urethritis in the male, which may also ascend and lead to epididymitis. *Chlamydia* infection is often present in conjunction with other sexually transmitted diseases. The same organism causes trachoma, an infection of the eye that is the primary cause of blindness world-wide (p. 210).

Gonorrhoea

This is the most commonly occurring venereal disease and affects men and women. It is caused by *Neisseria gonorrhoeae* which affects the mucosa of the reproductive and urinary tracts. In the male, suppurative urethritis occurs and the infection may spread to the prostate gland, epididymis and testes. In the female the infection may spread from vulvar glands, vagina and cervix to the body of the uterus, uterine tubes, ovaries and peritoneum. Healing by fibrosis in the female may cause obstruction of the uterine tubes, leading to infertility. In the male it may cause urethral stricture.

Non-venereal transmission of gonorrhoea may cause *neonatal ophthalmia* in babies born to infected mothers. The eyes are infected as the baby passes through the vagina.

Syphilis

This disease is caused by *Treponema pallidum*. There are three clearly marked stages although the third is now rarely seen in Britain. After an incubation period of several weeks, the *primary sore* (chancre) appears at the site of infection, e.g. the vulva, vagina, perineum, penis or round the mouth. In the female the primary sore may be undetected if it is internal. After several weeks the chancre subsides spontaneously. *Secondary lesions* appear 3 to 4 months after infection. They consist of skin rashes and raised papules (condylomata lata) on the external genitalia and vaginal walls. These subside after several months and are followed by a latent period of a variable number of years. *Tertiary lesions* (gummas) develop in many organs and in a few cases the nervous system is involved, leading to general paralysis.

Sexual transmission occurs during the primary and secondary stages when discharge from lesions contains microbes. Congenital transmission occurs when microbes from an infected mother cross the placenta to the fetus, often with fatal consequences. Accidental spread of infection may occur by blood transfusion if a donor's blood is taken during the incubation period after microbes have spread to the blood from the site of infection.

Trichomonas vaginalis

These *protozoa* cause acute vulvovaginitis. It is usually sexually transmitted and is commonly present in women with gonorrhoea.

Candidiasis

The yeast *Candida albicans* (see also p. 319) is frequently a commensal in the normal vagina and causes no problems. It is normally prevented from flourishing by, e.g., vaginal acidity, but in certain circumstances it proliferates, causing candidiasis (thrush). Common precipitating factors include:

- antibiotic therapy, which kills the bacteria that keep vaginal pH low
- pregnancy
- reduced immune function.

Acquired immune deficiency syndrome (AIDS) and hepatitis B infection

These viral conditions may be sexually transmitted but there are no local signs of infection. For a description of AIDS and HIV see page 385 and hepatitis B page 334.

DISEASES OF THE FEMALE REPRODUCTIVE SYSTEM

Learning outcomes

After studying this section, you should be able to:

- describe the causes and consequences of pelvic inflammatory disease
- discuss the disorders of the vulva
- define the term imperforate hymen
- outline the causes and effects of cervical carcinoma
- discuss the main pathologies of the uterus and uterine tubes
- describe the causes and effects of ovarian disease
- describe the causes of female infertility
- discuss the principal disorders of the female breast.

Pelvic inflammatory disease (PID)

This infection may be specific or non-specific. It usually begins as vulvovaginitis, including the vulvar glands, then it may spread to the cervix, uterus, uterine tubes and ovaries. Upward spread is most common when microbes are present in the vagina before a surgical procedure, childbirth or abortion, especially if some of the products of conception are retained.

Complications of PID include:

- infertility due to obstruction of uterine tubes
- peritonitis
- intestinal obstruction due to adhesions between the bowel and the uterus and/or uterine tubes
- bacteraemia which may lead to meningitis, endocarditis or suppurative arthritis
- Bartholin's gland abscess or cyst formation if the duct is blocked.

Vulvar dystrophies

Atrophic dystrophy

This is thinning of vulvar epithelium and the formation of fibrous tissue, occurring after the menopause due to oestrogen withdrawal. It predisposes to infection, especially in debilitated women, and to malignant epithelial neoplasia.

Vulvar intraepithelial neoplasia (VIN)

The extent of development of hyperplasia and dysplasia of cells of the skin of the vulva varies considerably. It is often associated with human papilloma virus infection. In the majority of cases the neoplasia are benign. Some VIN cases progress to invasive carcinoma while others regress spontaneously. In elderly women and immunosuppressed young women malignant tumours may develop which spread locally with early involvement of inguinal lymph nodes. Because of the anastomoses between lymph vessels on the two sides of the vulva, bilateral lymph node involvement is common.

Imperforate hymen

This is a congenital abnormality which may not be noticed until the onset of menstruation. When complete (imperforate), the hymen forms a barrier in the vagina. Blood accumulates in the vagina, uterus and uterine tubes with each menstrual cycle, and it may enter the peritoneal cavity and cause peritonitis. The uterine tubes may become obstructed by coagulated blood, leading to infertility.

Disorders of the cervix

Cervical carcinoma

Dysplastic changes referred to as cervical intraepithelial neoplasia (CIN) begin in the deepest layer of cervical epithelium, usually at the junction of the stratified squamous epithelium of the lower third of the cervical canal with the secretory epithelium of the upper two-thirds. Dysplasia may progress to involve the full thickness of epithelium, called *carcinoma-in-situ*. The cancer may develop further and spread locally to the vagina, uterine body and other pelvic structures. More widespread metastases occur late in the disease. Not all cases in which dysplastic changes are observed develop to the cancerous stage but it is not possible to predict how far development will go, and whether it will remain static or regress. Three degrees of dysplasia have been described, although clear distinction between them is not always possible:

- CIN 1 = mild dysplasia
- CIN 2 = moderate dysplasia
- CIN 3 = carcinoma-in-situ.

CIN 3 may progress to invasive carcinoma. Early spread is via lymph nodes and local spread is commonly

to the uterus, vagina, bladder and rectum. In the late stages spread via the blood to the liver, lungs and bones may occur.

The disease takes 15 to 20 years to develop and it occurs mostly between 35 and 50 years of age. It is likely that a significant proportion of cases are due to the transmission of some carcinogenic factor to the female during sexual intercourse. This is supported by the observations that the disease occurs more frequently in women who commence sexual activity at an early age, who have many partners, who have many pregnancies or have frequent sexual intercourse. Additionally, barrier contraceptives protect against the disease. The human papillomavirus (HPV), which causes genital warts, is strongly associated with this cancer (see also p. 369).

Disorders of the uterine body

Acute endometritis

This is usually caused by non-specific infection, following parturition or abortion, especially if fragments of membranes or placenta have been retained in the uterus. A variety of microbes may be involved, e.g. staphylococci, streptococci, *Escherichia coli* or *Pseudomonas*. The inflammation may subside after removal of retained products. The infection may spread to:

- myometrium, perimetrium and surrounding pelvic tissues which may lead to thrombosis of iliac veins
- uterine tubes, causing salpingitis, fibrosis, obstruction and infertility
- any of the above-mentioned areas, causing peritonitis and possibly adhesions.

Chronic endometritis

This may follow an acute attack or be due to spread of pelvic inflammatory disease. It may follow abortion or parturition and may be associated with chronic salpingitis, endometrial carcinoma or the use of intrauterine contraceptive devices.

Endometriosis

This is the growth of endometrial tissue outside the uterus, most commonly in the ovaries, uterine tubes and other pelvic structures. The ectopic tissue, like the uterine endometrium, is responsive to the fluctuations in sex hormone levels of the menstrual cycle, causing menstrual-type bleeding into the lower abdomen and, in the ovaries, the formation of coloured cysts, 'chocolate cysts'. There is intermittent pain due to swelling, and recurrent haemorrhage causes fibrous tissue formation. Ovarian endometriosis may lead to pelvic inflammation, infertility and extensive pelvic adhesions, involving the ovaries, uterus, uterine ligaments and the bowel. The cause is not clear but it has been suggested that there may have been:

- abnormal cell differentiation in the fetus
- regurgitation of menstrual material through the uterine tubes
- spread of endometrial cells in lymph and blood during menstruation.

Adenomyosis

This is the growth of endometrium within the myometrium. The ectopic tissue may cause general or localised uterine enlargement. The lesions may cause dysmenorrhoea and irregular excessive bleeding (menorrhagia), usually beginning between 40 and 50 years of age.

Endometrial hyperplasia

The hyperplasia may affect endometrial glands, causing cyst formation and/or focal hyperplasia of atypical cells. The focal type frequently undergoes malignant change. Both types are associated with a sustained high blood oestrogen level, which may be due to:

- failure of ovarian follicles to mature and release their ova
- oestrogen-secreting ovarian tumours
- prolonged oestrogen therapy.

Leiomyoma (fibroid, myoma)

These are very common, often multiple, benign tumours of myometrium. They are firm masses of smooth muscle encapsulated in compressed muscle fibres and they vary greatly in size. Large tumours may undergo degenerative changes if they outgrow their blood supply, leading to necrosis, fibrosis and calcification. They develop during the reproductive period and may be hormone dependent, enlarging during pregnancy and when oral contraceptives are used. They tend to regress after the menopause. Large tumours may cause pelvic discomfort, frequency of micturition, menorrhagia, irregular bleeding, dysmenorrhoea and reduced fertility. Malignant change is rare.

Endometrial carcinoma

This occurs mainly in nulliparous women (i.e. women who have never been pregnant) between 50 and 60 years of age. The cause is not known but there is some evidence

that oestrogen may be involved. The incidence of carcinoma is increased when an oestrogen-secreting tumour is present and in women who are obese, hypertensive or diabetic, because they tend to have a high level of blood oestrogen. The tumour may develop as a diffuse mass, a localised plaque or a polyp and there is often ulceration and bleeding. Endometrium has no lymphatics, so lymph spread is delayed until there is extensive local spread that involves other pelvic structures. Distant metastases, spread in blood or lymph, develop later, most commonly in the liver, lungs and bones. Invasion of the ureters leads to hydronephrosis and uraemia which is commonly the cause of death.

Disorders of the uterine tubes and ovaries

Acute salpingitis

Salpingitis is inflammation of the uterine tubes, and is usually due to infection spreading from the uterus, and only occasionally from the peritoneal cavity. The outcome may be:

- uneventful recovery
- chronic inflammation, leading to fibrous tubal obstruction and infertility
- pus formation (*pyosalpinx*) and further spread to the ovaries and peritoneal cavity, leading to fibrous tubal obstruction, infertility and/or pelvic adhesions.

Ectopic pregnancy

This is the implantation of a fertilised ovum outside the uterus, most commonly in a uterine tube. As the fetus grows the tube ruptures and its contents enter the peritoneal cavity, causing acute inflammation (peritonitis) and possibly severe intraperitoneal haemorrhage.

Ovarian tumours

The majority of ovarian tumours are benign, usually occurring between 20 and 45 years of age. The remainder occur mostly between 45 and 65 years and are divided between borderline malignancy (low-grade cancer) and frank malignancy. There are three main types of cells involved: epithelial cells, germ cells and hormone-secreting cells (sex-cord stroma cells).

Epithelial cell tumours

Most of these are borderline or malignant tumours. They vary greatly in size from very large to quite small and some are partly cystic. Large tumours may cause pressure, leading to gastrointestinal disturbances, frequency of micturition, dysuria and ascites. Those suspended by a pedicle may twist, causing ischaemia, necrosis, haemorrhage or rupture of a cyst. The principal methods of spread are invasion of local and peritoneal structures. Later lymph- and blood-spread metastases may develop.

The prevalence is higher in developed societies and in the higher socio-economic groups. Pregnancy and suppression of ovulation by medicinal contraceptives, e.g. the oral contraceptive pill, may have a protective effect due to a reduction in the number of ovulatory menstrual cycles.

Germ cell ovarian tumours

These occur mainly in children and young adults and only a few are malignant. Benign *dermoid cysts* are the most common type. These are thick-walled cysts containing a variety of uncharacteristic tissues, e.g. hair, skin, epithelium or teeth. They are usually small and have a tendency to twist on a pedicle, causing ischaemia and necrosis.

Sex-cord stroma cell tumours

These cells are the precursors of the ovarian follicle lining cells, luteal cells and fibrous supporting cells. Mixed tumours develop, some of which secrete hormones. Oestrogen-secreting tumours cause precocious sexual development in children. In adults the excess oestrogen may cause endometrial hyperplasia, endometrial carcinoma, cystic disease of the breast or breast cancer. Androgen-secreting tumours occasionally develop, causing atrophy of the breast and genitalia and the development of male sex characteristics.

Metastatic ovarian tumours

The ovaries are a common site of metastatic spread from primary malignant tumours in other pelvic organs, the breast, stomach, pancreas and biliary tract.

Female infertility

This may be due to:

- blockage of uterine tubes, often the consequence of pelvic inflammatory disease
- anatomical problems, e.g. retroversion (tilting backwards) of the uterus
- endocrine factors; any abnormalities of the glands and hormones governing the menstrual cycle can interfere with, for example, ovulation or the uterine cycle
- low body weight or severe malnourishment
- endometriosis.

Disorders of the breast

Mastitis (inflammation of the breast)

Acute non-suppurative mastitis
This occurs during lactation and is associated with painful congestion and oedema of the breast. It is of hormonal origin.

Acute suppurative (pyogenic) mastitis
The microbes enter through a nipple abrasion caused by the infant sucking. The most common causative microbes are *Staphylococcus aureus* and *Streptococcus pyogenes* usually acquired by the infant while in hospital. The infection spreads along the mammary ducts of a lobe causing localised swelling and redness. If it does not resolve it can become chronic and an abscess may form.

Tumours of the breast

Benign tumours
Most breast tumours (90%) are benign. Fibroadenomas are the commonest type and occur any time after puberty; incidence peaks in the third decade. Some are cystic and some solid and they usually occur in women nearing the menopause. They may originate from secretory cells, fibrous tissue or from ducts.

Malignant tumours
The most common types of tumour are usually painless lumps found in the upper outer quadrant of the breast. There is considerable fibrosis around the tumour that may cause retraction of the nipple and necrosis and ulceration of the overlying skin. It is increasingly common between 35 and 70 years.

Early spread beyond the breast is via lymph to the axillary and internal mammary nodes. Local invasion involves the pectoral muscles and the pleura. Blood-spread metastases may occur later in many organs and bones, especially lumbar and thoracic vertebrae. The causes of breast cancer are not known, but an important predisposing factor appears to be high oestrogen exposure. Women with an early menarche, a late menopause, and no pregnancies have a higher than normal risk because they experience more menstrual cycles in their lifetimes, and each monthly cycle brings with it the oestrogen surge seen in the proliferative phase of the cycle. A genetic component is also likely, with close relatives of cancer sufferers having a significantly elevated risk of developing the disease. One per cent of all breast cancer occurs in men.

DISEASES OF THE MALE REPRODUCTIVE SYSTEM

Learning outcomes

After studying this section, you should be able to:

- outline the causes and effects of penile and urethral infections
- describe the main pathologies of the testis
- discuss the principal disorders of the prostate gland
- list the main causes of male infertility.

Infections of the penis

Inflammation of the glans and prepuce may be caused by a specific or non-specific infection. In non-specific infections, or *balanitis*, lack of personal hygiene is an important predisposing factor, especially if *phimosis* is present, i.e. the orifice in the foreskin (prepuce) is too small to allow for its normal retraction. If the infection becomes chronic there may be fibrosis of the foreskin, which increases the phimosis.

Infections of the urethra

Gonococcal urethritis is the most common specific infection. Non-specific infection may be spread from the bladder (cystitis) or be introduced during catheterisation, cystoscopy or surgery. Both types may spread throughout the system to the prostate, seminal vesicles, epididymis and testes. If infection becomes chronic, fibrosis may cause urethral stricture or obstruction, leading to retention of urine.

Epididymis and testes

Infections

Non-specific epididymitis and orchitis are usually due to spread of infection from the urethra, commonly following prostatectomy. The microbes may spread either through the deferent duct (vas deferens) or via lymph.

Specific epididymitis. This is usually caused by gonorrhoea spread from the urethra.

Orchitis (inflammation of the testis). This is more commonly caused by mumps viruses, bloodborne from the

parotid glands. Acute inflammation with oedema occurs about 1 week after the appearance of parotid swelling. The infection is usually unilateral but, if bilateral, severe damage to germinal epithelium of the seminiferous tubules may result in sterility.

Undescended testis (cryptorchidism)

During embryonic life the testes develop within the abdominal cavity, but descend into the scrotum prior to birth. If they fail to do this and the condition is not corrected, infertility is likely to follow and the risk of testicular cancer is increased.

Hydrocele

This is the most common form of scrotal swelling and is accumulation of serous fluid in the tunica vaginalis. The onset may be acute and painful or chronic. It may be congenital or be secondary to another disorder of the testis or epididymis.

Testicular tumours

Most testicular tumours are malignant and are the commonest malignancies in young men. They occur in children and young adults in whom the affected testis has not descended or has been late in descending into the scrotum. The tumour tends to remain localised for a considerable time but eventually spreads in lymph to pelvic and abdominal lymph nodes, and more widely in blood. Occasionally hormone-secreting tumours develop and may cause precocious development in children.

Prostate gland

Infections

Acute prostatitis is usually caused by non-specific infection, spread from the urethra or bladder, often following catheterisation, cystoscopy, urethral dilatation or surgery in which part of the gland is removed. Chronic infection may follow an acute attack, but it may develop insidiously and is not associated with known microbes. Fibrosis of the gland may occur during healing, causing urethral stricture or obstruction.

Benign prostatic enlargement

Hyperplastic nodules form around the urethra and may cause constriction or obstruction to the flow of urine, causing retention of urine. Urethral stricture may prevent the bladder emptying completely during micturition, predisposing to infection which may spread upwards and cause pyelonephritis and other complications.

Prostatic enlargement is common in men over 50, affecting up to 70% of men aged over 70. The cause is not clear, but it may be an acceleration of the ageing process associated with the decline in androgen secretion which changes the androgen/oestrogen balance.

Malignant prostatic tumours

These are a relatively common cause of death in men over 50. The carcinogen is not known but changes in the androgen/oestrogen balance may be significant or viruses may be involved. Invasion of local tissues is widespread before lymph-spread metastases develop in pelvic and abdominal lymph nodes. Blood-spread metastases in bone are common and bone formation rather than bone destruction is a common feature. Lumbar vertebrae are a common site, possibly due to retrograde spread along the walls of veins. In many cases bone metastases are the first indication of malignant prostatic tumours.

Breast

Breast tissue in men consists of ducts and stroma only.

Gynaecomastia

This is proliferation of breast tissue in men. It usually affects only one breast and is benign. It is common in adolescents and older men, and is often associated with:

- endocrine disorders, especially those associated with high oestrogen levels
- cirrhosis of the liver (p. 335)
- malnutrition
- some drugs, e.g. chlorpromazine, spironolactone, digoxin
- Klinefelter's syndrome, a genetic disorder in which there is testicular atrophy and absence of spermatogenesis.

Malignant tumours

These develop in a small number of men, usually in the older age groups.

Male infertility

This may be due to:

- endocrine disorders
- obstruction of the deferent duct
- failure of erection or ejaculation during intercourse
- surgical vasectomy
- suppression of spermatogenesis by, e.g., ionising radiation, chemotherapy and other drugs.

Normal values

Note. *Some biological measures have been extracted from the text and listed here for easy reference. In some cases slightly different 'normals' may be found in other texts and used by different medical practitioners.*

Metric measures, units and SI symbols

Name	SI unit	Symbol
Length	metre	m
Mass	kilogram	kg
Amount of substance	mole	mol
Pressure	pascal	Pa
Energy	joule	J

Decimal multiples and submultiples of the units are formed by the used of standard prefixes.

Multiple	Prefix	Symbol	Submultiple	Prefix	Symbol
10^{6}	mega	M	10^{-1}	deci	d
10^{3}	kilo	k	10^{-2}	centi	c
10^{2}	hecto	h	10^{-3}	milli	m
10^{1}	deca	da	10^{-6}	micro	μ
			10^{-9}	nano	n
			10^{-12}	pico	p
			10^{-15}	femto	f

Conversion table for kPa/mmHg (for e.g. capillary pressures)

1 mmHg = 0.13 kPa
1 kPa = 7.5 mmHg
35 mmHg = 4.7 kPa
25 mmHg = 3.3 kPa
15 mmHg = 2.0 kPa
10 mmHg = 1.3 kPa

Hydrogen ion concentration (pH)

Neutral = 7 Acid = 0 to 7 Alkaline = 7 to 14

Normal pH of some body fluids	
Blood	7.35 to 7.45
Saliva	5.4 to 7.5
Gastric juice	1.5 to 3.5
Bile	6.0 to 8.5
Urine	4.5 to 8.0

Some normal plasma levels in adults

Calcium	2.12 to 2.62 mmol/l	(8.5 to 10.5 mg/100 ml)
Chloride	97 to 106 mmol/l	(97 to 106 mEq/l)
Cholesterol	3.6 to 6.7 mmol/l	(140 to 260 mg/100 ml)
Glucose	3.5 to 8 mmol/l	(63 to 144 mg/100 ml)
Fasting glucose	3.6 to 5.8 mmol/l	(65 to 105 mg/100 ml)
Potassium	3.3 to 4.7 mmol/l	(3.3 to 4.7 mEq/l)
Sodium	135 to 143 mmol/l	(135 to 143 mEq/l)
Urea	2.5 to 6.6 mmol/l	(15 to 44 mg/100 ml)

Arterial blood gases

PO_2	12 to 15 kPa	(90 to 110 mmHg)
PCO_2	4.5 to 6 kPa	(34 to 46 mmHg)
Bicarbonate	21 to 27.5 mmol/l	
H^+ ions	36 to 44 nmol/l	(7.35 to 7.45 pH units)