CHAPTER 8

The special senses

ANIMATIONS

The special senses of hearing, sight, smell and taste all have specialised sensory receptors that collect and transmit information to specific areas of the brain. Incoming nerve impulses from sensory receptors in the ears, eyes, nose and mouth are integrated and coordinated within the brain allowing perception of this sensory information. Up to 80% of what we perceive comes from external sensory stimuli. The first sections of this chapter explore the special senses, while the later ones consider the effect of ageing and problems that arise when disorders occur in the structures involved in hearing and vision.

Hearing and the ear

Learning outcomes

After studying this section, you should be able to:

- describe the structure of the outer, middle and inner parts of the ear
- explain the physiology of hearing.

The ear is the organ of hearing and is also involved in balance. It is supplied by the 8th cranial nerve, i.e. the *cochlear part* of the *vestibulocochlear nerve*, which is stimulated by vibrations caused by sound waves.

With the exception of the auricle (pinna), the structures that form the ear are encased within the petrous portion of the temporal bone.

Structure

The ear is divided into three distinct parts (Fig. 8.1): the outer ear, middle ear (tympanic cavity) and inner ear.

The outer ear collects the sound waves and directs them to the middle ear, which in turn transfers them to the inner ear, where they are converted into nerve impulses and transmitted to the hearing area in the cerebral cortex.

Outer ear

The outer ear consists of the auricle (pinna) and the external acoustic meatus (auditory canal).

The auricle (pinna)

The auricle is the visible part of the ear that projects from the side of the head. It is composed of fibroelastic cartilage covered with skin. It is deeply grooved and ridged; the most prominent outer ridge is the *helix*.

The *lobule* (earlobe) is the soft pliable part at the lower extremity, composed of fibrous and adipose tissue richly supplied with blood.

External acoustic meatus (auditory canal)

This is a slightly 'S'-shaped tube about 2.5 cm long extending from the auricle to the *tympanic membrane* (eardrum). The lateral third is embedded in cartilage and the remainder lies within the temporal bone. The meatus is lined with skin continuous with that of the auricle. There are numerous *ceruminous glands* and hair follicles, with associated *sebaceous glands*, in the skin of the lateral third. Ceruminous glands are modified sweat glands that secrete *cerumen* (earwax), a sticky material containing protective substances including the bacteriocidal enzyme lysozyme and immunoglobulins. Foreign materials, e.g. dust, insects and microbes, are prevented from reaching the tympanic membrane by wax, hairs and the curvature of the meatus. Movements of the temporomandibular

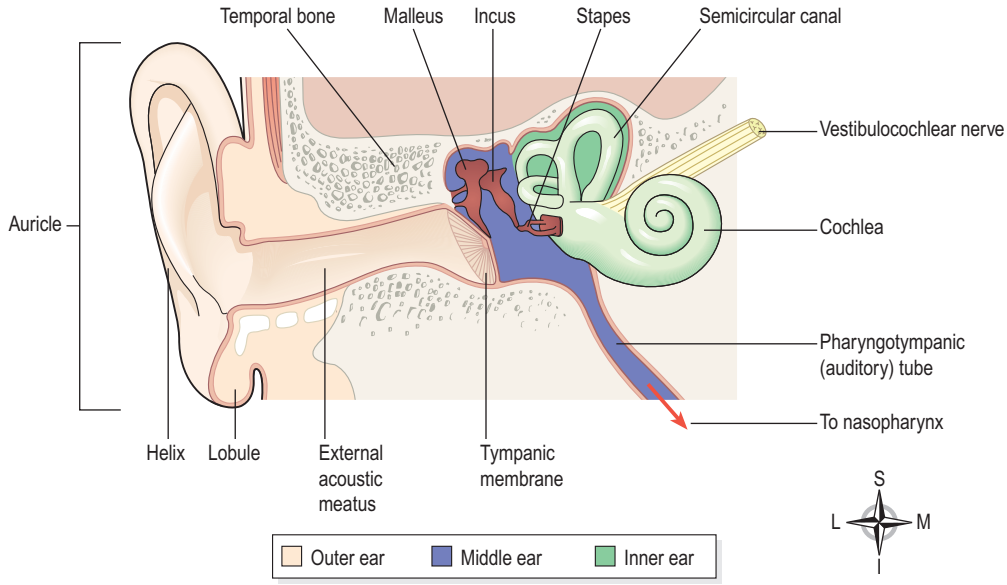

Figure 8.1 The parts of the ear.

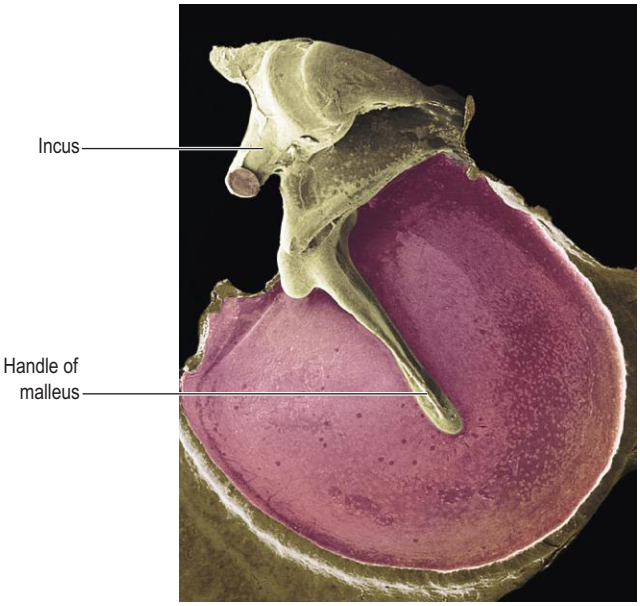

Figure 8.2 The tympanic membrane. Coloured scanning electron micrograph showing the malleus and the incus.

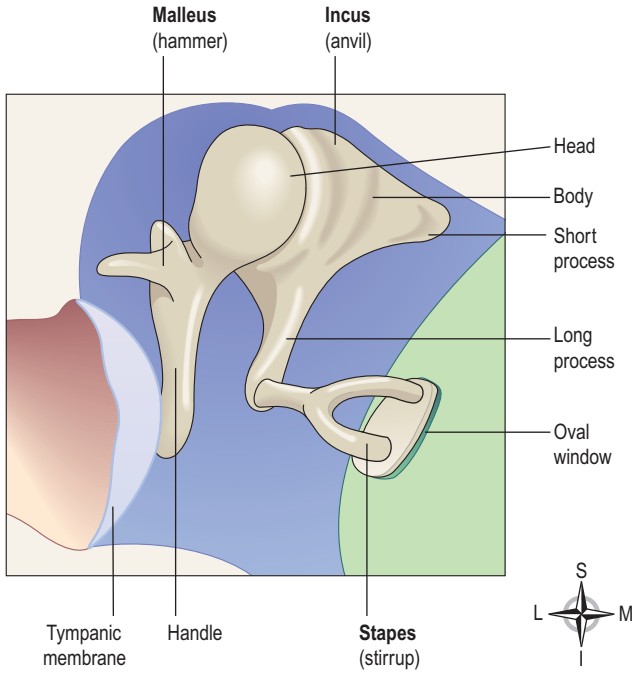

Figure 8.3 The auditory ossicles.

joint during chewing and speaking 'massage' the cartilaginous meatus, moving the wax towards the exterior.

The tympanic membrane (eardrum) (Fig. 8.2) completely separates the external acoustic meatus from the middle ear. It is oval-shaped with the slightly broader edge upwards and is formed by three types of tissue: the outer covering of hairless skin, the middle layer of fibrous tissue and the inner lining of mucous membrane continuous with that of the middle ear.

Middle ear (tympanic cavity)

This is an irregular-shaped air-filled cavity within the petrous portion of the temporal bone (Figs 8.1 and 8.3). The cavity, its contents and the air sacs which open out of it are lined with either simple squamous or cuboidal epithelium.

The *lateral wall* of the middle ear is formed by the tympanic membrane.

The *roof and floor* are formed by the temporal bone.

The *posterior wall* is formed by the temporal bone with openings leading to the mastoid antrum through which air passes to the air cells within the mastoid process.

The *medial wall* is a thin layer of temporal bone in which there are two openings:

- oval window
- round window (see Fig. 8.6).

The oval window is occluded by part of a small bone called the *stapes* and the round window, by a fine sheet of fibrous tissue.

Air reaches the cavity through the *pharyngotympanic* (*auditory* or *Eustachian*) *tube*, which links the nasopharynx and middle ear. It is about 4 cm long and lined with ciliated columnar epithelium. The presence of air at atmospheric pressure on both sides of the tympanic membrane is maintained by the pharyngotympanic tube and enables the membrane to vibrate when sound waves strike it. The pharyngotympanic tube is normally closed but when there is unequal pressure across the tympanic membrane, e.g. at high altitude, it is opened by swallowing or yawning and the ears 'pop', equalising the pressure again.

Auditory ossicles (Fig. 8.3)

These are three very small bones only a few millimetres in size that extends across the middle ear from the tympanic membrane to the oval window (Fig. 8.1). They form a series of movable joints with each other and with the medial wall of the cavity at the oval window. The ossicles are held in place by fine ligaments and are named according to their shapes.

The malleus. This is the lateral hammer-shaped bone. The handle is in contact with the tympanic membrane and the head forms a movable joint with the incus.

The incus. This is the middle anvil-shaped bone. Its body articulates with the malleus, the long process with the stapes, and it is stabilised by the short process, fixed by fibrous tissue to the posterior wall of the tympanic cavity.

The stapes. This is the medial stirrup-shaped bone. Its head articulates with the incus and its footplate fits into the oval window.

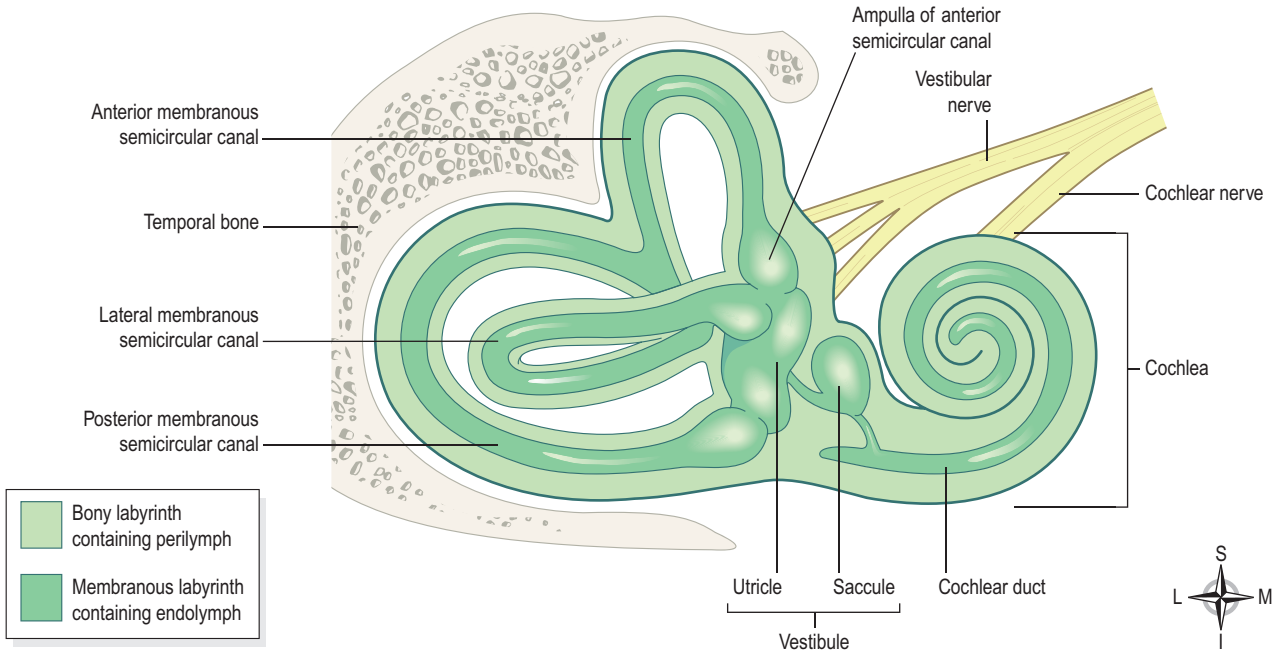

Figure 8.4 The inner ear. The membranous labyrinth within the bony labyrinth.

Inner ear (Fig. 8.4)

The inner ear or labyrinth (meaning 'maze') contains the organs of hearing and balance. It is described in two parts, the *bony labyrinth* and the *membranous labyrinth* and is divided into three main regions:

- the vestibule, containing the utricle and saccule
- three semicircular canals
- the cochlea.

The inner ear is formed from a network of channels and cavities in the temporal bone (the *bony labyrinth*). Within the bony labyrinth, like a tube within a tube, is the *membranous labyrinth*, a network of fluid-filled membranes that lines and fills the bony labyrinth (Fig. 8.4).

The bony labyrinth. This is lined with periosteum. Within the bony labyrinth, the membranous labyrinth is suspended in a watery fluid called *perilymph*.

The membranous labyrinth. This is filled with *endolymph*.

The vestibule

This is the expanded part nearest the middle ear. The oval and round windows are located in its lateral wall. It contains two membranous sacs, the utricle and the saccule, which are important in balance (p. 196).

The semicircular canals

These are three tubes arranged so that one is situated in each of the three planes of space. They are continuous with the vestibule and are also important in balance (p. 196).

The cochlea

This resembles a snail's shell. It has a broad base where it is continuous with the vestibule and a narrow apex, and it spirals round a central bony column.

A cross-section of the cochlea (Fig. 8.5) contains three compartments:

- the scala vestibuli
- the scala media, or *cochlear duct*
- the scala tympani.

In cross-section the bony cochlea has two compartments containing perilymph: the scala vestibuli, which originates at the oval window, and the scala tympani, which ends at the round window. The two compartments are continuous with each other and Figure 8.6 shows the relationship between these structures. The cochlear duct is part of the membranous labyrinth and is triangular in shape. On the *basilar membrane*, or base of the triangle, are *supporting cells* and specialised *cochlear hair cells* containing auditory receptors. These cells form the *spiral organ* (of Corti), the sensory organ that responds to vibration by initiating nerve impulses that are then perceived as hearing within the brain. The *auditory receptors* are dendrites of efferent (sensory) nerves that combine forming the cochlear (auditory) part of the vestibulocochlear nerve (8th cranial nerve), which passes through a foramen in the temporal bone to reach the hearing area in the temporal lobe of the cerebrum (see Fig. 7.20, p. 157).

Physiology of hearing 8.1

Every sound produces sound waves or vibrations in the air, which travel at about 332 metres per second. The

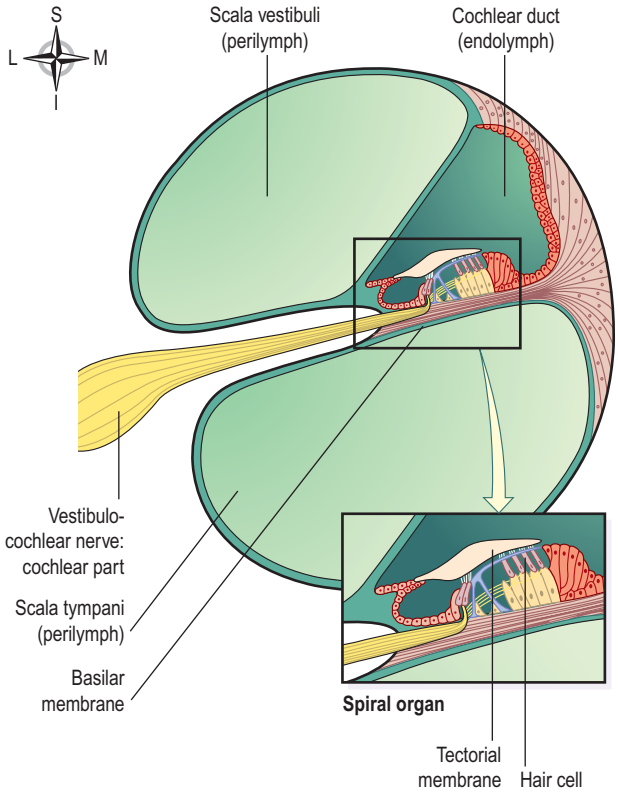

Figure 8.5 A cross-section of the cochlea showing the spiral organ (of Corti).

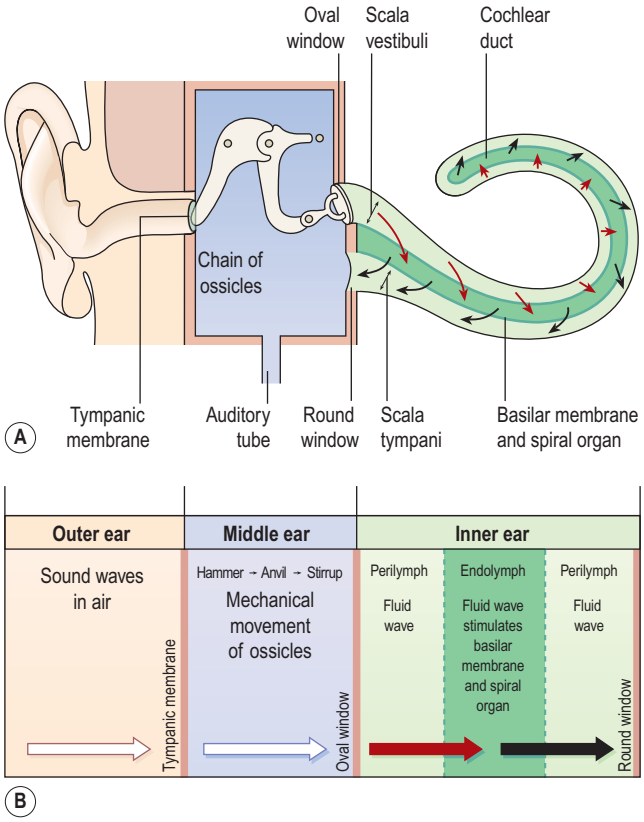

Figure 8.6 Passage of sound waves: A. The ear with cochlea uncoiled. **B.** Summary of transmission.

auricle, because of its shape, collects and concentrates the waves and directs them along the auditory canal causing the tympanic membrane to vibrate. Tympanic membrane vibrations are transmitted and amplified through the middle ear by movement of the ossicles (Fig. 8.6). At their medial end the footplate of the stapes rocks to and fro in the oval window, setting up fluid waves in the perilymph of the scala vestibuli. Some of the force of these waves is transmitted along the length of the scala vestibuli and scala tympani, but most of the pressure is transmitted into the cochlear duct. This causes a corresponding wave motion in the endolymph, resulting in vibration of the basilar membrane and stimulation of the auditory receptors in the hair cells of the spiral organ. The nerve impulses generated pass to the brain in the cochlear (auditory) portion of the vestibulocochlear nerve (8th cranial nerve). The fluid wave is finally expended into the middle ear by vibration of the membrane of the round window. The vestibulocochlear nerve transmits the impulses to the auditory nuclei in the medulla, where they synapse before they are conducted to the auditory area in the temporal lobe of the cerebrum (see Fig. 7.20, p. 157). Because some fibres cross over in the medulla and others remain on the same side, the left and right auditory areas of the cerebrum receive impulses from both ears.

Sound waves have the properties of *pitch* and *volume*, or intensity (Fig. 8.7). Pitch is determined by the frequency of the sound waves and is measured in Hertz (Hz). Sounds of different frequencies stimulate the basilar membrane (Fig. 8.6A) at different places along its length, allowing discrimination of pitch.

The volume depends on the magnitude of the sound waves and is measured in decibels (dB). The greater the amplitude of the wave created in the endolymph, the greater is the stimulation of the auditory receptors in the hair cells in the spiral organ, enabling perception of volume. Long-term exposure to excessive noise causes hearing loss because it damages the sensitive hair cells of the spiral organ.

Balance and the ear

Learning outcome

After studying this section, you should be able to:

- describe the physiology of balance.

The semicircular canals and vestibule (Fig. 8.4)

The semicircular canals have no auditory function although they are closely associated with the cochlea.

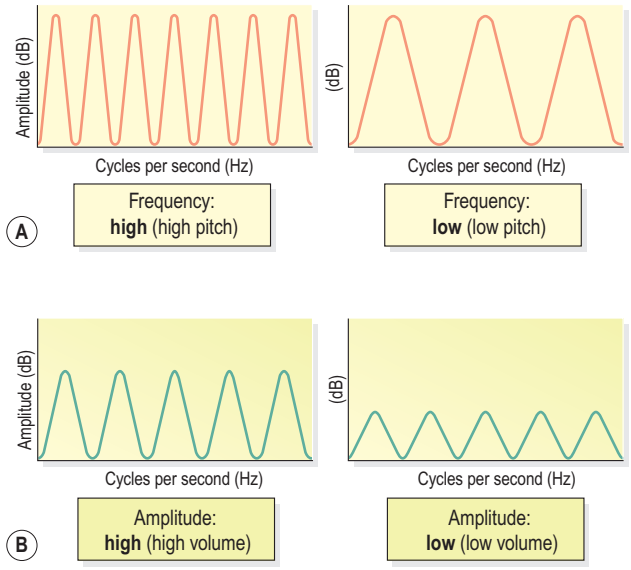

Figure 8.7 Behaviour of sound waves. A. Difference in frequency but of the same amplitude. **B.** Difference in amplitude but of the same frequency.

Instead they provide information about the position of the head in space, contributing to maintenance of posture and balance.

There are three semicircular canals, one lying in each of the three planes of space. They are situated above, beside and behind the vestibule of the inner ear and open into it.

The semicircular canals, like the cochlea, are composed of an outer bony wall and inner membranous tubes or *ducts*. The membranous ducts contain endolymph and are separated from the bony wall by perilymph.

The utricle is a membranous sac which is part of the vestibule and the three membranous ducts open into it at their dilated ends, the *ampullae*. The saccule is a part of the vestibule and communicates with the utricle and the cochlea.

In the walls of the utricle, saccule and ampullae are fine, specialised epithelial cells with minute projections, called *hair cells*. Amongst the hair cells there are receptors on sensory nerve endings, which combine forming the vestibulocochlear nerve.

Physiology of balance

The semicircular canals and the vestibule (utricle and saccule) are concerned with balance, or *equilibrium*. The arrangement of the three semicircular canals, one in each plane, not only allows perception of the position of the head in space but also the direction and rate of any movement. Any change of position of the head causes movement in the perilymph and endolymph, which bends the hair cells and stimulates the sensory receptors in the utricle, saccule and ampullae. The resultant nerve impulses are transmitted by the vestibular nerve, which joins the cochlear nerve to form the vestibulocochlear nerve. The vestibular branch passes first to the *vestibular nucleus*, then to the cerebellum.

The cerebellum also receives nerve impulses from the eyes and proprioceptors (sensory receptors) in the skeletal muscles and joints. The cerebellum coordinates incoming impulses from the vestibular nerve, the eyes and proprioceptors. Thereafter, impulses are transmitted to the cerebrum and skeletal muscles enabling perception of body position and any adjustments needed to maintain posture and balance. This maintains upright posture and fixing of the eyes on the same point, independently of head movements.

Sight and the eye

Learning outcomes

After studying this section, you should be able to:

- describe the gross structure of the eye
- describe the route taken by nerve impulses from the retina to the cerebrum
- explain how light entering the eye is focused on the retina
- state the functions of the extraocular eye muscles
- explain the functions of the accessory organs of the eye.

The eye is the organ of sight. It is situated in the orbital cavity and supplied by the *optic nerve* (2nd cranial nerve).

It is almost spherical in shape and about 2.5 cm in diameter. The space between the eye and the orbital cavity is occupied by adipose tissue. The bony walls of the orbit and the fat protect the eye from injury.

Structurally the two eyes are separate but, unlike the ears, some of their activities are coordinated so that they normally function as a pair. It is possible to see with only one eye (monocular vision), but three-dimensional vision is impaired when only one eye is used, especially in relation to the judgement of speed and distance.

Structure (Fig. 8.8)

There are three layers of tissue in the walls of the eye:

- the outer fibrous layer: *sclera* and *cornea*
- the middle vascular layer or *uveal tract*: consisting of the choroid, ciliary body and iris
- the inner nervous tissue layer: the *retina*.

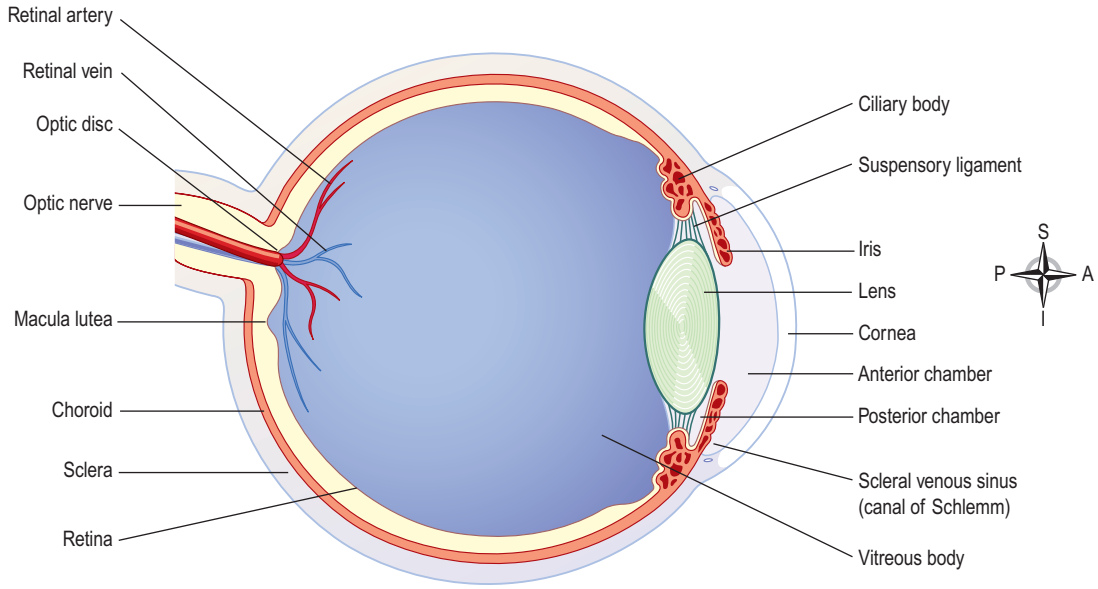

Figure 8.8 Section of the eye.

Structures inside the eyeball include the lens, aqueous fluid and vitreous body.

Sclera and cornea

The sclera, or white of the eye, forms the outermost layer of the posterior and lateral aspects of the eyeball and is continuous anteriorly with the cornea. It consists of a firm fibrous membrane that maintains the shape of the eye and gives attachment to the *extrinsic muscles* of the eye (see Table 8.1, p. 204).

Anteriorly the sclera continues as a clear transparent epithelial membrane, the cornea. Light rays pass through the cornea to reach the retina. The cornea is convex anteriorly and is involved in refracting (bending) light rays to focus them on the retina.

Choroid (Figs 8.8 and 8.9)

The choroid lines the posterior five-sixths of the inner surface of the sclera. It is very rich in blood vessels and is deep chocolate brown in colour. Light enters the eye through the pupil, stimulates the sensory receptors in the retina (p. 198) and is then absorbed by the choroid.

Ciliary body

The ciliary body is the anterior continuation of the choroid consisting of *ciliary muscle* (smooth muscle fibres) and secretory epithelial cells. As many of the smooth muscle fibres are circular, the ciliary muscle acts like a sphincter. The lens is attached to the ciliary body by radiating *suspensory ligaments*, like the spokes of a wheel (see Fig. 8.10). Contraction and relaxation of the ciliary muscle fibres, which are attached to these ligaments, control the size and thickness of the lens. The epithelial cells secrete *aqueous fluid* into the anterior segment of the eye, i.e. the space between the lens and the cornea (anterior and posterior chambers) (Fig. 8.8). The ciliary body is supplied by parasympathetic branches of the oculomotor nerve (3rd cranial nerve). Stimulation causes contraction of the ciliary muscle and accommodation of the eye (p. 202).

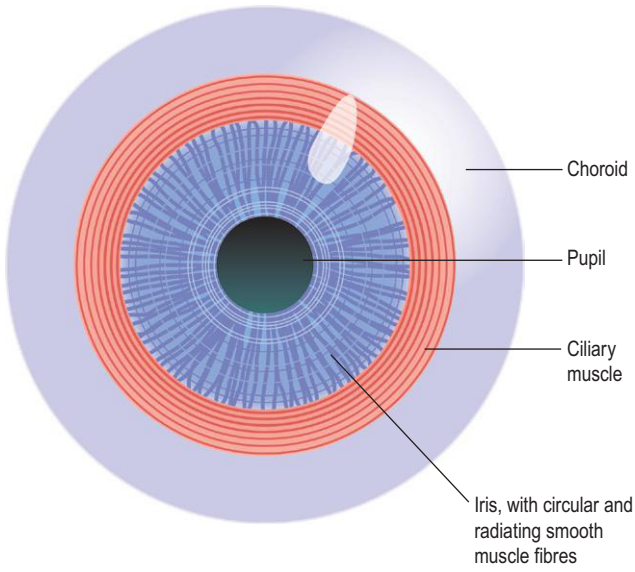

Figure 8.9 The choroid, ciliary body and iris. Viewed from the front.

Iris

The iris is the visible coloured ring at the front of the eye and extends anteriorly from the ciliary body, lying behind the cornea and in front of the lens. It divides the *anterior*

segment of the eye into anterior and posterior chambers which contain aqueous fluid secreted by the ciliary body. It is a circular body composed of pigment cells and two layers of smooth muscle fibres, one circular and the other radiating (Fig. 8.9). In the centre is an aperture called the *pupil*.

The iris is supplied by parasympathetic and sympathetic nerves. Parasympathetic stimulation constricts the pupil and sympathetic stimulation dilates it (see Figs 7.44 and 7.43, respectively, pp. 174 and 175).

The colour of the iris is genetically determined and depends on the number of pigment cells present. Albinos have no pigment cells and people with blue eyes have fewer than those with brown eyes.

Lens (Fig. 8.10)

The lens is a highly elastic circular biconvex body, lying immediately behind the pupil. It consists of fibres enclosed within a capsule and is suspended from the ciliary body by the suspensory ligament. Its thickness is controlled by the ciliary muscle through the suspensory ligament. The lens bends (refracts) light rays reflected by objects in front of the eye. It is the only structure in the eye that can vary its refractory power, which is achieved by changing its thickness.

When the ciliary muscle contracts, it moves forward, releasing its pull on the lens, increasing its thickness. The nearer is the object being viewed, the thicker the lens becomes to allow focusing (see Fig. 8.18).

Retina

The retina is the innermost lining of the eye (Fig. 8.8). It is an extremely delicate structure and well adapted for stimulation by light rays. It is composed of several layers of nerve cell bodies and their axons, lying on a pigmented layer of epithelial cells. The light-sensitive layer consists of sensory receptor cells, *rods* and *cones*, which contain photosensitive pigments that convert light rays into nerve impulses.

The retina lines about three-quarters of the eyeball and is thickest at the back. It thins out anteriorly to end just behind the ciliary body. Near the centre of the posterior part is the *macula lutea*, or yellow spot (Figs 8.11A and 8.12). In the centre of the yellow spot is a little depression called the *fovea centralis*, consisting of only cones. Towards the anterior part of the retina there are fewer cones than rods.

About 0.5 cm to the nasal side of the macula lutea all the nerve fibres of the retina converge to form the optic nerve. The small area of retina where the optic nerve

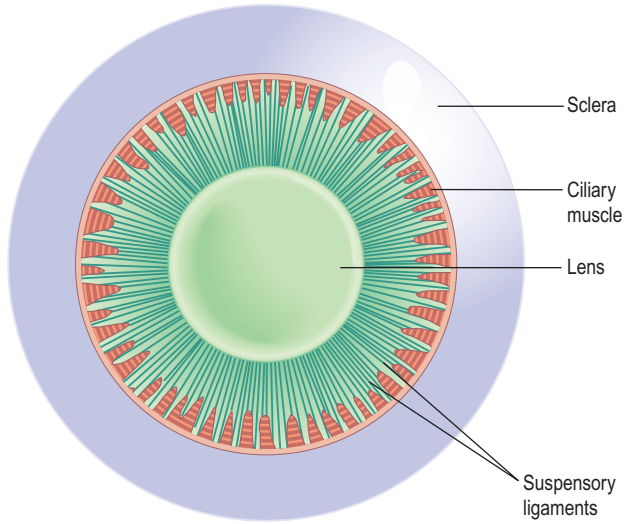

Figure 8.10 The lens and suspensory ligaments viewed from the front. The iris has been removed.

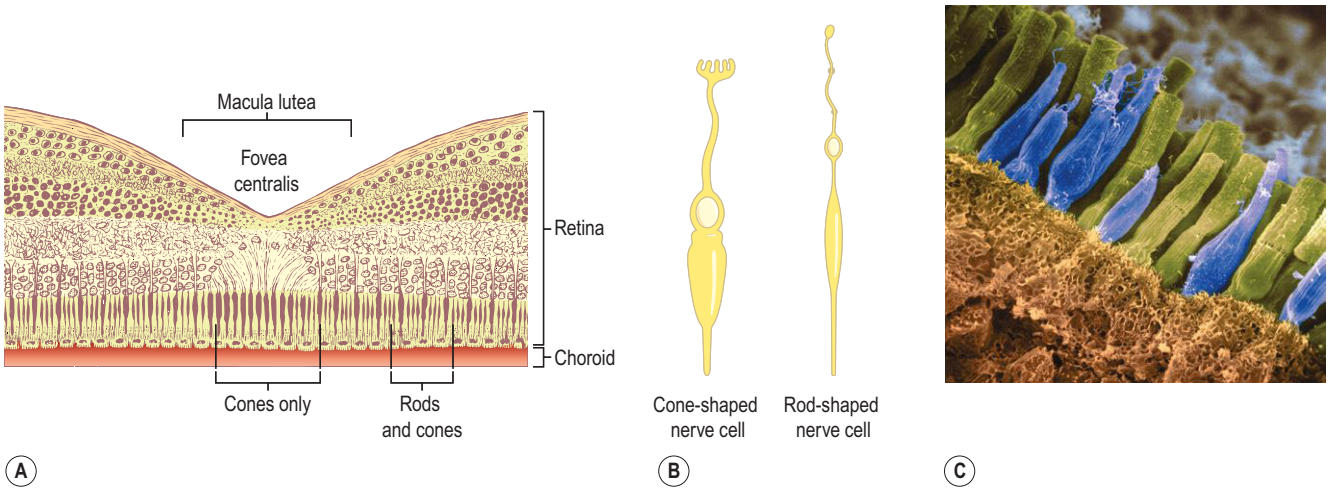

Figure 8.11 The retina. A. Magnified section. **B.** Light-sensitive nerve cells: rods and cones. **C.** Coloured scanning electron micrograph of rods (green) and cones (blue).

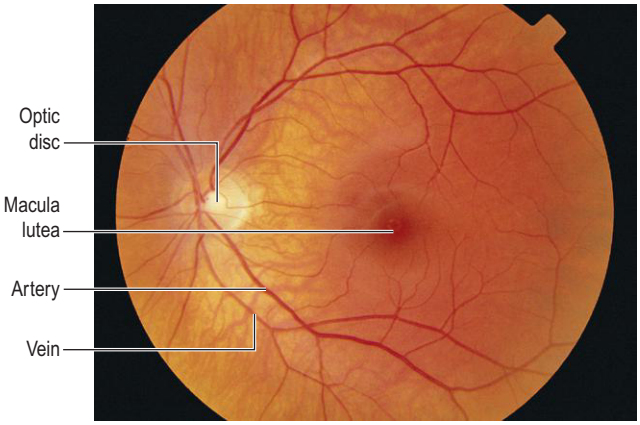

Figure 8.12 The retina as seen through the pupil with an ophthalmoscope.

leaves the eye is the *optic disc* or *blind spot*. It has no light-sensitive cells.

Blood supply to the eye

The eye is supplied with arterial blood by the *ciliary arteries* and the *central retinal artery*. These are branches of the ophthalmic artery, a branch of the internal carotid artery.

Venous drainage is by a number of veins, including the *central retinal vein*, which eventually empty into a deep venous sinus.

The central retinal artery and vein are encased in the optic nerve, which enters the eye at the optic disc (Fig. 8.8).

Interior of the eye

The anterior segment of the eye, i.e. the space between the cornea and the lens, is incompletely divided into anterior and posterior chambers by the iris (Fig. 8.8). Both chambers contain a clear aqueous fluid secreted into the posterior chamber by the ciliary glands. It circulates in front of the lens, through the pupil into the anterior chamber and returns to the venous circulation through the *scleral venous sinus* (canal of Schlemm) in the angle between the iris and cornea (Fig. 8.8). The intraocular pressure remains fairly constant between 1.3 and 2.6 kPa (10 to 20 mmHg) as production and drainage rates of aqueous fluid are equal. An increase in this pressure causes *glaucoma* (p. 211). Aqueous fluid supplies nutrients and removes wastes from the transparent structures in the front of the eye that have no blood supply, i.e. the cornea, lens and lens capsule.

Behind the lens and filling the posterior segment (cavity) of the eyeball is the *vitreous body*. This is a soft, colourless, transparent, jelly-like substance composed of 99% water, some salts and mucoprotein. It maintains sufficient intraocular pressure to support the retina against the choroid and prevent the eyeball from collapsing.

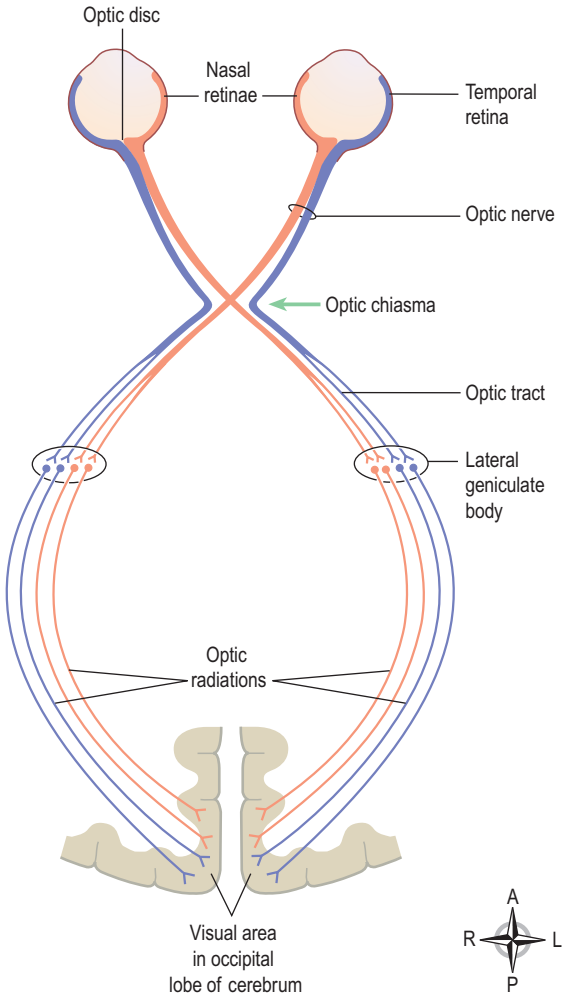

Figure 8.13 The optic nerves and their pathways.

The eye keeps its shape because of the intraocular pressure exerted by the vitreous body and the aqueous fluid. It remains fairly constant throughout life.

Optic nerves (second cranial nerves) (Fig. 8.13)

The fibres of the optic nerve originate in the retina and they converge to form the optic nerve about 0.5 cm to the nasal side of the macula lutea at the optic disc. The nerve pierces the choroid and sclera to pass backwards and medially through the orbital cavity. It then passes through the optic foramen of the sphenoid bone, backwards and medially to meet the nerve from the other eye at the *optic chiasma*.

Optic chiasma

This is situated immediately in front of and above the pituitary gland, which is in the hypophyseal fossa of the sphenoid bone (see Fig. 9.2, p. 217). In the optic chiasma the nerve fibres of the optic nerve from the nasal side of each retina cross over to the opposite side. The fibres from the temporal side do not cross but continue backwards

on the same side. This crossing over provides both cerebral hemispheres with sensory input from each eye.

Optic tracts

These are the pathways of the optic nerves, posterior to the optic chiasma (Fig. 8.13). Each tract consists of the nasal fibres from the retina of one eye and the temporal fibres from the retina of the other. The optic tracts pass backwards to synapse with nerve cells of the *lateral geniculate bodies* of the thalamus. From there the nerve fibres proceed backwards and medially as the *optic radiations* to terminate in the *visual area* of the cerebral cortex in the occipital lobes of the cerebrum (see Fig. 7.20, p. 157). Other neurones originating in the lateral geniculate bodies transmit impulses from the eyes to the cerebellum where, together with impulses from the semicircular canals of the inner ears and from the skeletal muscles and joints, they contribute to the maintenance of posture and balance.

Physiology of sight 8.2

Light waves travel at a speed of 300 000 kilometres (186 000 miles) per second. Light is reflected into the eyes by objects within the field of vision. White light is a combination of all the colours of the visual spectrum (rainbow), i.e. red, orange, yellow, green, blue, indigo and violet. This is demonstrated by passing white light through a glass prism which bends the rays of the different colours to a greater or lesser extent, depending on their wavelengths (Fig. 8.14). Red light has the longest wavelength and violet the shortest.

This range of colour is the *spectrum of visible light*. In a rainbow, white light from the sun is broken up by raindrops, which act as prisms and reflectors.

The electromagnetic spectrum

The electromagnetic spectrum is broad, but only a small part is visible to the human eye (Fig. 8.15). Beyond the long end are infrared waves (heat), microwaves and radio waves. Beyond the short end are ultraviolet (UV), X-rays and gamma rays. UV light is not normally visible because it is absorbed by a yellow pigment in the lens. Following removal of the lens (cataract extraction), it is usually replaced with an artificial one to prevent long term damage to the retina from UV light rays.

A specific colour is perceived when only one wavelength is reflected by the object and all the others are absorbed, e.g. an object appears red when it only reflects red light. Objects appear white when all wavelengths are reflected, and black when they are all absorbed.

In order to achieve clear vision, light reflected from objects within the visual field is focused on to the retina of each eye. The processes involved in producing a clear image are *refraction of the light rays*, changing the *size of the pupils* and *accommodation* (adjustment of the lens for near vision, see p. 202).

Although these may be considered as separate processes, effective vision is dependent upon their coordination.

Refraction of the light rays

When light rays pass from a medium of one density to a medium of a different density they are bent; for example, a glass prism (Fig. 8.14). In the eye, the biconvex lens bends and focuses light rays (Fig. 8.16). This principle is

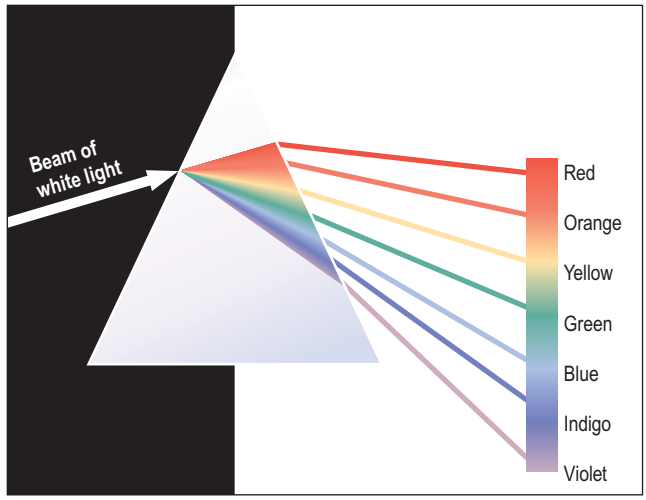

Figure 8.14 Refraction: white light broken into the colours of the visible spectrum when it passes through a glass prism.

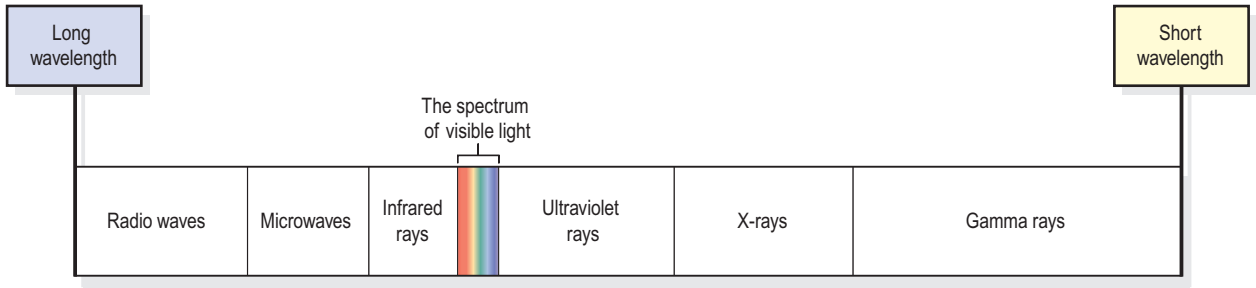

Figure 8.15 The electromagnetic spectrum.

used to focus light on the retina. Before reaching the retina, light rays pass successively through the conjunctiva, cornea, aqueous fluid, lens and vitreous body. They are all denser than air and, with the exception of the lens, they have a constant refractory power, similar to that of water.

Focusing of an image on the retina

Light rays reflected from an object are bent (refracted) by the lens when they enter the eye in the same way as shown in Figure 8.16, although the image on the retina is actually upside down (Fig. 8.17). The brain adapts to this early in life so that objects are perceived 'the right way up'.

Abnormal refraction within the eye is corrected using biconvex or biconcave lenses, which are shown on page 212.

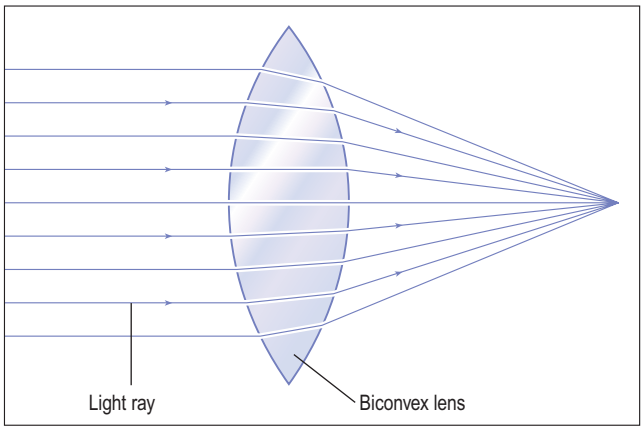

Figure 8.16 Refraction of light rays passing through a biconvex lens.

Lens

The lens is a biconvex elastic transparent body suspended behind the iris from the ciliary body by the suspensory ligament (Fig. 8.10). It is the only structure in the eye able to change its refractive power. Light rays entering the eye need to be refracted to focus them on the retina. Light from distant objects needs least refraction and, as the object comes closer, the amount of refraction needed increases. To focus light rays from near objects on the retina, the refractory power of the lens must be increased – by accommodation. To do this, the ciliary muscle (a sphincter) contracts moving the ciliary body inwards towards the lens. This lessens the pull on the suspensory ligaments and allows the lens to bulge, increasing its convexity and focusing light rays on the retina (see Fig. 8.18B).

To focus light rays from distant objects on the retina, the ciliary muscle relaxes, increasing its pull on the suspensory ligaments. This makes the lens thinner and focuses light rays from distant objects on the retina (see Fig. 8.18A).

Size of the pupils

Pupil size contributes to clear vision by controlling the amount of light entering the eye. In bright light the pupils are constricted. In dim light they are dilated.

If the pupils were dilated in bright light, too much light would enter the eye and damage the sensitive retina. In dim light, if the pupils were constricted, insufficient light would enter the eye to activate the light-sensitive pigments in the rods and cones, which stimulate the nerve endings in the retina enabling vision.

The iris consists of one layer of circular and one of radiating smooth muscle fibres. Contraction of the

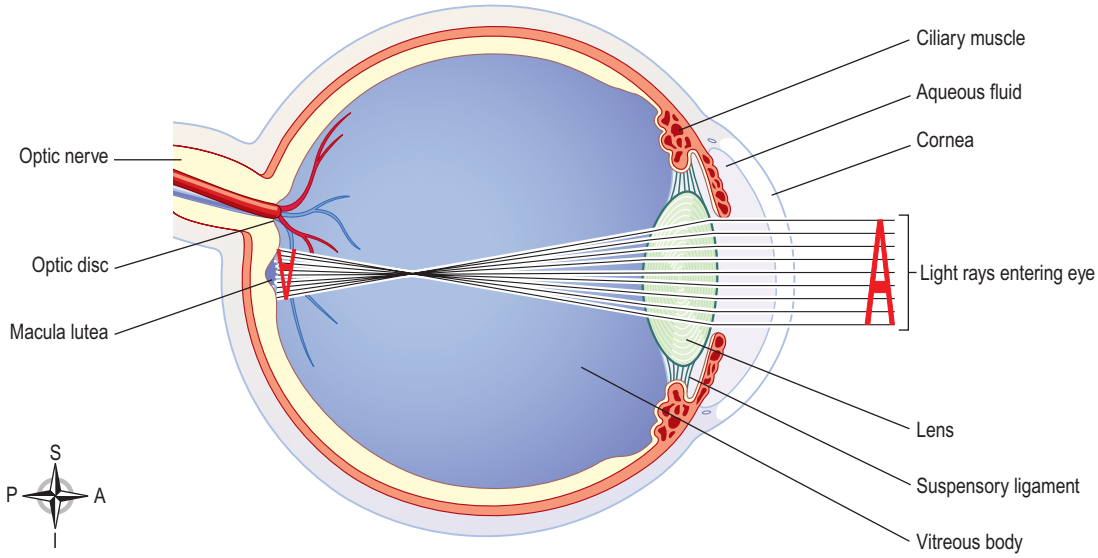

Figure 8.17 Section of the eye showing the focusing of light rays on the retina.

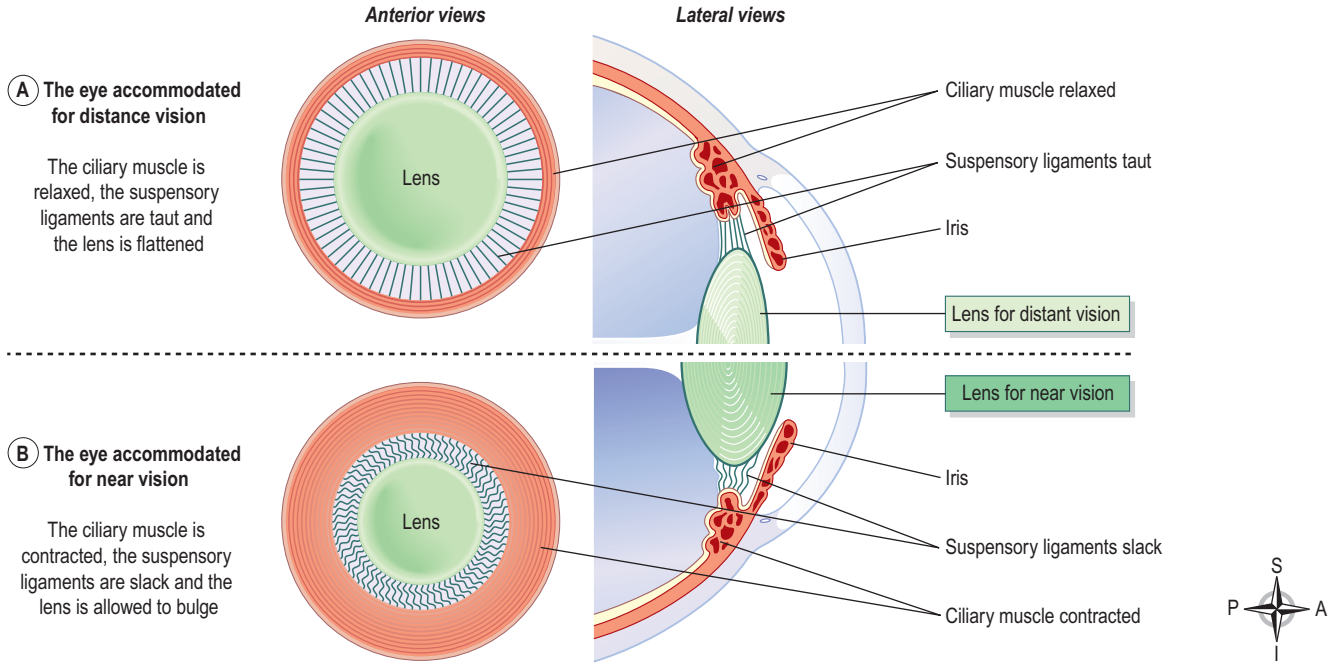

Figure 8.18 Accommodation: action of the ciliary muscle on the shape of the lens. A. Distant vision. **B.** Near vision.

circular fibres constricts the pupil, and contraction of the radiating fibres dilates it. The size of the pupil is controlled by the autonomic nervous system; sympathetic stimulation dilates the pupils and parasympathetic stimulation constricts them.

Accommodation

Near vision

In order to focus on near objects, i.e. within about 6 metres, accommodation is required and the eye must make the following adjustments:

- constriction of the pupils
- convergence
- changing the refractory power of the lens.

Constriction of the pupils. This assists accommodation by reducing the width of the beam of light entering the eye so that it passes through the central curved part of the lens (Fig. 8.17).

Convergence (movement of the eyeballs). Light rays from nearby objects enter the two eyes at different angles and for clear vision they must stimulate corresponding areas of the two retinae. Extrinsic muscles move the eyes and to obtain a clear image they rotate the eyes so that they converge on the object viewed. This coordinated muscle activity is under autonomic control. When there is voluntary movement of the eyes, both eyes move and convergence is maintained. The nearer an object is to the eyes the greater the eye rotation needed to achieve convergence, e.g. focusing near the tip of one's nose gives the appearance of being 'cross-eyed'. If convergence is not complete, the eyes are focused on different objects or on different points of the same object. There are then two images sent to the brain and this can lead to double vision, *diplopia*. If convergence is not possible, the brain tends to ignore the impulses received from the divergent eye (see Squint, p. 211).

Changing the refractory power of the lens. Changes in the thickness of the lens are made to focus light on the retina. The amount of adjustment depends on the distance of the object from the eyes, i.e. the lens is thicker for near vision and at its thinnest when focusing on objects more than 6 metres away (Fig. 8.18). Looking at near objects 'tires' the eyes more quickly, owing to the continuous use of the ciliary muscle. The lens loses its elasticity and stiffens with age, a condition known as *presbyopia* (p. 208).

Distant vision

Objects more than 6 metres away from the eyes are focused on the retina without adjustment of the lens or convergence of the eyes.

Functions of the retina

The retina is the light-sensitive (photosensitive) part of the eye. The light-sensitive nerve cells are the rods and cones and their distribution in the retina is shown in Figure 8.11A. Light rays cause chemical changes in light-sensitive pigments in these cells and they generate nerve impulses which are conducted to the occipital lobes of the cerebrum via the optic nerves (Fig. 8.13).

The *rods* are much more light sensitive than the cones (see Fig. 8.11), so they are used when light levels are low. Stimulation of rods leads to monochromic (black and white) vision. Rods outnumber cones in the retina by about 16:1 and are more numerous towards the periphery of the retina. *Visual purple* (*rhodopsin*) is a light-sensitive pigment present only in the rods. It is bleached (degraded) by bright light and is quickly regenerated, provided an adequate supply of vitamin A is available.

The *cones* are sensitive to light and colour; bright light is required to activate them and give sharp, clear colour vision. The different wavelengths of visible light light-sensitive pigments in the cones, resulting in the perception of different colours.

Colour blindness. This is a common condition that affects more men than women. Although affected individuals see colours, they cannot always differentiate between them as the light-sensitive pigments (to red, green or blue) in cones are abnormal. There are different forms but the most common is red–green colour blindness which is transmitted a by sex-linked recessive gene (see Fig. 17.11, p. 445) where greens, oranges, pale reds and browns all appear to be the same colour and can only be distinguished by their intensity.

Dark adaptation. When exposed to bright light, the rhodopsin within the sensitive rods is completely degraded. This does not affect vision in good light, when there is enough light to activate the cones. However, moving into a darkened area where the light intensity is insufficient to stimulate the cones causes temporary visual impairment whilst the rhodopsin is being regenerated within the rods, 'dark adaptation'. When regeneration of rhodopsin has occurred, normal sight returns.

It is easier to see a dim star in the sky at night if the head is turned slightly away from it because light of low intensity is then focused on an area of the retina where there is a greater concentration of rods. If looked at directly, the light intensity of a dim star is not sufficient to stimulate the less sensitive cones in the area of the macula lutea. In dim evening light, colours cannot be distinguished because the light intensity is insufficient to stimulate colour-sensitive pigments in cones.

Breakdown and regeneration of the visual pigments in cones is similar to that of rods.

Binocular vision 8.3

Binocular or stereoscopic vision enables three-dimensional views although each eye 'sees' a scene from a slightly different angle (Fig. 8.19). The visual fields overlap in the middle but the left eye sees more on the left than can be seen by the other eye and vice versa. The images from the two eyes are fused in the cerebrum so that only one image is perceived.

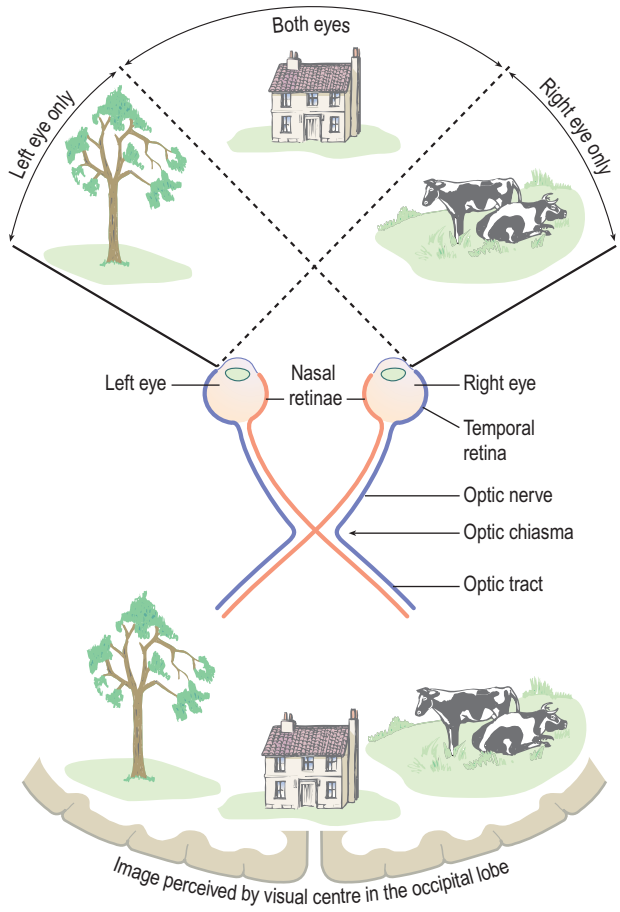

Figure 8.19 Parts of the visual field: monocular and binocular.

Binocular vision provides a much more accurate assessment of one object relative to another, e.g. its distance, depth, height and width. People with monocular vision may find it difficult, for example, to judge the speed and distance of an approaching vehicle.

Extraocular muscles of the eye

These include the muscles of the eyelids and those that move the eyeballs. The eyeball is moved by six *extrinsic muscles*, attached at one end to the eyeball and at the other to the walls of the orbital cavity. There are four *straight* (rectus) muscles and two *oblique* muscles (Fig. 8.20).

Moving the eyes to look in a particular direction is under voluntary control, but coordination of movement, needed for convergence and accommodation to near or distant vision, is under autonomic (involuntary) control. Movements of the eyes resulting from the action of these muscles are shown in Table 8.1.

Nerve supply to the muscles of the eye

Table 8.1 shows the nerves that supply the extrinsic muscles. The *oculomotor nerves* supply the *intrinsic eye muscles* of the iris and ciliary body.

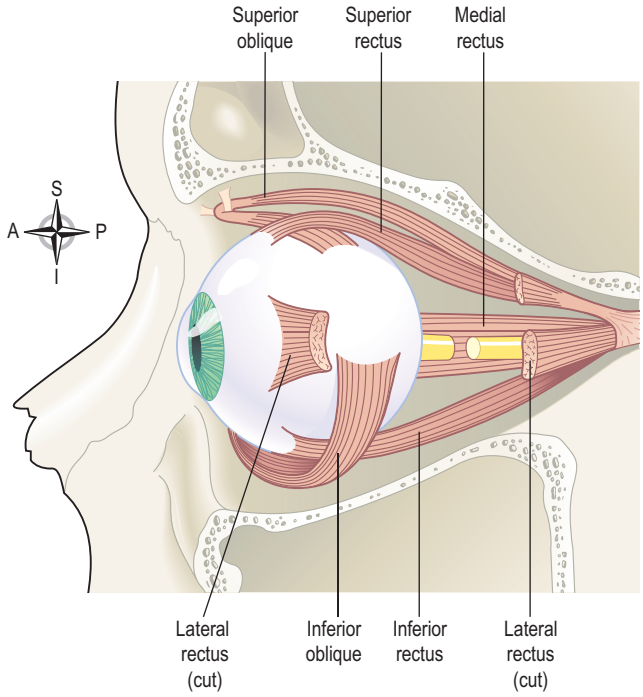

Figure 8.20 The extrinsic muscles of the eye.

Table 8.1 Extrinsic muscles of the eye: their actions and cranial nerve supply

Name	Action	Cranial nerve supply
Medial rectus	Rotates eyeball inwards	Oculomotor nerve (3rd cranial nerve)
Lateral rectus	Rotates eyeball outwards	Abducent nerve (6th cranial nerve)
Superior rectus	Rotates eyeball upwards	Oculomotor nerve (3rd cranial nerve)
Inferior rectus	Rotates eyeball downwards	Oculomotor nerve (3rd cranial nerve)
Superior oblique	Rotates eyeball downwards and outwards	Trochlear nerve (4th cranial nerve)
Inferior oblique	Rotates eyeball upwards and outwards	Oculomotor nerve (3rd cranial nerve)

Accessory organs of the eye

The eye is a delicate organ which is protected by several structures (Fig. 8.21):

- eyebrows
- eyelids and eyelashes
- lacrimal apparatus.

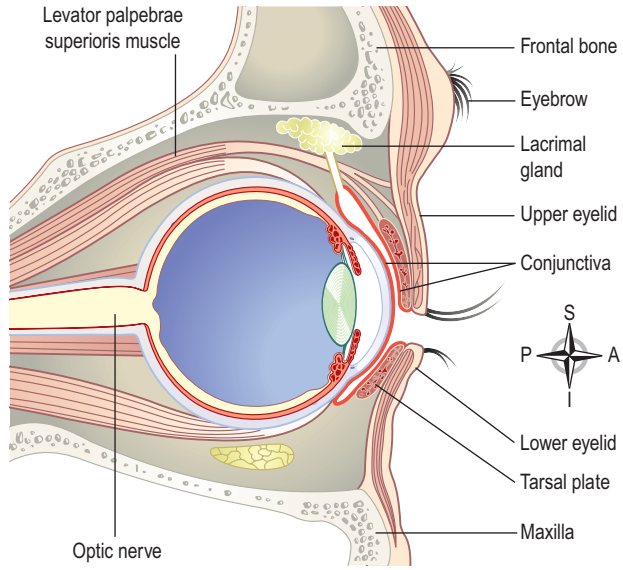

Figure 8.21 Section of the eye and its accessory structures.

Eyebrows

These are two arched ridges of the supraorbital margins of the frontal bone. Numerous hairs (eyebrows) project obliquely from the surface of the skin. They protect the eyeball from sweat, dust and other foreign bodies.

Eyelids (palpebrae)

The eyelids are two movable folds of tissue situated above and below the front of each eye. On their free edges are short curved hairs, the *eyelashes*. The layers of tissue forming the eyelids are:

- a thin covering of skin
- a thin sheet of subcutaneous connective (loose areolar) tissue
- two muscles – the *orbicularis oculi* and *levator palpebrae superioris*
- a thin sheet of dense connective tissue, the *tarsal plate*, larger in the upper than the lower eyelid, which supports the other structures
- a membranous lining, the *conjunctiva*.

Conjunctiva

This is a fine transparent membrane that lines the eyelids and the front of the eyeball (Fig. 8.21). Where it lines the eyelids it consists of highly vascular columnar epithelium. Corneal conjunctiva consists of avascular stratified epithelium, i.e. epithelium without blood vessels. When the eyelids are closed the conjunctiva becomes a closed sac. It protects the delicate cornea and the front of the eye. When eyedrops are administered they are placed in the lower conjunctival sac. The medial and lateral angles of the eye where the upper and lower lids come together are called respectively the *medial canthus* and the *lateral canthus*.

Eyelid margins

Along the edges of the lids are numerous *sebaceous glands*, some with ducts opening into the hair follicles of the eyelashes and some on to the eyelid margins between the hairs. *Tarsal glands* are modified sebaceous glands embedded in the tarsal plates with ducts that open on to the inside of the free margins of the eyelids. They secrete an oily material, spread over the conjunctiva by blinking, which delays evaporation of tears.

Functions

The eyelids and eyelashes protect the eye from injury:

- reflex closure of the lids occurs when the conjunctiva or eyelashes are touched, when an object comes close to the eye or when a bright light shines into the eye – this is called the *corneal reflex*
- blinking at about 3- to 7-second intervals spreads tears and oily secretions over the cornea, preventing drying.

When the orbicularis oculi contract, the eyes close. When the levator palpebrae contract, the eyelids open (see Fig. 16.58, p. 424).

Lacrimal apparatus (Fig. 8.22)

For each eye this consists of the structures that secrete tears and drain them from the front of the eyeball:

- 1 lacrimal gland and its ducts
- 2 lacrimal canaliculi
- 1 lacrimal sac
- 1 nasolacrimal duct.

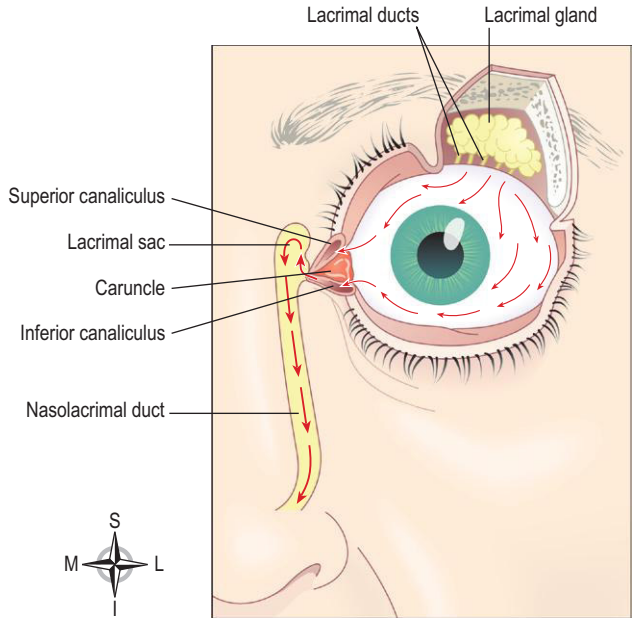

Figure 8.22 The lacrimal apparatus. Arrows show the direction of the flow of tears.

The lacrimal glands are exocrine glands situated in recesses in the frontal bones on the lateral aspect of each eye just behind the supraorbital margin. Each gland is approximately the size and shape of an almond, and is composed of secretory epithelial cells. The glands secrete *tears* composed of water, mineral salts, antibodies (immunoglobulins, see Ch. 15) and *lysozyme*, a bactericidal enzyme.

The tears leave the lacrimal gland by several small ducts and pass over the front of the eye under the lids towards the medial canthus where they drain into the *two lacrimal canaliculi*; the opening of each is called the *punctum*. The two canaliculi lie one above the other, separated by a small red body, the *caruncle*. The tears then drain into the *lacrimal sac*, which is the upper expanded end of the *nasolacrimal duct*. This is a membranous canal approximately 2 cm long, extending from the lower part of the lacrimal sac to the nasal cavity, opening at the level of the inferior concha. Normally the rate of secretion of tears keeps pace with the rate of drainage. When a foreign body or other irritant enters the eye the secretion of tears is greatly increased and the conjunctival blood vessels dilate. Secretion of tears is also increased in emotional states, e.g. crying, laughing.

Functions

The fluid that fills the conjunctival sac is a mixture of tears and the oily secretion of tarsal glands, which is spread over the cornea by blinking. The functions of this fluid include:

- provision of oxygen and nutrients to the avascular corneal conjunctiva and drainage of wastes
- washing away irritating materials, e.g. dust, grit
- the bactericidal enzyme *lysozyme* prevents microbial infection
- its oiliness delays evaporation and prevents friction or drying of the conjunctiva.

Sense of smell

Learning outcome

After studying this section, you should be able to:

- describe the physiology of smell.

The sense of smell, or *olfaction*, originates in the nasal cavity, which also acts as a passageway for respiration (see Ch. 10).

Olfactory nerves (first cranial nerves)

These are the sensory nerves of smell. They originate as *chemoreceptors* (specialised olfactory nerve endings) in the mucous membrane of the roof of the nasal cavity above

the superior nasal conchae (Fig. 8.23). On each side of the nasal septum nerve fibres pass through the cribriform plate of the ethmoid bone to the *olfactory bulb* where interconnections and synapses occur (Fig. 8.24). From the bulb, bundles of nerve fibres form the *olfactory tract*, which passes backwards to the olfactory area in the temporal lobe of the cerebral cortex in each hemisphere where the impulses are interpreted and odour perceived (see Fig. 7.20, p. 157). 8.4

Physiology of smell

The human sense of smell is less acute than in other animals. Many animals secrete odorous chemicals called *pheromones*, which play an important part in chemical communication in, for example, territorial behaviour, mating and the bonding of mothers and their newborn. The role of pheromones in human communication is unknown.

All odorous materials give off volatile molecules, which are carried into the nose with inhaled air and even very low concentrations, when dissolved in mucus, stimulate the olfactory chemoreceptors.

The air entering the nose is warmed, and convection currents carry eddies of inspired air to the roof of the nasal cavity. 'Sniffing' concentrates volatile molecules in the roof of the nose. This increases the number of olfactory receptors stimulated and thus perception of the smell. The sense of smell and the sense of taste are closely related; the sense of smell may affect the appetite. If the odours are pleasant the appetite may improve and vice versa. When accompanied by the sight of food, an appetising smell increases salivation and stimulates the digestive system (see Ch. 12). The sense of smell and the sense of taste are closely related; the sense of smell may create powerful and long-lasting memories, especially for distinctive odours, e.g. hospital smells, favourite or least-liked foods.

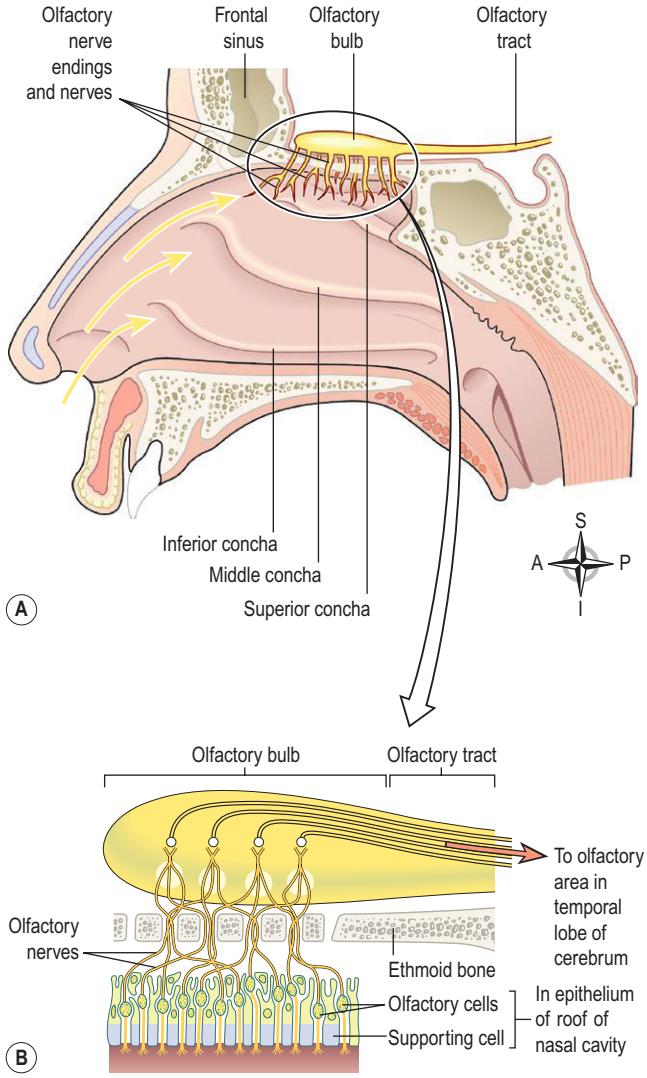

Figure 8.23 The sense of smell. A. The olfactory structures. **B.** An enlarged section of the olfactory apparatus in the nose and on the inferior surface of the cerebrum.

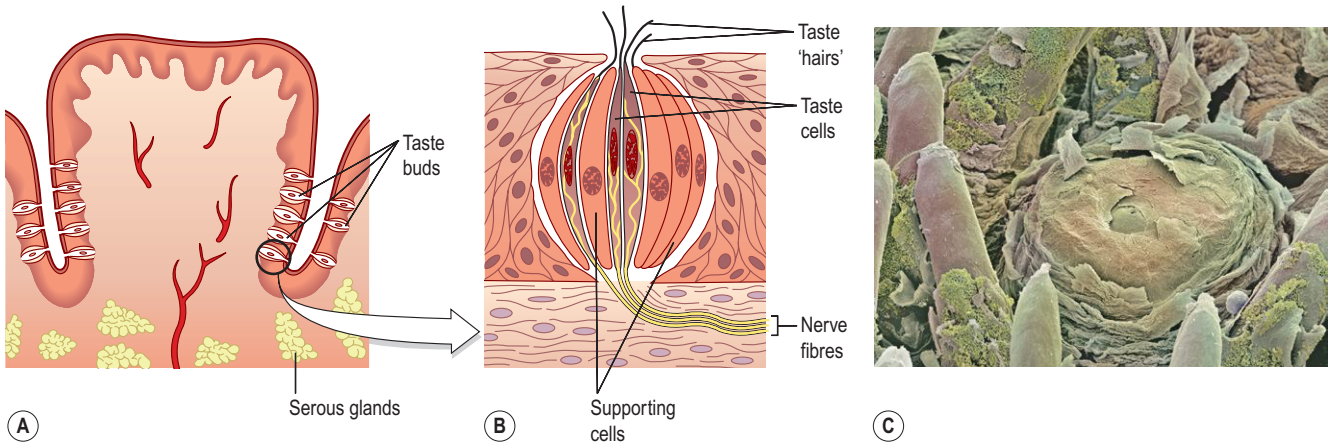

Figure 8.24 Structure of taste buds. A. A section of a papilla. **B.** A taste bud – greatly magnified. **C.** Coloured scanning electron micrograph of a taste bud (centre) on the tongue.

Inflammation of the nasal mucosa prevents odorous substances from reaching the olfactory area of the nose, causing loss of the sense of smell (*anosmia*). The usual cause is a cold.

Adaptation. When an individual is continuously exposed to an odour, perception of the odour decreases and ceases within a few minutes. This loss of perception affects only that specific odour.

Sense of taste

Learning outcome

After studying this section, you should be able to:

- describe the physiology of taste.

The sense of taste, or *gustation*, is closely linked to the sense of smell and, like smell, also involves stimulation of chemoreceptors by dissolved chemicals.

Taste buds contain chemoreceptors (sensory receptors) that are found in the papillae of the tongue and widely distributed in the epithelia of the tongue. They consist of small sensory nerve endings of the glossopharyngeal, facial and vagus nerves (cranial nerves VII, IX and X). Some of the cells have hair-like cilia on their free border, projecting towards tiny pores in the epithelium (Fig. 8.24). The sensory receptors are highly sensitive and stimulated by very small amounts of chemicals that enter the pores dissolved in saliva. Nerve impulses are generated and conducted along the glossopharyngeal, facial and vagus nerves before synapsing in the medulla and thalamus. Their final destination is the *taste area* in the parietal lobe of the cerebral cortex where taste is perceived (see Fig. 7.20, p. 157).

Physiology of taste

Four fundamental sensations of taste have been described – sweet, sour, bitter and salt; however, others have also been suggested, including metallic and umami (a Japanese 'savoury' taste). However, perception varies widely and many 'tastes' cannot be easily classified. It is thought that all taste buds are stimulated by all 'tastes'. Taste is impaired when the mouth is dry, because substances can only be 'tasted' when in solution.

The sense of taste is closely linked to the sense of smell. For example when one has a cold, it is common for food to taste bland and unappealing. In addition, taste triggers salivation and the secretion of gastric juice (see Ch. 12). The sense of taste also has a protective function, e.g. when foul-tasting food is eaten, reflex gagging or vomiting may be induced.

The effect of ageing on the special senses

Learning outcome

After studying this section, you should be able to:

- describe the impact of ageing on the special senses.

Changes in hearing and vision that occur as part of normal ageing are almost universal and often accompanied by diminished senses of taste and smell. The number of olfactory receptors reduces around the age of 50, diminishing the sense of taste; older adults may complain of their food being bland while children can find the same food too spicy. In a similar way, older adults may not smell (perceive) weak odours. The effect of changes associated with ageing on hearing and vision are considered below.

Presbycusis

This form of hearing impairment accompanies the ageing process and is therefore common in older adults. Degenerative changes in the sensory cells of the spiral organ result in sensorineural hearing loss (p. 209). Perception of high-frequency sound is impaired first and later low-frequency sound may also be affected. Difficulty in discrimination develops, e.g. following a conversation, especially in the presence of background noise.

Vision

Presbyopia and cataracts are common consequences of normal ageing.

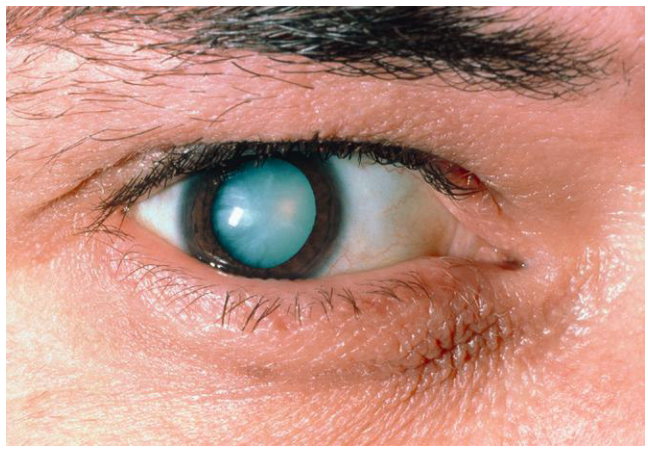

Figure 8.25 Cataract.

Presbyopia

Age-related changes in the lens lead to loss of accommodation as the lens loses its elasticity and becomes firmer. This prevents focusing of light on the retina, giving blurred vision. Correction is achieved using glasses with convex lenses for near vision, e.g. reading (see Fig. 8.27).

Cataracts

Cataracts arise when there is opacity of the lens (Fig. 8.25). Weak light rays cannot easily pass through a less transparent or cloudy lens and is the reason why many older adults use brighter light for reading and may also experience difficulty with night vision. It is most commonly age-related occurring as a result of exposure to predisposing factors which include UV light, X-rays and cigarette smoke. There are also other important causes of cataracts (p. 211).

Disorders of the ear

Learning outcomes

After studying this section, you should be able to:

- compare and contrast the features of conductive and sensorineural hearing loss
- describe the causes and effects of diseases of the ear.

Hearing loss

Hearing impairment can be classified in two main categories: *conductive* and *sensorineural*. Hearing impairment can also be mixed when there is a combination of conductive and sensorineural hearing loss in one ear.

Conductive hearing impairment

This occurs when an abnormality of the outer or middle ear impairs conduction of sound waves to the oval window; common examples are listed in Box 8.1.

Otosclerosis. This is a common cause of progressive conductive hearing loss in young adults that may affect one ear but is more commonly bilateral. It is usually hereditary, more common in females than males and often worsens during pregnancy. Abnormal bone develops around the footplate of the stapes, fusing it to the oval window, reducing the ability to transmit sound waves across the tympanic cavity.

Serous otitis media. Also known as *'glue ear'*, or secretory otitis media, this is a collection of fluid (*effusion*) in the middle ear cavity. Causes include:

- obstruction of the auditory tube by, for example, pharyngeal swelling, enlarged adenoids or tumour
- barotrauma (usually caused by descent in an aeroplane when suffering from a cold)
- untreated acute otitis media.

The air already present in the middle ear is absorbed and a negative pressure develops causing retraction of the tympanic membrane. Thereafter fluid is drawn into the low-pressure cavity from surrounding blood vessels causing conductive hearing loss.

Adults experience hearing loss and, usually painless, blockage of the ear. However, this is a common cause of hearing impairment in preverbal children which may manifest as delayed speech and/or achievement of developmental milestones. Secondary infection can complicate this condition in both adults and children.

Box 8.1 Common causes of hearing loss

Conductive	Sensorineural
Acute otitis media	Impacted earwax or foreign body
Serous otitis media	Presbycusis
Chronic otitis media	Long-term exposure to excessive noise
Barotrauma	Congenital
Otosclerosis	Ménière's disease
External otitis	Ototoxic drugs, e.g. aminoglycoside antibiotics, diuretics, chemotherapy
Injury of the tympanic membrane	Infections, e.g. mumps, herpes zoster, meningitis, syphilis

Sensorineural hearing impairment

This is the more prevalent form of hearing impairment and is the result of a disorder of the nerves of the inner ear or the central nervous system, e.g. the cochlea, cochlear branch of the vestibular nerve or the auditory area of the cerebrum. Noise-induced hearing loss is one cause of sensorineural hearing impairment which may arise as a consequence of:

- employment e.g. construction work, manufacturing or the music industry
- social activities e.g. listening to loud music on personal equipment or at nightclubs.

Other causes are listed in Box 8.1.

Risk factors for congenital sensorineural hearing impairment include family history, exposure to intrauterine viruses, e.g. maternal rubella and acute hypoxia at birth.

Ménière's disease. In this condition there is accumulation of endolymph causing distension and increased pressure within the membranous labyrinth with destruction of the sensory cells in the ampulla and cochlea. It is usually unilateral at first but both ears may be affected later. The cause is not known. Ménière's disease is associated with recurrent episodes of incapacitating dizziness (*vertigo*), nausea and vomiting, lasting for several hours. Periods of remission vary from days to months. During and between attacks there may be continuous ringing in the affected ear (*tinnitus*). Loss of hearing is experienced during episodes, which may gradually become permanent over a period of years as the spiral organ is destroyed.

Presbycusis. (see p. 207).

Ear infections

External otitis

Infection by *Staphylococcus aureus* is the usual cause of localised inflammation (boils) in the auditory canal. More

generalised inflammation may be caused by prolonged exposure to bacteria or fungi or by an allergic reaction to, e.g., dandruff, soaps, hair sprays, hair dyes.

Acute otitis media

This is inflammation of the middle ear cavity, usually caused by upward spread of microbes from an upper respiratory tract infection via the auditory tube. It is very common in children and is accompanied by severe earache. Occasionally it spreads inwards from the outer ear through a perforation in the tympanic membrane.

Bacterial infection leads to the accumulation of pus and the outward bulging of the tympanic membrane. Sometimes the tympanic membrane ruptures and pus discharges from the middle ear (*otorrhoea*). The spread of infection may cause *mastoiditis* and *labyrinthitis* (see below). As the petrous portion of the temporal bone is very thin, the infection may spread through the bone and cause meningitis (p. 184) and brain abscess.

Chronic otitis media

In this condition there is permanent perforation of the tympanic membrane following acute otitis media (especially when recurrent, persistent or untreated) and mechanical or blast injuries. During the healing process stratified epithelium from the outer ear sometimes grows into the middle ear, forming a *cholesteatoma*. This is a collection of desquamated epithelial cells and purulent material. Continued development of cholesteatoma may lead to:

- destruction of the ossicles and conductive hearing loss
- erosion of the roof of the middle ear and meningitis
- spread of infection to the inner ear that may cause labyrinthitis (see below).

Labyrinthitis

This complication of middle ear infection may be caused by development of a fistula from a cholesteatoma (see above). It is accompanied by vertigo, nausea and vomiting, and nystagmus. In some cases the spiral organ is destroyed, causing sudden profound sensorineural hearing loss in the affected ear.

Motion sickness

This occurs when the brain receives conflicting sensory information; the visual information received from the eye does not match the information from the semicircular canals of the inner ear about one's position in relation to the environment. It causes nausea and vomiting in some people, and is usually associated with travel, e.g. by car, train or aeroplane.

Disorders of the eye

Learning outcome

After studying this section, you should be able to:

- describe the pathological changes and effects of diseases of the eye.

Inflammatory conditions

Stye

Also known as hordeolum, this is an acute and painful bacterial infection of sebaceous or tarsal glands of the eyelid margin. The most common cause is *Staphylococcus aureus*. A 'crop' of styes may occur due to localised spread to adjacent glands. Infection of tarsal glands may block their ducts, leading to cyst formation (*chalazion*), which may damage the cornea.

Blepharitis

This is chronic inflammation of the eyelid margins, usually caused by bacterial infection or allergy, e.g. staphylococcal infection or *seborrhoea* (excessive sebaceous gland secretion). If ulceration occurs, healing by fibrosis may distort the eyelid margins, preventing complete closure of the eye. This may lead to drying of the eye, conjunctivitis and possibly corneal ulceration.

Conjunctivitis

Inflammation of the conjunctiva may be caused by irritants, such as smoke, dust, wind, cold or dry air, microbes or antigens and may be acute or chronic (Fig. 8.26). Corneal ulceration (see below) is a rare complication.

Infection. This is highly contagious and in adults is usually caused by strains of staphylococci, streptococci or haemophilus.

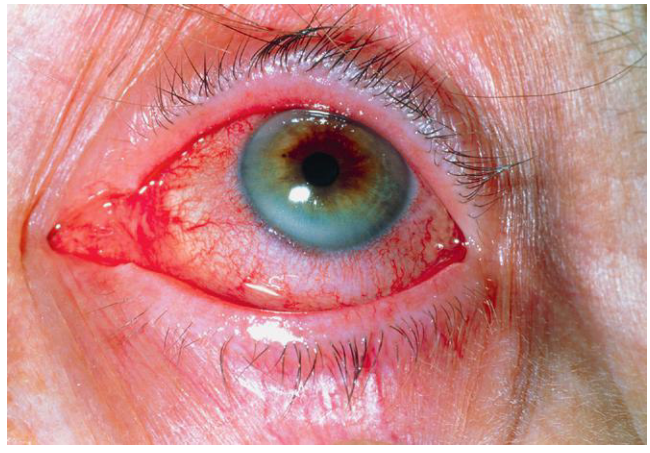

Figure 8.26 Conjunctivitis.

Neonatal conjunctivitis. Sexually transmitted disease in the mother, including gonorrhoea, chlamydia and genital herpes, can infect the newborn infant's eyes as the baby passes through the birth canal.

Allergic conjunctivitis. This may be a complication of hay fever, or be caused by a wide variety of airborne antigens, e.g. dust, pollen, fungus spores, animal dander, cosmetics, hair sprays, soaps. The condition sometimes becomes chronic.

Trachoma

This chronic inflammatory condition is caused by *Chlamydia trachomatis* and is a common cause of sight loss in developing countries. Deposition of fibrous tissue in the conjunctiva and cornea leads to eyelid deformity and corneal scarring as the eyelashes rub against the surface of the eye. The microbes are spread by poor hygiene, e.g. communal use of contaminated washing water, cross-infection between mother and child, or contaminated towels and clothing.

Corneal ulcer

This is local necrosis of corneal tissue, usually associated with corneal infection (*keratitis*) following trauma (e.g. abrasion), or infection spread from the conjunctiva or eyelids. Causative organisms include staphylococci, streptococci and herpes viruses. Acute pain, *injection* (redness of the cornea), photophobia and lacrimation interfere with sight during the acute phase. In severe cases extensive ulceration or perforation and healing by fibrosis can cause opacity of the cornea requiring corneal transplantation.

Glaucoma

This is a group of conditions in which intraocular pressure rises due to impaired drainage of aqueous fluid through the scleral venous sinus (canal of Schlemm) in the angle between the iris and cornea in the anterior chamber (Fig. 8.8). Persistently raised intraocular pressure may damage the optic nerve by mechanical compression or compression of its blood supply causing ischaemia.

Damage to the optic nerve impairs vision; the extent of which varies from some visual impairment to complete loss of sight.

In addition to the primary glaucomas below, it is occasionally congenital or secondary to other causes, e.g. anterior uveitis or a tumour.

Primary glaucomas

Primary open-angle glaucoma (POAG). There is a gradual painless rise in intraocular pressure with progressive loss of vision. Peripheral vision is lost first but may not be noticed until only central (*tunnel*) vision remains. As the condition progresses, atrophy of the optic disc occurs leading to irreversible loss of vision. It is commonly bilateral and occurs mostly in people over 40 years of age. The cause is not known but there is a familial tendency.

Acute closed-angle glaucoma. This is most common in people over 40 years of age and usually affects one eye. During life the lens gradually increases in size, pushing the iris forward. In dim light when the pupil dilates, the lax iris bulges still further forward, and may come into contact with the cornea, blocking the scleral venous sinus (canal of Schlemm) suddenly raising the intraocular pressure. Sudden severe pain, photophobia, headache, nausea and blurred vision accompany an acute attack. It may resolve spontaneously if the iris responds to bright light, constricting the pupil and releasing the pressure on the scleral venous sinus. After repeated attacks spontaneous recovery may be incomplete and vision is progressively impaired.

Chronic closed-angle glaucoma. The intraocular pressure rises gradually without symptoms. Later, peripheral vision deteriorates followed by atrophy of the optic disc and loss of sight.

Strabismus (squint, cross-eye)

In normal binocular vision, the eyes are aligned so that each eye sees the same image, meaning that both eyes send the same image to the brain. In strabismus only one eye is directed at the observed object and the other diverges (is directed elsewhere). The result is that two different images are sent to the brain, one from each eye, instead of one. It is caused by one-sided extrinsic muscle weakness or impairment of the cranial nerve (III, IV or VI) supply to the extrinsic muscles. In most cases the image from the squinting eye is suppressed by the brain, otherwise there is double vision (*diplopia*).

Presbyopia

(see p. 208).

Cataract

This is opacity of the lens which impairs vision especially in poor light and darkness when weak light rays can no longer pass through the cloudy lens to the retina (Fig. 8.25). Although most commonly age-related (p. 208) this condition also be congenital or secondary to other conditions e.g. ocular trauma, uveitis, diabetes mellitus.

The most common cause of visual impairment worldwide, cataracts can affect one or both eyes. The extent of visual impairment depends on the location and extent of the opacity.

Congenital cataract may be idiopathic, or due to genetic abnormality or maternal infection in early pregnancy, e.g. rubella. Early treatment is required to prevent permanent loss of sight.

Retinopathies

Vascular retinopathies

Occlusion of the central retinal artery or vein causes sudden painless unilateral loss of vision. *Arterial occlusion* is usually due to embolism from, e.g., atheromatous plaques, endocarditis. *Venous occlusion* is usually associated with increased intraocular pressure in, for example, glaucoma, diabetes mellitus, hypertension, increased blood viscosity. The retinal veins become distended and retinal haemorrhages occur.

Diabetic retinopathy

This occurs in type I and type II diabetes mellitus (p. 236) and is the commonest cause of blindness in adults aged between 30 and 65 years in developed countries. Changes in retinal blood vessels increase with the severity and duration of hyperglycaemia. Capillary microaneurysms develop and later there may be proliferation of blood vessels. Haemorrhages, fibrosis and secondary retinal detachment may follow and, over time, there may be severe retinal degeneration and loss of vision.

Retinopathy of prematurity (ROP)

This condition affects premature babies. Known risk factors include: birth before 32 weeks' gestation, birth weight less than 1500 g, requirement for oxygen therapy and serious illness. There is abnormal development of retinal blood vessels and formation of fibrovascular tissue in the vitreous body causing varying degrees of interference with light transmission. The prognosis depends on the severity and many cases resolve spontaneously. In severe cases there may also be haemorrhage in the vitreous body, retinal detachment and loss of vision.

Retinal detachment

This painless condition occurs when a tear or hole in the retina allows fluid to accumulate between the layers of retinal cells or between the retina and choroid. It is usually localised at first but as fluid collects the detachment spreads. There are visual disturbances, often spots before the eyes or flashes of light due to abnormal stimulation of sensory receptors, and progressive loss of vision, sometimes described as a 'shadow' or 'curtain'. In many cases the cause is unknown but it may be associated with trauma to the eye or head, tumours, haemorrhage, cataract surgery when intraocular pressure is reduced or diabetic retinopathy.

Retinitis pigmentosa

This is a group of hereditary diseases in which there is degeneration of the retina, mainly affecting the rods. Progressive impairment of peripheral vision, especially in dim light, usually becomes apparent in early childhood. Over time this leads to tunnel vision and, eventually, loss of sight.

Tumours

Choroidal malignant melanoma

This is the most common ocular malignancy in adults, occurring between 40 and 70 years of age. Vision is not normally affected until the tumour causes retinal detachment or secondary glaucoma, usually when well advanced. The tumour spreads locally in the choroid, and blood-borne metastases usually develop in the liver.

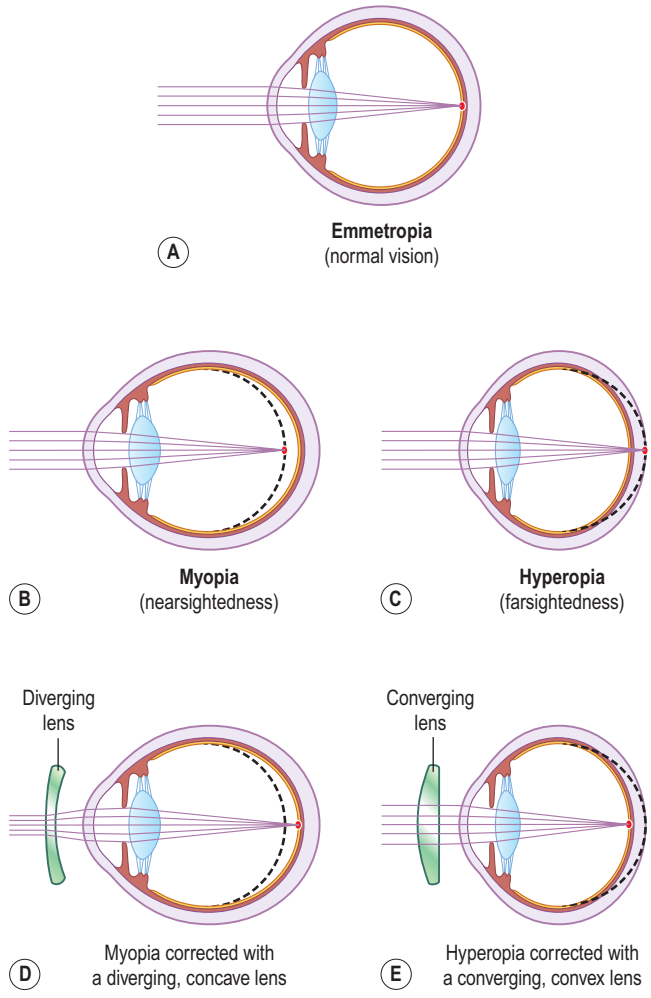

Figure 8.27 Common refractive errors of the eye and corrective lenses. A. Normal eye. **B.** Nearsightedness. **C.** Farsightedness. **D.** Correction of nearsightedness. **E.** Correction of farsightedness.

Retinoblastoma

This is the most common malignant tumour in children. A small number of cases are familial. It is usually evident before the age of 4 years and usually affects one side. The condition presents with a squint and enlargement of the eye. As the tumour grows visual impairment develops and the pupil looks pale. It spreads locally to the vitreous body and may grow along the optic nerve, invading the brain.

Refractive errors of the eye

Learning outcome

After studying this section, you should be able to:

- explain how corrective lenses overcome refractive errors of the eye.

In the *emetropic* or normal eye, light from near and distant objects is focused on the retina (Fig. 8.27).

In *myopia*, or nearsightedness, the eyeball is too long and distant objects are focused in front of the retina (Fig. 8.27B). Close objects are focused normally, but distant vision is blurred. Correction is achieved using a biconcave lens (Fig. 8.27D).

In *hyperopia*, or farsightedness, a near image is focused behind the retina because the eyeball is too short (Fig. 8.27C). Distant objects are focused normally, but close vision is blurred. A convex lens corrects this (Fig. 8.27E).

Astigmatism is the abnormal curvature of part of the cornea or lens. This interferes with the light path though the eye and prevents focusing of light on the retina, causing blurred vision. Correction requires cylindrical lenses. It may coexist with hypermetropia, myopia or presbyopia.

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