6/ Special Senses

Introduction. To fight out the environment, general sensations are essential but not enough. Because, unless the offending agent actually falls on the body, general sensations will fail to give any information to the organism. Hence, a special *information service* must be created, by which the existence of an object may be known from a distance. Out of this necessity the special senses evolved.

The organs of special senses have got special end organs for the reception of specific types of stimuli. The sense impressions are carried by sensory nerves to the brain where the sensation is received and interpreted. The particular type of sensations is carried by the sensory nerve from each specified organ. There are four varieties of special senses, viz., (1) **taste,** (2) **smell,** (3) **vision,** and (4) **hearing.** The first two are collectively known as **chemical senses.**

General characters. (1) All are located in the head. Two advantages: (*a*) better protection, (*b*) quicker response—being nearest the centre. (2) Subserved by special receptive apparatus situated in special regions. (3) Highly developed. Fine discriminations are possible. (4) Greater part of the impulse goes to cerebral cortex and is conscious. A small part used for reflex purposes. (5) Excepting taste, *all are projected outside the body, on to the source of the impulse.* Such as, light is localised not on the retina but on the luminous object. Sound is localised not on the ear but on the source of the sound. Obviously, taste is an exception to this rule.

TASTE (GUSTATION)

The senses of taste and smell are closely interrelated and there is probably no such thing as a pure taste. They are mainly concerned with nutrition. In the nose and mouth, common chemical sensibility is closely associated with senses of smell and taste respectively. For example, smells of the vapours of onions are caused by stimulation of the olfactory apparatus and by stimulation of the V nerve endings subserving common chemical sense.

Sensation of taste does not evolve solely from the stimulation of taste buds. It is also due to sensation of common senses like heat, cold, touch and specially of olfaction. In the absence of olfaction taste sensation is remarkably altered. Discrimination of taste is attributed to the palate by the help of olfaction. Flavour is the complex sensation of taste, odour, hotness or coldness, pungency or blandness, roughness or smoothness.

Tongue is mainly concerned with taste sensation. Taste buds are the end organs of taste.

Primary taste sensations (FIG. 6.1 A). Four primary tastes: (1) **sweet,** (2) **bitter,** (3) **sour** (acid), (4) **salt.** Some hold that there are two others: (5) **metallic,** (6) **alkaline.** Taste sensations often become complicated due to three factors: (*a*) Blending of two or more primary tastes, (*b*) Blending with general sensations in the mouth, (*c*) Blending with the sensation of smell.

Tongue

Papillae and histology of tongue. [*Vide* Vol. I]

Taste buds

Taste buds are the end organs of taste (FIG. 6.1 C_1 & C_2). These are

located in the mucosa of the epiglottis and pharynx and in the lateral walls of the fungiform and vallate papillae of the tongue (FIG. 6.1 B). There are about 10,000 taste buds in man. In old age it decreases in number.

Histology. Oval, about 70 μ long and 50 μ broad, with the long axis perpendicular to the surface. Vessels and nerves enter through the bottom.

Taste buds were formerly classified into two—**gustatory** or **taste cells** and **supporting** or **sustentacular cells** on the basis of shape, size and staining properties. But recent *electron microscopic studies* have shown that many of the cells are **transitional** in character and this is due to different stages of development of the taste buds. The taste cells are developed from the epithelial cells around the taste buds and during the process of maturity the cells thus migrate towards the centre of the bud and then degenerate. Life span of taste cells is very limited and it is about 3–5 days in rats. Each taste cell has got microvilli that are projected in the taste pore.

Each bud (FIG. 6.1 C_1) contains a number of receptor cells whose apical tips project through a pore in the surface epithelium. Nerve fibres enter the base of the taste bud, lose their myelin sheaths and either invaginate the receptor cells or lie between them.

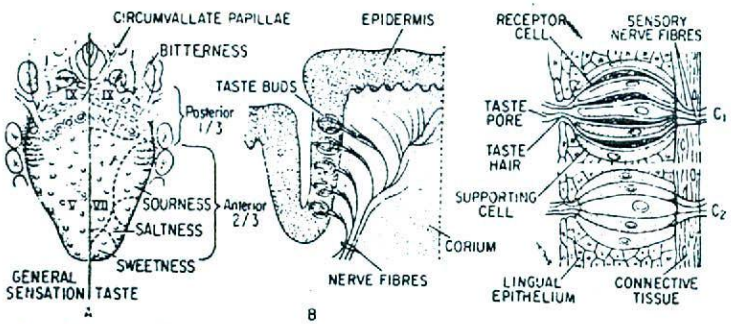


FIG. 6.1. A = Diagrammatic representation of chief distribution of primary taste sensations on different areas of tongue. Impulses from taste receptors carried by V, VII and IX cranial nerves. The sides and tip of the tongue are served by facial (VII) and trigeminal (V) nerves whereas glossopharyngeal (IX) nerve serves the back and sides. B = A vallate papillae cut lengthwise. C_1 & C_2 = Sectional representation of taste bud showing taste cell, taste pore and taste hair that protrude from the free surface of the taste cell.

Distribution. (1) Mainly on the tongue. Circumvallate and fungiform papillae contain taste buds. Also in (2) soft palate, (3) pharynx, (4) epiglottis, (5) inner surface of arytenoid process, (6) anterior pillars of fauces, etc.

Varieties. Probably there are as many varieties of taste buds as there are primary taste sensations. *Proof:* (1) Differential distribution of taste on the tongue. (2) Decoction of or chewing the leaves of *Gymnema sylvestre* selectively abolishes at first the sweet and then the bitter sensations, leaving sour and salt unaffected. (3) Cocaine abolishes at first the bitter, then sweet, then salt and lastly the sour. (4) Some substances produce different taste sensations according to the area of tongue stimulated, e.g., magnesium sulphate tastes salty in front but bitter behind. (5) Action potentials: Three types of nerve fibres were identified in the cat by Pfaffmann. Action potentials were detected only when acid, salt and bitter

substances were placed over the tongue. Other workers recorded action potentials when sweet substances were placed over the tip of the tongue.

Nature of stimuli. The substance to be tasted must be in solution. Normally saliva acts as the solvent.

Receptor stimulation. Taste receptors are considered as *chemoreceptors* that respond to substances dissolved in the oral fluids bathing them. Receptors have microvilli covered with a polyelectrosurface film. Binding of ions to this film causes a distortion of the film with initiating the generator potential which would in turn depolarise the sensory nerves. The sensation of taste is mediated by nerve fibres which innervate the taste buds. The anterior two-thirds of the tongue is innervated by the lingual nerve and the posterior third of the tongue is supplied by the glossopharyngeal nerve.

modalities of taste. There are about four basic modalities of taste, sweet, sour, bitter and salt. *Sweet* chiefly at the tip, *salt* on the dorsum anteriorly, *sour* at the sides, and *bitter* at the back of the tongue (FIG. 6.1A). Mid-dorsum has got no taste sensation. The taste buds in each of these areas are not histologically different from those in other areas.

Electrophysiological studies have shown that the taste receptor cells do not always belong to basic types corresponding to the basic taste qualities. Individual sensory cells are differentially sensitive to chemicals as for instance a receptor cell can be excited by low concentrations of *sodium chloride* or by high concentrations of *sucrose*. Thus any taste cell is capable of stimulation to a varying degree by different chemical stimuli.

Recently, attempts have been made to record from the receptor cells of rats. Electrodes are pushed into a taste bud pore until they abruptly record a negative potential of 50–90 mV. This is assumed to indicate cell penetration. Application of an appropriate chemical to the taste bud causes the potential to shift towards zero. With any one substance, the magnitude of the potential change is proportional to the concentration. No spikes can be recorded. These potential changes are believed to be generator potentials which activate nerve endings in an unknown way. If they are, experimental results present difficulties for the concept of separate taste modalities. Each receptor cell commonly responded to substances of all four classes, though it was most sensitive to one of them. This might be explained if the receptor cells were merely protective devices for transporting substances to the nerves without exposing the delicate nerve endings on the surface of the tongue. The *generator* potentials might then be irrelevant to the production of impulses by axons. Alternatively, although generator potentials might be produced by all four types of substance, only the one to which the cell was most sensitive might produce a response great enough to excite the axon. On observation of the axon discharge using single fibre recording, the receptor would then appear to be absolutely specific. No decision between these or any other hypothesis can be made at present.

Chemical constitution related to taste. (1) **Sweet taste** is caused by some organic compounds, e.g., sugars, certain polysaccharides, alcohols, aldehydes, ketones, saccharine, chloroform, etc. But certain inorganic substances also arouse sweet sensations, e.g., lead acetate, highly diluted alkalis, etc. It is held that (Oertly and Myers), every sweet substance possesses two radicles. One is called *glucophore* and the other *auxogluc*. Any

stuff having both the radicles will taste sweet. (2) **Salty taste** is caused by inorganic compounds, such as chlorides, sulphates, bromides, iodides, etc. *The saline sensation is attributed to the anions of these compounds.* Certain organic compounds also give saline taste, e.g., hydrochlorides of monomethyl and dimethyl amine. (3) **Sour taste** is caused by acids or acid salts. *It is attributed to the hydrogen ion concentration* upon which the acidity depends. Curiously enough, certain organic acids (with little ionisation), such as acetic acid, has more acid taste than mineral acids, e.g., HCl. This is due to greater power of penetration of acetic acid. (4) **Bitter taste** is caused by some organic compounds, such as alkaloids (strychnine and quinine), certain glucosides, bile salts, etc. Some inorganic substances also produce bitter taste, e.g., salts of magnesium, ammonium and calcium. *The bitterness of these inorganic compounds is attributed to the cations,* that of the organic compounds is somewhat related to the NO_2 group.

Threshold of primary taste sensations. It is the minimum concentration of different substances necessary to arouse a primary taste sensation. Most *sensitive to bitter* and least to *salt*. The threshold concentration, for HCl is at a pH of about 4 and for NaCl the least concentration is about 0·12 per cent. (0·02 M). On the other hand, for strychnine the threshold concentration is about 0·00006 per cent. and the least concentration of cane sugar is about 0·5 per cent., but for saccharin is only about 0·001 per cent.

Successive contrast. After taking bitter, sweet taste is enhanced. So the bitter taste enhances the sweet taste sensation. Similarly sweet taste enhances the bitter taste sensation.

Simultaneous contrast. After applying salt on one border of the tongue other border of the tongue becomes hypersensitive to sweet.

Factors influencing taste sensations

Area. Intensity of taste sensation depends upon the total surface area stimulated, as for instance stimulation of a single papilla by a drop of solution produces weaker sensation that does tasting of the same solution by the whole of tongue.

Temperature. Taste sensation is modified with temperature and in a range of 30–40°C, maximum sensation is observed.

Olfaction. Taste sensation depends upon olfaction. In condition of diseases of olfactory receptors, the acquity of taste sensation is defective.

Individual variation. There is individual variation of taste sensation. A concentration of a substance which can be tasted by one individual may not be detected by another. These differences are usually *quantitative* but can be *qualitative*. The most interesting example of qualitative variation is the ability to taste PTC. (*phenyl thiocarbamide*). The human race can be divided into those who cannot taste this at all and those to whom it tastes sour. The ability to taste it is inherited as a **Mendelian recessive trait.**

Adaptation. Taste sensation is quickly adapted and threshold of stimulus for a particular substance is increased. As for instance after taking sweets if anyone takes tea with usual sugar then the sweet sensation is somewhat decreased.

Familial dysautonomia. There is a widespread familial disorder of sensation, in which the subject cannot identify even a saturated solution of NaCl by taste.

Selective taste blindness. In this case the subject does lose taste sensation of a particular substance and however does not lose completely. Threshold for stimulation is increased. The subject possesses normal taste sensation of other modalities and thus this defect is less selective. Caucasians are affected by this type of disease.

Acceptance and rejection of foods. Taste sensation is also related with food habit. Some one likes sweet and other likes salt. Besides this, certain substances may be accepted or may be rejected. Sweet is mostly accepted by all but the noxious substances are rejected. Acid and salt in mild concentration is accepted but in higher concentration is rejected.

Rejection and acceptance are also dependent upon the metabolic states of the individual. Hypoglycaemic patients want to take more sugar. Subject having adrenocortical insufficiency wants much salt.

Path of taste impulses

From anterior two-thirds of tongue taste sensations are carried by the VII cranial nerve, **from posterior third** by the IX cranial nerve (FIG. 6.2).

From the anterior two-thirds, the sensory taste fibres first enter the lingual nerve, then the chorda tympanic branch of the facial (VII) nerve and then join the trunk of the facial in the aqueductus Fallopii. Their cell station is situated in the **geniculate ganglion** (first-order neurone). The axons of these cells enter the pars intermedia and end in the dorsal nucleus of the VII cranial nerve (upper part of the nucleus of tractus solitarius). The chorda tympani also contains efferent fibres for salivation.

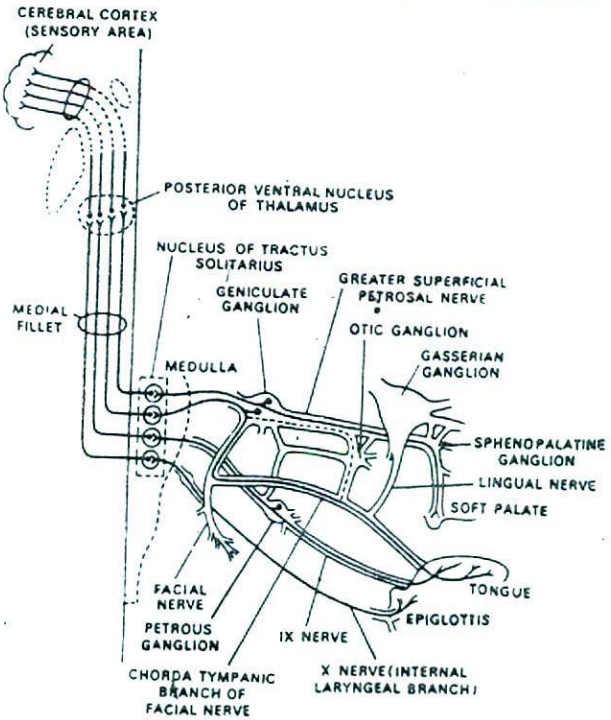


FIG. 6.2. Path of taste impulses.

From the posterior third, including the vallate papillae, taste fibres pass along the glossopharyngeal (IX) nerve and enter their cell station in the **petrous ganglion** first-order neurone). The axons of these

cells pass along the IX cranial nerve and end in the dorsal nucleus of the IX cranial nerve (lower part of the nucleus of tractus solitarius).

The greater superficial petrosal nerve supplies gustatory fibres to the palate. Taste buds near the epiglottis are innervated by the vagus.

From the nucleus of tractus solitarius, second-order neurones arise, cross the middle line, pass through the medial lemniscus or fillet and end in the posterior ventral nucleus of the thalamus. Here, third-order neurones arise, pass through the posterior limb of internal capsule and end in the inferior part of the postcentral gyrus of the cerebral cortex. The sensations of touch, hot, cold, pain (common sensibility) are carried by the lingual branch of the V cranial nerve (cell bodies which are situated in the semilunar ganglion) and pursue the similar course and end in the inferior part of the postcentral gyrus.

Electrical stimulation of the inferior part of the postcentral cortex can produce hallucinations of taste, and destructive lesions of this area reduce gustatory and tactile sensibility of the tongue. When a taste-evoking substance be placed on the tongue, it (e.g., quinine) produces changes in the electrocorticogram of the inferior part of the postcentral cortex (sensory area).

SENSE OF SMELL (OLFACTION)

Introduction. Like taste, smell is also a chemical sensation. For taste, the substance must be in liquid form; for smell it must be in gaseous form. Volatile substances have generally strong odours. After reaching the nose the vapour gets dissolved in the local secretion and stimulates the olfactory epithelium. Olfactory sensation is the most primitive of all special senses and is much more acute than taste. A man can detect mercaptan and artificial musk in a dilution of one part in several billion parts of air. In many animals the sensation of smell is much more acute than in man. Such animals are called *macrosmatic*. In **macrosmatic animals** the olfactory sense plays an important role in protecting the animals from enemies, search for food and in the reactions of sex. In comparison to them man is *microsmatic*. Like primary taste sensations, attempts have been made to find out whether there is any primary smell sensation. Such attempts have not yet been successful. Attempts have also been made to classify olfactory sensations. But no such classification is satisfactory. Smell sensations are often blended with simultaneous taste and general sensations and thus often become a complex admixture, viz., sweet smell (chloroform), pungent smell (ammonia), etc. An instrument called **olfactometer** is used to determine the *minimum identifiable odour* (MIO) of a substance.

Olfactory receptors

The olfactory receptors themselves are quite different from the taste buds. These receptor cells are supported by tall sustentacular cells. They superficially resemble taste receptor cells in having micro-projections from their apical ends, but they are, in fact, nerve cells and themselves give rise to axons (Fig. 6.5). These axons are very fine having about only 0·2 μ m in diameter and several run together within a single Schwann cell sheath. Because of this, it is very difficult to record from single axons and it is debatable whether this has ever been done successfully.

The axons pass through the **cribriform plate** to reach the olfactory

bulb. The two main cell types are the **tufted** and **mitral cells** (FIG. 6.5). Both give off innumerable bushy dendrites on the side nearest the bulb surface and it is with these dendrites that the olfactory nerve axons make connections. The *tufted cells* send their axons *via* the medial olfactory tract to the opposite side of the brain. The *mitral cell* axons enter the lateral olfactory tract which is primarily ipsilateral. There has been considerable controversy about the precise central destinations of these tracts.

The **olfactory epithelium** is that part of nasal epithelium which is sensitive to smell and confined to the nasal mucosa of the olfactory area.

Smelling sensation is developed mainly from the stimulation of receptors in the *yellow-brown olfactory mucosa* that lines the surface of the *superior turbinate* and the upper third of the nasal septum. In man the total area of olfaction on each side is about 250 mm². The **olfactory area** is formed by the superior nasal concha, the upper part of the septum and the roof of the nose (FIGS. 6.3 & 6.4).

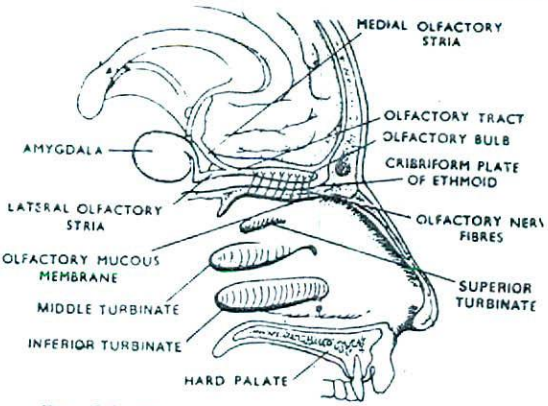


FIG. 6.3. Diagram represents the anatomy of the olfactory mucous membrane.

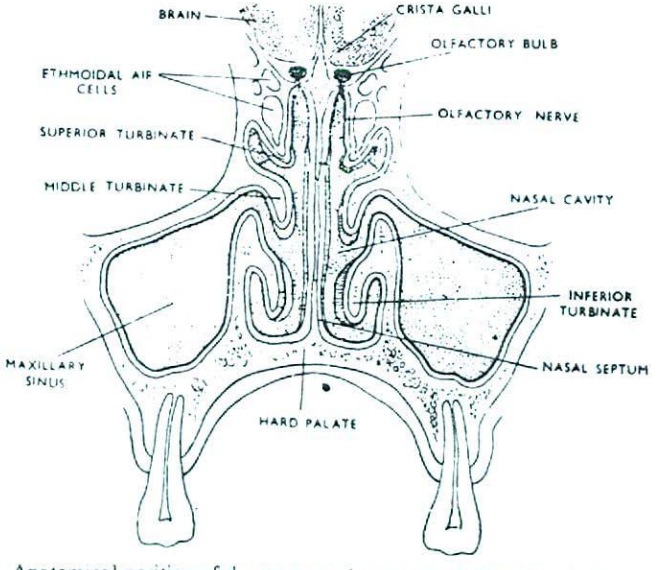


FIG. 6.4. Anatomical position of the receptors for the sense of smell in the interior of the nose in between the median nasal septum and the superior turbinate (*after Grollman*).

Olfactory mucous membrane. The olfactory receptors are located in a small specialised portion of the nasal mucosa which is called the **olfactory area.** This area differs from the rest of the nasal mucosa both in its gross appearance as well as in its histological structure. The *dog* is a **macrosmatic animal** and its olfactory area is large. The *human being* is **microsmatic** and his olfactory area is comparatively very small.

The olfactory epithelium (FIG. 1072) is composed of mainly two types of cells. These are **supporting (sustentacular) cell** and **receptor cell.** There is a third type of cell which is called the **basal cell.** This basal cell is *not* numerous but mainly parent cell and gives rise to supporting cell.

The *supporting cell* is highly columnar with large oval nuclei and ends in microvilli that secrete mucus. Cytoplasm contains granules of a golden-brown pigment that gives the golden-yellow colour of the nasal mucosa. Bowman's glands also secrete golden-yellow pigment.

The **receptor cells are bipolar neurones** and are about 10-20 millions in number in man. They function both as receptor and ganglion cells. *Dendrite* arises from superficial pole and the *axon* arises from the deeper pole of the fusiform receptor cell. The dendrite projects out through the space in between the supporting cells and expand slightly to form the olfactory rods. The *olfactory rods* contain numerous vacuoles, mitochondria as well as vesicles. Unmyelinated cilia project out from the rod. There are about 10-20 cilia per receptor cell.

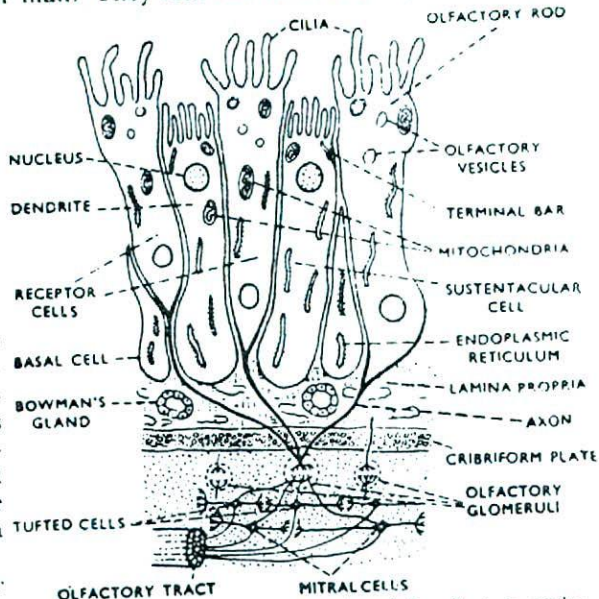


FIG. 6.5. Histological representation of the olfactory epithelium and connections of olfactory nerve fibres (diagrammatic)

Beside these, the lamina propria contain certain glands—**Bowman's glands,** which secrete fluid containing the watery and oily substances (FIG. 6.5).

Olfactory bulb. In the olfactory bulb there are a large number of nerve cells called granules intermingled in an interlacement of nerve fibrils, mitral cells and tufted cells with their dendrites and axons forming the layer of olfactory glomeruli. Electron micrograph has revealed the presence of intranuclear rodlet, mitochondria and endoplasmic reticulum in the cytoplasm of the granule cells.

Olfactometry. The most well-known method of investigation of the olfactory sensation is the **olfactometer of Zwaardemaker**

(FIG. 6.6). This instrument consists of a glass-tube sliding over another tube. The inner tube A is graduated into 10 equal divisions of 0·7 cm each. Both ends of the inner tube are open but one outer end C is curved and tapering. This tapering end is introduced into the nostrils. The inner wall of the outer tube B contains the odorous substance. The subject inhales the substance as he breathes within the tube because other nostril is kept closed The intensity of smell is increased with the gradual withdrawal of the outer tube because this process facilitates the dilution with increasing concentration in the inspired air. This method gives only an approximate result.
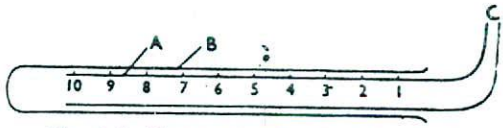


FIG. 6.6. Diagram showing Zwaardemaker's Olfactometer.

Another method is the *blast method of Elsberg and Levy*. Here a controlled volume of odour-laden air is forced into the nose. Blast of such air is given successively so as to just perceive the odour. The minimum volume of odour-laden air necessary for identification is called the **minimum identifiable odour (MIO)** or the **olfactory coefficient.** This method has been a great aid in localisation of tumours in the anterior part of the skull.

Classification of odours by Zwaardemaker

Class	*Subdivisions*
I. Alliaceous	(*i*) Arsine, (*ii*) Chlorine, (*iii*) Hydrogen sulphide.
II. Ambrosial	(*i*) Amber, (*ii*) Musk.
III. Aromatic	(*i*) Bitter almond, (*ii*) Camphor, (*iii*) Cloves, (*iv*) Lavender, (*v*) Lemon.
IV. Caprillic	(*i*) Cheese, (*ii*) Rancid fat.
V. Empyreumatic	(*i*) Benzene, (*ii*) Coffee.
VI. Ethereal	(*i*) Beeswax, (*ii*) Ether, (*iii*) Fruit.
VII. Fragrant	(*i*) Flowers, (*ii*) Vanilla.
VIII. Nauseating	(*i*) Carrion, (*ii*) Faeces.
IX. Repulsive	(*i*) Bedbug, (*ii*) Belladonna.

PHYSIOLOGY OF OLFACTION

The substances which remain in the gaseous state, e.g., turpentine, essential oil, etc., produce strong odours, whereas non-volatile substances, viz., the heavy metals, etc., are inodorous. The odoriferous substances after inhalation do not reach the olfactory areas immediately but remain in the middle of the nose, e.g., below the level of the superior concha. The odorous particles of molecular size emitted by the odoriferous substances ascend upwards through the air and reach the olfactory areas either by eddy current or by diffusion. Diffusion is a slow process and so eddy current is the main factor. After reaching the olfactory area the odorous particles get dissolved in the fluid secreted by **Bowman's glands.** The secretion of Bowman's glands contains both water and oil. The odoriferous substances, which are soluble in water and oil, produce strong odours. The high solubility in oil is of greater importance than that of water-solubility for olfactory stimulation. The odorous particles after

getting into solution become adsorbed on the surface of the cilia. There is some specificity or selectivity in the adsorption of odorous substances on to the olfactory receptors. After the odorous substances being adsorbed on to the olfactory mucosa, generator potentials of 4–6 second durations are developed. These generator potentials when reached to its threshold value, initiates action potential which is then propagated along the axons to the olfactory bulb.

The odour producing molecules react with chemical groupings on the surface protein film of the receptor thereby increasing the surface area of the proteins which would distort the receptor surface and thus a generator potential is initiated.

Olfactory discrimination. Man can distinguish between 2000 and 4000 different odours, and different parts of the olfactory mucous membrane respond differently to the same odours and receptors classification according to the different odours is still unsuccessful.

Olfactory adaptation. The olfactory receptors become insensitive to a particular odorous substance after exposure to a specific period of time. As for instance the odour of the oil of lemon is not perceptible after an exposure varying from 2·5 to 10 minutes.

This is due to sensory adaptation. It is partly a central phenomenon, but it is also due to a change of the receptors themselves.

Threshold of olfactory sensation. It is the minimum concentration of different odorous substances to arouse olfactory sensation. Artificial musk, iodoform, butyric acid, etc., is perceptible in minimum concentration.

Chemical compounds and their relation to olfactory sensation

The intensity of olfactory sensation varies with different chemical compounds. If the chemical compounds belong to homologue series then the intensity of stimulation will increase from the lower members to higher one. In alcoholic series the odours increase in strength (from lower to higher one), i.e., from methyl, ethyl, propyl, butyl to amyl.

Relation of odorous substances. Weaker odours are masked by the stronger ones. If the odorous substances are of equal strength then the odours of both are perceived. Some of the odorous substances are antagonistic to each other, e.g., iodoform antagonises balsam of Peru.

Action potentials have been recorded from the olfactory bulb of the frog. In the frog there is constant rhythmical discharge from the olfactory bulb. Adrian and Ludwig recorded the action potentials in the olfactory stalk of the cat-fish and found that although the resting discharge is of low frequency but after stimulation of olfactory nerve fibres there was burst of impulses at high frequency.

No successful intracellular receptor recordings have yet been made. The attempts at single axon recordings show strong responses to some odours and weak responses to others. Receptors, recording to a single odour at low threshold and not to other odours, have not been neurophysiologically identified. This may be because various odoriferous agents are very complex molecules and can activate several different types of receptors. On the other hand, it may mean that the recordings were from axon bundles coming from several receptors, rather than from true single axons.

As with taste, various attempts have been made to correlate smell with chemical structure. None of these can be regarded as established,

but one of the most successful theories is that of Amoore. He studied description of all known odoriferous agents and came to conclusion that smells were all mixtures of seven primary smells, which he called camphoraceous, musky, floral, peppermínty, ethereal, pungent and putrid (Fig. 6.7). From a study of the structure of molecules, he concluded that each smell was evoked by a characteristic structure. For instance, camphoraceous materials have spherical or egg-shaped molecules while musky molecules have flat discs. Compounds which evoked mixtures of primary

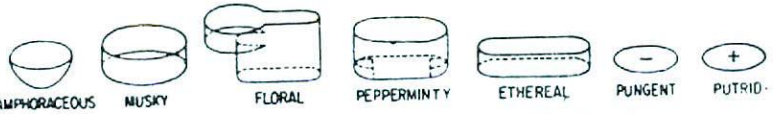


Fig. 6.7. Amoore's proposed molecular shapes, capable of activating seven primary types of olfactory receptors.

smells had structural features of both. Amoore therefore postulated that there must be sites somewhere on the receptor cells into which these structures could fit and thus initiate impulses. Seven types of receptor required have not yet been found but this is no reason for denying their existence since experimental techniques at the moment are poor. Probably the most convincing aspect of Amoore's work is that it has been used to predict accurately smells which will be possessed by new compounds which have never before been synthesised.

There is a mass of intricate neurophysiological work on the olfactory bulb. Unfortunately it has as yet given us little information as to how the olfactory information is processed and so it is debatable.

Pathways of olfactory impulses

Bipolar nerve cells of the olfactory epithelium are the first-order neurones. Each receptor cell gives rise to only one axon which joins with those derived from other receptors forming collectively the *fila olfactoria* or the olfactory nerves. The olfactory nerves are unmyelinated and have got neurolemmal sheath. The sheath is continuous with the subarachnoid space. Virus of acute anterior poliomyelitis probably passes through this olfactory route and reach the central nervous system. The nerves pierce the cribriform plate of the ethmoid bone and enter the **olfactory bulb.** Here, the axons make synapses with the dendrites of the mitral cells and the tufted cells to form the complex globular structures which are known as **olfactory glomeruli.** Approximately 26,000 receptor cells converge on each glomerulus and each glomerulus again passes impulses to about 24 mitral cells and 68 tufted cells. The mitral and tufted cells are the second-order neurones and their axons constitute the olfactory tract. The olfactory tract proceeds towards the anterior perforated substance and divides in the olfactory trigone into well-defined olfactory striae—*medial*, *intermediate* and *lateral* (Figs. 6.8 & 6.9). Regarding further course of this tract opinions differ. The commoner view is given below.

The axons of the tufted cells leave the tract and most probably end in the opposite bulb *via* the anterior commissure through the **medial olfactory stria.** This tract after entering the anterior commissure passes bilaterally to the nucleus of the stria terminalis; olfactory tubercle and to

amygdaloid nuclear complex. The granule cells are the terminals of the tufted cells of the opposite bulb. Functions of this fibrous arrangement are not debatable. If the olfactory tract is served, the axons of the tufted cells do not degenerate. Furthermore, if the anterior commissure is sectioned

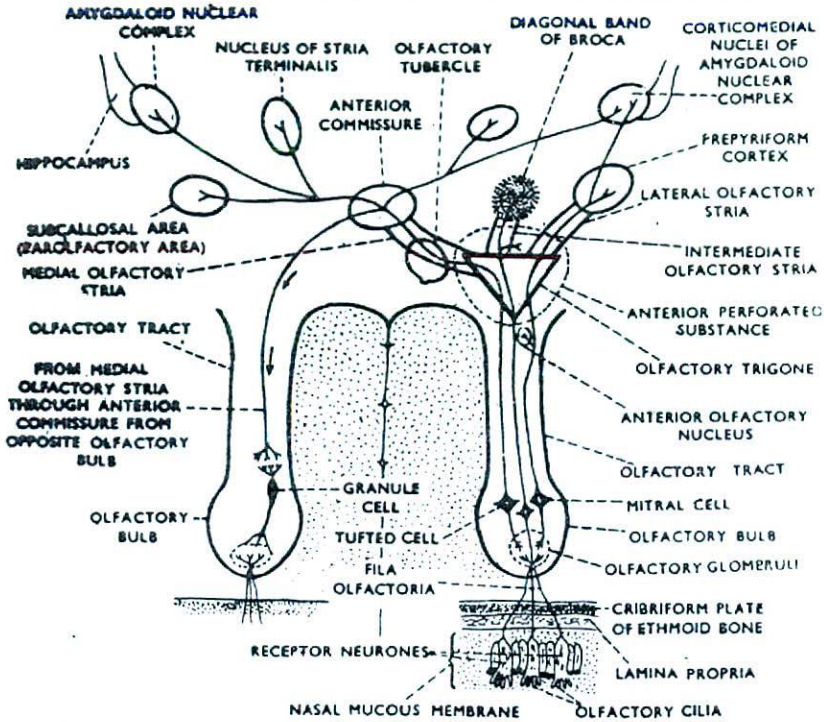


FIG. 6.8. Diagrammatic representation of path of olfactory impulses showing olfactory rods (receptor cells) and supporting cells in the olfactory mucosa.

then it produces a considerable olfactory deficit but if one olfactory bulb is removed then only unilateral loss of olfaction occurs.

The axons of the mitral cells pass through the **lateral olfactory stria** and ends in the anterior olfactory nucleus. Cortical and medial protions of the ipsilateral amygdaloid nucleus and in the prepyriform cortex and periamygdaloid cortex. The prepyriform cortex and the periamygdaloid cortex are the primary olfactory cortex. Entorhinal cortex (area 28) is the secondary olfactory area because it receives fibres from

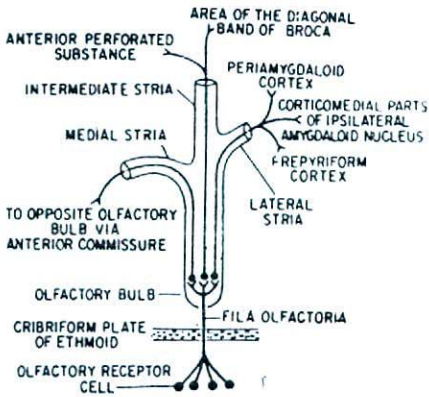


FIG. 6.9. Simplified diagram of the olfactory bulb and its connections.

primary olfactory cortex or adjacent neocortex. It is doubt that there is any direct connections with the hippocampal region. There is also no thalamic representation to the olfactory system.

Another groups of fibres which are less significant in man pass through the **intermediate stria** and ends within the anterior perforated substance and the area of the diagonal band of Broca.

Olfactory pathways, as described above, are not clear and further studies are required.

Abnormalities of olfactory sensation

Loss of the sense of smell is known as **anosmia.** It occurs due to congenital abnormalities of olfactory bulbs or nerves. During inflammation of nasal mucosa there is temporary loss of smell.

Hyperosmia is the morbid sensitiveness to odours. It occurs in hysteria, in raised intracranial pressure, etc.

VISION

Anatomical considerations. Human eyeball (Figs. 6.10 & 6.11). is roughly spherical, being a little flat from above downwards (25 mm

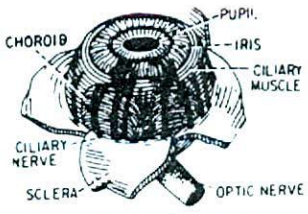


Fig. 6.10. It shows the eyeball, sclera cut and turned back, pupil, iris, ciliary muscle, choroid, ciliary nerves and optic nerve.

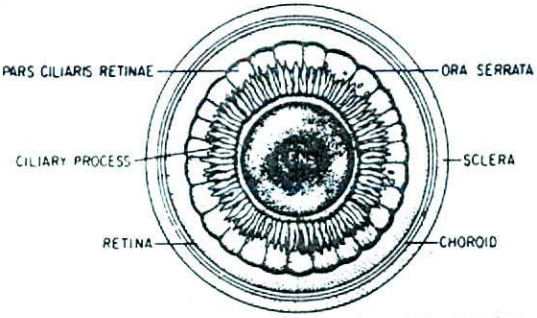


Fig. 6.11. Diagrammatic representation of the interior of the anterior half of the eyeball.

horizontal, 23·5 mm vertical). The **optic nerve** enters the eyeball (Fig. 6.12) a little inside the posterior pole, through the **optic disc. Tunics**—From outside inward, the wall has three coats: (1) **fibrous coat,** (2) **vascular coat,** (3) **nervous coat. Fibrous coat**—Two parts: (*i*) Posterior (5/6ths) is opaque and called the *sclera.* (*ii*) Anterior (1/6th) is transparent, called the *cornea.* The **vascular coat (uvea,** or **uveal tract)**—Three parts: From behind forward—(*i*) the *choroid*—remains just behind the retina forming the posterior 5/6ths of the middle coat. Composed of numerous blood vessels and pigmented cells containing melanin. Hence, its colour is dark. Its **function** is to convert the eyeball into a dark chamber. (*ii*) The *ciliary body*—includes orbicularis ciliaris, ciliary processes, ciliaris muscle. (*iii*) The *iris* (*vide* later). **Nervous coat**—The nervous coat is called the **retina.** It contains the photosensitive receptors, where the visual impulses are generated.

Conjunctiva. The exposed part of the eyeball is covered by a thin stratified mucous membrane which is reflected on to the inner surface of the eyelids. It is called *conjunctiva.* Its **function** is protection and lubrication.

Lacrimal apparatus. The lacrimal gland is an almond-shaped racemose gland remaining inside the upper and outer parts of the orbit

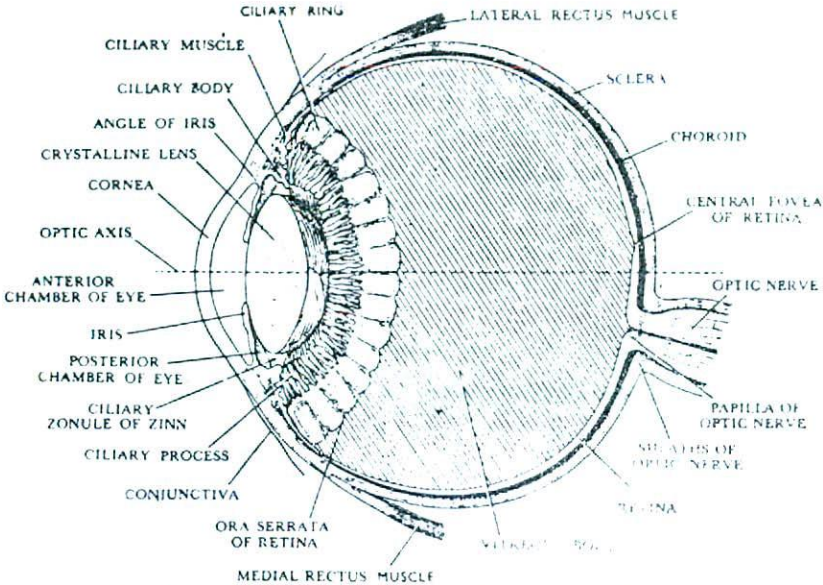


FIG. 6.12. Anatomical representation of the structure of the eye.

and secreting a watery fluid, the *tears*. Smaller accessory glands are common (FIG. 6.13). The secretions are delivered into the conjunctival sac through six to ten fine ducts. The movements of the eyelids help to spread the tears over the conjunctival surface. The tears ultimately collect into a small triangular area (*lacrimal lake*) at the inner angle of the eye. From here the fluid passes through *puncta lacrimalia* and is carried through two small *lacrimal canaliculi* into the *lacrimal sac* inside the nose, where the sac opens (FIG. 6.14). The normal **function** of tears is to keep the exposed surface moist. Irritation or emotion increases secretion. **Nerve supply**—Sympathetic from superior cervical ganglion, parasympathetic from the facial. **Composition of tear**—closely resembles aqueous humour:

Water—98·2%; solids—1·8%; Organic elements: protein—0·67%; sugar—0·65%; NaCl—0·66%;

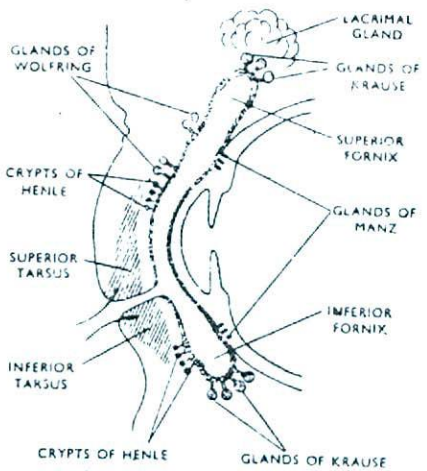


FIG. 6.13. Sectional representation through eyelid and eyeball showing the conjunctival sac and positions of its glands (*after Adler*).

N.P.N.—0·05%; urea—0·03%; other mineral elements—sodium, potassium and ammonia—0·79%.

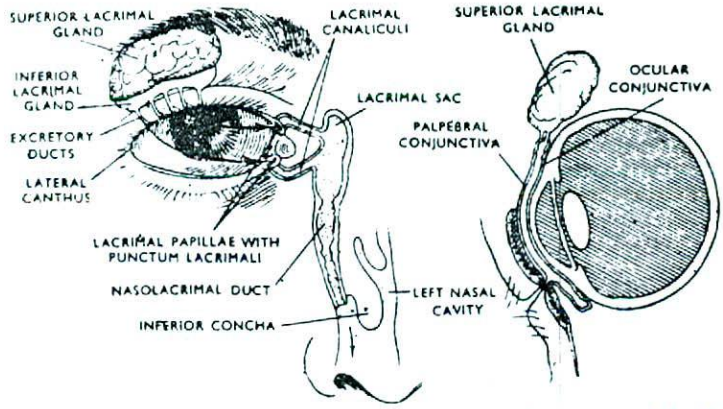


FIG. 6.14. Diagrammatic representation of the lacrimal apparatus. Left-sided diagram showing the direction of drainage of lacrimal fluid from the excretory ducts of the lacrimal glands across the eye to the nasolacrimal duct *via* lacrimal sac and right-sided diagram representing relation of the lacrimal gland to conjunctival surfaces with the eye closed (*after Langley & Others*).

Electrophoretic analysis of tears shows three types of proteins—*albumin*, *globulin* and *lysozyme*. The lysozyme can dissolve many air-borne saprophytes rapidly and completely. Lysozyme along with other unidentified labile agents seems to inhibit the growth of many pyogenic cocci (staphylococcus, haemolytic streptococcus, diplococcus pneumoniae, etc.).

Eyeball. Its interior is divided into two compartments by a partition. The centre of this partition is occupied by the **lens.** The peripheral part, by which the lens remains attached to the wall of the eyeball, is called the **suspensory ligament.** The posterior compartment contains **vitreous humour** (vitreous body). The anterior compartment contains **aqueous humour.** Aqueous humour and vitreous humour create the **intraocular pressure.** This pressure tends to hold the eyeball round and firm (FIG. 6.15). The anterior compartment is further subdivided by the iris into an *anterior* and a *posterior chamber*. Both contain aqueous humour. **Intraocular pressure**—25–30 mm of Hg. It varies with general blood pressure. Pressure on the ocular veins or on eyeball from outside, disturbed drainage of aqueous humour and exposure to darkness raise the pressure.

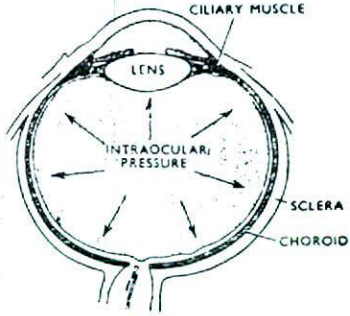


FIG. 6.15. Diagrammatic representation of the mechanism of the intraocular pressure (*after Langley & Others*).

Refractive media of the eyeball

Physics of vision. Light is a form of radiant energy. This energy travels in waves through air at a speed of about 3,00,000 Km per second.

The wave length of light visible to the human eye differs from about 3850 m μ to 7200 m μ.

Refraction denotes 'to bend' or 'to turn aside'. As the speed of light is different from one medium to another medium, the beam of light travels in glass more slowly than in air.

Refractive index is a relative measure of transmission of light. This measure is relative to the rate of light ray in air. If light travels through a medium at a rate of 1,50,000 Km per second, this medium is said to have a refractive index about 2·0 (3,00,000÷1,50,000). As the lens of the human eye transmits light at the rate of about 2,14,285 Km per second, it has a refractive index of about 1·40.

If a transparent medium be at a right angle to its surface there is no bending of light rays. Therefore, the degree of refraction depends upon the *difference between refractive idices of two media* and the *angle at which the beam of light traverses the interface between the media.*

A **lens** is a transparent substance. This substance bends rays of light and causes rays to converge or diverge. Man-made lens (FIG. 6.16) is

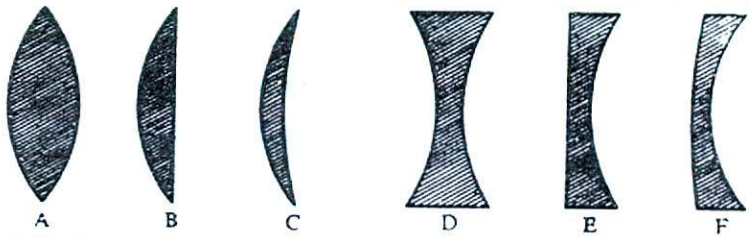


FIG. 6.16. Various types of lenses. A=biconvex, B=planoconvex, C=concavoconvex, D=biconcave, E=planoconcave. F=convexoconcave.

generally of glass. If the rays of light are parallel to the axis (line through the middle) of a lens (or curved mirror) they come to the **principal focus.** If the lens has sufficient convexity, these diverging rays of light will be bent and ultimately converge on a **conjugate focus** (FIG. 6.17). Therefore, the distance to the conjugate focus is greater than to the principal focus for any lens.

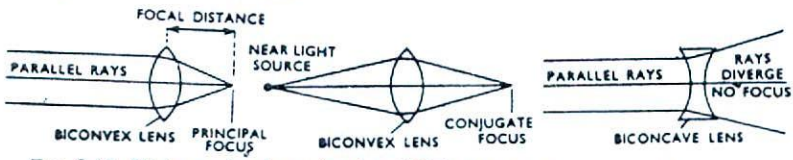


FIG. 6.17. Diagram showing refraction of light rays by biconvex and biconcave lenses (*after Langley & Others*).

The **power of a lens to bend light** depends upon the *refractive index of the lens* and the *curvature of the surfaces. Refractive power* of a lens is expressed in **diopters** and **focal distance** is the distance between a biconvex lens and its principal focus. *The shorter the focal distance be, the greater the refractive power of the lens will be.* When the focal distance is 100 cm, the lens is said to get a power of one diopter (100÷100). And, when the focal distance is 20 cm, a lens has a power of 5 diopters (100÷20). As the parallel

rays of light are caused to diverge by a biconcave lens and there is no focal point, the power of a biconcave lens would be expressed in terms of its ability to counteract converging ability of a biconvex lens. Therefore, the power of a biconvex lens is termed in **plus diopters** and the power of a biconcave lens is expressed in **minus diopters** (FIG. 6.18).

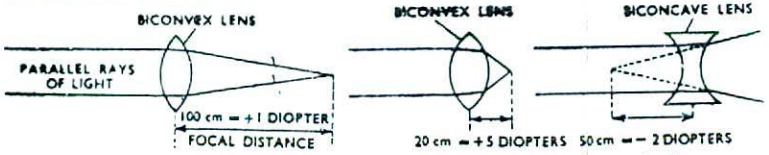


FIG. 6.18. Diagrams representing the refractive power of the lenses in diopters.

Optical arrangement of the eye. The beams of light first strike the cornea and then pass through the aqueous humour. Afterwards these strike the lens, then vitreous humour and finally the retina.

The normal eyeball acts as a camera. Inverted images of external objects are formed exactly on the retina, and generate appropriate visual impulses. These impulses are carried through the optic nerves to the brain where visual impressions are produced. There are four refractive media in the eye which help to converge the light rays and focus them on the retina. The media are: (1) **cornea,** (2) **aqueous humour,** (3) **crystalline lens,** and (4) **vitreous humour.** Maximum refraction occurs at the air-corneal surface—42 diopters; at lens—20 diopters, with full accommodation 36 diopters. The media are briefly described below.

CORNEA

It is the round, transparent convexity in the anterior part of the eyeball. **Diameters**—11 mm (vertical) × 12 mm (lateral). **Thickness**—0·5–1 mm. **Nerve supply**—*rich* and *non-medullated.* Only free nerve terminals—subserving pain. There are no other sensations and no other endings. **Nutrition**—from lymph and probably aqueous humour. **Refractive index**—1·38. **Function.** (*a*) Allows free entry of light. (*b*) Acts as a refractive medium. **Intraocular pressure or tension**—The aqueous humour is under a pressure of 25 to 30 mm of Hg. The tension of the eyeball can be felt by palpating the eyeball with two fingers. The pressure is measured with the help of **Tonometer.** The intraocular pressure* becomes high in **glaucoma.**

Histology (FIG. 6.19). From outside inwards there are as follows:

1. **Corneal epithelium** or **External stratified epithelium.** This layer is about 50 μ m thick. Several layers of cells continuous with the conjunctiva: (*i*) The superficial four or five layers are stratified *squamous.* (*ii*) The deepest (*basal*) layer is *columnar.* (*iii*) The intermediate two or three layers are *polyhedral.*

* **Pressures of the vascular system.** According to Duke-Elder the following values (mm of Hg) are associated with an intraocular pressure of 20 mm of Hg.

Retinal vein	22·0	Ophthalmic artery	99·5
Intrascleral vein ..	21·5	Retinal arteries	75·5
Episcleral vein ..	13·0		

The values are most important in the anterior ciliary or episcleral veins; these are probably dependent of the intraocular pressure. Thus in human subjects Weigelin and Löhlein found the following empirical relationship:

$Pev = 0{\cdot}48 \times Poc + 3{\cdot}1$, where *Pev* is the episcleral venous pressure and *Poc* the intraocular pressure.

2. **Bowman's membrane** or **Anterior elastic lamina.** Under light microscope this layer is homogeneous and structureless. But in electron microscope it shows the presence of a network of relatively fine collagenous fibres with an irregular arrangement. This homogeneous layer is 5–10 μ m thick.

3. **Substantia propria** or **Corneal stroma.** 90% thickness of the cornea is dependent upon this layer. It is composed of connective tissue fibres and cells. This layer is held together by mucoprotein and rich in keratin sulphate. As the fibres and mucosubstance have the refractive index and are highly hydrated, the whole cornea is transparent. The transparency of the cornea is dependent upon the regular arrangement of its microscopic and submicroscopic elements. The collagen fibrils in the lamellae are arranged parallelly with the surface of the cornea. Again within one lamella, the fibrils are strictly arranged parallel to one another but with adjacent lamella the same is arranged in different directions roughly at right angles to the fibres in the next layer.

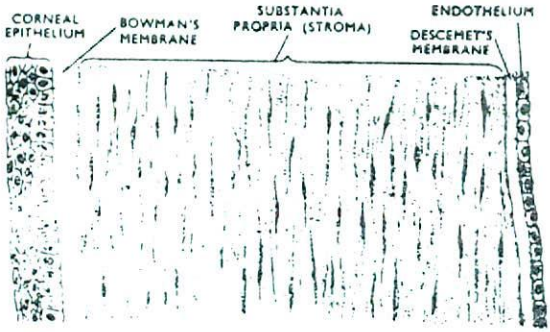


FIG. 6.19. It shows meridional section through the cornea.

4. **Descemet's membrane** or **Posterior elastic lamina.** Under light microscope this layer is highly homogeneous and has a highly refractive structure. Early concept was that this membrane contained fine elastic tissue which stained with resorcin-fuchsin. But X-ray diffraction studies and also chemical studies show the presence of chiefly fibrous protein of collagen type. E/M structure shows the presence of dense granules connected by fine filaments. This homogeneous layer is about 5–10 μ m thick.

5. **Corneal endothelium** or **Inner layer of squamous epithelium.** A single layer of endothelial cells that extends over the inner surface of Descemet's membrane and is continuous with the epithelium covering the anterior surface of the iris. In cross-section the cells are rectangular with oval nuclei.

Nutrition of cornea. It is avascular and its living materials like epithelium, corneal corpuscles and endothelium get nutrition from aqueous humour and from superficial marginal plexus of blood vessels. Transitional between the cornea and the adjacent sclera and conjunctiva there is a zone of about 1 mm wide. This zone is known as **limbus.** The superficial marginal plexus which nourishes the cornea, are present in the limbus. Cornea is devoid of blood vessels for 1 to 2 mm inside the limbus.

Nutrition of the cornea depends upon the diffusion of substances into it from the film of tears covering its epithelial surface, from capillaries in the tarsal conjunctiva when the lids are closed, from the limbal capillaries and from the aqueous humour bathing its endothelial surface.

Theoretically, O_2 need of the cornea is met through four processes:

(*i*) the aqueous humour, (*ii*) the capillaries at the limbus, (*iii*) the capillaries in the tarsal conjunctiva, and (*iv*) O_2 dissolved in the layer of tear film on the epithelial surface. Most probably the aqueous humour supplies much of its O_2. During sleep when lids (FIG. 6.20) are closed the cornea gets deprived of O_2 supply from the air. Thus tarsal conjunctiva fails to supply O_2 from the air and during such state the cornea suffers from interference with its normal metabolism, resulting in a slight turgescence of the cornea. Ofcourse when the lids are closed some O_2 becomes available to the cornea from the blood in the tarsal capillaries.

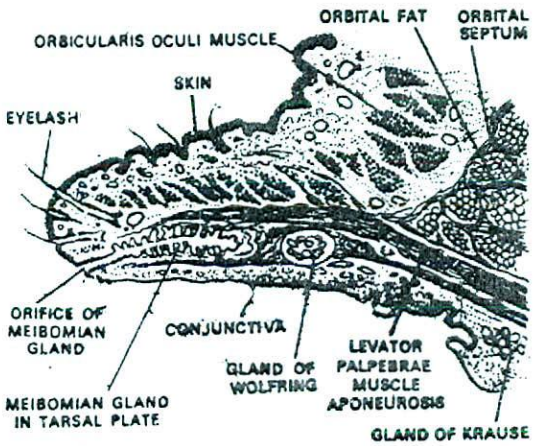


FIG. 6.20. It shows transverse section of the upper eyelid.

Corneal grafting. This method of surgical procedure has been a great aid in rectification of vision, where the cornea has become opaque. In most cases eye of donor (subject just has expired) is incised and kept in the refrigerator for use when necessary. But due to refrigeration, the cornea absorbs aqueous humour and becomes opaque due to oedema. But if the eye is kept under 37°C. in the incubator, then the water is expelled from the cornea and the transparency is thus maintained. The fact is that cornea absorbs water at low temperature and expels water at high temperature (37°C.). In corneal grafting there is no sloughing effect which is most unlikely in case of skin grafting from one human to another (homograft). In corneal grafting, the damaged cornea of the recipient is removed and replaced by similar-sized healthy corneal piece of the donor and held with suture. By rearrangement of layers and mitotic divisions the cute dges of the recipient's epithelium rapidly cover up the graft. The corneal corpuscles seal the graft firmly by laying down collagen bridges towards the junction which ultimately becomes indistinguishable from the surrounding cornea.

AQUEOUS HUMOUR

It is a clear watery fluid occupying both the anterior and posterior chambers of the eye. It is known to be the only secretory product of epithelium covering the **ciliary body.** It is not a stagnant fluid but continuously secreted and passed over the lens through the pupil into the anterior chamber and then drained into the vascular system—the anterior ciliary veins (FIGS. 6.21 & 6.22). **Reaction**—Alkaline (pH 7·1–7·3), **Specific gravity**—1·002–1·004. **Viscosity**—1·029. **Refractive index**—1·34. **Composition** — *Water* — 98·69%. *Solids*—1·31%. Further details, as compared with serum, are as follows: (*a*) The diffusible, non-ionisable substances, viz., urea, N.P.N., sugar, etc., same as in serum. (*b*) Colloids—only traces, i.e., much less than serum. (*c*) Chlorides—much higher

than serum.* On the whole, it closely resembles cerebrospinal fluid. **Blood-aqueous barrier**—Like cerebrospinal fluid, the aqueous humour also maintains a blood-aqueous barrier. Because large water-soluble molecules like penicillin, sucrose, *p*-aminohippurate, etc., cannot pass from blood to the aqueous humour or if pass, it is very slow indeed. **Formation**—Formed by the enzymatic activity of the ciliary processes. **Three theories:** (*a*) ***ultrafiltration***, (*b*) ***dialysis***, and (*c*) ***secretion***. The first two cannot explain the higher chloride content. Hence, it is chiefly secretion and the concept of secretion is now generally referred to as **active transport which has** been applied as the formation of aqueous humour.

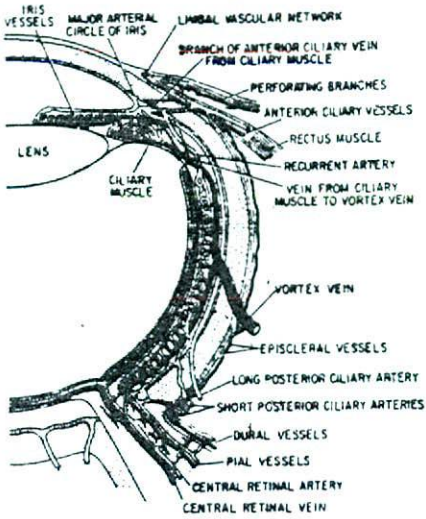


FIG. 6.21. Diagram showing vascular supply to eye. The ophthalmic artery gives rise to all arterial branches and venous drainage is through the cavernous sinus and pterygoid plexus.

Canal of Schlemm. This is a circular canal in the *corneosclera of the limbus* which comes into relation with the aqueous humour on the one hand and the *intrascleral venous plexus* on the other. Its structure is more complex, since it divides into several channels which, however, later reunite. Hence its appearance of a cross-section varies with the position of section. In the antero-posterior section, it appears as a flat vesicle. The lumen of this channel is separated from the aqueous humour by a meshwork of endothelium-covered trabeculae—**the trabecular meshwork or pectinate ligament.** Aqueous humour, to enter the canal, must percolate between the trabeculae and finally cross the endothelial wall. The meshwork has been divided into (*a*) **uveal meshwork** (innermost portion), (*b*) **corneoscleral meshwork** (outer portion), and (*c*) **pore area** or **pore tissue** immediately adjacent to the canal. Each lamella

*** Chemical composition of aqueous humour**

Protein. The most obvious chemical difference between aqueous humour and blood plasma is to be found in the protein contents of two fluids. In the plasma it has the order of 6·0–7·0 gm/100 ml, whereas in the aqueous humour it is only 5·0–15·0 mgm/100 ml in man and about 50·0 mgm/100 ml in the rabbit.

Non-colloidal constituents. The crystalloidal composition of the aqueous humour is similar to that of plasma. But the concentrations of ascorbate (vit. C), pyruvate and lactate are much higher than in plasma, whilst those of urea and glucose are much less.

COMPOSITION OF AQUEOUS HUMOUR AND PLASMA OF RABBIT (*after Davson*, 1962)

	Aqueous humour	Plasma		Aqueous humour	Plasma
Na	143·50	151·50	Lactate	7·40	4·30
K	5·25	5·50	Pyruvate	0·66	0·22
Ca	1·70	2·60	Ascorbate	0·96	0·02
Cl	109·50	108·00	Urea	7·00	9·10
Mg	0·78	1·00	Reducing value (as glucose)	6·90	8·30
HCO_3	33·60	27·40			

Concentrations are expressed as millimoles per kilogram of water.

of the uveal meshwork is attached anteriorly to the corneal tissue at the end of **Descemet's membrane,** constituting **Schwalbe's ring,** and posteriorly to the meridional fibres of the ciliary muscle. A pull of these fibres may well operate to open up the meshes of this trabecular structure, thereby favouring passage of fluid (FIG. 6.22).

The canal, which is commonly full of aqueous humour, drains by a series of small connecting vessels (**collectors**) into the vessels of **intrascleral venous plexus.** This plexus is largely made up of ramifications of branches of the anterior ciliary veins. But according to Maggiore here is also an arterial contribution, branches of the anterior ciliary arteries passing directly to the plexus and breaking up into fine vessels that come into direct continuity with fine vessels of the plexus. It connects with the canal of Schlemm by some 20–30 fine vessels—collectors—which thus transport aqueous humour into the venous system. The blood from the plexus is drained away by large trunks encircling the globe into the **episcleral plexus;** from here large veins, also draining the more superficial plexuses—**conjunctival** and **Tenon's capsule** (*subconjunctival*) **plexuses**—carry the blood to the insertions of the rectus muscles into the sclera and accompany the muscular veins (FIG. 6.22). Thus the aqueous humour drains essentially into the anterior ciliary veins.

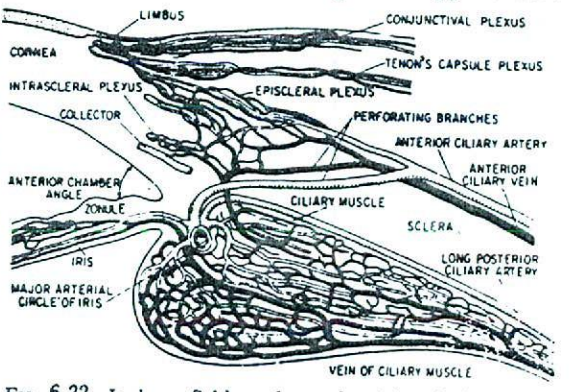


FIG. 6.22. It shows Schlemm's canal and its relation to the plexuses of antero-posterior segment of the eye (*after Davson*).

Flow of aqueous humour

Experiment. In 1921 Seidel established that the aqueous humour was continually formed and drained away. He inserted a needle, connected to a reservoir containing a solution of indigocarmine into the anterior chamber of a rabbit. By lowering the reservoir below the intraocular pressure a little aqueous humour was drawn into the needle and when the reservoir was raised, this fluid, mixed with the dye, was returned to the anterior chamber. As soon as the dye mixed with the rest of the fluid, Seidel observed that the dye stained blood vessels in the surface of the globe—**episcleral vessels**—showing that aqueous humour was being carried into the venous system.

Aqueous humour flows from the posterior chamber into the anterior chamber through the pupil. From the anterior chamber, the aqueous humour gets exit chiefly at the so-called **iridocorneal** or **filtration angle** (FIG. 6.23). At the filtration angle, the aqueous humour comes in contact with the trabecular tissue and makes its way through the meshwork to reach the canal of Schlemm. From here it goes to the scleral and episcleral venous plexus and thence to the aqueous veins and out of the eye.

It was long known that Schlemm's canal did act as a safety valve during elevations of the pressure (Duke-Elder). But at present it is considered to be a continuous outflow from the anterior chamber through the canal of Schlemm and the drainage mechanism functions constantly as

overflow mechanism rather than safety valve mechanism. Furthermore, the increased formation of aqueous humour is readily compensated by a rapid escape through the canal of Schlemm.

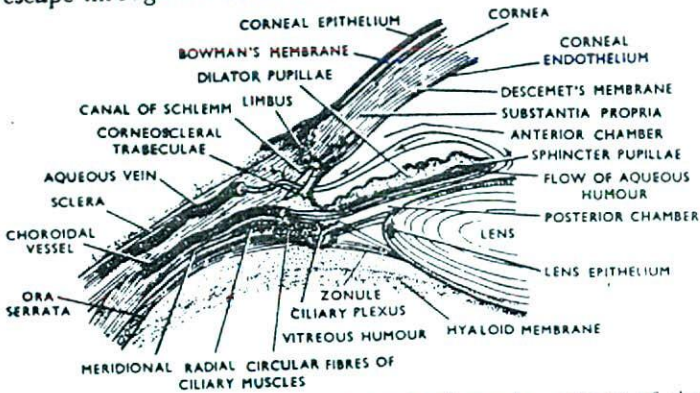


FIG. 6.23. Diagram shows sectional details of anterior segment of the human eye and also flow of the aqueous humour.

A small amount may pass through the suspensory ligament to the anterior surface of the vitreous, then through the hyaloid canal to the optic disc and then drains away through the perineural lymphatics of the optic nerve. A little may be absorbed through the veins and lymphatics of the interior of the eyeball. **Functions**—(*a*) Maintains intraocular pressure and the shape of the eyeball. (*b*) Acts as a refractive medium. (*c*) Supplies nutrition to and drains the metabolites from the surrounding structures.

Aqueous veins. In 1942 Ascher, when examining the superficial vessels of the globe with the slit-lamp microscope, observed what appeared to be empty veins but they turned out to be full of aqueous humour, and he called them **aqueous veins.** Usually one of these veins could be followed till it joined a blood vessel, in which event the contents of two vessels, aqueous humour and veins, did not mix immediately but often ran in parallel streams constituting a **laminated** aqueous vein (FIG. 6.24). If the blood vein beyond the junction was compressed, either the blood drove the aqueous humour out of its channel or the aqueous humour drove the blood out (FIG. 6.25). The latter was described as *positive glass-rod phenomenon* and the former situation (with the blood driving aqueous humour) was called the *negative glass-rod phenomenon*. These superficial vessels, full of aqueous humour, are presumably derived from deeper vessels of the intrascleral plexus, the pressure-relationships in the collectors from Schlemm's canal being such as to favour displacement of blood throughout the course of the fluid to the surface of the globe.

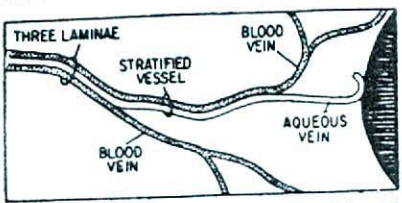


FIG. 6.24. Diagrammatic representation of formation of laminated aqueous vein.

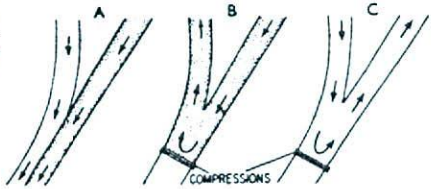


FIG. 6.25. Diagrammatic representation of aqueous veins. A shows the formation of laminated vein. In B blood drives aqueous humour from its vessel (C) due to compression of laminated vein (*after Starling*).

Goldmann proved conclusively that these vessels contained aqueous humour by injecting *fluorescein* intravenously. This substance passes very slowly from blood into

aqueous humour, so that one would not expect any vessel containing aqueous humour to fluoresce immediately after injection, while vessels full of blood or plasma should do so; in fact only the blood vessels did fluoresce.

Later, Ashton identified an aqueous vein in the human eye that was about to be enucleated, and after enucleation, the canal of Schlemm was injected with Neoprene. After digesting the tissue away a cast of the canal and its connecting vessels remained, and the aqueous vein did, indeed, take origin from the canal of Schlemm.

Autonomic regulation of the formation of the aqueous humour. The innervations of the ciliary processes are both sympathetic and parasympathetic running mostly along the vessels. Stimulation of the parasympathetic accelerates whereas stimulation of the sympathetic depresses the formation of aqueous humour. The sympathetic stimulation constricts the filtering and secretory parts of the ciliary process and thus the intraocular pressure is reduced.

Autonomic regulation of the outflow of the aqueous humour. The rate of outflow is mostly facilitated by the contraction of the iris and the ciliary muscle. Dilatation of the trabecular network and the canal of Schlemm also facilitates the outflow.

Following sympathetic stimulation the rate of flow is decreased because the intraocular pressure falls due to vasoconstriction in the filtering and the secretory parts of the ciliary process. But the outflow from the anterior chamber remains unaltered either by sympathetic stimulation or by administration of sympathomimetic drugs.

Parasympathetic stimulation increases the outflow from the anterior chamber and the rate of flow is increased. Parasympathomimetic drugs like pilocarpine and physostigmine also increase the outflow mechanism through contraction of the ciliary muscle; dilatation of the trabecular network, of Schlemm's canal and of the episcleral veins. These drugs are very important in the treatment of **glaucoma.**

Parasympatholytic drug like **atropine** has got opposite effect. It affects the outflow of the aqueous humour and thus the intraocular pressure is elevated.

CRYSTALLINE LENS

It is the chief refracting medium of the eyeball having the maximum refractory power. It is a transparent, elastic and biconvex lens, enclosed in a capsule. Posteriorly, it is more convex. It is circular, about 11 mm in diameter. The thickness at the centre is about 3·6–3·9 mm. **Refractive index**—1·40 at the centre. Less in the periphery. It is held *in situ* by the suspensory ligament.

Histology (FIG. 6.26). Composed of concentric layers of elongated modified cells. The peripheral parts are soft and nucleated, while the central part forms a dense non-nucleated mass with a higher refractive index. A single layer of columnar epithelial cells lies on the anterior surface, just behind the capsule. From the central part of the anterior surface to the equator of the lens, the columnar cells undergo a gradual transition into the **attenuated cells** of the lens.

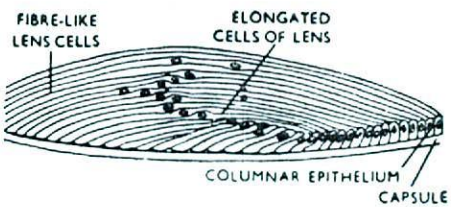


FIG. 6.26. Structure of lens.

Function. To refract light and focus it exactly on the retina. **Metabolism of lens.** Isolated lens, when placed in a suitable microspirometer, can be shown to use O_2 and eliminate CO_2. Oxygen lack, CO_2 excess and other pathological conditions cause degeneration of the lens. In the young, the lens is rich in cystine, which may be a part of glutathione, necessary for local tissue oxidation As age advances, the cystine content falls.

Nutrition of lens and cataract. Nutrition of the lens is maintained by the aqueous humour over the lens. Transparency of the lens is maintained by the normal nutrition and if this nutritional status is altered due to some dietary deficiencies then there comes a certain pathological changes in the lens leading to opacity. This condition is known as *cataract*. Experimentally, this condition can be produced in animals by feeding galactose which interferes with normal glucose metabolism of the lens. This cataract condition in the lens is also observed in deficiency of sulphur-containing amino acid or with X-ray irradiation over the lens. Glutathione content in the cataract lens is greatly decreased and has got direct relationship with the degree of opacity in the lens.

Vitreous humour (vitreous body)

It is a jelly-like material covered by a homogeneous membrane, the **hyaloid membrane,** and occupying the posterior compartment. It is made up of a series of lamellae arranged concentrically round the **hyaloid canal** (FIG. 6.27). The lamellae are composed of flat cells. The spaces

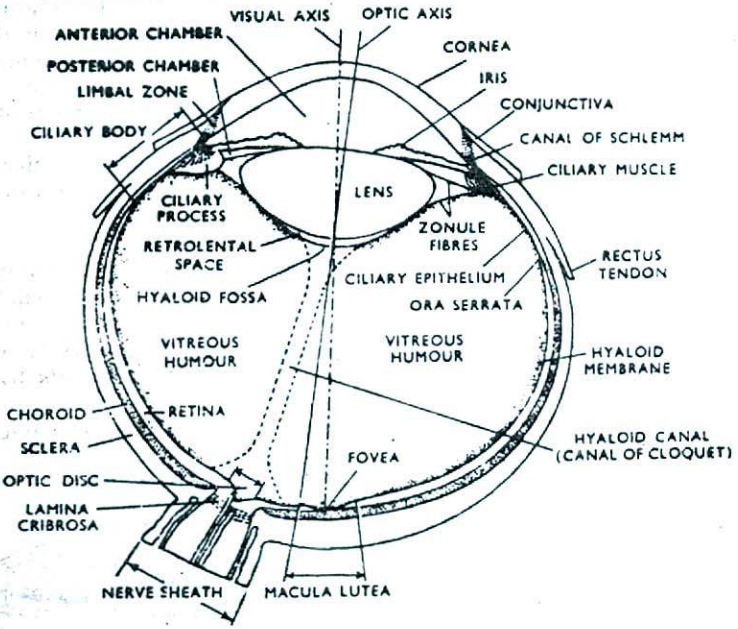


FIG. 6.27. Diagram represents the horizontal section of right human eyeball showing hyaloid canal, fovea, hyaloid fossa, retrolental space, etc.

between the lamellae are filled up with fluid. The hyaloid canal runs forward through the vitreous body from the entrance of the optic nerve to the

posterior surface of the lens. The vitreous body has a depression in front (**hyaloid fossa**) which lodges the posterior convexity of the lens. The *retrolental space* (**posterior lenticular space**) containing a little fluid, separates the lens from the vitreous. A fine canal (**hyaloid canal**) passes through the vitreous from the posterior pole of the lens up to the centre of the optic disc. In the embryo, the hyaloid artery passes through this canal. The **composition** of the vitreous humour is mostly similar to that of the aqueous humour except (*i*) its glucose content is considerably less than that in either aqueous humour or plasma, and (*ii*) its concentration of pyruvic acid and lactic acid are higher than that in the aqueous humour. Retina liberates these acids greatly. **Refractive index**—1·34. **Function**—(1) Maintains shape and pressure of the eyeball, (2) Acts as a refractive medium. **Retinal detachment** is the cause of localised liquefaction of the vitreous body and as a result a part of the retina floats in the vitreous cavity.

Blood-vitreous barrier (Fig. 6.28). Early studies on the kinetics of the blood-aqueous barrier tended to ignore the influence of the vitreous body on the penetration of material into the aqueous humour. Usually, the vitreous body comes into equilibrium with blood much more slowly than the aqueous humour and it may be called a **blood-vitreous barrier.** The vitreous body presumably receives material from the choroidal and retinal circulations. But, with many substances, equilibrium with plasma takes a long time to be achieved. The vitreous body must also receive material from the aqueous humour in the posterior chamber, the concentration tending to be higher in this fluid. Thus the blood-vitreous barrier is complex.

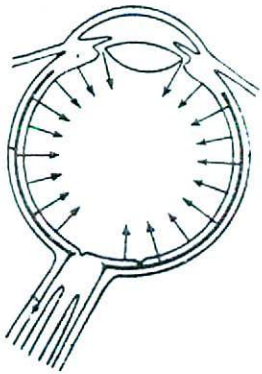


Fig. 6.28. It shows the blood-vitreous barrier (*after Dawson*).

Control of eye movements

There are six external ocular muscles for the movement of each eyeball (Figs. 6.30 & 6.31). Their names, **nerve supply** and **actions** are:

Muscle	*Direction*	*Interconnection*
Medial rectus or Internal rectus	Inward	Oculomotor (C. N. III)
Lateral rectus or External rectus	Outward	Abducens (C. N. VI)
Superior rectus	Up and in	Oculomotor (C. N. III)
Inferior rectus	Down and in	Oculomotor (C. N. III)
Superior oblique	Down and out	Trochlear (C. N. IV)
Inferior oblique	Up and out	Oculomotor (C. N. III)

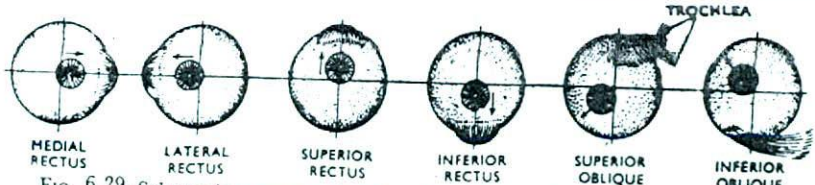


Fig. 6.29. Schematic representation of action of the extrinsic eye muscle showing direction of movements of the eyeball by arrows (*after Langley & Others*).

Co-ordination of eye movements. The external ocular muscles move the eyeballs in such a way that the two images are formed on the

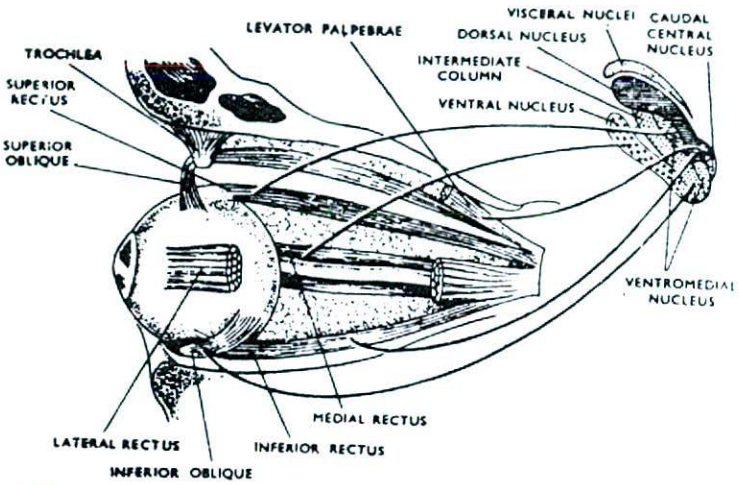


FIG. 6.30. Schematic representation of the localisation of specific extraocular muscles which are innervated by cell groups within oculomotor nuclei (*after Warwick*).

physiologically corresponding points on the retinae, so that, only one visual impression is produced. These movements may be either voluntary or reflex. Ocular movements are of three kinds: (1) Those in which the eye axes move to the same side, e.g., *right*, *left*, *up*, or *down*. (2) Those in which the axes move in opposite directions, e.g., *convergence* or *divergence*. (3) Those in which the eyeballs rotate round their axes clockwise or anticlockwise (FIGS. 6.29, 6.31 & 6.32).

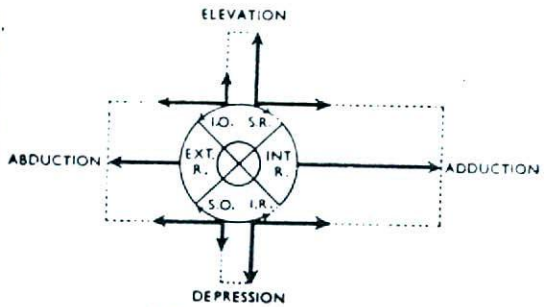


FIG. 6.31. Action of ocular muscles. S.R. = superior rectus; I.R. = inferior rectus; Ext. R. = external or lateral rectus; Int. R. = internal or medial rectus; I.O. = inferior oblique; S.O. = superior oblique (*from Best & Taylor*).

Nervous control

The co-ordinated movements of the eyeballs are controlled in several ways:

1. **Bilateral nerve supply.** Nerve of one side may supply muscles on both sides. For instance, internal and inferior recti and the inferior oblique muscles of one side receive fibres from III cranial nerve nuclei of both sides.

2. **Intercommunications** between the different parts of the III

cranial nerve nucleus and between the III, IV and VI cranial nerve nuclei of the same and opposite side (FIG. 6.33).

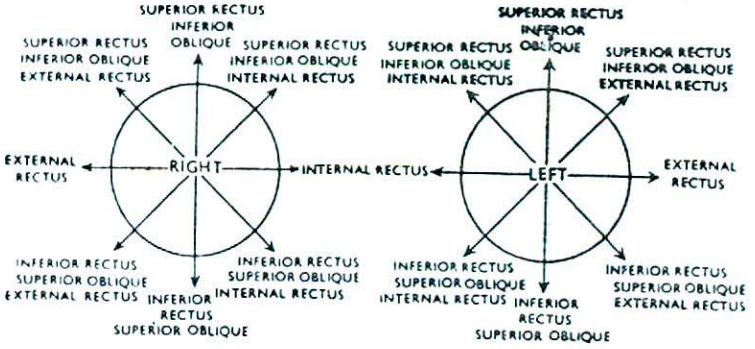


FIG. 6.32. Illustrates the action of the different ocular muscles and co-ordinated movements of the two eyeballs (*Savill*).

Intercommunication of the III, IV and VI cranial nerve nuclei

The III cranial nerve nucleus is supposed to contain seven component nuclei—four for the four external ocular muscles it supplies, and one each for the levator palpebrae superioris (upper lid), ciliary muscles (accommodation) and constrictor pupillae (Edinger-Westphal nucleus).

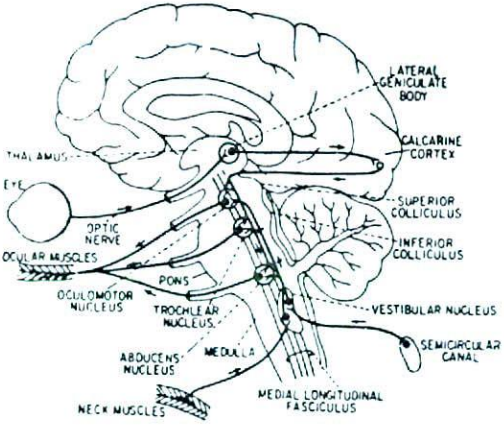


FIG. 6.33. Diagrammatic representation of reflex innervation of the ocular muscles.

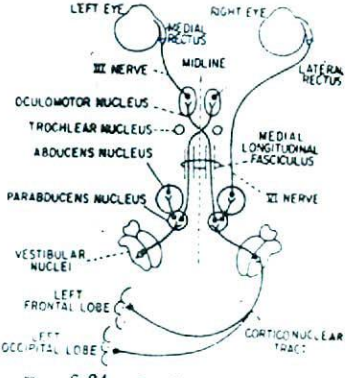


FIG. 6.34. It shows conjugate right gaze. Impulses for conjugate movements in the left frontal lobe (voluntary) and the left occipital lobe (involuntary).

The chief intercommunications are as follows:

(*a*) Nuclei for lateral rectus of one side and medial or internal rectus of other (FIG. 6.34) (conjugate deviation).

(*b*) ,, ,, constrictor pupillae, ciliary muscles and internal rectus (accommodation).

(*c*) ,, ,, superior rectus of both sides (FIG. 6.35) —upward movement.

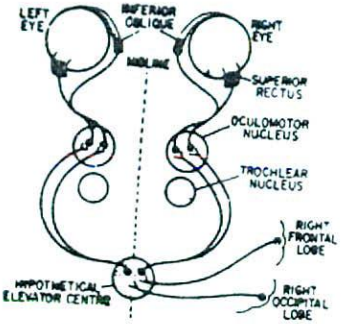


FIG. 6.35. It shows central organisation for conjugate elevation of eyes.

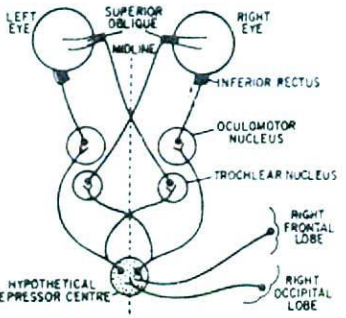


FIG. 6.36. It represents central organisation for conjugate depression of eyes.

(*d*) Nuclei for inferior rectus of both sides (FIG. 6.36).—downward movement.

(*e*) ,, ,, superior oblique of one side and inferior oblique of other (conjugate rotation).

(*f*) ,, ,, superior rectus and inferior oblique of same side (FIG. 6.37B) (deviation of one corrected by other).

(*g*) ,, ,, inferior rectus and superior oblique of same side (FIG. 6.37A) (deviation of one corrected by other).

(*h*) ,, ,, superior rectus and levator palpebrae superioris of same side (raises the lid when eyes move up).

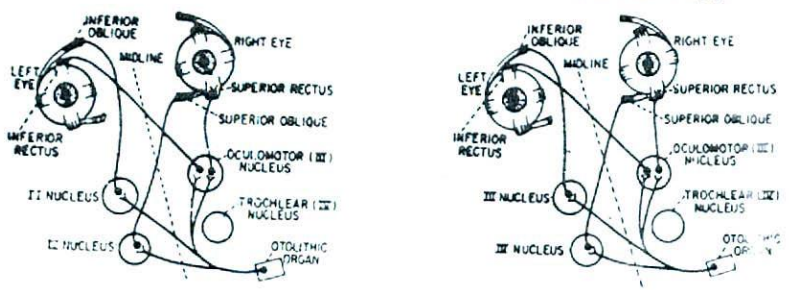


FIG. 6.37. A represents effects of head tilting towards the right shoulder. Du lack of any compensating mechanism, the vertical meridians of the corneas are t towards the right. B shows the otolithic organ which compensates for this and vertical meridians become perpendicular (*after Adler*).

Squint or Strabismus. Anatomical or functional defects of exter or lateral ocular muscles cause various types of squint.

3. **Cerebellar control.** Through vestibular nuclei and posterior longitudinal bundle.

4. **Midbrain control.** Through superior corpora quadrigemina and possibly others.

5. **Cerebral control** (FIG. 6.38) Concerned with both voluntary and reflex eye movements. Co-ordinates the action of antagonistic muscles (reciprocal innervation). A number of areas are concerned: (*a*) Frontal

eyefield (*vide* page 5-233). (*b*) Occipital eyefield (*vide* page 5-239), (*c*) Parietal and temporal adversive fields (*vide* pages 5-237 – 5-238).

Effects of section of III cranial nerve. Muscles supplied by the III cranial nerve will be paralysed. **Effects:** (1) Ptosis (drooping of the upper lid), (2) External strabismus, (3) Crossed diplopia, (4) Slight protrusion of the eyeball, (5) Loss of accommodation, (6) Dilatation of pupil, (7) Loss of light reflex, (8) Inability to move eyeball inwards. Downward and upward movements are also restricted.

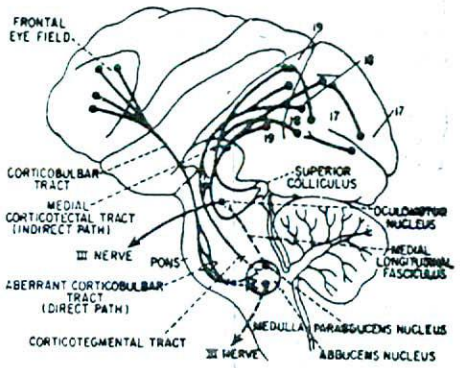


FIG. 6.38. Diagram showing the pathways for the cortical control of eye movements (*after Crosby* and *drawn from Dawson's*).

Effects of section of VI cranial nerve. (1) Internal strabismus, (2) Homonymous diplopia, (3) Inability to move the eyeball outwards.

Effects of section of IV cranial nerve. (1) Slight upward deviation of cornea due to the rolling up of the eyeball, (2) Homonymous diplopia while looking downwards.

IRIS

Iris is a circular partition just in front of the lens. It has a central round aperture—**pupil.** The diameter of pupil can be altered (2 mm to 8 mm) and in this way iris acts as an adjustable diaphragm. The centre of the pupil is exactly against the centre of the lens.

Histology. Iris consists of the following layers:

1. **Squamous endothelium**—It is continuous with that of the posterior surface of the cornea.

2. **Anterior border layer**—It is present immediately after endothelial layer. The colour of the iris depends mostly upon the thickness of this layer and also of the amount of pigments present in this layer. In subjects having brown iris, the cells are heavily pigmented but in case of subjects having blue iris, this layer is thin and faintly pigmented.

3. **Vessel layer** or **stroma**—This layer consists of (*a*) blood vessels, (*b*) collagen fibrils, (*c*) elastic fibres, (*d*) pigmented stroma cells. There is a circular band of smooth muscle in the stroma of the pupillary zone. This circular smooth muscle is known as **sphincter pupillae** which encircles the pupil and on contraction reduces the diameter of the opening.

4. **Dilator pupillae**—It extends from the ciliary border of the sphincter muscle to the root of the iris. Langley and Anderson first proved the presence of this muscle through their experiment. Contraction of dilator muscle produces dilatation of the pupil.

5. **Pigment epithelium**—This layer is continued from the ciliary body over the posterior surface of the iris. This layer is densely pigmented and the cell bodies and the nuclei are not deciphered.

Thus the iris has got two types of muscles—the *sphincter pupillae* and the *dilator pupillae*. These two muscles along with the ciliary body, comprise the intrinsic muscles of the eye. The sphincter is innervated by the parasympathetic whereas the dilator pupillae is by the sympathetic. The efferent fibres subserving light reflex are lying in the parasympathetic division of the autonomic nervous system.

The **parasympathetic nerve fibres** supplying the sphincter pupillae originate from the oculomotor nucleus (probably the Edinger-Westphal nucleus) and run *via* the III cranial nerve, ciliary ganglion and short ciliary nerve (FIG. 6.39).

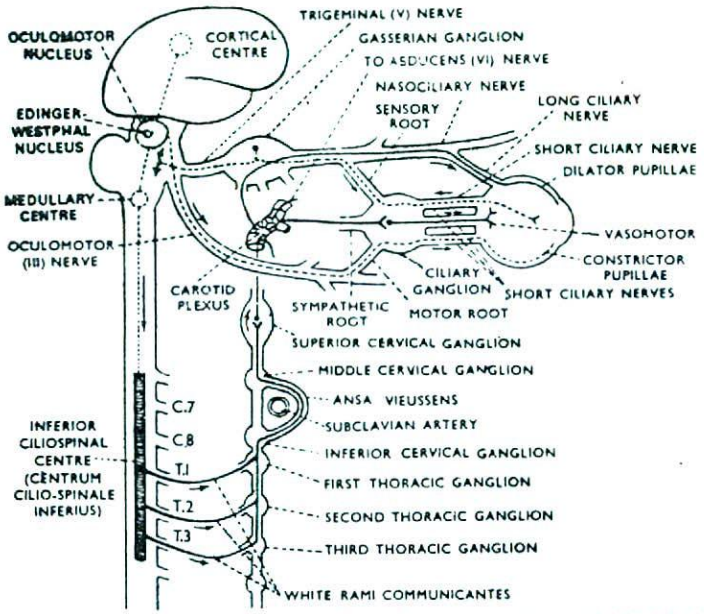


FIG. 6.39. Diagram showing ciliary ganglion with its connections and also origin and course of sympathetic and parasympathetic nerves supply to the muscles of the iris (*after Adler*).

Sympathetic fibres supplying the dilator pupillae originate from the first and second thoracic segments of the spinal cord and sometimes from the eighth cervical or the third thoracic segment. From here the fibres, through white rami, pass into the cervical sympathetic chain and end in the superior cervical ganglion. From here the postganglionic fibres arise and run along the internal carotid artery and *via* the Gasserian ganglion and nasociliary branch of the first division of the V cranial nerve ultimately end in the dilator pupillae in the long ciliary nerve.

Contraction of the sphincter pupillae alone or in association with relaxation of the dilator pupillae causes constriction of the pupil. Constriction of the pupil is called **miosis.** Contraction of the dilator pupillae along with relaxation of the sphincter pupillae causes dilatation of the pupil. The dilatation of the pupil is called **mydriasis.** Drugs that cause

constriction of the pupil are called *miotics* (Fig. 6.40). Parasympathomimetic substances are miotics. These are acetylcholine, eserine, prostigmin, pilocarpine. Drugs that dilate the pupil are *mydriatics*. Mydriatics are (*i*) *parasympatholytic* drugs—atropine (Fig. 1107), homatropine, scopolamine;

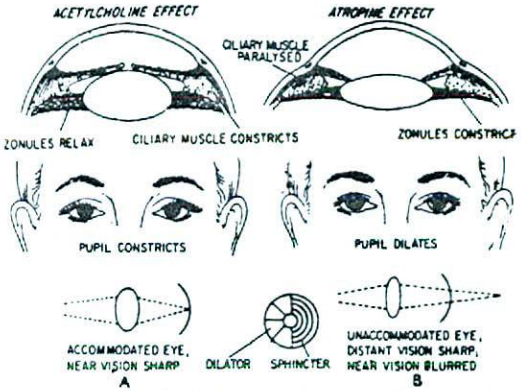


Fig. 6.40. Drawing shows accommodation. A = the eye is shown with the ciliary muscle and the sphincter muscle of the pupil relaxed by atropine. B = the ciliary and the sphincter muscles are contracted by a cholinergic drug or by parasympathetic nerve activity. Contraction of both the circular and radial fibres of the ciliary muscle moves the ciliary body toward the optical axis, loosening the fibres of the suspensory ligament and allowing the lens to become more convex. Radial fibres are fixed to the scleral spur, and shortening (contraction) has the same effect as contraction of the circular fibres. Under parasympathetic influence, the eye is thus accommodated for near vision. The pupil is constricted by the contraction of the sphincter muscle of the pupil. Constriction of the pupil—movement of the iris toward the optical axis—pulls the structures away from the angle and facilitates drainage of aqueous.

(*ii*) *sympathomimetic* drugs—catecholamines, norepinephrine-like substances, such as paredrine, amphetamine and amine oxidase inhibitor—cocaine.

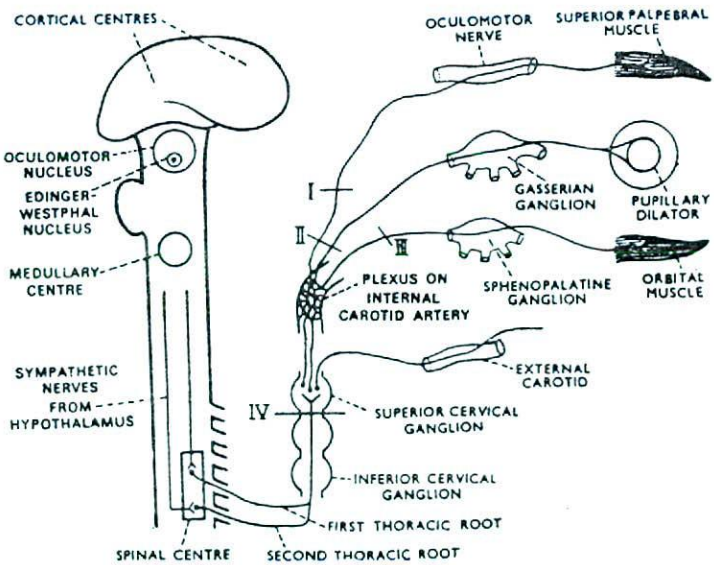


Fig. 6.41. Distribution of the cervical sympathetic nervous system. Lesions at I = ptosis, II = ptosis and narrowing of the pupil, III = enophthalmos (theoretically) and IV = symptoms of Horner's syndrome produced (*after Adler*).

Effects of sections of the nerves at different levels produce (*a*) ptosis, (*b*) ptosis and narrowing of the pupil, (*c*) enophthaimos (theoretically), and (*d*) symptoms of Horner's syndrome as shown in the FIG. 6.41.

Functions of iris (1) It adjusts the amount of light falling on the retina. (2) By cutting off the peripheral rays it helps to avoid errors of refraction (such as spherical aberration), and thus produces better *definition* of the image. (3) Increases the depth of focus.

PUPIL

Pupil, the central round aperture of the refractive system of the eye; is controlled by the iris. The iris behaves like a diaphragm. The normal size of the pupil is 3–4 mm. The size of the pupil varies with ages. It is small in new-born infant. In childhood and also in adolescence the pupils are at maximum size and in advanced age it is often *miotic*. Besides this, the **pupil in woman is larger than that in man.**

If the two pupils are unequal then the condition is described as **anisocoria.** Anisocoria is harmless but should not be considered as normal. Unilateral or bilateral lesions may produce anisocoria.

Functions of pupil. There are three main functions of the pupil:

1. Pupil modifies the amount of light entering the eye. The amount of light that enters the eye is directly proportional to the area of the pupil. In nocturnal animals, pupil size is of great importance in permitting proper light during night and daytime.

2. Pupil controls the depth of focus of the optical system of the eye. Smaller pupil increases the depth of focus (FIG. 6.42).

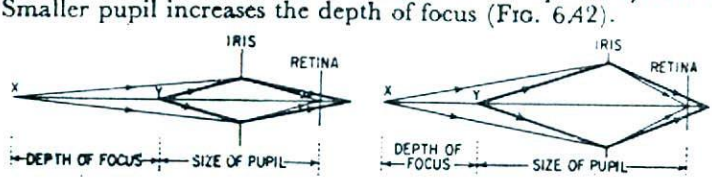


FIG. 6.42. It shows the influence of the size of the pupil on the depth of the focus. Smaller pupil increases the depth of focus (A) and larger pupil decreases the depth of focus (B).

3. Acuity of vision is dependent upon pupillary size. Spherical aberrations are minimised by the reduction of the pupillary size.

Different factors may alter the size and appearance of the pupil and these have been presented in the following tabular form:

Factors constricting pupil	Factors dilating pupil
1. When light falls on retina (light reflex).	1. When eyes exposed to dark.
2. Accommodation for near vision.	2. When eyes set for distant vision.
3. During sleep.	3. Paralysis of III cranial nerve.
4. Stimulation of III cranial nerve.	4. Stimulation of cervical sympathetic.
5. Paralysis of cervical sympathetic.	5. Emotions—fear, anxiety, fatigue.
6. Severe pain.	6. Exhaustion of the central nervous system, e.g., deep anaesthesia, asphyxia, coma, etc.
7. Loss of aqueous humour or fall of intraocular pressure. Hence, vessels dilate and pupils constrict.	7. Rise of intraocular pressure, e.g., glaucoma.

Factors constricting pupil	Factors dilating pupil
8. Drugs (*myotics*): (*a*) eserine, pilocarpine—stimulate III cranial nerve endings, (*b*) opium and morphine—stimulate III cranial nerve nucleus.	8. Concussion or compression of brain.
9. During induction of inhalation anaesthetic—ether, chloroform, etc.	9. Hyperactive thyroid or adrenal medulla (also epinephrine injection).
10. *Orbicularis reflex* (*lid-closure reflex*). Pupil is constricted when the eyelid is kept open although the effort of closure is made.	10. Stimulation of the skin of the neck (*ciliospinal reflex* or pupillary neck reflex).
	11. Drugs (*mydriatics*). (*a*) Atropine, homatropine, belladonna—paralyse the III cranial nerve endings, (*b*) cocaine—sympathetic stimulation.
	12. *Oculosensory reflex*. Irritation of the eyelids or conjunctiva causes dilatation followed by constriction of the pupils.
	13. *Psychosensory reflex*. Emotional upset (fear) may dilate the pupil.
	14. *Rapid rotation of the body* around its long axis may dilate the pupil (stimulation of labyrinthine receptor).

PUPILLARY REFLEXES

I. Reflexes constricting the pupil

1. **Light reflex.** If bright light falls on the eye and the intensity of illumination is increased gradually and reaches above a threshold value then pupils constrict. This is known as *light reflex*. Light reflex is of two types, such as **direct** and **consensual** (*indirect*). **Direct light reflex**—Constriction of the pupil due to fall of light directly on the eye is the *direct light reflex*. **Consensual light reflex**—This is also called *indirect light reflex*. If light is directed on one eye then constriction also occurs on the other eye. This is dependent upon fibres which cross to the pupillary constrictor centre of the opposite side.

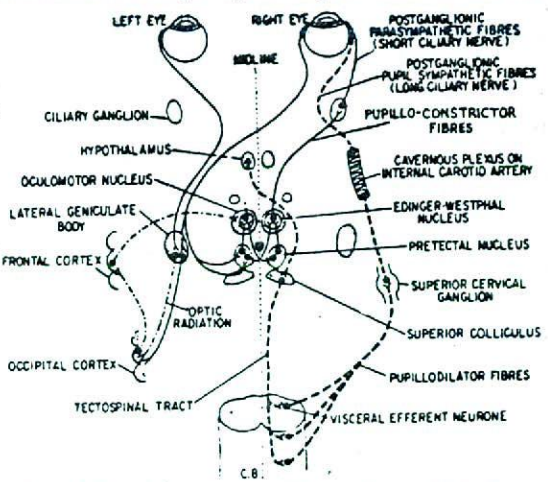


Fig. 6.43. It illustrates pupillary reflexes. *Plain lines* indicate pupillo-constrictor reflex. *Thin dotted lines* show path of near reflex from lateral geniculate body of the thalamus to the cortex and oculomotor nucleus. *Deep dotted lines* represent pupillo-dilator reflex.

Nerve path (Fig. 6.43). Visual impulses arise in the rods and cones, pass through the different layers of retina, enter the optic nerve and then the optic chiasma. Here the nerve partially decussates and the fibres enter the optic tracts of both sides. From the optic

tract fibres pass out and end in the *pretectal area* (some hold—superior colliculus), where a new relay arises (colliculonuclear fibres), crosses both in front and behind the aqueduct and ends in III cranial nerve nucleus (most anterior part—Edinger-Westphal nucleus). Fibres of III cranial nerve relay in the ciliary ganglion from where postganglionic fibres arise and supply the sphincter pupillae through short ciliary nerves.

The reflex arc for the direct and indirect light reflexes has been presented in FIG. 6.43.

Functions of light reflex. (1) To adjust the amount of light entering the eye. (2) To prevent spherical aberration by cutting off the peripheral rays. (3) To increase the depth of focus.

2. **Near reflex (Accommodation-convergence reaction).** Constriction of the pupil during focussing on a near object. This is a part of the mechanism of accommodation to near vision. This is also accompanied by convergence of the eyes on to a near object and accommodation of the lens. All the reactions—pupillary constriction (miosis), convergence and accommodation are mediated by the oculomotor nerve (C.N. III) (*vide* Accommodation).

3. **Lid-closure reflex.** It is also called *orbicularis reflex*. Constriction of the pupil occurs when the effort for closure of the eyelid is made though the eyelids are kept open. The mechanism is central in origin and oculomotor nucleus may be a factor in the reflex.

4. **Oculopupillary reflex (Oculosensory reflex).** It is also called **trigeminal reflex.** Pupil is constricted if the conjunctiva or cornea is irritated. The pathway for this reflex is the ophthalmic branch of the trigeminal nerve (C.N. V) to the Gasserian ganglion and the nucleus of V cranial nerve. By way of medial longitudinal bundle, connecting fibres finally run to the sphincter nucleus.

The constriction of the pupil is sometimes happened due to reflex vasodilatation of the iris capillaries through local axon reflex.

II. REFLEXES DILATING THE PUPIL (FIG. 6.43)

1. **Psychosensory reflex.** Emotion like fear and anxiety may cause dilatation of the pupil. It is caused by stimulation of the sympathetic nerve.

2. **Withdrawal reflex.** Withdrawal of light from the eyes causes dilatation of the pupil.

3. **Labyrinthine pupillary reflex.** Dilatation of the pupil due to rotation of the body around its long axis. This is due to stimulation of the labyrinthine receptors.

Argyll-Robertson pupil

It is a condition when light reflex (direct or indirect) is lost, but the near reflex (accommodation-convergence reflex) is retained. It is commonly seen in syphilitic degeneration of the central nervous system and in other conditions. The pupils are generally smaller than normal (miotic) and frequently unequal in size. The pupil does not dilate to a painful stimulus and even does dilate poorly with atropinisation or cocainisation. Even if the patient is kept for a long time in the dark, the pupils do not dilate. Though usually bilateral it sometimes occurs unilaterally.

The seat of lesion is not precisely known, but the evidence supports the theory of its being in the intercalated fibres just before they reach the

sphincter nucleus. It is also suggested that lesion may be close to the oculomotor nucleus so that both the crossed and uncrossed pretectal fibres are affected. Possibly the lesion of fibres from pretectal nucleus to Edinger-Westphal nucleus may cause typical symptoms of Argyll-Robertson pupil (FIG. 6.44). This will interrupts the light reflex (direct and indirect) without affecting the near reflex as the fibres descend to the oculomotor nucleus away from the viscinity of the intercalated fibres.

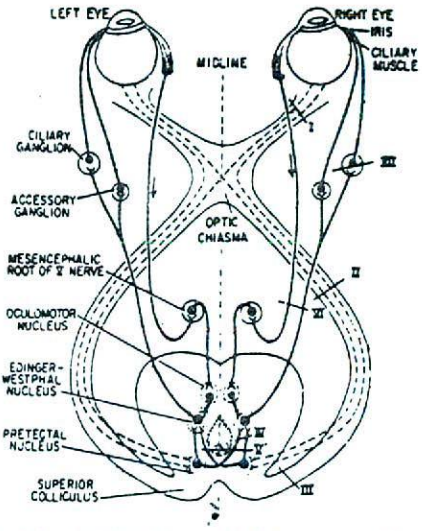


FIG. 6.44. It shows the pupillary afferent (*broken lines*) and efferent (*solid lines*) pathways for light reflex and miosis of accommodation.
I=Lesion at optic nerve. Loss of direct and opposite consensual reaction. Retention of near reflex.
II=Lesion at optic tract. Contralateral hemianopsic loss of reaction (Wernicke's).
III=Lesion at optic tract beyond the exit of the pupillary fibres. No pupillary reaction loss (right homonymous hemianopsia present),
IV=Lesion between decussation and Edinger-Westphal nucleus. Loss of ipsilateral direct and opposite consensual reaction. Near reflex intact (unilateral Argyll-Robertson pupil),
V=Lesion of all fibres from pretectal nucleus to Edinger-Westphal nucleus. Loss of all light responses. Retention of near reflex (complete Argyll-Robertson pupil).
VI=Lesion of oculomotor nerve. Absolute ipsilateral pupillary paralysis.
VII=Lesion of ciliary ganglion. Ipsilateral loss of light reflex with retention of near reflex (unilateral Argyll-Robertson pupil) (*after Duke-Elder;* and *Vaughan et al*).

A bilateral lesion affecting the pathway from the cortex to the centre for accommodation in the oculomotor nucleus will cause a loss of the near reflex, but light reflex (direct and indirect) will remain intact. This is just the reverse of the Argyll-Robertson pupil. It is happened sometimes in postdiphtheritic paralysis (FIG. 6.44).

Just the reverse condition is called **Wernicke's hemianopsic pupillary reaction**—where light, falling on the blind half of retina, elicits light reflex but not accommodation reflex.

ACCOMMODATION

Definition. Adjustment of the optical apparatus so as to change the refractive power of the eye, when the image of a near object is brought into focus on the retina is described as accommodation. Such mechanisms involve an increase in dioptric power of the lens. The normal resting eye (**emmetropic**) is set for parallel rays (20 ft. or beyond—far point) and is capable of focussing the distant object without accommodation, but from nearer objects are divergent and unless the refractive power of the media be adequately raised, they will not be focussed on the retina but behind it. Normally, this is done by increasing the curvature of the lens and thus raising its refractive power. *Accommodation is not a pure reflex process. It is to some extent a 'willed' movement.* Because, the object must be inten-

tionally looked at before the changes can occur. Hence, it is called **accommodation reaction (convergence-accommodation reaction).**

If object is brought closer to closer then a point is reached at which in spite of strong contraction of the ciliary muscle the object is not focussed. The rays from the object is so divergent that the object cannot be focussed (FIG. 6.45). The nearest point at which the subject is clearly focussed with full accommodation is the **near point.** Distance between the near point and the eyes increases with age. It is increased very rapidly in early forty and then slowly in after fifty. **Presbyopia** or **old-sightedness** is the loss of accommodative power of a near object. This is detected only when reading is interfered with at the age of 40–45 years.

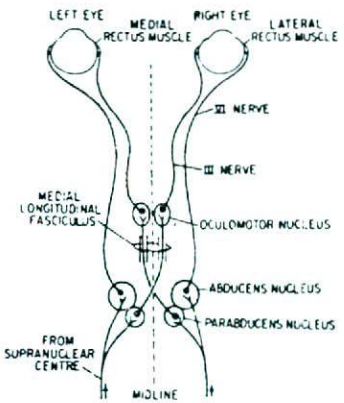


FIG. 6.45. Mechanisms of convergence and divergence (diagrammatic).

In normal **emmetropic eye** parallel rays are focussed on the retina from infinity (more than 20 ft.) without accommodation (rest). Thus 20 ft. is the **far point** of the normal eye.

The 'far point' of the eye is at infinity (6 m or 20 ft. and beyond). The 'near point' varies from 9–50 cm (average 6") according to age. As age advances, the power of accommodation becomes lowered and the near point recedes. The difference between far and near points is called the *range of accommodation.* The difference of refractive powers between a resting eye and a fully accommodated one is called the *amplitude of accommodation.*

Mechanism of accommodation

Following changes take place:

1. **Increased curvature of lens.** Helmholtz explained the changes in the curvature of lens in the following manner: When the eyes are accommodated for **distant vision,** the ciliary muscle is relaxed; and the

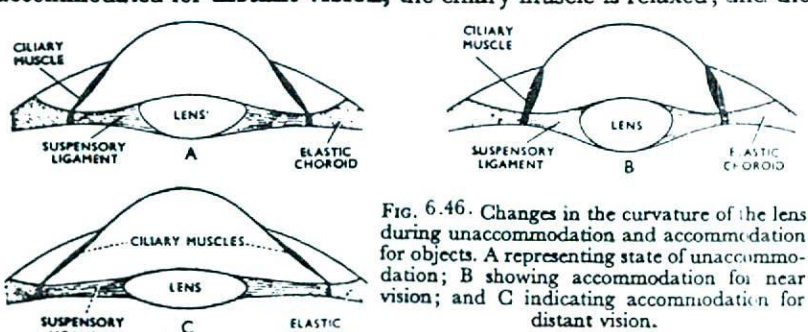


FIG. 6.46. Changes in the curvature of the lens during unaccommodation and accommodation for objects. A representing state of unaccommodation; B showing accommodation for near vision; and C indicating accommodation for distant vision.

choroid, due to its elastic property, recoils and pulls the suspensory ligament peripherally (FIG. 6.46 C). Due to peripherally directed traction of the suspensory ligaments, the anterior surface of the lens is flattened and the object is correctly focussed on the retina.

When the eyes are accommodated for **near vision** contraction of ciliary muscles draws the choroid inward, causing relaxation of the suspensory ligaments. As a result there is relaxation of the capsule of the lens. Due to relaxation of the capsule, the lens assumes a more convex form (Fig. 6.64B). The anterior surface bulges more than the posterior, because the latter is prevented by the vitreous humour.

Helmholtz's hypothesis has been explained by Gullstrad through a mechanical model as shown in Fig. 6.47. The lower spring represents the elastic choroid and the upper one represents the lens with its capsule. The string in between the two springs is the **zonule of Zinn.** A cord from the lower spring passes over a pulley supporting a weight (W), represents the pull of the ciliary muscle. At rest or when the eye is not accommodated, the weight (W) is at rest and there is no pull (Fig. 6.47A). But during accommodation the weight moves down —pulling the lower spring (elastic choroid) forward and thus slackens the zonule of Zinn. The slackening of the zonule allows the upper spring (capsule and lens) to contract. The contraction of the upper spring represents the relaxation of the lens capsule (Fig. 6.47B). These phenomena actually happen during accommodation for near vision by the relaxation of the zonule of Zinn (Fig. 6.48).

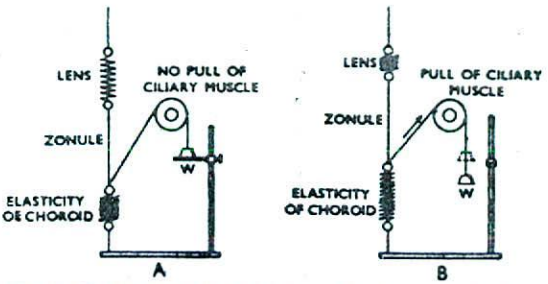


Fig. 6.47. Mechanical model shows the forces producing accommodation according to Young-Helmholtz's theory.

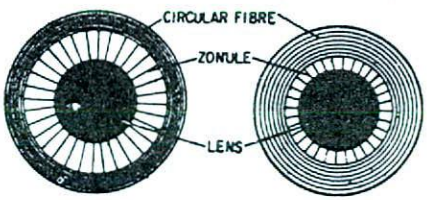


Fig. 6.48. It shows the action of the circular fibres in relaxing the tension of the zonule (diagrammatic).

The changes in the curvature of the lens, i.e., the increased convexity of the lens during accommodation can be determined by the **phakoscope of Helmholtz** (Fig. 6.49). With the help of this instrument following experiment can be performed.

When a lighted candle is kept on the outer side of a subject's eye, three images (**Purkinje-Sanson images**—Fig. 6.50). of the flame within

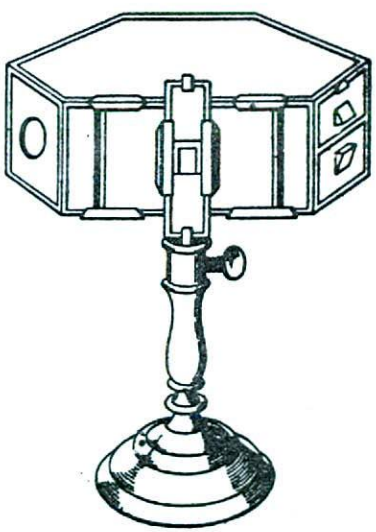


Fig. 6.49. Helmholtz's phakoscope.

the subject's pupil are seen by an observer. (*a*) A small, erect and bright image formed by the cornea. (*b*) A larger, erect and less distinct image formed by the anterior convex surface of the lens. (*c*) A smaller, indistinct and inverted image formed by the posterior surface of the lens which acts like concave mirror.

When the eye is focussed to a near object the second image (*b*) becomes smaller and moves towards the first (FIG 6.50B). The second

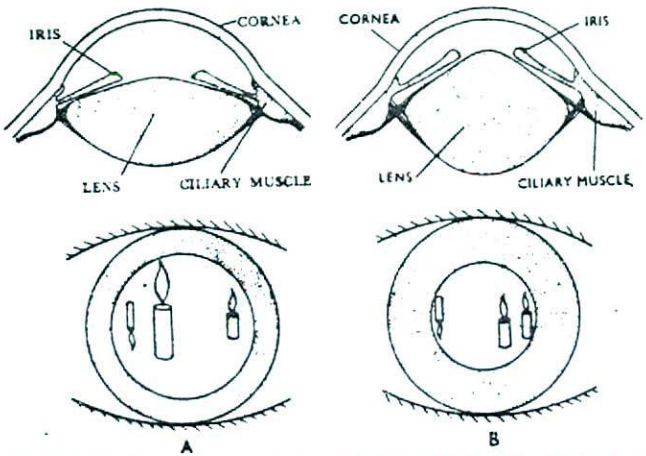


FIG. 6.50. Purkinje-Sanson images. A showing the reflections of a candle in the pupil with the subject looking at a distant object and B also the same at a near object. The erect image *on the right* is (*a*), *on the middle* large and erect is (*b*) the inverted image *on the left* is (*c*).

image again becomes larger when the eye is focussed to a *far point* (FIG. 6.50A). There is no change of the first and third images. This experiment thus shows that during accommodation no change occurs either in the curvatures of the cornea or in the posterior surface of the lens, but there is increased curvature of the anterior surface of the lens.

2. **Constriction of pupils**—caused by the contraction of the sphincter pupillae by the III cranial nerve.

3. **Convergence of two optical axes**—due to internal rotation of eyeballs caused by contraction of internal recti by the III cranial nerve (FIG. 6.41).

Nerve path for accommodation reflex (FIG. 6.51). The nerve pathway for the accommodation reflex consists of the optic nerve, optic tract, lateral geniculate body of the thalamus, optic radiation and

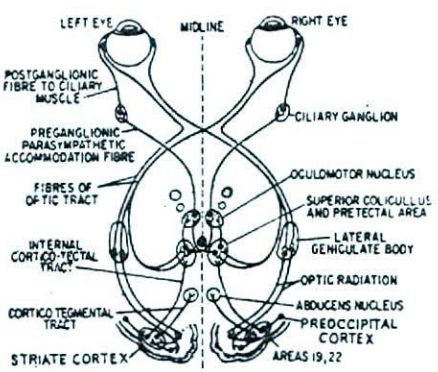


FIG. 6.51. It shows nerve pathways for the accommodation reflex (diagrammatic).

the visual area of the cerebral cortex. From the occipital cortex impulses are relayed to the frontal eyefield (area 8) by the superior longitudinal association tract, whence nerve fibres descend through the anterior limb of the internal capsule to the oculomotor nuclei of the opposite side. From the Edinger-Westphal nucleus nerve fibres pass to the ciliaris and sphincter pupillae relaying in the ciliary ganglion and from the ventral nucleus of the oculomotor nuclei supply the medial recti for the action of convergence. These pathways are still debatable as those for the pupillary light reflex and it has been suggested by Wilkinson (1927) that the contraction of the pupil in the accommodation reflex is secondary to the convergence of the eyes and that the afferent impulses arise in the proprioceptor nerve endings in the orbital muscles and travel in the oculomotor nerve direct to the Edinger-Westphal nucleus.

Near response. When a person looks at a near object, his pupil constricts and along with the accommodation the visual axes converge. There are three responses: (*i*) *pupillary constriction*, (*ii*) *convergence*, and (*iii*) *accommodation*—together three are called the *near response*.

COMMON ERRORS OF REFRACTION

The normal eye with correct refraction is called **emmetropic.** Emmetropic eye is capable of focussing the distant object without accommodation. Thus parallel rays from distant objects are brought to focus on the retina when the eye is at rest. Any deviation from the condition of emmetropia is called **ametropia.** But refraction may be defective in a number of ways. They are briefly given below.

Hypermetropia (long-sightedness) (Fig. 6.52)

It can see distant objects but not near ones. Because, parallel rays (from distant objects—6 metres (20 ft.) or beyond) are focussed on retina, but divergent rays (from near objects) behind the retina. **Two varieties:** (*i*) **The childhood variety.** Here the eyeball is very short. Because, the optical system sometimes develops much faster than the size of the eyeball. As the child grows, the eyeball becomes longer and the defect disappears (in a few cases it persists). (*ii*) **The old age variety** (between 40 and 45 age group) or **presbyopia.** Due to reduced power of accommodation; caused by less elasticity of the lens and weakness of the ciliary muscles.

Remedy. *Convex glasses* (convergent—positive power) *

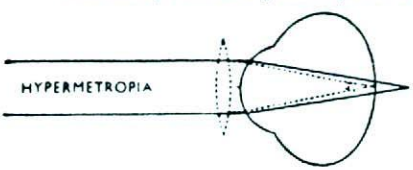


Fig. 6.52. Hypermetropia—correction by convex lens.

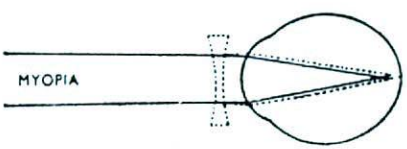


Fig. 6.53. Myopia—correction by concave lens (*Winton & Bayliss*).

Myopia (short-sightedness) (Fig. 6.53)

It can see near objects but not distant ones. The eyeball is elongated, so that parallel rays are focussed in front of retina but divergent rays are focussed on it.

Remedy. *Concave glasses* (divergent—negative power).

* Light is always refracted towards the thicker part of the lens.

Astigmatism

Astigmatism (Gr. *a-*, privative or negative; *stigma*, a point) is the condition, where the rays of light are not brought into sharp focus at the retina. It is the refractive error of the lens system of the eye due to (*i*) irregular or

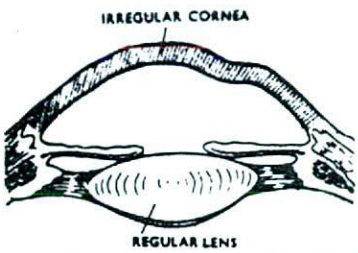


FIG. 6.54. Schematic representation of astigmatism from an irregular cornea.

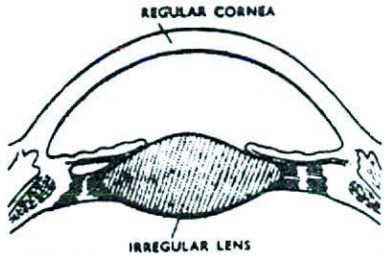


FIG. 6.55. Schematic representation of astigmatism from an irregular lens.

oblong shape of the cornea (commonest) (FIG. 6.54) or (*ii*) also of the lens (FIG. 6.55). The curvature of an astigmatic lens along one plane is not similar with the curvature at other plane. For this reason light rays falling on one plane or on other plane of an astigmatic lens do not fall at a common focal point. In an astigmatic lens with greater curvature in vertical plane (A–C) and lesser curvature in horizontal plane (B–D) (FIG. 6.56), light rays in the vertical plane are refracted more greatly than in the horizontal plane. Thus the light rays passing through astigmatic lens do not converge on a common focal point due to the unequal curvature as well as unequal refractive power of the lens at different planes.

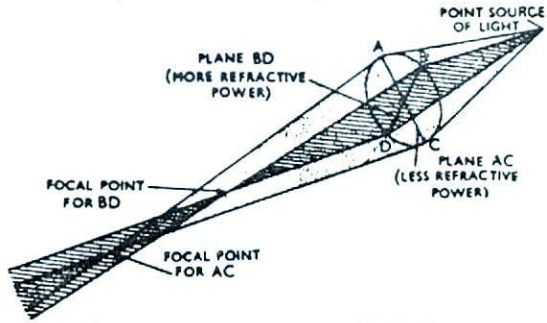


FIG. 6.56. Diagram showing rays of light focus at one focal distance in one focal plane and at another focal distance in the plane at right angles (*after Guyton*).

Remedy. Astigmatism may be corrected by a cylindrical lens or by combination of spherical and cylindrical lenses of such strength and so placed that they equalise the refraction in the meridians of the greatest and least curvature.

Contact lens. It consists of a glass shell fitted close apposition to the cornea and sclera, and accompanies the eye in its movements. It has got different portions. The **corneal segment** which covers the cornea corrects optical defects by eliminating the cornea as a refracting surface and, in effect, replacing it by a perfectly spherical one. The common principle of the use of a contact lens is indicated in FIG. 6.57A.

The corneal segment is separated from the cornea by a thin film of saline or tears. Refraction of light takes place at the anterior and also posterior surfaces of the contact glass since the film of saline has got approximately

the same refractive index as that of the cornea, so further refraction does not take place in the saline film until the lens is reached. Thus the cornea will be replaced as a refracting surface. If there are irregularities (astigmatism, etc.) they are not capable of influencing the refraction of light since the saline layer fills with these irregularities and gives a surface corresponding exactly in shape with that of the posterior surface of the contact glass.

In the case of emmetropic eye apart from these corneal defects the contact glass need only be designed by making the curvatures of the two surfaces of this glass equal to that of the cornea and in this event the glass is *afocal*.

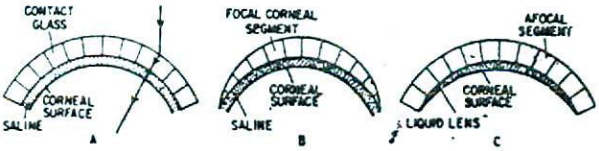


FIG. 6.57. A=showing refraction of a ray by an afocal contact glass, B=glass lens, and C=liquid lens.

For the correction of a spherical refractive error, modification of the curvature of the anterior surface of the contact glass be needed. When this curvature is made greater, the contact glass behaves as a concavo-convex lens and corrects hypermetropia.

Glass and Liquid lens systems. The *glass lens system* is that system when the spherical correction in a contact lens is achieved by modifying the curvature of the anterior surface (FIG. 6.57B), the increased dioptric power being provided by the extracurvature of the anterior surface of the contact glass. When the glass is kept afocal by making its surfaces concentric, correction of optical defects can be made by a so-called liquid lens system by modifying the effective curvature of the cornea (FIG. 6.57C). But the term, liquid lens is somewhat of a misnomer. The liquid between the glass and the cornea is lens-shaped, but since the refractive index of the liquid is taken as being equal to that of the cornea, the liquid behaves optically, not as a lens, but as a single spherical refracting surface—a modified cornea.

Advantages of a contact lens. Apart from the cosmetic value, the contact lens is of prime importance in correcting the irregular *astigmatism*. It is also very important in correcting *hypermetropia* without producing retinal image of different sizes in subjects having unilateral *aphakia* (conditions having unilateral removal of the lens).

Spherical aberration

The peripheral rays in a convex lens are focussed at a nearer point than the central rays, so that the margins of the image become blurred. In the normal eye it is **corrected** in two ways: (*a*) The iris shuts off the peripheral rays. (*b*) The central portion of the lens has a higher refractive power than the peripheral portion. Hence, all the rays are brought to the same focus.

Chromatic aberration

Lights of different colours (e.g., of different wave lengths) undergo different degrees of refraction. Red light (shortest) is refracted least. Violet rays (longest) are refracted most. Hence, the margin of the image may show rainbow colours. The lens of the human eye has the same defect. It is

normally rectified in two ways: (*i*) The difference of refractive powers of the various refractive media of the eyeball partly rectifies it. (*ii*) The colour fringes are ignored by the brain.

RETINA

Anatomy. It is the light sensitive nervous layer situated between the choroid and vitreous. It ends just behind the ciliary body in a serrated border—the *ora serrata*. But the pigment layer is prolonged further on to the inner surface of the ciliary body and the iris. Opposite the pupil, lies the yellow spot (*macula lutea*) having a central depression (0·44 mm)—the *fovea centralis*. The yellow colour is due to a pigment which is bleached by light. A little medial to the macula (3·5 mm) lies the optic disc (FIG. 6.64). It is a pinkish-white oval area (1·5 mm average) through which the optic nerve fibres pass out.

Histology (FIGS. 6.58, 6.59 & 6.60). Retina is composed of *one pigment layer, seven nervous layers* and *two limiting membranes—external* and *internal*. The nervous layers are supported by elongated modified neuroglial cells, the *Müller's fibres*. Their outer ends fuse around the bases of the rods and cones and form the **external limiting membrane.** Similarly, the inner ends form the **internal limiting membrane** forming the innermost layer. The layers of the retina from the choroidal side towards the vitreous humour are as follows:

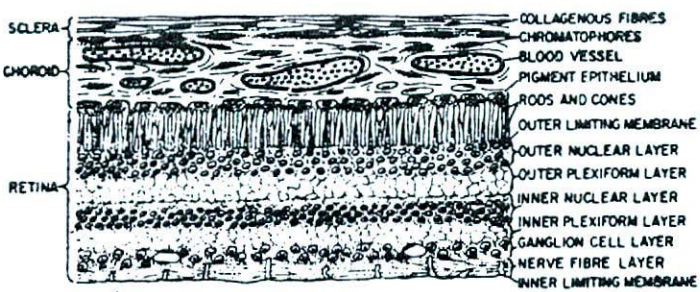


FIG. 6.58. Histological structures showing panoramic view of layers through the thickness of the retina and the choroid (diagrammatic).

1. **Pigment layer.** The retinal layer just near the choroid is known as pigmented layer. This layer consists of large hexagonal epithelial cells. The cells send processes in between rods and cones. The pigment layer contains pigment which is the inert derivative of tyrosine, **melanin.** This black melanin pigment in the pigment layer and also in the choroidal layer acts like a black paint inside a camera. This black pigment prevents reflection of light throughout the eyeball and it is very important for acute vision. The light which is not absorbed by the rods and cones is absorbed by the pigment layer and the choroidal layer. For this reason only a very small part of the entering light escapes absorption and is **reflected back.**

Bruch's membrane is present immediately against the basement membrane of the pigment epithelium (FIG. 6.59).

2. **Layer of rods and cones.** Rods and cones are the photoreceptors and are separated from one another by radially arranged **glial**

fibres (Müller's fibres). *Cones are pyramidal in shape; rods are cylindrical.* The cones vary in shape in different parts of the retina. In the fovea they are long, narrow and rod-shaped and in the periphery they are shorter and broader (Fig. 6.66).

The elongated projected parts of the rods and cones extend beyond the **external limiting membrane.** The *cones are concerned with bright light vision, acuity of vision and colour vision.* The cones contain a pigment known as **iodopsin.** The rods contain a pigment called **rhodopsin** and are concerned with dim light

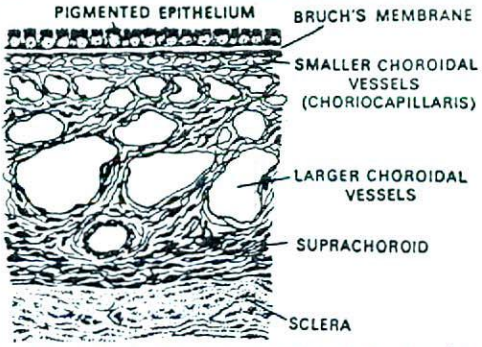


Fig. 6.59. It shows cross-section of the choroid (diagrammatic).

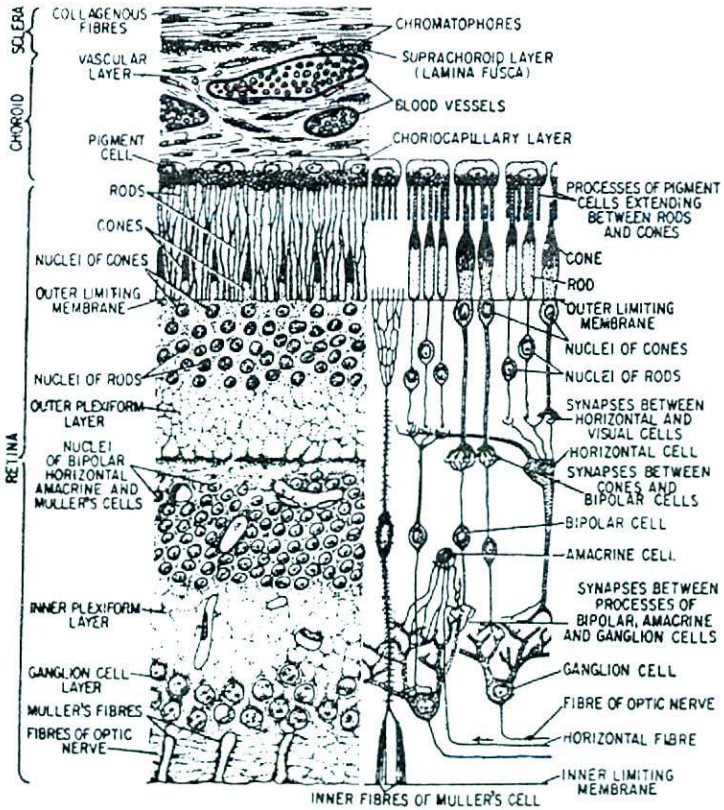


Fig. 6.60. Diagrammatic representation of the layers of the retina and choroid in detail.

vision. Both are set perpendicular to the surface and possess a smaller outer segment and a larger inner segment (*vide* under 'Rods and Cones').

3. **External limiting membrane.** This is a thin sieve-like membrane formed by the lateral processes of the radially arranged glial fibres at the bases of the rods and cones. The glial cells are very rich in **glycogen.**

4. **External nuclear layer.** This layer is composed solely of nuclei of rods and cones (also called rod granules and cone granules). The rod nuclei are smaller, round and are stained intensely whereas the cone nuclei are larger, oval and are stained faintly. They constitute the *first-order neurones* in the path of visual impulses. Their axons form synapse in the next layer.

5. **External plexiform layer.** It consists of synapses formed by the axons of the rod cell and cone cell and the dendrites of the bipolar cells. Here the several rods make connection with dendrites of one bipolar cell but in case of cones, one cone axon is connected with a single bipolar cell—separately in the region of fovea (FIG. 6.61).

6. **Internal nuclear layer.** This layer contains the cell bodies and nuclei of the bipolar cells—the *second-order neurone* of the visual pathway. Beside this bipolar cell there are certain association neurones—the **horizontal cells** and **amacrine cells.** These cells serve to interconnect various regions of the retina and play an important role in the integration and summation of the responses.

Bipolar cells are of two types (Polyak). The most common one is the *diffuse, polysynaptic rod and cone bipolar cells.* This type of cell receives axons from multiple rods and also of a cone. The another type is the *individual, monosynaptic cone cell* or *midget bipolar cell.* This type of bipolar cell only connects one cone cell (FIG. 6.61).

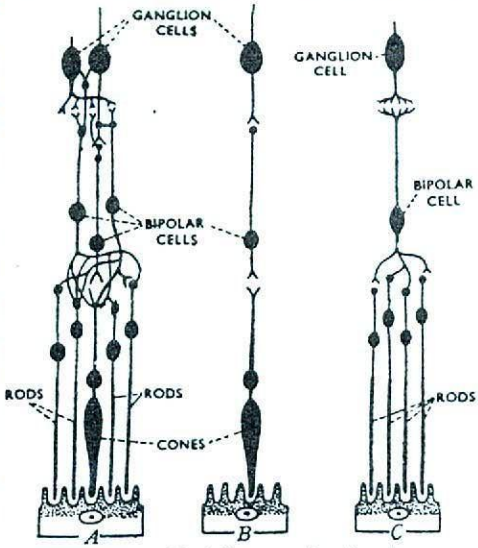


FIG. 6.61. Simplified diagram showing three probable connections between rods and cones and the ganglion cells. *A* showing connections of 4 rods and 1 cone *via* 3 bipolar cells to 2 ganglion cells; *B* representing connections of 1 cone *via* a midget bipolar cell to a single ganglion cell; *C* indicating connections of 4 rods *via* one bipolar cell to a single ganglion cell (*after Bradbury*).

7. **Internal plexiform layer.** This is composed of synapses formed by the axons of the bipolar cells and the dendrites of the ganglion cells and also processes of amacrine cells.

8. **Layer of the ganglion cells.** This layer consists of large pyriform cells with Nissl granules. Their axons form the *stratum opticum* and dendrites pass into the inner plexiform layer. **Ganglion cells** are also of two types: One is the diffuse type and connects with many bipolar cells. Another type is the monosynaptic or individual ganglion cell which

connects synaptic connection by way of **midget bipolar cells** with only one or two cone cells. This type is only seen in the foveal region, hence foveal vision is very accurate.

9. **Stratum opticum** or **Optical nerve fibre layer.** This is made up of axons of the ganglion cells which converge at the optic disc and form the optic nerve. On the inner side of this layer lies the internal limiting membrane. Retinal blood vessels are also present here.

10. **Internal limiting membrane.** This layer separates the retinal layer from the vitreous humour. It is formed by the apposition of the **glial fibres.** It is thus the innermost layer of the retina.

The light rays falling on the eye pass through cornea→pupil→aqueous humour→lens→vitreous humour→blood vessels→nerve fibres and cell bodies to reach photoreceptor cells—the rods and cones. The rods and cones are the first-order neurones and then electrical impulses from these neurones proceed, in reverse direction of rays of light, to the bipolar cells (second-order neurones) and thence to the ganglion cells (third-order neurones) and finally through the optic nerve.

Regional peculiarities of the retina

I. **Fovea.** At the posterior pole of the eye there is a yellowish pigmented spot—is called the **macula lutea** in which the fovea centralis is situated (FIG. 6.62). This is: (1) Very thin (0·1 mm), due to the absence of stratum opticum, ganglion cell, inner plexiform, inner nuclear, and outer plexiform layers. (2) *Rods—absent.* (3) *Only cones present.* (4) Cones are highly developed, longer, thinner and closely packed. They are set obliquely so that their fibres communicate with bipolar cells at the margin of the fovea. (5) One cone communicates with one bipolar cell and one ganglion cell. (6) Pigment layer—highly developed and thus the reflected light is well suppressed in the fovea. It is the point where visual acuity is maximum. (7) There is no blood vessels that cross the fovea.

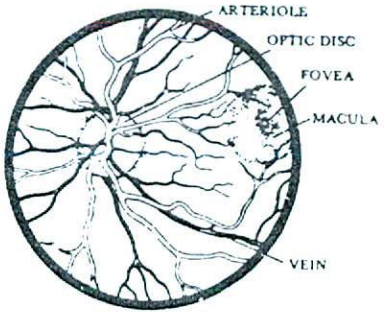


FIG. 6.62. Diagrammatic representation of the retina seen through the ophthalmoscope in a normal human eye.

Polyak has shown that the connections of the foveal cones are different from those of the other cones and of the rods. At the foveal region, each cone connects only with one small bipolar cell. Each of these midget bipolar cells connects with only one ganglion cells so that each cone of the fovea seems to have a private line in the optic nerve. In the rest of the retina, while there are some midget bipolar cells, many of them receive inputs from a number of both rods and cones. Similarly many of the ganglion cells receive inputs from a number of bipolar cells though again there are some which receive impulses from only a few cones.

II. **Peripheral retina.** Chiefly rods. From the fovea to the periphery—cones diminish and rods increase in number. Hence, in the periphery—*only twilight vision and no colour vision.*

III. **Optic disc.** The optic nerve leaves the eye and the retinal blood vessels enter through a point 3 mm medial to and slightly above at the posterior pole of the globe. Through the ophthalmoscope this region is visible as *optic disc*. There are no photoreceptors in this area and hence no vision (FIG. 6.62). Thus called **blind spot** (FIG. 6.63).

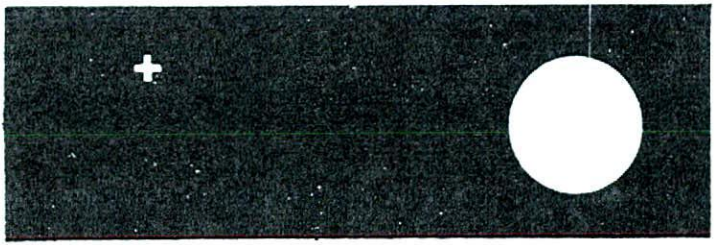


FIG. 6.63. Illustrates the presence of *blind spot*. Hold the book at about 22 cm from the eye and look at the cross with the right eye only. The white disc will not be visible. Because the image falls on the blind spot (*after Winton & Bayliss*).

Blood supply of the retina. The **retinal blood supply** is maintained through retinal circulation and choroidal circulation. The central artery forms the retinal circulation. It enters the eye at the optic disc and gives off smaller branches that course over the retinal surface. Corresponding veins return over the retinal surface to form the central retinal vein and then leaves the eye at the optic disc (FIG. 6.64). The major

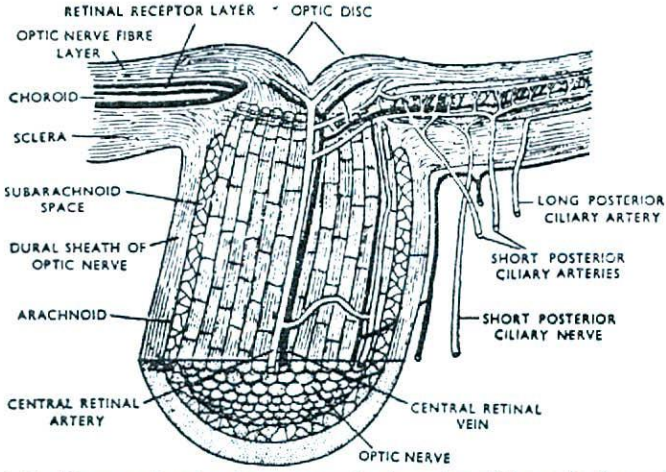


FIG. 6.64. Diagram showing the structure in the region of the optic disc and the entrance of the optic nerve and blood vessels.

retinal blood vessels, artery and vein run mostly in pairs. Capillaries of the retinal circulation are present at different retinal levels and deepest capillary plexus is seen in the outer margin of the inner nuclear layer.

The **choroidal circulation** is maintained by the vessels that penetrate the sclera to one side of the optic disc. A *rich capillary plexus*, lying

immediately against the basement membrane, is called the **choriocapillary.** This is derived from the choroidal circulation.

It is presumed that the ganglionic cell layer and inner nuclear layer are supplied by retinal circulation whereas the receptors are supplied partly by retinal circulation and partly by choroidal circulation.

Functions of the retina

1. **Vision.** Retina reacts to light of wave lengths between 390 m μ and 750 m μ. Fovea due to the presence of cones is responsible for acuity of vision, bright light or daylight or **photopic** vision and colour vision. The peripheral retina due to preponderance of rods is responsible for dim light or twilight or **scotopic** vision. The duality of the retinal receptors has been collectively known as *duplicity theory of vision.*

2. **Reflexes.** It is concerned with various reflexes: (*a*) *light reflex*, (*b*) *accommodation reflex*, (*c*) *fixation reflex*, (*d*) *visuospinal reflex*, etc.

3. **Tone, posture and equilibrium.** Retinal impulses also help to maintain tone, posture and equilibrium.

RODS AND CONES

The cones which are responsible for colour vision and the rods which cannot detect colour are not evenly distributed over the retina. Each eye contains well over 100 million rods and about 7 million cones. The cones are most densely packed in the fovea. There are no rods in the fovea and the cones themselves are finer than the cones found elsewhere. On moving out from the fovea the proportion of cones to rods gradually diminishes until at the edge of the retina no cones are found.

Fine structure. Actually, photosensitive cells (rods and cones) are situated in the outermost layer of the retina next to the pigment epithelium. Light reaching the photoreceptors first passes through the other layers of the retina, except in the foveal region. Over most of the retina, rods and cones are found side by side, although in the central fovea there are only very closely packed (rod-shaped) cones while in most peripheral regions of the retina, where the receptors are markedly separated, very few cones are found.

Rods and cones form synaptic connections with bipolar cells

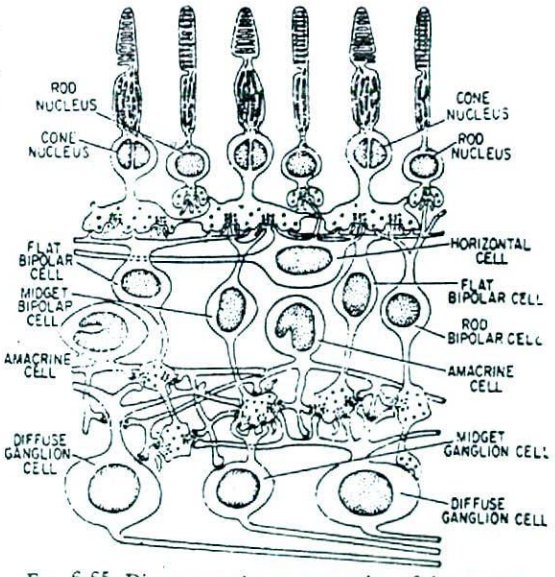


FIG. 6.65. Diagrammatic representation of the contacts in the retina (*after Boycott & Dowling*).

and they in turn connect with ganglion cells. The ganglion cells give rise to optic nerve fibres that run toward the optic nerve head as the innermost layer of the retina. Ganglion cells and to a lesser extent bipolar cells possess quite extensively spreading dendrites so that the activity of a large number of rods and cones influences each ganglion cell. Amacrine cells and horizontal cells mediate further lateral interactions (FIG. 6.65). In all parts of the retina, except the central fovea, rods and cones are connected with the ganglion cells through intermediate bipolar cells. Though in the fovea the proportion of ganglion cells to cones may be more nearly one, but over most of the retina there are many more photoreceptors than ganglion cells. However, this does not mean that in the fovea each nerve fibre is connected to only a single cone.

Photoreceptors of the retina are situated just inside the stratum pigmenti. The principle underlying the generation of visual impulses seems to be that, these receptors contain certain photosensitive pigments which are changed on exposure to light. These chemical changes stimulate the nerve cells to produce visual impulses. When light is withdrawn these pigments are quickly resynthesised. They are briefly described below:

Rods (FIG. 6.66). (1) **Function.** Dim light vision. No colour vision. (2) **Number.** About 110–25 million. (3) **Distribution.** *Vide* above. (4) **Size.** 40–60 μ long and about 2 μ in diameter. (5) **Parts.** Consist of outer segment and inner segment. The *outer segment* consists of two parts: (*a*) Outer part—thin cylindrical and transversely striated. Composed of a myelin-like substance and contains rhodopsin. (*b*) Inner part is broad (thickness double) with longitudinal striations and is protoplasmic. The *inner segment* is thinner and consists of a nucleus and a long fibre terminating in end bulb or a spherule. The outer segment is the most sensitive part and contains the visual purple of the photosensitive pigment which absorbs light and the inner segment is concerned with the metabolic activity of the rod cell. (6) **Neural connections.** 10 to 100 rods are connected with one single optic nerve fibre.

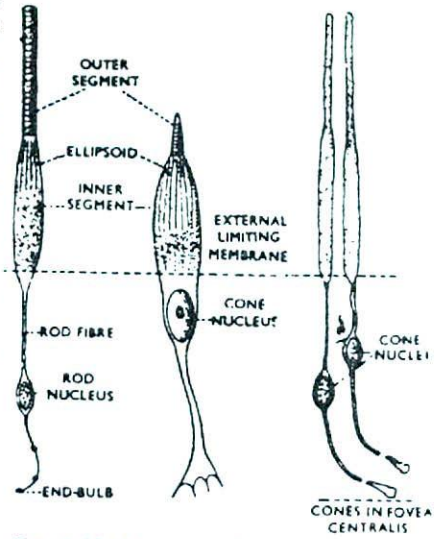


FIG. 6.66. Diagrammatic representation of a retinal rod (*on the left*), a cone (*on the middle*) and cones in the fovea centralis (*on the right*) indicating their component parts; and *ellipsoid* which is the aggregation of mitochondria in the inner segment.

Cones (FIG. 6.66). (1) **Function.** Concerned with bright light vision, colour vision and acuity of vision. It is suggested that there are possibly three types of cones for the three primary colours, containing special varieties of pigment sensitive to the three-coloured light. (2) **Number.** About 6–7 million. (3) **Distribution.** Fovea—only cones, periphery—only rods, intermediate—both. (4) **Size.** 28–85 μ long and 2–5 μ in

diameter. (5) **Parts.** Consist of outer and inner segments. The *outer segment* consists of two parts: (*a*) Outer part—smaller and transversely striated. (*b*) Inner part—larger and longitudinally striated. The *inner segment* is thinner and consists of a nucleus and a fibre. (6) **Neural connections.** In the central part of the retina one cone is connected with one optic nerve fibre.

Recent observation by **electron microscope** has revealed that the outer segments of the rods and cones consist of flattened vesicles or sacs and the inner segments contain metabolic enzymes, e.g., succinic dehydrogenase and large number of mitochondria known as **ellipsoid.** There is a plasma membrane covering the outer and inner segments. The outer segment is connected with the inner segment by the cilium.

The cross-section of cilium shows the outer membrane and nine pairs of peripheral fibrils. These intracellular peripheral fibrils are originated from a basal body which is known as **centriole.** These fibrils also penetrate into the outer segment and proceed to a considerable length. FIG. 6.67

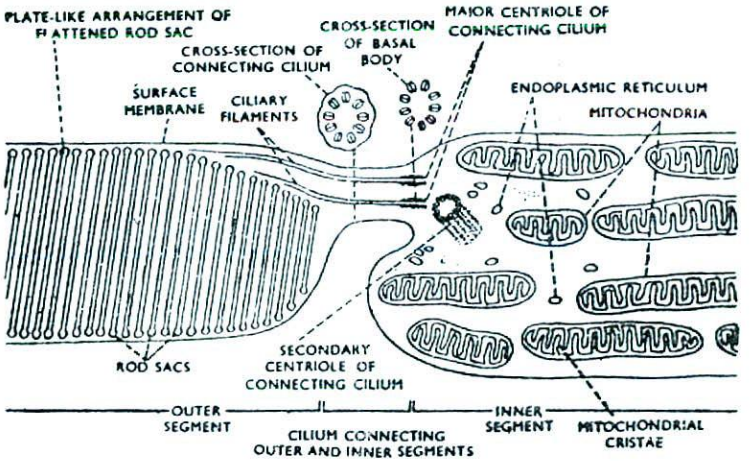


FIG. 6.67. Electron microscopic structure showing connecting zone (diagrammatic) between the inner segment and the outer segment of rod (*after Mountcastle*).

shows in detail an electron microscopic structure of outer and inner segments of rod and its connection through the cilium. De Robertis has claimed that the entire outer segment is derived from a cilium.

There is a structural differences in between the outer segments of the rods and cones. In the outer segment of the rod (FIG. 6.67), the double layer discs—the sacs are arranged one above the other like a stack of plates and all are enclosed within the plasma membrane. The double layer sacs look like a rim. The discs or sacs are cut into lobules by longitudinal fissures (FIG. 6.68C & D).

In the outer segment of the cone there are certain differences. The plasma membrane is repeatedly infolded on the side opposite from the cilium to give the double layer disc (sac) structure. This double layer disc structure does not look like a rim and the cone lacks the fissures and lamellar micelles (FIG. 6.68A & B).

The outer segments of the rods which contain the visual purple or rhodopsin absorb light and due to the piling up of the sacs this photochemical reaction takes place over an wider area. In the cones the pigment is **iodopsin.** In many animals at the junction of the inner and outer segments of the cones there are oil globules. In human cones no such oil

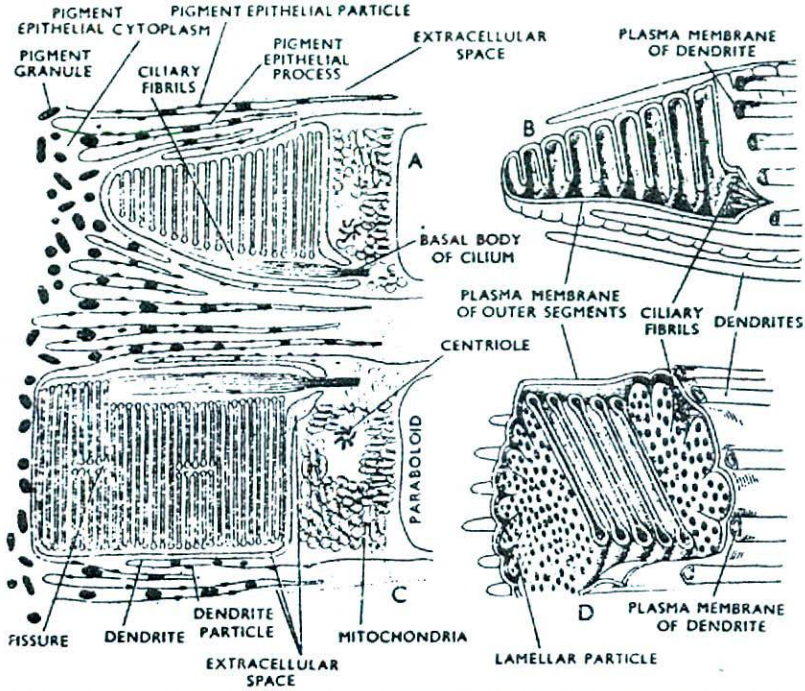


FIG. 6.68. Electron microscopic structure indicating relations between rods (C & D) and cones (A & B). C & D show the cut discs or sacs arranged in stack and covered by a separate plasma membrane. A & B represent infolding of repeated common plasma membrane on the side opposite from the cilium (*after Best & Taylor*).

globules are seen. Presence of xanthophyll has been demonstrated in the cytoplasm of the cones. Rod cells have got smaller nuclei without definite nucleoli. The nuclei of the cone cells are found near the external limiting membrane and each nucleus has got a nucleolus. The rods have got knobs at their endings where they make synaptic connections with the bipolar cells whereas the cones have got 'pedicles' at their endings. Since there are approximately 6–7 million cones, 110-25 million rods in the retina of each eye and about 0·4–1·0 million nerve fibres in each optic nerve of the human being—main convergence of receptors takes place through bipolar and ganglion cells.

Formation of an image on the retina. Rays of light which traverse through the centre of the convex lens fall on the retina without any bend, but rays from the peripheral region of the pupil are bent back to a focus on the retina. The image that is formed in the retina is inverted.

The cerebral cortex interprets the inverted image on the retina as an upright one.

Effects of light on the retina. *Chemical structures* of the different photochemical substances of the retina have been shown in FIG. 6.69.

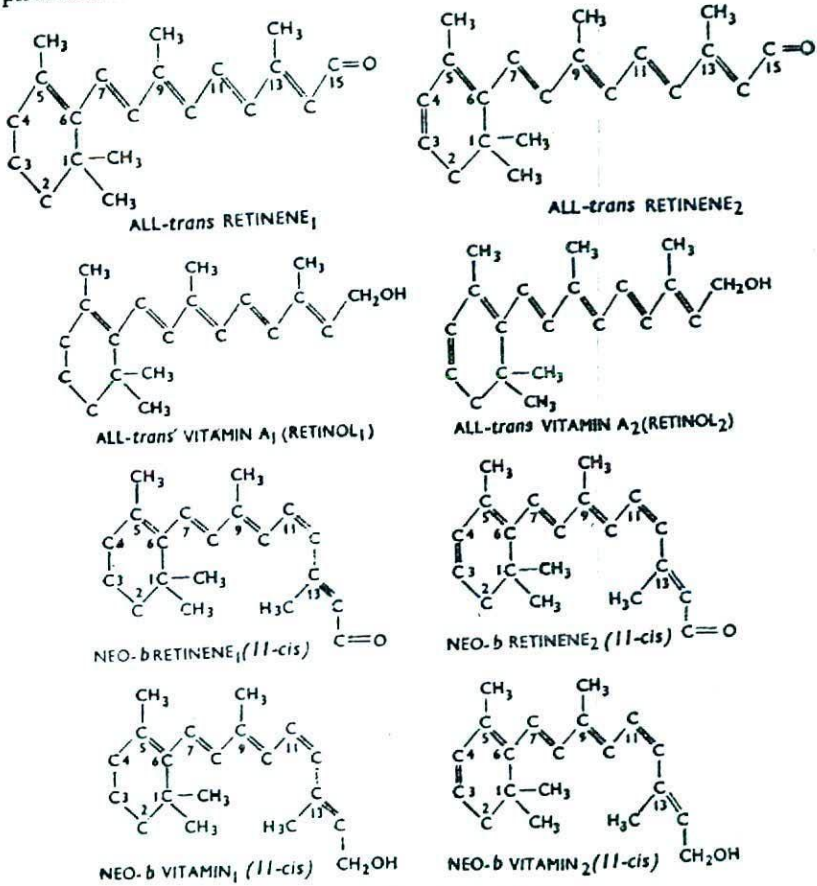


FIG. 6.69. Description in text.

When light falls on the retina following **changes** take place:

Photochemical changes. Visual purple or **rhodopsin** is a chromoprotein containing a carotenoid pigment (*chromophore group*) as a prosthetic group. This group is the specific part of the molecule and is a derivative of vitamin A. It is present in the outer segments of the rods only. It is bleached on exposure to light. Its main steps have been described in FIG. 6.70. When light falls on the retina, the rhodopsin passes rapidly through several stages. It is first converted into **lumirhodopsin** and then to **metarhodopsin.** The metarhodopsin is then split into **retinene$_1$ (retinene)** and **opsin.** Retinene$_1$ is the oxidative product of vitamin A_1 (vitamin A). Opsin is the protein of rhodopsin.

The retinene is then reduced to vitamin A_1 by enzyme **alcohol dehydrogenase** (*retinene reductase*) in presence of NAD. Vitamin A_1 is then stored in the pigment epithelium. The photopigment must be regenerated after being bleached by light so as to prepare for another response. The retinene$_1$ is ***trans* isomer** whereas the retinene of rhodopsin is 11-*cis* **isomer** (neo-*b* retinene$_1$). So for the quick synthesis of rhodopsin in the dark, the all-*trans* retinene$_1$ is isomerised rapidly by *retinene isomerase* into its 11-*cis* form the neo-*b* retinene$_1$ (*cis* retinene$_1$). This reaction consumes energy and takes place in *light of short wave length*. The combination of the 11-*cis* retinene$_1$ with opsin gives rise to rhodopsin which occurs spontaneously.

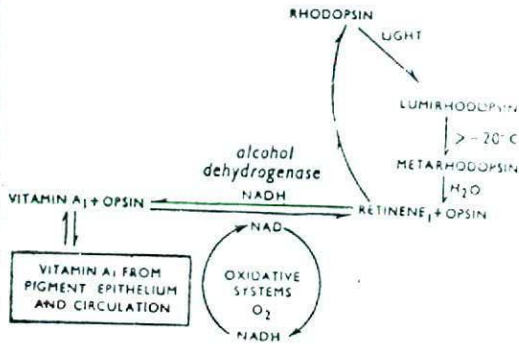


FIG. 6.70. The bleaching and synthesis of rhodopsin. In the absence of light, retinene and opsin can recombine rapidly, but in its absence retinene is further converted to vitamin A (*after Mountcastle*).

Thus *trans* **retinene cannot condense with opsin efficiently to form rhodopsin.** In the light, the 11-*cis* isomer of rhodopsin is converted into the all-*trans* isomer that is present in lumirhodopsin. All-*trans* retinene$_1$ is converted reversibly into *trans* vitamin A_1 by *alcohol dehydrogenase* in presence of NAD. The 11-*cis* retinene is also reversibly converted into 11-*cis*

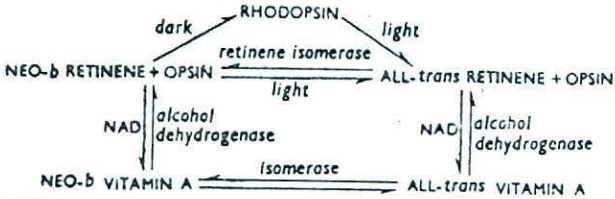


FIG. 6.71. Diagram of photochemical process in the rhodopsin and vitamin A system (*after Field, Magoun & Hall*).

vitamin A_1. The *trans* vitamin A_1 is likewise enzymatically isomerised into *cis*-form which then is oxidised to *cis* retinene$_1$ (neo-*b* retinene$_1$). The above photochemical changes following a fall of light on the retina have been presented schematically in FIG. 6.71.

The *trans*-form of vitamin A (retinol), which is abundant in the liver and the blood, is the most active isomer in all physiological roles of the vitamin other than that of the vision. For vitamin A to give rise to a visual pigment, it must be converted in the eye to a *cis*-isomer of *retinal* by *retinal reductase* and *retinal isomerase*.

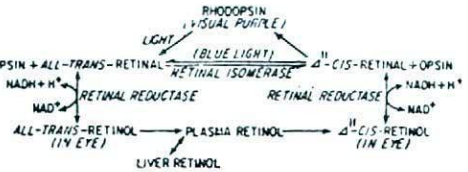


FIG. 6.72. It shows the visual cycle.

The *cis*-retinal combines with opsin to form rhodopsin. Light falling on the retina splits retinal from opsin by isomerising it to the *trans*-configuration. The liberated *trans*-retinal then either may be changed back to the *cis*-form by the enzyme retinal isomerase to regenerate rhodopsin after completion of an isomerisation cycle in the eye or may be reduced vitamin A by retinal reductase (FIG. 6.72).

Rhodopsin is not the only pigment isolated from vertebrate rod but another purple light-sensitive pigment—**porphyropsin** has been isolated from the rods of the vertebrates which live in fresh-water fishes, spawning lampreys, adult and certain larval amphibia, etc. The series of changes undergo following absorption of light are precisely parallel with those of the rhodopsin system. Porphyropsin after bleaching splits into (*i*) inactive all-*trans* retinene$_2$ the aldehyde (oxidative products) of all-*trans* vitamin A$_2$, and (*ii*) **scotopsin.** The rhodopsin and scotopsin are entirely similar and share entirely the same protein. Like rhodopsin system, the all-*trans* retinene$_2$ is reversibly converted to neo-*b* retinene$_2$ (11-*cis* isomer) by the **specific isomerase.** Neo-*b* retinene$_2$ and all-*trans* retinene$_2$ are also reversibly transformed into neo-*b* vitamin A$_2$ and all-*trans* vitamin A$_2$ respectively by *alcohol dehydrogenase* in presence of NAD. The porphyropsin system has been presented in FIG. 6.73.

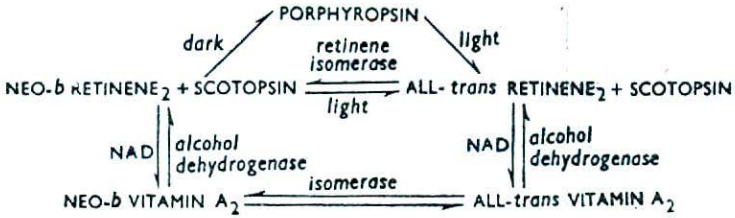


Fig. 6.73. Diagram showing porphyropsin system (*after Field et al*).

In the cone there is photosensitive pigment which is known as **iodopsin.** Wald and his colleagues (1955) isolated iodopsin from cones of chicken. Hubbard and his followers (1965) have observed the iodopsin to undergo the same series of changes which happen in case of rhodopsin after absorption of light. Only the difference is in its opsin. Cone opsin is called **photopsin** (FIG. 6.74).

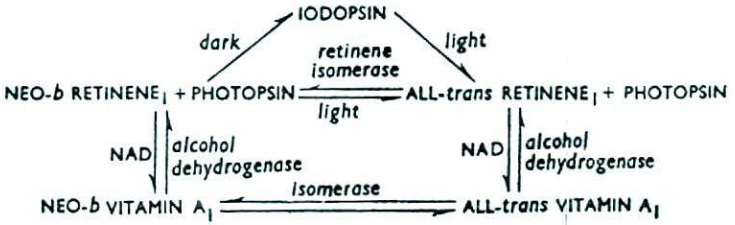


FIG. 6.74. Diagram showing the series of changes in the photochemical substances of cone after absorption of light (*after Field et al*).

Wald and his associates (1953) synthesised in the laboratory a blue photosensitive pigment, known as **cyanopsin** which absorbs at about

620 m μ. This pigment has not been identified in the retina, but there is little doubt that cyanopsin is the cone pigment for fresh-water fishes as compared with Granit's electrophysiological measurements of the spectral sensitivity of cone vision in fresh-water fishes, leeches, etc.

In summary, it can be described that vitamin A_1 (vitamin A) takes part in the synthesis of *rhodopsin* or *iodopsin* whereas vitamin A_2 takes part in the synthesis of *porphyropsin* or *cyanopsin* (FIG. 6.75).

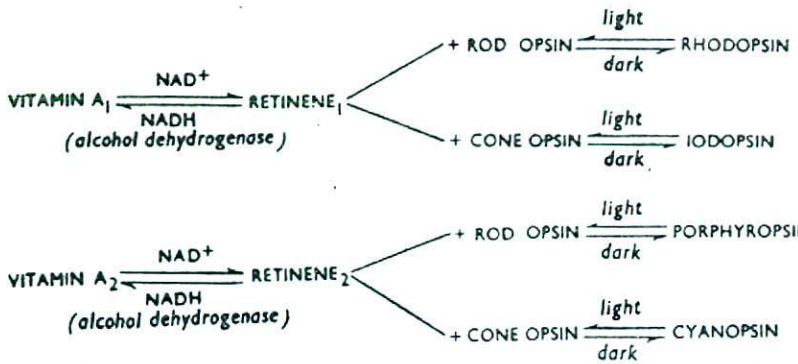


FIG. 6.75. Recapitulations of visual system in the synthesis and breakdown of different photosensitive pigments and *vice versa* through interactions of rod opsin or cone opsin, with vitamin A–retinene system in presence of enzyme alcohol dehydrogenase *(after Field et al)*.

Bleaching and regeneration of photosensitive pigments are independent of nerves, but occur only so long as the rods remain in contact with the stratum pigmenti. It is held that either the bleaching process itself or some product formed during bleaching, stimulates the rods to generate the visual impulses. In other words, radiant energy is converted into nerve impulses through this *photochemical* process.

Neurophysiology of vision

Electrical activity in the retina. When the photopigment absorbs light in a retinal rod or cone, light causes the cell membrane to become hyperpolarised. Although the effect of this mechanism is not clear, it is suggested that the action of light decreases the permeability of the cell membrane to sodium ions. This change in permeability could be directly related to the light-induced change in the structure of photopigment because the photopigment is a constituent of the membrane of the outer segment of the photoreceptor cells (rods and cones).

The potential changes which are generated in the outer segment of a photoreceptor are transmitted electrotonically to synapses on both bipolar and horizontal cells (FIG. 6.65). The bipolar cells in turn transmit a graded electrical signal to the ganglion and amacrine cells with which they are in contact. On the other hand, the horizontal cells may be capable of spreading an inhibitory effect to surrounding photoreceptors. In the ganglion cells the generated action potential passes along optic nerve fibres and optic tract fibres to the lateral geniculate body and the superior colliculus.

Electrophysiological changes. Sequence of potential changes in the retina occurs when light strikes the eye. This record of the sequence of

potential changes is known as **electroretinogram** (ERG). The most conventional method of recording the ERG is to put an electrode in contact with the retina and an indifferent electrode on the back of the eye.

Responses recorded by this method are very small in amplitude and give only a summated response of the whole retina but not of its individual layer. Recently it has been possible to record ERG by introducing microelectrode into the vitreous humour of an experimental animal and the indifferent electrode on the back of the eye.

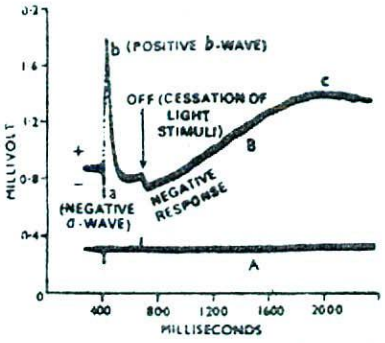


FIG. 6.76. Electroretinogram of cat with major deflection in B and the onset and termination of the stimulus indicated in A by vertical lines (*after Brown*, 1968).

FIG. 6.76 represents ERG recorded by the above method in cat (Brown, 1968) The ERG shows different waves. Immediately after the flashing of light there occurs a *negative a-wave* followed by larger *positive b-wave*. The *c-wave* also rises slowly even after the cessation of light stimulus. Just after the onset of 'off-signal' there is a negative response. This method also fails to explain the functional significance of each component (wave) or to determine the site of generation of each component. So analysis of each ERG component by introducing the microelectrode at different layers of the retina is necessary.

Analysis of ERG. Since human retina contains both rods and cones, analysis of ERG through recording each from rod-dominated area and also from cone-dominated area is essential. FIG. 6.77 shows the ERG recorded through microelectrode technique by Brown (1968) from rod and from cone area separately.

The *c-wave* has been omitted from both rod and cone analyses as the same wave is originated from the pigment epithelium. Each component of general ERG except the *c*-wave can be analysed and explained by the three major neural components (the receptor potential, D. C. component, and *b-wave*). The rod receptor potential recorded directly from the rod has a rapid onset but with a very slow decay at the termination of the stimulus. The D. C. component is so named as it looks like a direct current pulse and is originated from the inner nuclear layer. The *b-wave* also arises from the nuclear layer. The *a-wave* is due to the rising phase of rod receptor potential and the rise of wave is abruptly terminated by the onset of the D. C. component and large *b-wave*. The D.C. component and *b-wave* have got opposite in polarity to the receptor potential. The *b-wave* maintains its positivity only for a short period and becomes negative within a short period and maintains this stage though the stimulus is continued. This abrupt onset of negativity is due to the negative receptor potential having larger amplitude than the positive D. C. component. After discontinuing stimulation the D. C. component abruptly terminates and gives a negative deflection called the 'off-response' accompanied by a slowly decaying negativity. This slowly decaying negativity is known as **remnant negativity** which is due to very slow decay of the rod receptor potential.

The cone ERG is more or less similar to that of the rod ERG until the stimulus is terminated. The pure cone ERG shows *d-wave* which occurs just after termination of the stimulus. This *d-wave* has got both positive and

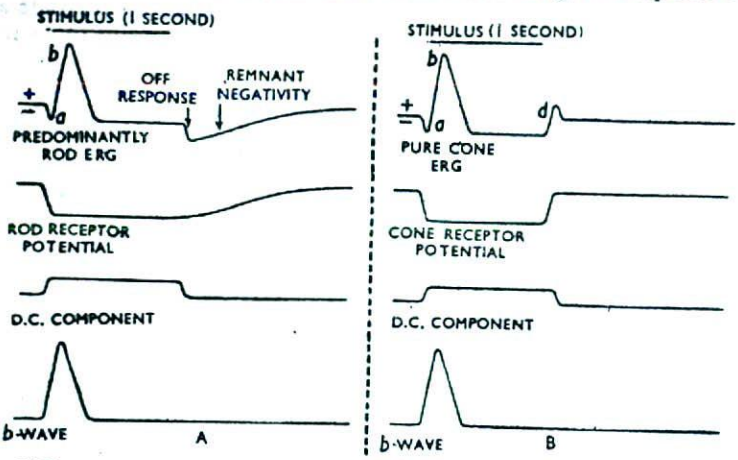


FIG. 6.77. Component of analysis of electroretinogram. A showing ERG from rod and B indicating ERG from cone. 'Off-response' means the stimulation is discontinued (*after Brown*, 1968).

negative waves and there is no remnant negativity. The cone receptor potentials decay very abruptly after termination of the stimulus. Thus the main differences in form of the cone and rod ERGs appear to be the differences in rates of decay of the rod and cone receptor potentials.

Spectral sensitivity: Scotopic vision and Photopic vision

Scotopic (*dim light*) vision and photopic (*daylight*) vision are the function of the rods and cones respectively. The sensitivity of the eye to a flash of light varies with the wave length of the light. The colour of light depends upon its wave length and the visible spectrum extends from wave length 400 m μ to 750 m μ. Thus the eye responds to light of wave length from 400 m μ to 750 m μ. Beyond this range the receptors are not stimulated. For this reason **ultraviolet rays** (below 400 m μ) and infra red (above 750 m μ) are not visible. If the relative energy against variable wave length for sensation of a constant brightness is determined, then the reciprocals of these energies will give a curve which is known as **luminosity curve.** This luminosity curve actually shows the relative luminosity of various wave lengths of light. The scotopic luminosity is obtained at **dark-adapted eye** and photopic luminosity at **light-adapted eye.** The amount of **radiant energy** required to have a

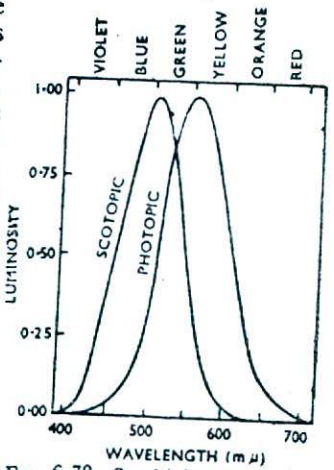


FIG. 6.78. Sensitivity curves for scotopic (rod) and photopic (cone) vision.

constant sensation of brightness varies with the wave lengths of the **incident light.** In scotopic vision, maximum luminosity is observed at a wave length of 507 m μ (blue-green) and in photopic vision the maximum luminosity is observed at a wave length of 550 m μ (yellow-green). *The rods are much more sensitive than cones* (FIG. 6.78).

This shows that the yellow-green photopic function is shifted towards longer wave length than the blue-green scotopic function. This shift of spectral sensitivity was first observed by Purkinje in 1825 and is thus known as **Purkinje shift** and the phenomenon is known as **Purkinje phenomenon.** He also noted that in bright illumination the yellow colour appears brighter than other but in dim light or fading light blue-green colour appears brighter than other.

In the garden with gradual advent of dark, the red and blue flowers look equally bright only before the light fades but after then red flowers appear black and the blue flowers are seen only as coloured objects.

As the scotopic vision is due to rod and photopic is due to cone, Purkinje's shift can also explain partly the **duplicity theory of vision.**

LIGHT AND DARK ADAPTATION

If the eye, after being exposed to dark, be rapidly exposed to strong light or *vice versa*, the eyes take a little time to get adapted. Thus there are two types of adaptation: (*a*) **light adaptation** (*photopic eye*), (*b*) **dark adaptation** (*scotopic eye*). Dark adaptation involves two separate processes: (1) Removal of after-images from the retina. (2) Resynthesis of the bleached visual purple in the rods. In strong light, the first method predominates. In dim light, the second method is the chief process. Time taken for dark adaptation is directly proportional to the intensity of light and duration of exposure and inversely proportional to vitamin A content. Dark adaptation is not possible in vitamin A deficiency and as a matter of fact—*depressed dark adaptation is taken as the most sensitive test for vitamin A deficiency.*

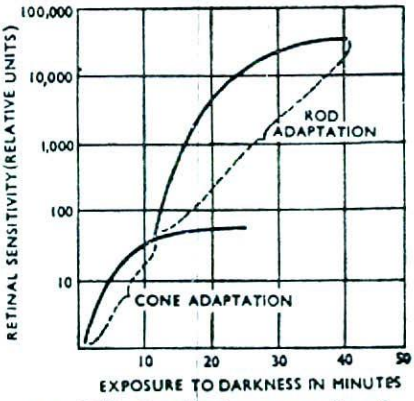


FIG. 6.79. Graphical representation of relative retinal sensitivity during dark adaptation.

It is a common experience that after entering from brightly illuminated area into a dark area, the vision at first is very poor but after sometimes the vision is improved. This is known as **dark adaptation.** The dark adaptation can be studied quantitatively by determining the time interval that requires for dark adaptation of a subject who was previously exposed to bright light, now in the dark. Dark adaptation is completed within an hour. FIG. 6.79 shows the course of dark adaptation when a person is exposed to total darkness after having been exposed to bright light for several hours. The curve has got two distinct phases with a kink in the middle. The first phase of the curve is due to dark adaptation of the cone, because if this test is made

with the foveal retina then the similar curve is obtained and no kink is observed. The second curve is due to dark adaptation of the rod.

This shows that at first the sensitivity of vision rises quickly to reach a fairly steady value within 4–5 minutes and maintained this state for another 5–6 minutes (**cone adaptation**). After that the sensitivity is further increased though slowly but with much greater degree (**rod adaptation**). This shows that both cones and rods take part in dark adaptation. *The cone adapts more earlier than the rod.*

In deficiency of vitamin A or in congenital absence of rod, this secondary increased sensitivity (rod adaptation) is not observed.

Dark adaptation is very slow if the subject is previously exposed to intense bright light. The radiologist, pilot, etc., use red goggles only to avoid bleaching of visual purple because the rod is insensitive to wave length of above 640 m μ. The red goggles allow light having longer wave length (650 m μ to 750 m μ).

Light adaptation. If a person suddenly passes from a dim to brightly illuminated environment, intense and uncomfortable light is felt by the subject but after a while (5 minutes) this stage is passed over and the visual threshold rises. This is known as **light adaptation.** The light adaptation is nothing but the disappearance of the dark adaptation.

VISUAL ACUITY

The **visual acuity** is the power of the eye to resolve two stimuli separated in space. It is the sharpness to which the details and contours of objects are perceived. If a series of black lines on a white paper is moved gradually far and far away from the observer then a point will come when the observer will fail to distinguish the objects on such uniformly grey sheet of paper. Thus the resolving power of the eye is the angle subtended at the eye by the spacing between the lines at the point where they are just resolvable. The visual acuity must not be confused with the **visual threshold.** The visual threshold is the *minimal* amount of light that elicits light sensation. The visual acuity is usually defined in terms of *minimum separable* or **resolution threshold**—the shortest distance by which the two lines will be perceived separately.

The visual acuity depends upon the sensitivity of retina to light, illumination of the surface, the time of exposure and ability to recognise the distance of parallel rays. Visual acuity helps in determining shape, form, outline and minute details of the surroundings. *It is expressed as the reciprocal of the angle subtended at the nodal point of the eye—visual angle.* The visual angle is generally one *minute* (60 seconds) when the retinal images are separated by 4·5 μ. Visual acuity is found to be maximum at the *fovea centralis* where there are large number of cones; and minimum at the peripheral part of the retina where the number of cones are very few. The visual acuity increases with monochromatic light. Errors of refraction reduce the visual acuity.

In determining the visual acuity (acuteness of vision), figures such as the broken circle **O** of Landolt or Snellen's prong, **E**, printed black on a white ground and in graded sizes are employed (FIG. 6.80). The Landolt-**C** is almost universally used in scientific studies and is distinguishable from a circle by a white gap; and the power to recognise the position of the gap depends upon the angle it subtends at the eye. The Landolt-**C** can be

rotated into eight separate positions. The subject is seated at a distance of 6 metres (20 ft.) and a letter (say s) is placed with the gap of the **O** or the prong of the E turned to the right or left; he is asked to say in which position the letter is directed. The width of the lines composing the letters and the gap in the **C** or the spaces between the prongs of the E, subtend angles of different degrees, depending on the size of the letter, when placed at a distance of 6 metres. The width of the whole figure is five times the thickness of its parts. By finding the smallest letter whose position can be recognised the subject's visual acuity in terms of the visual angle is ascertained. The maximum acuity measured with a Landolt-**C** subtending 0·5 minute of arc, is about 2·1. In testing the visual acuity for fitting of glasses, Snellen's test type is most commonly employed.



FIG. 6.80. It represents Landolt-**C** (A) and Snellen's test type (B & C) employed in measurement of visual acuity. In determining the power of the eye to resolve the detail, the distance, *s*, seems to be critical factor (*after Starling*).

Snellen's test type. Clinically Snellen's test is employed to measure the ability of the subject in discriminating different letters which are constructed so that their details (as for instance s in FIG. 6.80), subtend a known angle at a given distance from the eye. This test type is devised upon the basis that two points or lines separated by a space having a visual angle of 1 minute can be resolved by the average normal eye. This test comprises nine rows of block letters printed in black upon a white background (FIG. 6.81). The rows of these letters are arranged in descending order of size from above down. The width of the lines forming the letters of the first row subtends an angle of 1 minute at 60 metres from the eye, whereas that of the letters in the two to nine rows, have a visual angle of 1 minute at 36, 24, 18, 12, 9, 6, 5, and 4 (not shown) metres respectively. The top letter is constructed in such a fashion that its details subtend 1 minute at 60 metres. The subject stands at a distance of 6 metres (20 ft.) and reads these letters with one eye closed. The acuteness of vision is expressed by a fraction of which the numerator is 6 (distance of 6 metres from the letters) and the denominator is the distance at which the smallest letters can be read by the eye. If the subject cannot read beyond '12 metre' line then his visual acuity is said to be 6/12. The normal visual acuity is 6/6 and subject having 6/5 visual acuity is considered to be better than normal vision. A subject having 6/30 visual acuity is considered to be subnormal vision.



FIG. 6.81. It illustrates Snellen's test chart reduced in size (*after Starling*).



FIG. 6.82. It shows Snellen's test types constructed on a rectangular grid.

Snellen's test types of rectangular grid (FIG. 6.82) are also widely used. Each line of letters of a different size is marked with the distance in metres at which the small squares subtend 1 minute of arc. A normal individual at a distance of 6 metres can

distinguish the letters of the 6 metre line and his visual acuity will be 6/6. Subjects, having defective vision, distinguish letters standing 60 metres from a well-lit chart then his acuity will be 6/60.

FIG. 6.83 also demonstrates the acuteness of vision of a subject having the visual acuity 6/12. The subject can resolve details that subtend 1 minute at his eye when the letter E is held 12 metres away and if he

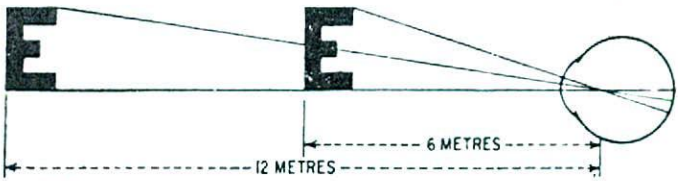


FIG. 6.83. It demonstrates the visual acuity of a subject having 6/12 vision at a distance of 6 metres and 12 metres.

reads the letter at 6 metres then it will subtend twice the angle they will subtend at 12 metres, i.e., 2 minutes. So the resolving power of the eye will be 2 minutes.

Development of (approximate) visual acuity

Age	Visual acuity
2 months	20/400 (6/122)
6 months	20/200 (6/61)
1 year	20/100 (6/30)
2 years	20/ 60 (6/18)
3 years	20/ 30 (6/9)
4–5 years	20/ 20 (6/6)

OPHTHALMOSCOPY

The retina of the living eye can be examined with the aid of an instrument known as **ophthalmoscope** which allows light to be projected into the eye through the pupil along the visual axis of the observer's eye. The term **ophthalmoscopy** *is applied for the process by which the condition of the retina (fundus) itself is examined to see whether it is healthy or diseased.* Details are as follows:

Ophthalmoscopy. When light enters our eye through the pupil, a portion of it is reflected back and traces its original course. If the observer and the source of light are in the same position, rays reflected from the fundus will now be received by the observer and the pupil of the subject appears as a *red glow*, which is called **fundus reflex.** The above principle is taken advantage of in ophthalmoscopic examination. In ophthalmoscopy light from a source is reflected from a plane or concave mirror into the subject's eye. If the observer's eye is placed just behind a hole in the mirror, then the rays of light travelling from the subject's eye reaches the observer's eye and the observer sees the **fundus glow.** In ophthalmoscopy the details of the **fundus oculi** are seen.

The optical principle of ophthalmoscopy is illustrated in FIG. 6.84. When the fundus of a normal eye is illuminated, an observer E will see only one point of the light from the retina of the subject (namely B). But if the observer comes closer to the subject and assumes a position as at E_1, then the rays from several points as in the figure A, B and C, will pass through his pupil and a much larger area of the retina will be visible.

An outline of the **ophthalmoscopic examination** as done in clinical practice is as follows:

1. *Examination at a distance of 1 metre from the subject with the plane mirror.*
2. *Examination at a distance of 9 inches (or 22 cm.) from the subject.*
3. *Ophthalmoscopic examination by indirect method.*
4. *Ophthalmoscopic examination by direct method.*

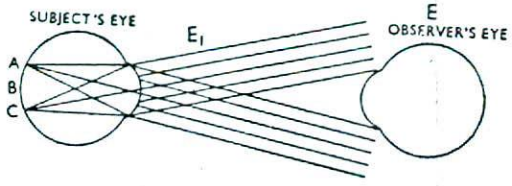


FIG. 6.84. Direct ophthalmoscopy.

First examination reveals the uniform red glow of the fundus (any opacity in the media will produce darkness). In this method the observer also notes the state of refraction of the eye.

Second examination reveals any opacity in the media.

The third and the fourth methods, i.e., examinations by ophthalmoscope are described below:

Indirect ophthalmoscopy. The eye to be examined is made highly **myopic** by placing a strong convex lens in front of the eye. In this case a *real inverted image* of the fundus is formed in the air between the observer and the convex lens. In FIG. 6.85 the rays from the area P Q

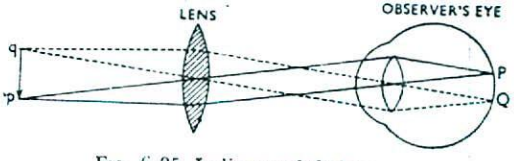


FIG. 6.85. Indirect ophthalmoscopy.

from the fundus form a real inverted image between the convex lens and the observer. The observer sees this *aerial image*. The lens used in clinical practice is +13D.* Magnification in this case is about 5. This method requires some practice than the direct ophthalmoscopy. The most important advantage of this method is that a large retinal field is visible.

Direct method. As described in the FIG. 6.84, in order to see a large area of the fundus to be seen, the observer approaches the subject very closely, so that the rays from the points A, B and C enter the observer's eye. The image in the direct ophthalmoscopy is much more magnified than the indirect ophthalmoscopy. The image is erect in this case. Direct ophthalmoscopy is done with reflecting ophthalmoscope or with self-illuminating ophthalmoscope.

The **reflecting ophthalmoscope** is Morton's type of ophthalmoscope (FIG. 6.86) in which *four* perforated mirrors are so arranged that by rotating the mirror, the perforation in the mirror will be opposite the hole

* The diopter is an unit which denotes the refractive power of an optic lens and is the reciprocal of focal length of the lens expressed in metres. If D = the diopter of a lens and f = the focal length of the lens in metre, then the formula is $D = (1/f)$.

in the centre of the instrument. There are two large mirrors and two small mirrors. *Large mirrors are used for indirect ophthalmoscopy and small mirrors are used for direct ophthalmoscopy.* The focal length of the large concave mirror



FIG. 6.86. Morton's type of or reflecting ophthalmoscope.
By courtesy of Dr. Gopal Sen, M.B., D.O.M.S., N. R. S. Medical College, Calcutta.

is 25 cm. Light is placed behind the subject's head. Immediately behind the *sight hole* there are lenses which can be placed according to the power of the subject or the observer.

The **modern ophthalmoscope,** as in the FIG. 6.87, is a self-illuminating one, the illumination is made by dry battery cells. A condenser condenses the light which passes through the lens. The lens forms

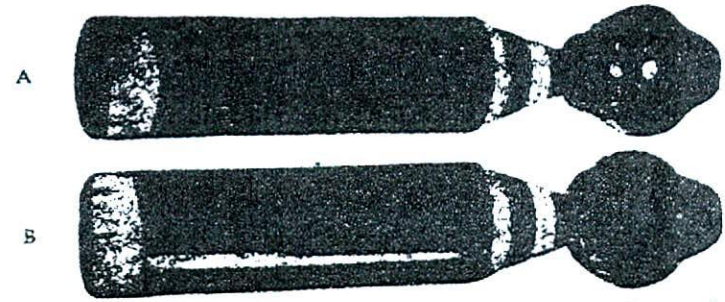


FIG. 6.87. Modern ophthalmoscope ; A=showing *anterior view*, B=*posterior view*. '+' indicates the power of convex lenses. '—' indicates the power of concave lenses.
By courtesy of Dr. Gopal Sen, M.B., D.O.M.S.

an image of the source of light at the top of the half mirror. The light then passes through the subject's eye and the observer can see the details of the *fundus oculi* by placing different powers of lenses behind the sight hole (as described in Morton's ophthalmoscope).

The first thing to look is the **optic disc.** The subject is asked to look at a distance with his accommodation fully relaxed. Normal optic disc is circular with 1·5 mm in diameter. The colour of the disc is pinkish. The centre of the disc is slightly white and the vessels emerge from the centre of the disc (FIG. 6.62).

Retinal blood vessels divide into numerous branches after emerging from the centre of the disc. Veins are slightly broader than the arteries which are narrower.

The next is to look at the **macula lutea** which is situated about 3 mm from the temporal side of the optic disc. The area is slightly redder than the general fundus and in the centre of macula, there is a very bright reflex which is called **foveal reflex.** The macula is brought to focus by asking the subject to look at the light of the ophthalmoscope.

RETINOSCOPY

Retinoscopy *is a process by which the refractive error of an eye is detected.*

The theory of retinoscopy is based on the principle that if a point of light is placed in front of an eye, the rays of light entering the eye through the pupil are brought to a focus on the retina so that a *circular area of the fundus* will be illuminated. In the FIG. 6.88, light is entering the eye through the nodal point (N) so that an illuminated area is formed in

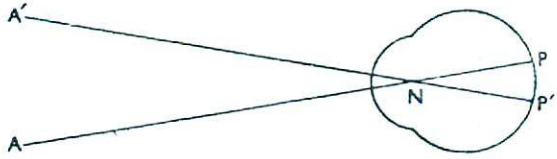


FIG. 6.88. Illuminated area of the fundus.

the retina at P. Now let us think that the point source of light moves upward from A to A', from the diagram it will be seen that the image moves down to P' in the *opposite direction*. So it is seen that when the object is moved, the image will always move in the opposite direction.

In the practice of retinoscopy the illuminated area of the fundus of the subject acts as a source. In the *hypermetropic eye*, the rays of light entering the eye are brought to a focus behind the retina. Conversely, the rays of light coming from the fundus of a hypermetropic eye will be **divergent** because these are coming from a focus behind the eye (FIG. 6.89). In this case when the illuminated area moves down, the circle of light in the fundus of the subject will move down also.

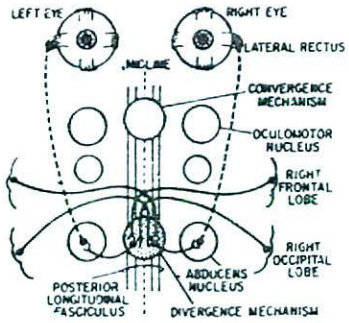


FIG. 6.89. Diagrammatic representation of divergent mechanism (*after Adler*).

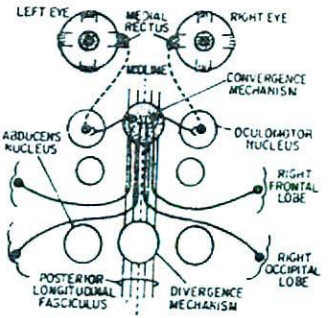


FIG. 6.90. Diagrammatic representation of convergent mechanism (*after Adler*).

In the case of the *myopic eye*, the rays of light entering the eye are brought to a focus in front of the retina. Naturally, the rays of light coming out of a myopic eye will be **convergent** (FIG. 6.90) and will be brought to a focus in front of the eye. The movements of the illuminated eye will be opposite of a hypermetropic eye. In this case object and apparent image move together.

In the FIG. 6.91, S is the source of light, the positions of the plane mirror being indicated by P and Q. When the position of the mirror is at

P, light entering the eye will appear to come from L and will converge towards *p*. When the mirror is turned to the position Q, light entering the eye will appear to come from L' and will converge towards *q*. In the FIG. 6.91, H, E and M are the positions of retina in *hypermetropia*, *emmetropia* (*normal*) and *myopia* respectively. In the retinoscopic examination, the observer does not see the real movement of the light on the retina but the apparent movement through the *pupillary area*. In the emmetropic eye far point is at infinity. In the case of an emmetropic eye the two movements

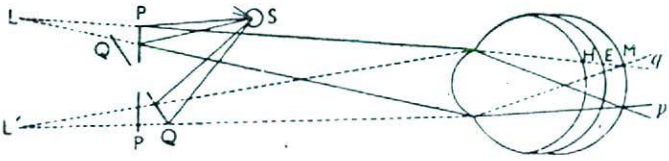


FIG. 6.91. Convergence and divergence of rays of light in hypermetropia, emmetropia and myopia.

will be neutralised so that the point of reversal would occur. This occurs when the far point of the subject corresponds to that of the observer's eye.

In the practice of retinoscopy the subject and the observer are one metre distance away. The examination is carried out in the dark room with the source of light behind the subject, so that the face is not illuminated. The observer stands or sits at a distance of one metre and holds a plane mirror, so that the rays of light reflected from the plane mirror enter the subject's eye through the pupil.

The observer notes a *circular red glow of the fundus*. In practice when the mirror is tilted to the right and the illuminated area moves in the same direction as the mirror, the eye is hypermetropic, emmetropic (normal) or myopic whose **refractive error** is less than −1D. If the movement is in opposite direction, the myopia is more than −1D.

Retinoscopy is done with or without mydriatic.

FIELD OF VISION

Definition. On looking straight ahead, with the eyeball fixed, that part of the external world which can be seen with each eye is called the **visual field of that eye.**

Extent. Laterally, it extends up to 104° (i.e., 14° behind the horizontal plane), on the nasal side

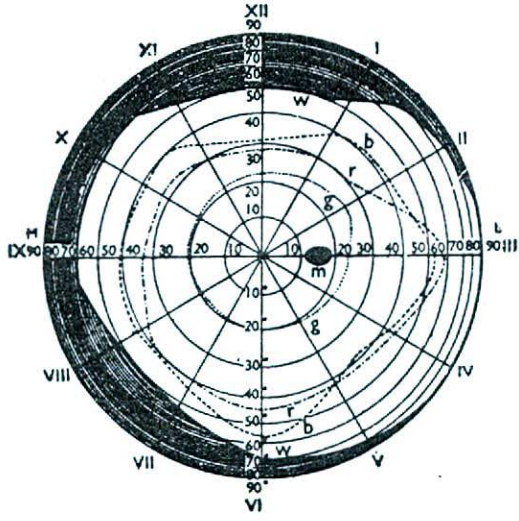


FIG. 6.92. Field of vision of left eye. m—blind spot; w, b, r, g—fields for white, blue, red, green respectively (*after Best & Taylor*).

about 65°. In front there is a cone-shaped area in which the two fields overlap and enjoy binocular vision. The visual fields for *blue*, *red* and *green* are progressively smaller (FIG. 6.92).

Mapping of the field of vision (*Perimetry*). Done by **Perimeter** (FIG. 6.93). It consists of a metal piece shaped like an arc of a circle, the centre of which is always marked by a fixed pointer attached to the base of the instrument. The subject's head is supported on the chinrest and the eye to be examined is placed very close to the metal point indicating the centre of the arc. The other eye is covered. An index mark, white in colour with a diameter of about 2 mm, is made to slide along the arc to find the limits of visual field in that meridian. The index mark can be blue, red or green to find out the corresponding colour fields. The arc can be rotated around a horizontal axis through a full circle, and at each new situation the test is repeated to find out the field in that meridian. These results when plotted give the visual field.

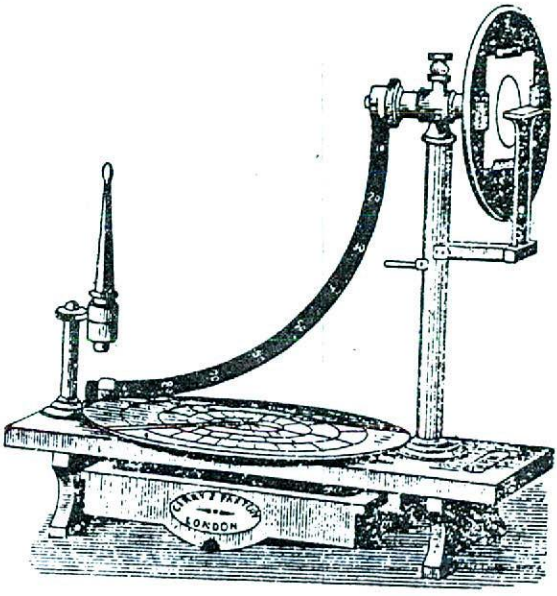


FIG. 6.93. Perimeter (*Priestley Smith*).

BINOCULAR VISION

Although we have two eyes, two optic nerves and two visual centres, yet we don't see two objects. This *phenomenon of seeing one object with two eyes is called* **binocular vision.** The impulses set up by light rays from an object when transmitted from the two retinae are simultaneously fused at the cortex into single image.

Conditions for binocular vision. (1) The two visual fields must overlap (FIG. 6.94). (2) Approximately similar images must be formed on the two retinae. (3) The retinae must possess physiologically corresponding points, so that images formed on them may cause a single visual impression. (4) The external ocular muscles must contract in such a way

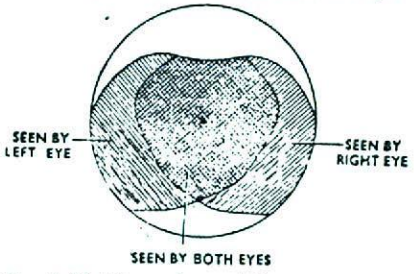


FIG. 6.94. Monocular and binocular visual fields. Heart-shaped common area (seen by each eye) is occurred with binocular vision.

that the axes of the two eyeballs converge on the object to be seen. (5) The oblique muscles must rotate the eyeballs, so that the physiologically corresponding points of retinae may occupy the corresponding meridians.

Advantages of binocular vision. (1) Optical defects of one eye are corrected by the other. (2) The combined field of vision of two eyes is much wider than that of single eye. (3) Binocular vision provides a very accurate perception of depth, size and distance **(Stereoscopic vision).**

Monocular depth perception

People with one eye can also judge accurately the length, breadth and thickness (depth or distance) of an object. Monocular depth perception (FIG. 6.94) depends on the following factors:

1. **The apparent size of known objects.** The further away the object, the smaller will be the size. Consequently, if the size be known, the distance can be judged.
2. **The apparent change of colour** of an object at a distance. Distance modifies colour. For instance, green trees appear blue at a distance. Colours of many other objects appear to fade more and more as the distance increases. The details of their outlines also become dim. These changes help to guess the distance.
3. **Partial blocking** of a distant object by a nearer object also gives idea of depth.
4. **The distribution of light and shade** is an important factor.
5. **The shadows** which one plane casts upon the other and upon its surroundings are also very helpful.
6. **Mathematical perspective,** for instance, parallel lines running to a distance seem to be convergent.
7. **Parallax.** When the head moves in one direction, the nearer object moves in the opposite direction, while the distant object moves in the same direction.

Stereoscopic vision

Stereoscopic vision is binocular perception of depth (FIG. 6.94). All the factors mentioned under monocular vision, are involved here. The following additional factors are also involved: (1) Convergence of the eye axes to produce images on the fovea simultaneously. (2) There must be some *dissimilarity* between the two images formed on the retinae. If a man looks at an object with one eye at a time, he will easily note the difference. This difference is of great help for stereoscopic vision.

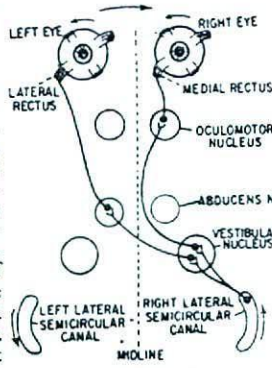


FIG. 6.95. It shows the deviation of eyes and displacement of the endolymph with rotation of the head to the right. Rotation of the head to the right results in counterrotation of the endolymph during the rotation and consequent deviation of eyes (slow phase of vestibular nystagmus) to the left. Since the movement of endolymph is towards the ampulla on the right and away from the ampulla on the left side, the right ampulla would be strongly stimulated during rotation of the head towards the right side. The reverse mechanism occurs with sudden cessation of rotation (postrotary deviation of eyes and nystagmus) (*after Adler*).

Fixation

Fixation is the maintenance of a fixed gaze on a particular object. It is caused by the co-ordinated contraction of the external ocular muscles so that the images may remain steadily on the two foveae. The point where the visual axes meet is called the *fixation point*. The lines joining the point to the foveae are called the *fixation lines*. The widest area through which fixation may be carried out is called **field of fixation.** It is circular with a diameter of about 100°, remaining well within the binocular field of vision. Fixation is partly voluntary and partly reflex. That it is voluntary

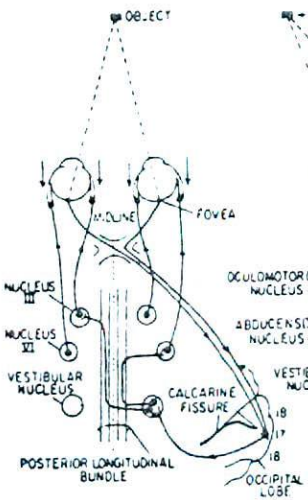


FIG. 6.96.

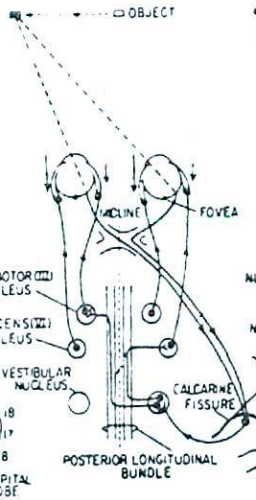


FIG. 6.97

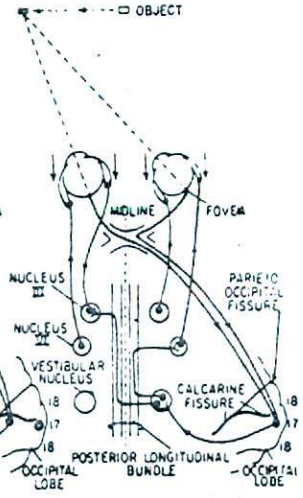


FIG. 6.98.

These demonstrate clearly the mechanism of fixation. Whenever the image of an object tends to move off the macula in one direction, an increase in tonus occurs in those muscles which move the eyes in that direction. Thus the eyes are turned reflexly to follow the object (FIGS. 6.96 & 6.98).

FIG. 6.96 The image of an object straight ahead of the eyes falls on the foveae. Visual impulses are carried up to the extreme tip of the occipital lobe along the calcarine fissure. From here visual impulses are carried to the vestibular nuclei by optomotor fibres and distribute tone to ocular muscles. The ocular muscle tone turning the eyes to the right is equal to that of the ocular muscles turning the eyes to the left, as long as the images are on the foveae. This figure purposely begs the question of bilateral representation of the fovea. The same stimuli go to the left calcarine cortex, as shown to the right.

FIG. 6.97. Object has moved to the left and therefore its image has moved off the foveae on to right halves of the both retinae. The visual impulses are carried up to the occipital cortex, to cells that lie farther forward along the calcarine fissure than those which come from the foveae. From these cells an optomotor impulse passes to the vestibular nuclei diminishing the tone of the ocular muscles which turn the eyes to the right and increasing the tone of those muscles which turn the eyes to the left. Until the image of the object once more falls on the foveae, the eyes are turned to the left. [It is assumed that the same stimuli go to the left occipital cortex as is shown to the right.]

FIG. 6.98 By means of the visual fixation reflex the eyes have been turned to the left to follow the movement of the object. Now the images that fall on the foveae again send their impulses up to the foveal localisation of vision in the occipital cortex. The optomotor impulses once again becomes equal to the tone of the ocular muscles turning the eyes to the right and the tone of those muscles turning the eyes to the left, so that the eyes remain fixed as long as the image of the object remains stationary.

The lengths of vertical arrows indicate amount of muscle tone (*after Adler*).

is proved by the fact that the eyes can be fixed at any imaginary object at 'will'. That it is reflex is proved by the facts that (*a*) no voluntary effort is required to maintain the fixation position, (*b*) when the head is moved or when the object is moved, the eyeballs rotate keeping a fixed gaze on the object (**rotation fixation**). All *psycho-optical reflexes* may be regarded as manifestations of the more general *fixation* reflexes—the reflex which brings the images of an object on to the foveae of two eyes and maintains them there. The reflex develops very early, as early as a few days after birth. Two sets of nerve paths are involved: (1) From the retina to calcarine cortex (visual path); (2) From calcarine cortex to superior corpora quadrigemina and thence to the nuclei of III, IV and VI cranial nerves (Figs. 6.95, 6.96, 6.97 & 6.98).

Strabismus or squint. Binocular vision requires a co-ordinated action of the eye muscles of both the eyes to move the eyeballs in one direction under united direction. If this balance is lost or the co-ordinating mechanisms are damaged owing to the disease, injuries or congenital abnormalities, the visual axes from both the eyes are not united upon the object or point looked at—this condition is called **strabismus** or **squint.** Such defects of long standing cases or caused by any actual muscle weakness may be remedied by careful surgical shortening of some of the eye muscles or by use of proper prismatic glasses which would direct the light upon the fovea.

THE VISUAL PATH

Visual impulses arise in the rods and cones which remain in the posterior part of retina. *So light has to pierce the whole depth of retina before it can arouse visual impulses.* The impulse then passes through the different layers of retina towards the ganglionic layer. The rod and cone cells constitute *first-order neurones*, the bipolar cells—*second-order neurones*, while the ganglion cells—*third-order neurones*.

Reactivity and behaviour of ganglion cells of the retina. There are extensive interconnections between the cells of the retina. Each ganglion cell responds to the pattern of illumination falling on a considerable area of the retina. Most retinal ganglion cells have a slow rate of discharge in the absence of any obvious light stimulus. Stimulation may alter this discharge in the following ways:

(*a*) There is an increase of discharge rate on switching on the light, This adapts rapidly and may temporarily fall below the original level when the light is switched off. (*b*) The sequence of events may be precisely the opposite. (*c*) There may be an increase in discharge at the beginning of the stimulus and at the end of the stimulus (Fig. 6.99).

Though the behaviour of all ganglion cells in the primate retina is less elaborate, some ganglion cells respond specifically to retinal images of the rabbit and the squirrel, moving in a particular direction across the receptive field. In the retina of the cat, monkey and probably of man, ganglion cells possess roughly circular receptive fields (Fig. 6.100). These fields comprise functionally distinct concentric *centre* and *surround* regions. Light, falling in one of these regions, excites the ganglion cell, causing its discharge frequency to increase. Whereas light, falling in the outer region, has an inhibitory effect causing the discharge frequency to decrease (Fig. 6.99).

Due to this antagonism, light falling uniformly over the whole receptive field has little effect upon the activity of the ganglion cell. It has been observed that the discharge of *off-centre* ganglion cell is inhibited by light falling on the centre of its receptive field (FIG. 6.100). It is also demonstrated that the response to a patterned stimulus can be maintained markedly for many minutes despite the occurrence of the initial adaptation (FIG. 6.99). The ganglion cells specially in the peripheral retina have much lower frequency of discharge at the time of uniform illumination. On the other hand, the ganglion cells respond with a transient increase in the frequency of discharge whenever the illumination is changed and adapt much more rapidly and completely to patterned stimulus. These types of ganglion cells seem to send information to the brain about the occurrence of changes in the visual environment requiring attention, rather than about the exact nature of the stimulating pattern (FIG. 6.99). For instance, when attention is suddenly transferred from a distant to a near object, the external ocular muscles contract to converge the eye axes, the iris constricts and the ciliary muscles contract.

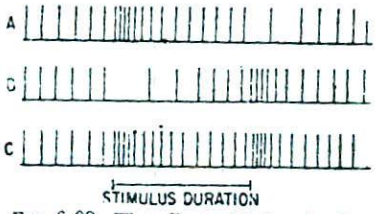


FIG. 6.99. The effects of light stimulation on three different types of retinal ganglion cells. A = this type of discharge fires with increased frequency at the beginning of the stimulus and its frequency falls below the resting level at the end. B = this type of discharge falls below the resting level at the beginning and rises above it at the end. C = this type of discharge both at the beginning and at the end.

When the retinal illumination is reduced to scotopic levels the behaviour patterns of the retinal ganglion cells commonly changes, while the surround mechanism becomes relatively less effective in antagonising the action of the central region. This effect is accompanied by an increase in the diameter of the central region of the receptive field and a change in the spectral sensitivity of the central mechanism. A ganglion cell at high light levels has a spectral sensitivity typical of cones and at low light levels may change to having one typical of rods.

In certain receptive fields, different spectral sensitivities are produced in the antagonistic regions though the significance of this mechanism in colour vision is unknown.

Retinal ganglion cells possess receptive fields of widely differing sizes. Although the receptive fields in the central area of the retina are smaller than those in the peripheral retina, yet in each area of the retina there are overlapping receptive fields with a range of sizes in the ganglion cells. The actual sizes of the receptive fields in the various retinal areas

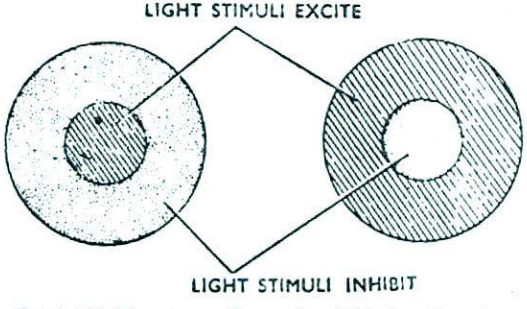


FIG. 6.100. Two types of receptive fields found at the retinal ganglion cells and in the neurones of the lateral geniculate body.

may differ but their range stays much the same. When a ganglion cell is much sensitive to the dimensions of the stimulus pattern, which are closely related to the size of its receptive field.. The existence of the receptive field of varying sizes denotes the transmission of information about spatial details of different dimension by different ganglion cells.

Recent study has revealed that the size of the cortical receptive field becomes larger and larger on moving away from the fovea. A few recordings from the foveal ganglion cells show that the central excitatory or inhibitory field may be the size of one cone.

In general, responses of lateral geniculate cells are similar to those of ganglion cells (Fig.6.100). The inhibitory fields are even more marked, thus tending to cut out weak responses and to leave strong responses clear cut.

There is some difference of opinion as to the number of neurones involved in the visual path. The *three-neurone* school believes that the bipolar cells are the first, ganglion cells the second and the lateral geniculate body the third-order neurones. The *four-neurone* school holds that the rod and cone cells are first-order neurones and from the lateral geniculate body the fourth-order neurones arise.

Optic nerve and tract. The impulses then pass through the stratum opticum and enter the **optic nerve** or **nerve of sight** (Fig. 6.102). In the optic nerve the impulses from different quadrants of retina occupy the corresponding quadrants. Nearly all its fibres are afferent and originate in the nerve cells of the retinal ganglion layer, but a few are efferent, their source of origin being debatable. In the **optic chiasma,** partial decussation takes place. Fibres coming from the temporal halves of retinae (nasal field) remain uncrossed, while those from the nasal halves (temporal field) cross. The macular fibres also decussate in the same way. After decussation they form the **optic tract.**

When each *optic tract* passes between the anterior perforated substance and the tuber cinereum, the tract becomes flattened and winds round the upper region of the cerebral peduncle. Here it is hidden from view on the basal surface of the brain by the uncus and the parahippocampal gyrus. When the tract reaches the lateral geniculate body, its course

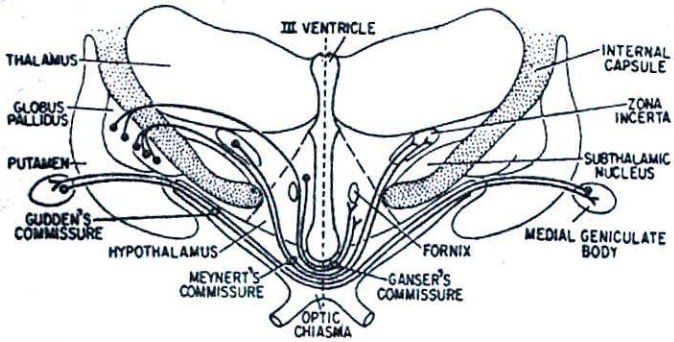


Fig. 6.101. Diagrammatic representation of the supra-optic commissures of Gudden, Ganser and Meynert. Fibres of Gudden's commissure are related to the medial geniculate bodies and possibly to the inferior colliculi. Ganser's commissure is derived from the anterior hypothalamic nucleus. Fibres of Meynert's commissure can be traced mainly into the globus pallidus and pass into the zona incerta.

is divided into a medial and a lateral root. The medial root is believed to contain the fibres of Gudden's commissure (FIG. 6.101). The lateral root comprises commonly the *afferent fibres* which arise in the retina and undergo partial decussation in the optic chiasma. But the lateral root contains a few fine *efferent fibres* which are passing forwards to terminate in the retina. Most of the fibres of the lateral root end in the lateral geniculate body, and some sweep medially below the pulvinar and reach the superior colliculus and the pretectal nucleus. The macular fibres end superiorly. New fibres arise from the cells in the lateral geniculate body and pass through the posterior root of the internal capsule. Emerging from the capsule as the optic radiation, the fibres of the second-order neurones curve backwards and medially to reach the cortex of the occipital lobe, where the higher visual centres are situated.

Some fibres in the optic radiation take an opposite course, arising from the cells of the occipital cortex and passing to the *superior colliculus*. Therefore the superior colliculus receives retinal as well as cortical fibres. From the superior colliculus new fibres arise and travel through the tectobulbar and tectospinal tracts to reach the nuclei of the oculomotor (III), trochlear (IV), abducens (VI) and superior spinal accessory (XI) nerves and the anterior gray column of the spinal cord (FIG. 6.102A & B). So the superior colliculus forms a *lower visual centre*. This lower visual centre is concerned, through its cortical fibres, with the reflex movements of the head and eyes, which occur in response to visual stimuli.

Arrangement of fibres. The fibres are grouped in the optic nerve in three bundles according to the retinal fields from which they originate:

1. Uncrossed temporal (lateral) fibres. 2. Crossed nasal (medial) fibres. 3. Macular fibres.

Close to the eyeball, the macular fibres (pupillo-macular bundle) occupy the lateral part of the nerve. Fifty per cent. of the macular fibres undergo decussation in the optic chiasma, so that half of the macula is represented in each tract. The fibres, being divided roughly into coarse and fine, vary in size; since the speed of conduction in a nerve fibre increases with its diameter, this variation in size must be associated with different conduction rates in the visual pathway.

The optic nerve, about 4 cm long, consists of finely myelinated fibres. The fibres from the temporal (lateral) side of each retina remain on the same side, but the nasal (medial) fibre cross. Of these about one-half are 7–10 μ in diameter and the other about 2–3 μ. The large fibres, having an average conduction rate of about 60 m/sec., are destined for the lateral geniculate body where they are relayed to the striate cortex. The smaller fibres, having conduction rate of about 25 m/sec., pass to the superior colliculi and are concerned with optical reflexes.

The optic tract fibres synapse with the fourth elements of the pathway in the lateral geniculate body. The lateral geniculate neurones carry the visual information up to the occipital pole of the cortex, where the receiving area is on the medial side of each hemisphere, associated with the calcarine fissure. In the tract from the thalamus to the cortex, fibres from the upper and lower halves of the retina tend to be separated by a large mass of macular (foveal) fibres.

Fibres of the optic tract *terminate in three ways:* (1) One set ends in the **pretectal area** from which fresh relay arises, crosses both in front and behind the aqueduct of Sylvius and ends round the III cranial

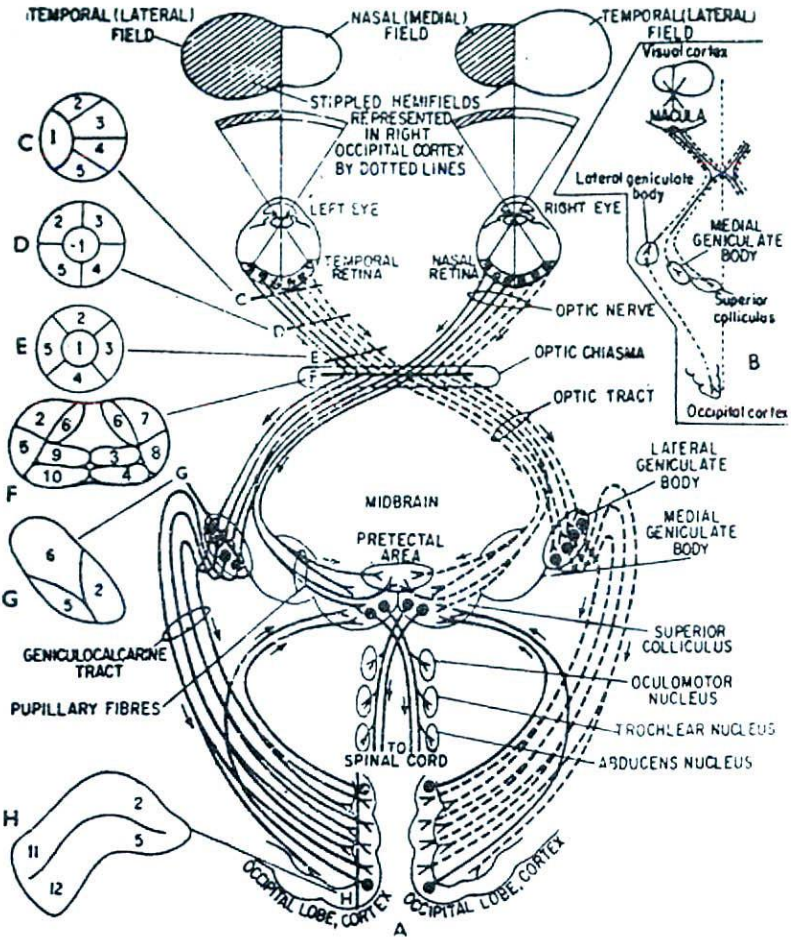


FIG. 6.102. Diagram of the central visual pathways. Fibres from the nasal retina, including the nasal half of the macula, decussate in the chiasma to join the uncrossed fibres of the temporal half of the retina (A & B). The broken lines in A represent nerve fibres from the retina to the occipital cortex that carry visual and pupillary afferent impulses from the left half of the visual field. Insets C–H showing distribution of the nerve fibre bundles at different levels of the visual pathway. C = optic nerve behind the eye. D = optic nerve behind the entrance of the central vessels into the nerve. E = optic nerve in front of its entrance into the optic chiasma. F = optic chiasma. G = lateral geniculate body. H = calcarine cortex. 1 = fibres from the macula. 2 = fibres from the temporal superior quadrant of the extramacular retina. 3 = fibres from the nasal superior quadrant of the extramacular retina. 4 = fibres from the nasal inferior quadrant of the extramacular retina. 5 = fibres from the temporal inferior quadrant of the extramacular retina. 6 = fibres from the temporal half of the macula. 7 = fibres from the temporal superior quadrant of the extramacular retina of the other eye. 8 = fibres from the temporal interior quadrant of the extramacular retina of the other eye. 9 = fibres from the nasal superior quadrant of the extramacular retina of the other eye. 10 = fibres from the nasal inferior quadrant of the extramacular retina of the other eye. 11 = fibres from the temporal superior quadrant of the macula. 12 = fibres from the temporal inferior quadrant of the macula.

nerve nucleus of the opposite side. *This pathway subserves light reflex.* (2) Some fibres end in the superior colliculus and are known as **mesencephalic root** of the optic tract. From here tectospinal and tectobulbar tracts arise. This pathway mediates tone, posture and equilibrium and also visuospinal reflexes (eye protection movements). Superior colliculi are considered as the highest visual centres in submammalian forms. In fishes and birds these structures are large as that of cerebral hemisphere. (3) The main set of fibres (**diencephalic root**) ends in the lateral (external) geniculate body and subserves vision. Hence, the lateral geniculate body is called the *primary visual centre. The macular fibres as a whole terminate here.*

Recent studies show that lateral geniculate body has six layers of nerve cells. Fibres coming from the retina of the contralateral side end in layers 1, 4 and 6. Those from equivalent spots of the ipsilateral retina end in same region but in layers 2, 3 and 5 (FIG. 6.103) [Le Gros Clark suggests that this layered arrangement may be concerned with colour vision.]

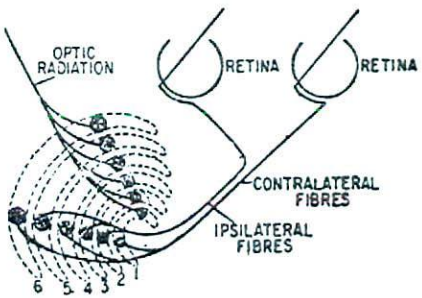


FIG. 6.103. It illustrates the segregation of the optic tract fibres in the separate laminae of the lateral geniculate body of the primate. The crossed impulses (fibres from the contralateral retina) relay in layers 1, 4 and 6 and uncrossed impulses (fibres from the ipsilateral retina) terminate in laminae 2, 3 and 5. These fibres terminate in a reception unit in the lateral geniculate body which constitutes a band of cells radiating from the hilus. The optic radiation (projection unit from the lateral geniculate body to the visual cortex forms a band of cells involving all the laminae (*after Le Gros Clark*).

Lateral geniculate body (nucleus). Ganglion cells of the retina send their axons to the lateral geniculate body and the superior colliculus. Although the cells of the lateral geniculate body are influenced by impulses in many fibres of the optic tract, they are usually very strongly influenced by discharges from one specified ganglion cell. This influence has been observed by simultaneous records from single cells in the retina and the lateral geniculate body and it is also observed that the behaviour of cells of the lateral geniculate body is not very different from that of the retinal ganglion cells that drive them, though they generally adapt quickly and completely. Like ganglion cells they possess concentrically organised receptive fields of different sizes many of which have *colour-specific centre* and *surround regions*. Although the significance of the difference of the antagonistic surround regions of the receptive fields of geniculate neurones and those of retinal ganglion cells is obscure, the receptive fields of the lateral geniculate body are rather more diffuse than those of retinal ganglion cells. Cells in the primate lateral geniculate body respond briskly to moving visual stimuli, but they do not show any directional effect nor any specific response to moving.

Lateral geniculate body receives afferent nerve fibres from both eyes, but stimulation of one eye can only excite individual cells. Recently it has been observed that most cells can be inhibited by stimuli falling on the corresponding regions of the retina of the other eye: It is also found

that the activity of cells in the lateral geniculate body can be influenced by stimulation of the vestibular and other sensory systems.

FEEDBACK MECHANISM. Traditionally the lateral geniculate body has been regarded as a relay-station from the retina to the cerebral cortex, and as such, doing little more than pass the visual information on. However, the message is modified, and this modification is effected not only by activity on the part of geniculate cells, but also corticofugal fibres, i.e., by fibres originating in the occipital cortex which, by either excitatory or inhibitory activity, modify the responses of the geniculate cells to the retinal impulses. By this feedback arrangement the cortex is able to modify and control the information it receives. Inhibition of geniculate cells following light stimuli may also be achieved by activity on the part of the geniculate cells themselves. The principal cell, activated by an optic tract fibre, sends its message up to the cerebral cortex but also, by a collateral fibre, it activates a short-axon neurone in the lateral geniculate body, and this in turn inhibits the activities of neighbouring principal geniculate cells (FIG. 6.104). According to Vastola, most strongly inhibited cells receive the weakest excitatory stimuli from the optic tract. Such an inhibition of the weakly stimulated cells by the more strongly excited cells has obvious value in sharpening up the retinal image, when considering the basis of visual activity. The lateral geniculate body continues a process that has already been begun in the retina itself.

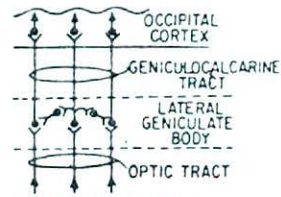


FIG. 6.104. It illustrates inhibition within the lateral geniculate body. The middle one of three geniculate cells relays an excitatory influence to the cerebral cortex, and to short-axon inhibitory cells which inhibit the neighbouring geniculate cells (*after Dawson*).

Central representation of visual fibres. From the lateral geniculate body the *fourth-order neurones* arise and constitute the **optic radiations.** These fibres pass through the posterior limb of internal capsule and end in the granular layer of the occipital cortex. The granular layer is also known as **striate area** (area 17). Area striata forms the walls and lips of the calcarine fissure on the medial aspect of the occipital lobe. The different retinal areas project on the definite area of the cerebral cortex and there is a point-to-point projection on to the striate area. The *nasal* (*medial*) *half* of the right retina and the *temporal* (*lateral*) *half* of the left retina are projected on to the left occipital cortex. The nasal half of the left retina and the temporal half of the right retina are projected on to the right striate area. It is supposed that the macula is represented bilaterally on the visual cortex since macular vision of both eyes is relieved even after extensive lesion of an occipital lobe (*vide* Macular sparing). Small lesions in the striate area lead to blindness. Lesions of the visual cortex in monkey may involve degeneration of the lateral geniculate body and even involve cells in all six layers of the geniculate body. Since each centre represents the opposite half of the visual field, it is also called the *half-vision centre*. The upper and lower quadrants of each half of the retina occupy the upper and lower regions of the centre. The area for peripheral retina is smaller and situated anteriorly. While the macular area is larger, mainly occupy the posterior part, but also project forward between the peripheral areas. In man, the

centre stops at the occipital pole and does not go on to the outer convex surface. But in monkey it spreads on to the convexity. Just above this centre on the convexity lies the visuopsychic centre (area 18). Here the 'meaning' of visual impression is understood.

Activity of cells in the visual cortex. Like other parts of the cortex, the visual cortex appears to contain six layers of cells. It is also called the **striate cortex** because, on taking a section at right angles to the surface, a white layer can clearly be seen. This is due to the almost complete absence of large pyramid-shaped cells. Most of the axons from the lateral geniculate nucleus end in fourth layer on cells which then make connections with other layers. The cells of third and fifth layers send their axons out of the visual cortex. Most of the connections are perpendicular to the cortical surface. Single unit recording has shown that the physiological organisation is in columns as in the somatic sensory and auditory areas. All the cells encountered in one track perpendicular to the surface respond to the same sort of stimulation.

The ability of cortical cells to respond rather selectively to particular spatial stimulus patterns is probably determined by the interplay of excitatory and inhibitory influences from various regions of their receptive fields. In this respect the receptive fields of visual cortical cells are linearly, rather than circularly, organised, the excitatory and inhibitory influences coming from adjacent strip-like regions of the receptive field. It has been shown that some receptive fields are made up of several such antagonistic strips side by side.

The **cortical receptive fields** are quite different from those of the lateral geniculate and retinal ganglion cells. Two categories are observed:

(*a*) Simple cells respond to line stimuli, such as a dark line on a light background or an edge between light and dark areas. The line must be of a certain minimum length, it must fall on a certain part of the retina, and it must be orientated at a certain angle to produce the maximum response (FIG. 6.105).

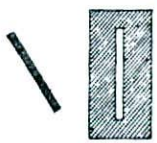


FIG. 6.105. It shows stimuli which effectively excite simple cortical cells (diagrammatic).

(*b*) With complex cells, the line must also be of a certain minimum length and a certain specific orientation, but the cells are activated no matter where the line falls on the retina.

According to Hubel and Wiesel, a group of the lateral geniculate cells which are all of one kind (either excitatory or inhibitory centres) and whose receptive fields fall in a line, all feed into one single cortical cell (FIG. 6.106). For maximum activity of the cortical cell, all of the lateral geniculate cells can be activated. This will be achieved only by a line stimulus which lies along all of the receptive field centres. Cells with inhibitory centres will be maximally activated by a dark line on a light background. On the other hand, cells with excitatory centres will be activated by a light line on a dark background. All the lateral geniculate units are not activated by a short line. A line which is not precisely the right orientation will activate fewer of the receptive field centres. If the line will lie across more of the surround areas, the effects of its stimulation will oppose the effects of central stimuli. Furthermore, if the line is very precisely oriented, the maximal response will only be seen. Each cortical column contains hundreds of these simple cortical

cells responding to lines of this orientation falling on different parts of the retina (Fig.6.107). These simple cells feed into the complex cells which will therefore recognise a line of that particular orientation. There is no matter where on the retina it falls.

Some cortical cells, known as **hypercomplex** cortical cells, exist and may be capable of recognising shapes. Fig. 6.108 represents how three complex cortical cells would feed into a hypercomplex cortical cell which could fire whenever a triangular stimulus be shone on to the retina. Hubel and Wiesel have pointed out that there are 'at least some parts of the brain can be followed relatively easily without necessarily requiring higher mathematics, computers, or a knowledge of network theories'.

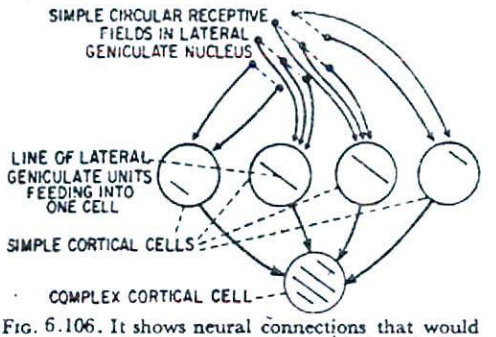


Fig. 6.106. It shows neural connections that would account for simple and complex cortical cells. Simple cortical cells cover a certain area of the retina. A line feeding into one cortical cell could lead to that cell responding to a stimulus which activated all the units in the line. The stimulus would have to have a precise orientation and would have to fall on a particular area of the retina. A number of simple cortical cells, all responding to stimuli of similar orientation, feed into a complex cell which will then respond to a stimulus of that orientation no matter where on the retina it falls.

Though one eye is commonly dominant, most cells in the visual cortex can be excited by stimuli applied to either eye. Simultaneous records from both eyes after stimuli have shown that the response of the cell may be much greater than when either eye is stimulated alone. The stimulus pattern must be appropriately positioned on both retinae in order to obtain maximum response. When both eyes are in their normal positions, the optimal positioning of both

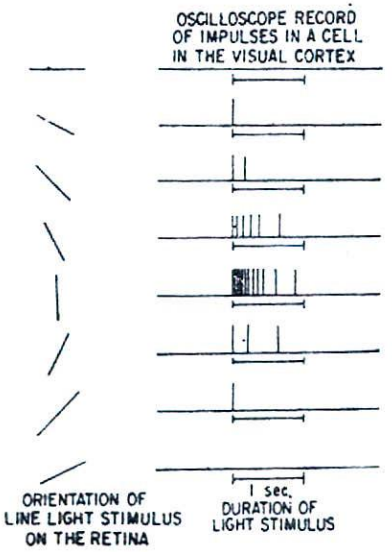


Fig. 6.107. It shows the effect of orientation of a stimulus on the discharge of a simple cortical cell. A high-frequency discharge gives rise precisely correct orientation (*after Hubel & Wiesel*).

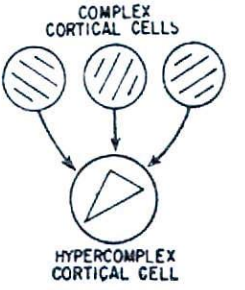


Fig. 6.108. It represents hypercomplex cells, capable of recognising shapes.

retinal images of a real object can only be achieved if the object is at a certain distance from the eyes. Probably different cortical cells respond best to objects at different distances, thus providing a basis for stereoscopic vision which depends upon the presence in the visual cortex of cells responding specifically to retinal images with particular degrees of disparity.

Effects of injury at different levels of visual path (FIG. 6.109)

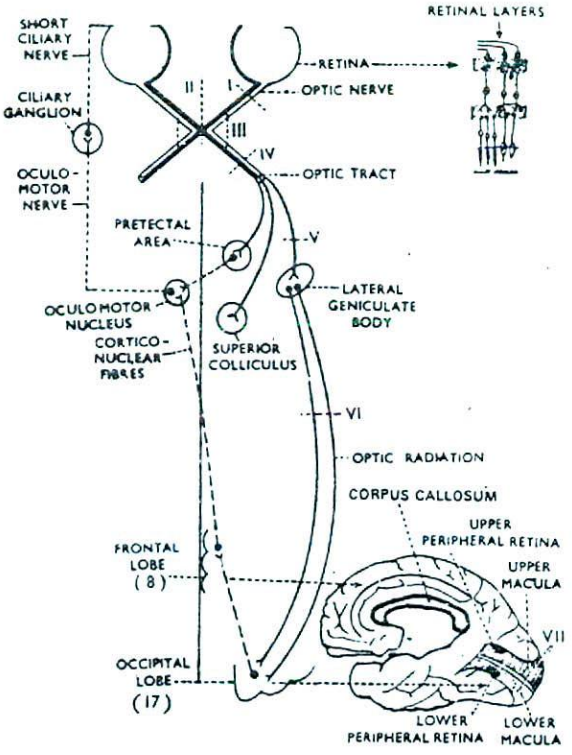


FIG. 6.109. Visual pathways in the CNS and the effects on the visual fields of lesions (I, II, III, IV, V, VI, VII) at various points.

(*a*) Injury to optic nerve at the level I blindness of the affected eye.
(*b*) ,, ,, crossed fibres at the level II bitemporal hemi-anopia.
(*c*) ,, ,, uncrossed fibres at the level III binasal hemi-anopia.
(*d*) ,, ,, optic tract before light reflex fibres leave at the level IV (*i*) Homonymous hemi-anopia. (*ii*) Light reflex lost.
(*e*) ,, ,, optic tract after light reflex fibres leave at the level V (*i*) Homonymous hemi-anopia. (*ii*) Light reflex persists.
(*f*) ,, ,, optic radiation at the level VI same as V.
(*g*) ,, ,, area striata of one occipital lobe causes homonymous hemi-anopia. Injury at the level VII to the upper or lower part of the area striata on one side causes quadrantic homonymous hemi-anopia.

Macular sparing. When there is intact macular vision but peripheral vision is lost. This is common with occipital lesion (at the level VII) because the macular representation is wide (bilateral) enough to cover a very large fields of the occipital lobe.

Bilateral destruction of the occipital cortex causes complete blindness in human beings.

COLOUR VISION

Appreciation of colour is a function of the light-adapted eye and is entirely the property of cones. A normal man can easily distinguish the seven colours of spectrum and an infinite number of intermediate shades. The human eye can recognise as many as 160 different colours in the visible spectrum (400–750 m μ) and difference of less than 3 m μ wave length can be detected. The limiting wave lengths of commonly named colours are:

Violet	397–424 m μ
Indigo	424–455 m μ
Blue	455–492 m μ
Green	492–575 m μ
Yellow	575–585 m μ
Orange	585–647 m μ
Red	647–723 m μ

White means a colour mixture in which the seven component colours remain in the same proportion as in the sunlight. Black is the sensation caused by the withdrawal of light. It is a sensation connected definitely with retinal activity and one must have a retina in order to see the black because in the **blind spot** where there is no retinal receptor one sees not black but nothing.

When the intensity of light is gradually reduced, at first the red objects lose their colour, then the yellow, then the green and lastly the blue. In the dim light, the surroundings appear to have no definite colour and seem to be of various shades of grey or bluish-black. All the colours of spectrum can be obtained by the fusion of the following three colours in varying proportions—**red, green** and **violet.** These are termed the **primary colours. Complementary colours** are those (*a*) which appear in the negative after-image, (*b*) which when blended in proper proportions, give the grey impression. *Red* and *green*, *yellow* and *blue*, *black* and *white* are complementary pairs. The same colour may be primary as well as complementary to another colour. The field of vision for the different colours is different.

In *colour fusion* one colour more or less may extinguish another colour, viz., fusion of yellow and blue produces white. *Maxwell's coloured disc* with rotating device is used for demonstration of the fusion of colours. If a piece of blue paper is placed against a yellow paper then intensity of colour becomes heightened (**colour contrast**).

Theories of colour vision

There are numbers of theories, but none can explain the full details. The first important theory (**trichromatic theory**) explaining the colour vision was that of Young in the year 1801. In the middle of the same century Helmholtz elaborated colour vision in detail.

Young-Helmholtz theory. This theory holds that there are *three types* of cones with three fundamental sensations—red, green and violet. These three types of cones contain three photochemical substances corresponding the three fundamental colour sensations. The theory also suggests the presence of specific nerve fibres and specific cortical cells corresponding specifically to each photochemical substance for each colour sensation—red or green or violet. The different photoreceptors, after being stimulated, transmit different types of messages to the visual cortex.

The theory of Helmholtz also describes that though the photochemical substances excite the nerve fibre but the quality of sensation developed depends on the ending of the fibre in the brain.

Helmholtz hypothesis of zonal representation of different colours in the cerebral cortex has not been confirmed but Le Gros Clark has suggested the presence of three layers in the lateral geniculate body which are somehow rather related to the **trichromatic theory of colour vision.**

It is assumed that each of the photoreceptors is stimulated to some degree by all of the spectral frequencies but for a particular colour sensation, the specific photoreceptor is chiefly affected by the specific wave length.

The sensations of many colours are produced by combined stimulation of the three receptors at different intensities. White sensation arises when all the three are equally stimulated.

Young and Helmholtz trichromatic theory of colour vision also states that any colour can be matched in terms of three stimuli. Fig. 6.110 represents the spectral mixture curves indicating the relative amounts of three primary colours that are required for matching any spectral wave length. From the three curves we can get the amounts of red, green and blue required to match any spectral hue. As for example, for a blue at 5000A, the relative contributions of green will be 0·33 unit, that of blue 0·01 unit (actual value in the figure is 0·1 but it has divided by 10 only because the luminosity value is very low and has been multiplied by ten) and of red 0·05 unit. The relative contributions of three primary colours for green of 5500Å will be 0·88 unit green, 0·12 unit red and −0·001 unit blue (approximately).

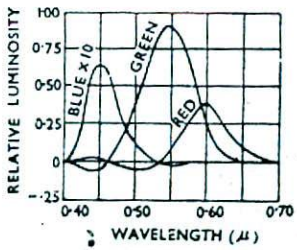


Fig. 6.110. Graphical representation of spectral mixture curves indicating the relative amounts of the three primary colours for matching any spectral hue (*after Wright*).

These curves lead to the hypothesis that any colour sensation can be made by stimulating three receptors of Young-Helmholtz to a varying degree. That is for the sensation of blue, either a pure blue light of 5000Å or a mixture of quite different lights of different wave lengths is required. A colour of certain wave length and a colour mixture of different wave lengths are physically different but in the retinal receptors, the sensation will be same and the wave length of pure colour and that of the mixture cannot be deciphered.

The **three receptor hypotheses** are the fundamental basis of Young and Helmholtz trichromatic theory of colour vision. Accordingly the three receptors will respond differently to the same wave length. The blue

receptor will be stimulated maximally at the short end of the spectrum, but minimally at the extreme long end of the spectrum. If the frequency of discharge from the single fibre of the optic nerve is recorded against the different wave lengths of light, then a characteristic wave length response curve (FIG. 6.111) may be obtained. The intensity of the light will be indicated by the absolute values of the frequencies of discharge in the receptors and the quality will be indicated by the relative values of frequencies.

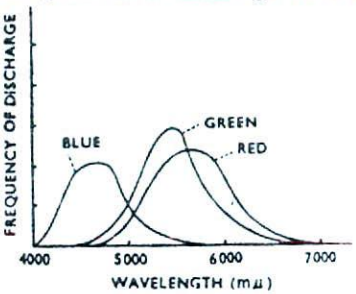


FIG. 6.111. Wave length response curve of three hypothetical receptors sensitive maximally to red, green and blue respectively (*after Dawson*, 1963).

Absorption spectra of cones: Photochemistry of colour vision. Young and Helmholtz theory of colour vision requires three pigments. Iodopsin, the violet-sensitive substance has been extracted by Wald in 1937 from chicken retina. The absorption spectrum of iodopsin corresponds with the photopic sensitivity curve.

Hanoaka and Fujimoto in 1957 spectrophotometrically studied the absorption spectra in fish cone and showed that different cones had different absorption spectra with maximum in the region of 5000Å, 5350Å, 5750Å and 6200Å. But Marks in 1965 also studied in fish and observed the absorption spectra maxima at 4550Å, 5300A and 6250Å, i.e., in the blue, green and red parts of the spectra respectively. Brown and Wald in 1964 also showed the presence of three types of cones having maximum absorption spectra at 4550Å, 5250Å and 5550A.

Marks and his colleagues in 1964 have also demonstrated through microspectroscopical studies the presence of blue receptor substance in the tiny cones of human and monkey retina. Wald in 1964 also obtained the evidence for the presence of the blue pigment in man. It has been strongly supported with proof that there are three pigments corresponding three cone receptors in the retina. These studies also confirm the validity of trichromatic theory derived from colour mixing.

Granit's modulator and dominator theory. Granit in 1945 inserted micro-electrode into the single ganglion cell and investigated the sensitivity to light of different wave lengths. He obtained the sensitivity curve in the light-adapted eye and also in the dark-adapted eye similar to that in photopic and scotopic visibility respectively.

The shift of scotopic curve to photopic curve suggests that as the rods and cones converge on the single ganglion cell, then the rod will possibly cease functions at light intensities which will stimulate the cones. On the basis of his findings Granit has described that there are ganglion cells which are stimulated by the whole of the visual spectrum. These are described by him as **dominator.** There are two types of dominator—**dominator for cone** and **dominator for rod.** In light-adapted eye the dominator cones in general give broad sensitivity curve with maximum response around wave length of 550 m μ. In dark-adapted eye the dominator rods only response and give maximum response at wave length of 500 m μ. **The dominator cone indicates intensity of light, but not the**

colour. Dominator rod is probably related to the sensation of brightness.

In the light-adapted eye Granit found also certain ganglion cells which are stimulated only at a narrow wave length band. These are described as modulators and are of three groups. One group is affected by blue light of wave length 450-470 m μ, one that are affected by green light of wave length 520-540 m μ, and other is affected by red-yellow light of 580-600 m μ (Fig. 6.112).

Granit has also described that during light adaptation, the modulators also come into action. The spectral composition of light stimulus determines at what degree the three groups will be stimulated. As for example, if the green light falls on the retina then the green modulators will be stimulated maximally while the red and blue modulators will be affected little. Thus **the modulators are concerned with different colour sensations.**

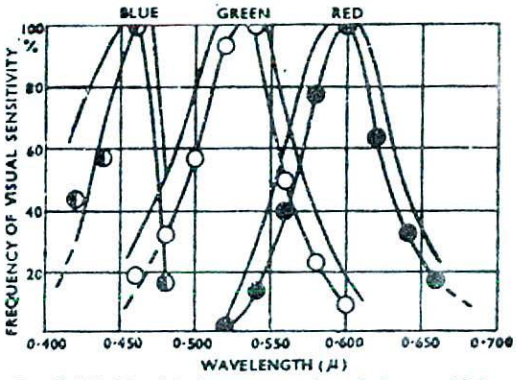


Fig. 6.112. Graphical representation of the sensitivity curves for blue, green and red receptors (*after Granit*).

Electrophysiological aspects of colour vision: Wave length discrimination. That human retina is trichromatic can be proved by recording the frequency discharge in single fibre of the optic nerve after stimulating the eye of Limulus containing one kind of receptor only. In such preparation, if the eye is stimulated at light of different wave lengths but with of same intensity, then a wave length response curve is obtained (Fig. 6.113). This curve looks like a typical sensitivity one having its maximum discharge at 5200Å corresponding to the wave length of maximum absorption of its photopigment.

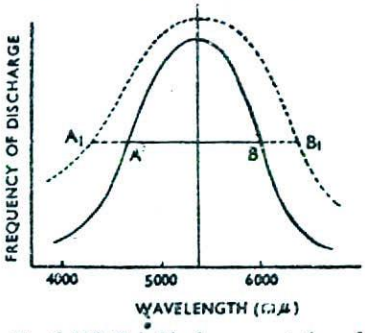


Fig. 6.113. Graphical representation of wave length response curve of Limulus retina with single receptor (*after Dawson*, 1963).

In Fig. 6.113, A and B in the curves show the same frequency of discharge though the wave length is different (4900Å and 6200Å). So there will be a confusion in between the blue (4200–5000Å) and orange (6000–6500Å). If similar lines are drawn parallel with the abscissa, then several other pairs of spectral hues can be obtained giving same responses. Fig. 6.113 was obtained by stimulating the eye with equal light intensity at different wave lengths, but if the light intensity is altered keeping the wave length constant then several curves like dotted lines will be obtained showing the responses

(frequency of discharges) of all other wave lengths of the spectrum simply by varying the intensity of the single wave length.

Thus by varying the intensity of one **monochromatic light,** all frequencies of discharges can be obtained or on the other hand by an appropriate intensity of monochromatic light, the response of any coloured stimulus can be matched.

This shows that the retina of Limulus having a single type of receptor fails to discriminate changes in wave length and intensity. This retina can be described as **achromatic** which is similar to the retina of the totally colour blind and also to human retina under dark adaptation.

Hering theory. Though the Hering theory is antagonistic to that of Young and Helmholtz yet it is simply the modification of the latter. Hering also assumes the existence of three photochemical substances in the retina but they are of such a nature as to give six different qualities of sensations.

According to this theory the photochemical substances (white-black, red-green and yellow-blue) give one sensation on breakdown and a different one on resynthesis.

Photochemical substances giving different colour sensations during breakdown and synthesis (*after Adler*)

Photochemical substance	*Retinal process*	*Sensation*
White-black	Breakdown Resynthesis	White Black
Red-green	Breakdown Resynthesis	Red Green
Yellow-blue	Breakdown Resynthesis	Yellow Blue

According to this theory, the complementary colours become antagonistic to its respective primary colours. This theory also does not accept the presence of specific nerve energies because it assumes that the same fibre can carry two different sensations to the brain.

Cortical representation of colour sensation. Recent evidences suggest that the colour sensation of the retina is largely concerned with cerebral cortex. But the rod function is not so dependent on the cortical function. In destruction of the cortex, the cone function is greatly affected, but the rod is totally unaffected. The visual impulses are carried from the different types of cones to the occipital cortex. It is in this area the differentiation of the visual impulses takes place and there is perception of various coloured lights. The perception of complementary colours also takes place in the same area.

COLOUR BLINDNESS

Colour blindness means defect or lack of appreciation of one or more colours. About 8% of males and 0·4% of females show some defect of colour vision.

Classification. Different ways of classification. But none is completely satisfactory. Because, (1) classification depends upon one or the other theory of colour vision, hence varies widely. (2) various anomalous types are often found which may not fit in with a particular classification.

The **following classification**—depending upon Young-Helmholtz theory of colour vision—is widely accepted. According to this theory, normal colour vision depends upon the presence of three primary colour substances—red, green and blue. So that the colour blindness is divided into three following broad classes:

I. **Anomalous trichromatism.** This is the commonest variety—5·6%. Anomalous trichromats can see all the three colours, but appreciation of one particular colour is subnormal. For instance, to match a particular shade of red, green or blue he will require a greater amount of that particular colour than a normal subject. This class has *three subclasses:* (*a*) **Protanomaly (partial protanopia)**—1%. Red vision is subnormal. Generally hereditary. (*b*) **Deuteranomaly (partial deuteranopia)**—Green vision is subnormal. Commonest variety—4·6%, generally hereditary. (*c*) **Tritanomaly (partial tritanopia)**—Subnormal blue vision. Rarest form—0·0001%. An instrument called *Anomaloscope* is used for testing these defects.

II. **Dichromatism.** Less common than the first group—2·6%. Dichromats can see two colours but fail to see one. Consequently, there are *three types:* (*a*) **Protanopia** or red blindness—1·2%. (*b*) **Deuteranopia** or green blindness—1·4%. (*c*) **Tritanopia** or blue blindness. The first two types are generally hereditary, whereas tritanopia is the rarest form and is generally acquired. It is found in detachment of retina, in jaundice (Bilirubin in the eye absorbs all blue colour), heavy doses of santonin, etc.

III. **Monochromatism.** This is the reverse of night blindness where the rods are all right but the colour sensation of the cones is lost. It may be regarded as total colour blindness. In the daytime the surroundings appear to be dark, grey or pale bluish-white (rod vision).

Tests for colour blindness

This test is essential for members of those profession who have to distinguish between different colours, such as drivers, signalmen, seamen, air pilots, etc. The following three types of tests are generally employed:

I. **Spectroscopic test.** In this test, the limits of visibility of spectrum, distinction between different spectral colours, their exact positions, etc., are tested.

II. **Colour-matching test** (Holmgren's wool test). The subject is given a set of coloured wools. He is required to select from the set only those specimens which will match another given sample of a particular colour.

III. **Use of pseudo-isochromatic plates** (Ishihara's test). These plates consist of a series of cards on each of which is printed a coloured background in spots of different sizes. A letter, figure or a number is printed against this background—also in spots of same size. These spots are of such a colour that it is likely to be confused with the colour of the background. To a normal subject the figure or letter at once becomes clear, but the colour-blind subject fails to distinguish it from the background. The numerical figures are read correctly by a normal subject, but the colour-blind subject may read these differently.

IV. **Green and red light test.** This device is in practice for testing the engine-drivers, signalmen, etc. The subject has to identify the green and red light focused from a small illuminated area.

AFTER-IMAGE

Visual impressions persist even after the removal of the object. This is called **after-image.** After-images may be of two types: (1) **positive,** and (2) **negative.**

1. **Positive after-image.** If after looking at an object the eyes be closed or turned to a dark surface or the object be quickly removed, the image of the same object with the same colours can be seen for a brief while. It is due to the continuation of the physicochemical changes in the rods and cones even after the stimulus is removed. This phenomenon is also called *persistence of vision.* It is due to this phenomenon that rapidly intermittent stimuli (as in the cinema) become fused into a smooth continuous visual impression.

2. **Negative after-image.** After looking at a coloured object if the gaze be turned against a white background, the image of the same object will be seen but with the complementary colour. If the object be white, red or yellow, the image will be black, green or blue respectively.

Young-Helmholtz theory holds that fatigue of one or more of the three varieties of cones (for three primary colours) is caused by the first stimulus and these fatigued cones will not respond for a brief while to a second stimulus of the same type. When the subject looks against a white surface, the white light stimulates the whole retina except the tired cones. So that the negative after-image of a white object will be black. If the original object was coloured, the white light will fail to stimulate the tired cones but will stimulate the others, giving the complementary colours.

Flicker. Observing the effects of intermittent stimuli, it is found that if the second stimulus falls during the after-image of the previous one, its character is altered. If the two stimuli be similar (black or white, etc.) in nature, the second stimulus is suppressed. But if of dissimilar nature, the second stimulus is heightened. The flicker phenomenon depends upon the latter condition. If a disc with alternate black and white sectors (Maxwell's disc) be rotated slowly—the stimuli heighten each other and an unpleasant glittering sensation is felt. This is flicker. This may be compared to 'clonus' of muscles. If the rate of rotation be further raised, flicker passes off, producing a complete fusion (like tetanus of muscle). [A static object in the dark may be invisible, but if it moves, it may be visible due to flicker.]

Contrast phenomenon

There are two types of contrast: (1) **simultaneous contrast,** and (2) **successive contrast.**

1. **Simultaneous contrast.** When an object with a particular colour is viewed against a background of the complementary colour, such as red against green, blue against yellow, black against white—both the colours appear to be more vivid. This is due to negative after-image.

2. **Successive contrast.** If one looks for some time at a coloured object in strong light and then at another object with the complementary colour, the latter appears brighter. For instance, if after looking at a red object a green object is seen, the colour of the latter seems to be more intensified. This is also due to negative after-image.

HEARING

Anatomy (FIGS. 6.114 & 6.115). Ear has three parts: **external ear, middle ear, internal ear.**

I. **External ear.** Two parts: (1) **pinna,** and (2) **external auditory meatus. Pinna** collects and reflects sound waves into the meatus. The

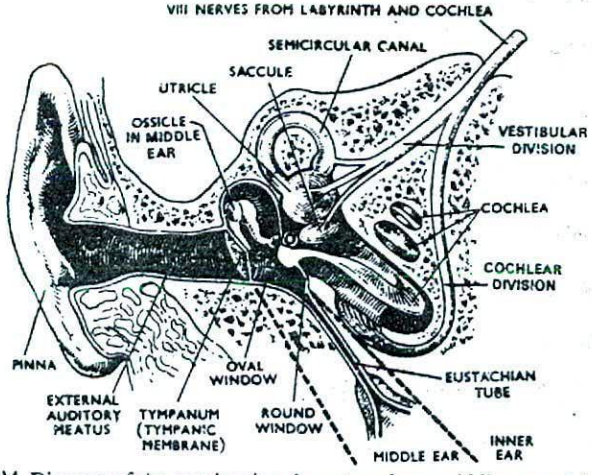


FIG. 6.114. Diagram of the ear showing the external ear, middle ear and internal ear and also showing the communication of the middle ear with the nasopharynx by means of the Eustachian tube.

external auditory meatus is a tortuous canal, about 2·54 cm long, looking at first inwards forwards and upwards and then inwards forwards and downwards. It is shut off medially by the **tympanic membrane** (FIG. 6.116). It transmits the sound waves perpendicularly to the membrane. A straight rod will hit the roof. Thus, tortuosity of the canal prevents mechanical injury to the tympanic membrane from outside.

II. **Middle ear.** Contains **air, auditory ossicles, tensor tympani muscle** (fifth cranial nerve) and **stapedius muscle** (seventh cranial nerve). **Tympanic membrane** (FIGS. 6.116 & 6.117) forms the outer wall being elliptical (10 mm vertical × 9 mm horizontal) and ear

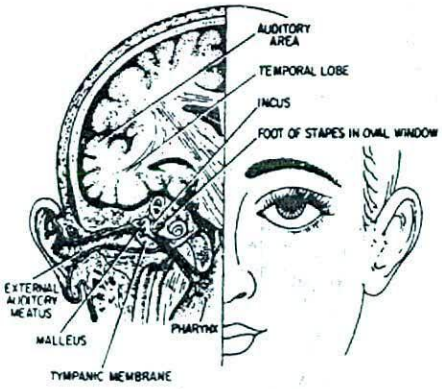


FIG. 6.115. It shows the principal features of the human ear. The external ear consists of the pinna and external meatus. The tympanic membrane is situated at the medial end of the meatus. The middle ear is situated within the temporal bone between the tympanic membrane and the lateral bony wall of the internal ear. The middle ear communicates with the pharynx through the Eustachian tube. The

and forwards It is concave from outside (FIG. 6.120). The point of maximum concavity is called **umbo.** To this point the handle of the malleus is attached on the inner side. In the medial wall there are two

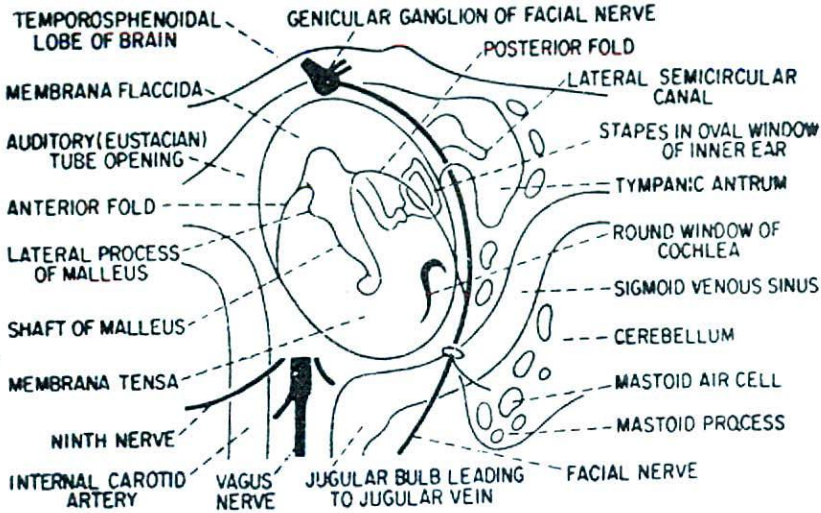


FIG. 6.116. Diagrammatic representation of the tympanic membrane and important anatomical structures around the middle ear.

windows: the **oval window (fenestra ovalis)** and the **round window (fenestra cochleae or rotunda).** The latter is closed by a membrane. To the former is fitted the foot plate of the stapes and sealed in by means of a membrane. The posterior wall communicates with air cavities in the mastoid process. The largest cavity being known as the **mastoid antrum.**

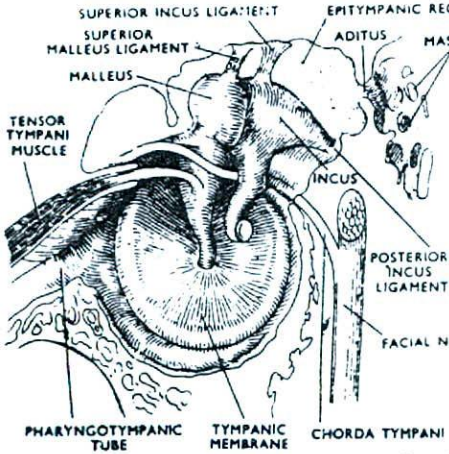


FIG. 6.117. Diagrammatic representation of the tympanic membrane and its attachment with the malleus.

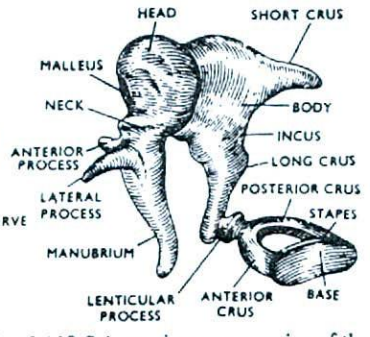


FIG. 6.118. Schematic representation of the ossicles of the ear showing the attachment of malleus, uncus and stapes.

Through the anterior wall pass *two canals.* The upper one lodges the **tensor tympani** (attached to the handle of the malleus), the lower one is known as the **Eustachian tube,** which opens into the pharynx (Fig.6.114).

The *Eustachian tube normally remains closed, but opens during chewing, yawning and sneezing.* Its **main function** is to equalise pressure on the two sides of the eardrum (the tympanic membrane). Any imbalance of the static pressure affects the transmission of the middle ear structure. This effect is generally felt while landing an aircraft. During diving or going up in an aeroplane, there is change of air pressure and this may produce a considerable stress on the tympanic membrane if the pressure in the middle ear is not equalised with the outside pressure. This is achieved through the Eustachian tube.

The auditory ossicles are three minute bones, known from outside inwards as **malleus** (hammer), **incus** (anvil) and **stapes** (stirrup) (Fig.6.118). The stapedius muscle remains attached to the stapes. They remain articulated together forming a complex system of levers stretching across the middle ear—the handle of the malleus remaining attached to the tympanic membrane and the foot plate of the stapes fitting into the oval window in the internal wall. The malleus, incus and stapes are bound by ligaments. When the sound waves impinge on the tympanic membrane, these chains of ossicles vibrate as a single unit.

Figs. 6.119 & 6.120 show the link between the inner side of the tympanic membrane to the liquid-filled cochlea at the oval window. The

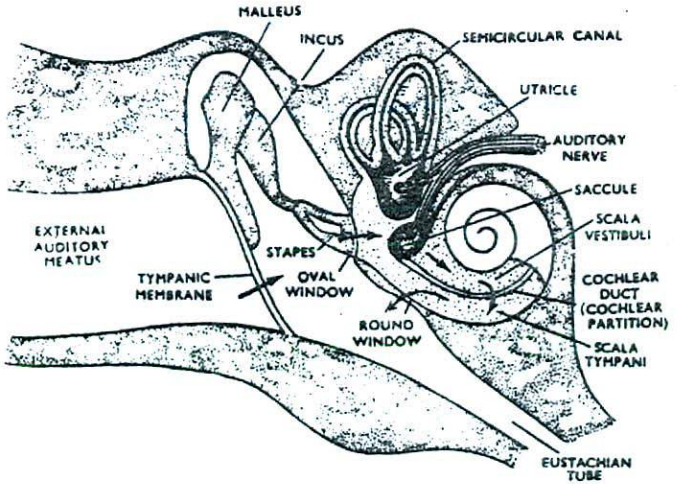


Fig. 6.119. Schematic representation of the link between the inner side of the tympanic membrane to the liquid-filled cochlea at the oval window and also the paths of acoustic transmission.

mechanical **joint and bearing systems** of the single unit of the bony ossicles are such that displacements at the tympanic membrane by air will be three times higher in the oval window. Thus it acts like a **hydraulic press.** The force in the oval window is spread over an area of 3·2 mm^2

but at the tympanic membrane this is spread over 65 mm². Accordingly the pressure changes in the liquid of inner ear just inside the oval window will be greater than that of outside the tympanic membrane.

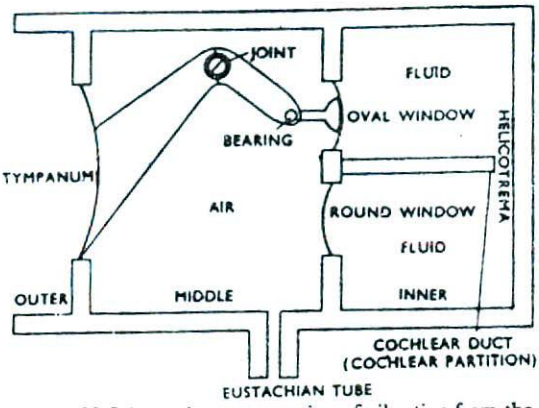


FIG. 6.120 Schematic representation of vibration from the external ear to the internal ear.

The base of the stapes produces rocking rather than push-pull movement. Tensor tympani and stapedius muscles modify the transmission of sounds of different intensities and provide a protective mechanism.

III. **Internal ear.** Two parts: (1) **cochlea**—in front, (2) **vestibular apparatus**—behind (FIG. 6.121).

1. **Cochlea.** It is made up of a bony canal, arranged spirally (two and three-fourth turns) like the shell of a snail. [Hence, the name.] The spirals wind round a central bony pillar, the **modiolus,** through the axis of which passes the auditory nerve. A tape-like bony projection winds round the modiolus like the edges of a screw and makes an incomplete partition. It is completed by the **basilar membrane** extending from

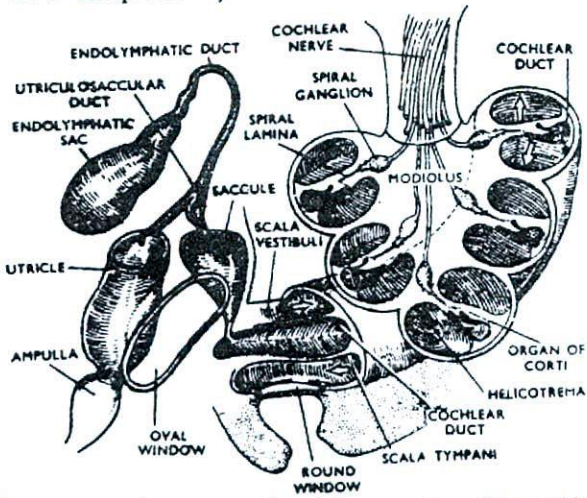


FIG. 6.121. Diagrammatic representation of the connection of the cochlea and the vestibular apparatus and directions of arrows showing transmission of sound waves.

the tip of the spiral lamina to the outer wall of the canal. A second membrane, **Reissner's membrane** stretches from the upper surface of the spiral lamina to the bony wall of the canal a little above the attachment of the basilar membrane.

The triangular fluid-filled space (**space of Nuel**) whose base is covered by fibrous, elastic membrane extending from the inner bony core of the cochlea, the modiolus, to the spiral ligament which lines the outer wall of the cochlear canal, and the other two sides being covered by Reissner's membrane and the **stria vascularis,** is called **cochlear duct** or **cochlear partition** (Fig. 6.122). Though the cochlea is spiral, yet it can be regarded as a long straight tube divided down its length by a *membranous partition*, the

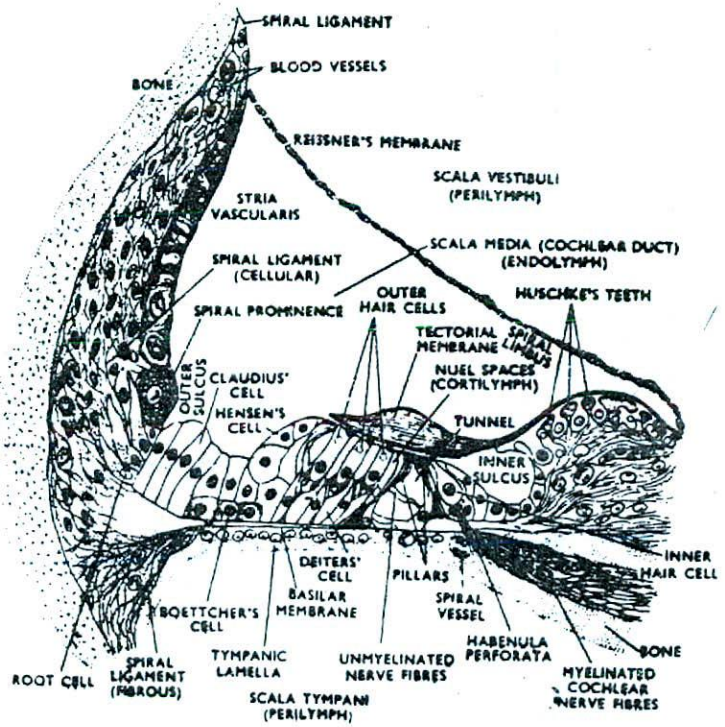


Fig. 6.122. Diagram showing cross-section of cochlear partition of the guinea-pig in the lower part of the second turn (*after Best & Taylor*).

cochlear partition. Thus the bony cochlea is partitioned down its length by the triangular duct, the cochlear duct into two halves—(*i*) scala vestibuli, and (*ii*) scala tympani. This partition is actually containing sense organs situated on the basilar membrane and other *accessory organs* and *endolymph*. The length of the cochlear partition (duct) in man from its origin between oval window and round window to the **helicotrema** is roughly about 35 mm. *The cochlear partition has been regarded as the* **mechanical frequency analyser of the ear.**

Thus the original bony canal is subdivided into three spiral canals. That below the basilar membrane is called the **scala tympani,** that between the two membranes is called the **scala media** (canal of cochlea), and that above Reissner's membrane the **scala vestibuli.** The scala media (membranous cochlea) are filled up with *endolymph*. It ends at the apex in

a blind sac, but at the base it communicates with the vestibular apparatus through the **duct of Hensen** (canalis reuniens). The scala vestibuli and scala tympani are filled up with *perilymph* and communicate with each other at the apex through a small opening, the *helicotrema* (FIG.6.123).

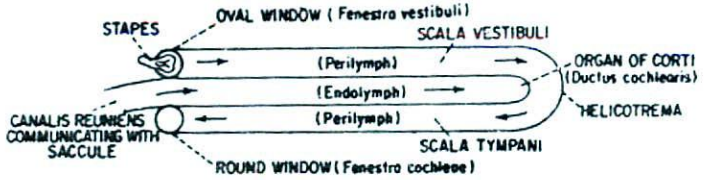


FIG.6.123. Diagrammatic representation of the bony and membranous cochlea, uncoiled. End organ of Corti projecting into the endolymph contained in the membranous cochlea. The perilymph indicated above the endolymph occupies the scala vestibuli. The perilymph in the lower compartment lies in the scala tympani.

On the basilar membrane lies the **organ of Corti** which is the *sense organ for hearing.*

The **basilar membrane** is about 32 mm long. It is composed of fibres that run laterally from the osseous spiral lamina to the spiral ligament. It is narrower at the base (0·17 mm) and broader at the apex (0·47 mm). It is not under tension. It consists of parallel fibres in a gelatinous sheet. The number of parallel fibres in man is about 24,000. The range of fibre length lies between 64–128 μ (basal) and 352–480 μ (apical). These fibres act as resonators. The vibration of higher frequency causes the maximal amplitude of vibration of the basal fibres. The vibration of lower frequency causes the maximal amplitude of vibration of the apical fibres. The vibration of the basilar membrane takes the form of an wave and may be compared with that of arterial pulse wave.

2. **Vestibular apparatus** has been described in pages 205–209–222

Organ of Corti

It is situated on the basilar membrane and consists of **hair cells,** separated into two rows by the **tunnel of Corti** (FIGS.6.124&6.125). Tunnel

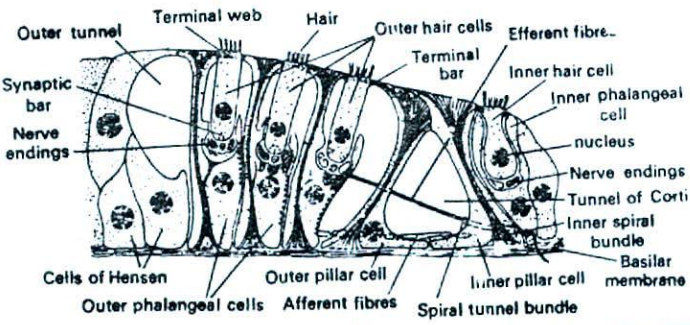


FIG.6.124. Diagrammatic representation of the sensory portion of the organ of Corti under various E/M studies in monkey and man (*after Ham*).

of Corti is formed when the inner rods of Corti and outer rods of Corti unite at the apex. Thus the tunnel of Corti separates the hair cells into a single row of inner hair cells and three or four rows of outer hair cells, each cell

having about twenty tiny hairs. There are about all total 3500 *inner hair cells* and 20,000 *outer hair cells*. Outer hair cells are much more specialised in their structure than inner hair cells. This fact has laid to the concept that outer hair cells are possibly more related with hearing than the inner hair cells.

Inner hair cells resemble mostly type I cells(FIG.5.206)in the vestibular labyrinth. The cells are flask-shaped (FIG. 6.126) and contain numerous

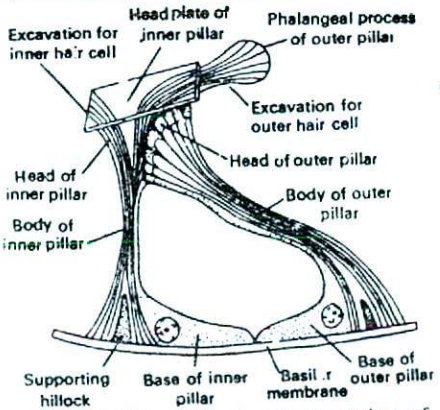


FIG. 6.125. Diagrammatic representation of inner and outer pillars of the organ of Corti (*after Bloom & Fawcett*).

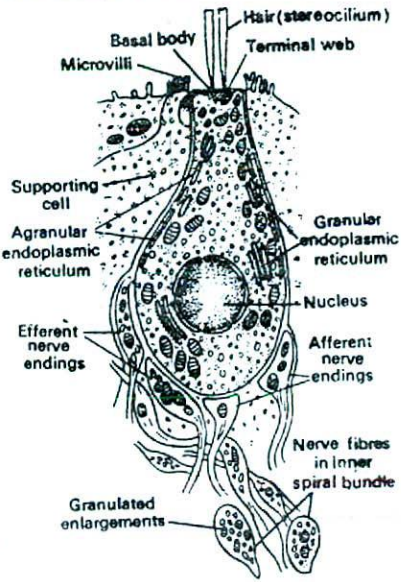


FIG. 6.126. Schematic diagram of an inner hair cell.

mitochondria with no cilia. The nucleus is larger than that of an outer hair cell and situated at the base. The flask-shaped cells are enclosed from its constricted neck inferiorly with a chalice-like nerve terminals. The hairs of the inner hair cells are arranged on the cell apex in the form of a letter W or U with the base of the letter directed towards the centrioles.

Outer hair cells are not flask-shaped and nuclei are mostly situated at the centre. Mitochondria are also aggregated at the base. Unlike inner hair cells only the base of this outer hair cells are enclosed by the afferent nerve endings. No cilium is present and hairs are arranged on the apex of the cells in a more distinct form of a distinctive 'long-horned W' pattern (FIG.6.127). Here there are more rows of hairs and the length of the hairs varies from long at the periphery to the short centrally (FIG.6.128).



FIG. 6.127. Electron microscopic structure of the W configuration of the hairs on the external sensory cells of the human organ of Corti (*after Bloom & Fawcett*).

In between the hair cells lie the **supporting cells of Deiters** (FIG. 6.129), their phalangeal processes project upwards and form part of the **reticular lamina.** The hairs of the hair cells project through the pores of the reticular lamina. Overhanging the hair cells lies the **membrana tectoria.** Membrana tectoria is attached

to the outer border of the *spiral lamina*. It is made up of gelatinous substance. The outer hair cells are surrounded by the **supporting cells of**

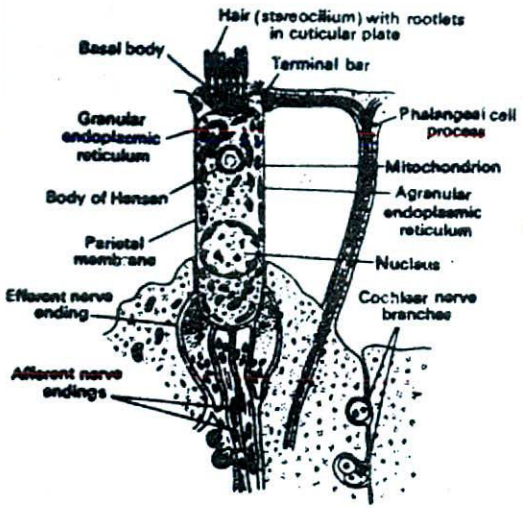


FIG. 6.128. Schematic drawing of an outer hair cell.

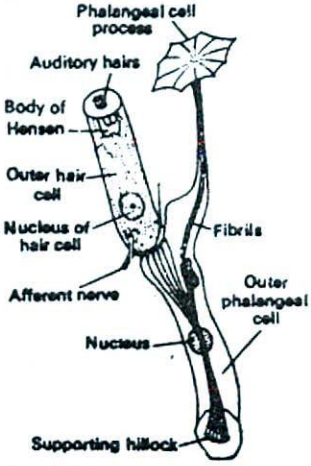


FIG. 6.129. It shows the supporting cells of Deiters and the associated outer hair cells (*after Bloom & Fawcett*).

Hensen. These cells also send off processes which help in the formation of reticular lamina. **Cells of Claudius** remain on the outer side of the Hensen cells. At the base of each hair cells two types of cochlear nerve endings are present—*smaller* and *larger*. The *smaller* nerve endings are *thinner* and *less granulated*. The *larger* ones are *thicker* and *densely granulated*. The cochlear nerve fibres after losing their sheaths enter the organ of Corti. The nerve fibres from the outer hair cells cross the tunnel of Corti and meet the nerve fibres from the inner hair cells and reach the bipolar cells of the **spiral ganglion** situated on the osseous spiral lamina. Auditory impulses arise in the hair cells and are carried by the auditory nerve.

Properties of sound and range of hearing

Sound waves. Sound is the vibration of air molecules, i.e., alternate phases of condensation and rarefaction of the molecules. When these air molecules strike the tympanic membrane sound is heard.

The vibrations imparted to the conducting medium by the sound-producing mechanism are generally spoken of as **sound waves,** which comprise a series of alternate condensations (compressions) and rarefactions propagated in a pendular manner in all directions. In order to get a visual image of the meaning of *condensation* and *rarefaction*, it may be analysed by a vibrating reed, such as a steel rod, one end of which is tightly held in a block, while the other end is vibrating back and forth. As the free end of the rod moves towards the right, it pushes the air molecules in front of it somewhat closer together than normal, creating a compressed region, or **condensation,** in the air. As the rod goes back towards the left, a partial vacuum is created behind, which causes the air molecules to be pulled farther apart than normal. Such a region is known

as a **rarefaction** (FIG. 6.130). When these alternating compressed and rarefied portions of the atmosphere strike the eardrums, they cause them to vibrate at the same rate as the iron rod, and the sound is heard. Thus a sound wave in air consists of an abnormal distribution of air molecules in which compressed regions alternate with regions of partial vacuum. When a normal distribution of air molecules exists, no sound waves are travelling through the air.

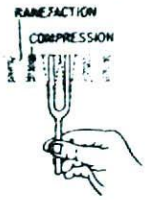


FIG. 6.130. Sound waves as produced by a tuning fork or vibrating rod showing the production of condensations (compressions) and rarefactions.

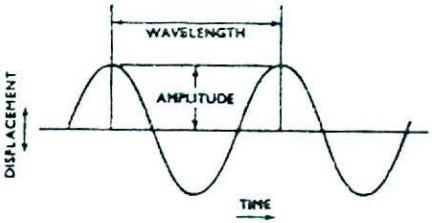


FIG. 6.131. Diagram showing the longitudinal oscillation of air molecules during passage of a sound wave. The loudness of sound is determined by the amplitude of vibration and the frequency of oscillation determines the pitch of the sound.

1. **Properties of sound.** In an electronic device, a sound wave can be seen in the form of a sine wave (FIG. 6.131). **Amplitude** of the waves determines the intensity of the sound and the **pitch** of the sound is determined by the frequency of the waves. Frequency also plays a part in subjective sensation of **loudness** because threshold of sound which the ear can just detect varies with frequency. Sounds of same intensity but of different frequencies, one may be heard and other may not be heard. One may be louder, other may not be.

The amplitude of a sound is generally compared with a standard sound and the relative scale commonly used is the intensity of a sound in **bel which is logarithm of the ratio of the intensities of that sound and a standard sound.**

$$1 \text{ bel} = \log \frac{\text{intensity of sound}}{\text{intensity of standard sound}}$$

The bel is a large unit and so **decibel** (one-tenth of a bel) is generally used. Acoustic Society of America has adopted the standard sound reference level corresponding to 0 decibel at a pressure level of 0·000204 dyne per sq. cm, a value which is just at the auditory threshold for the average human subject.

In the decibel (dB) scale the faintest audible sound is taken as the unit, and called 0. All other sounds are measured as multiples of 0. Briefly the scale is as follows:

	Decibels		*Decibels*
Faintest audible sound ..	0	Pneumatic drill	80
Whisper at 122 cm (4 ft.) ..	20	Boiler shop	100
Quiet street	30	Loud motor-horn at 7 m (23 ft.)	100
Average office	40	Thunder	120
Noisy street	50–80	Limit of endurance ..	130
	Jet engine .. 140 decibels		

It is to be noted that the actual scale is *logarithmic*. For instance, 1 *bel* is 10^1, i.e., 10 times 0, 2 *bels* are 10^2, i.e., 100 times 0, 3 *bels* are 10^3, i.e., 1000 times of 0, and so on.

Quality—same note produced by different instruments varies in quality. Depends on the peculiarity of the waves.

2. **Range of hearing.** Extends over 10–11 octaves. *Limits of audibility* lies from 20–20,000 cycles per second. Frequencies outside this range are not usually audible. But there are wide individual variations. The range is wider in certain animals.

AUDITORY ACUITY

Threshold of audibility. If the intensity of sound is gradually decreased then it reaches a certain value, when there will be no sound. The lowest intensity which will just give a sound sensation is known as *threshold of audibility*. It varies considerably with its frequency. Sound frequencies well above 20,000 cycles per second (c/s or CPS) are audible in dog, but not in man.

If the intensity of sound is increased gradually above the threshold value then the magnitude of sensation is increased and it is not only heard but felt by the ear often with a painful sensation. It is called the **threshold of feeling.**

Difference, or discrimination, threshold. Small differences between the frequencies of two tones can be detected by a normal individual. The minimum perceptible difference is known as **absolute difference threshold.** It is about 3 cycles per second independent of the frequency up to about 500 cycles per second. This will also rise more or less in proportion to the frequency above about 1000 cycles per second. The ability of discrimination between sounds of different frequencies improves with the gradual increase in the intensity of sound. Again the acuity of the ear for differences in sound frequency is higher than its acuity for differences in sound intensity.

Localisation of sound. In man, the power of localisation of sound is poorly developed. But in animals this power is well developed, because the animals can localise the source of sound by moving the pinna towards the sound. In human being, the localisation of sound is higher where the direction of sound is in vertical plane.

In the horizontal direction, this localisation of power is less as the head acts as a shadow. In horizontal direction of sound wave, one ear gets sound more earlier than the other ear. Besides this, the sound of high frequency is felt by one ear facing the sound at higher intensity than the other ear due to shadowing effect of the head.

Auditory fatigue. It is the transient loss of sensitivity due to prolonged hearing of a loud sound. This loss of sensitivity is known as auditory fatigue. The fatigue is binaural because if one ear is subjected to prolonged sound the other is not, then both the ears suffer the loss of sensitivity. If the frequency of sound is 1000 cycles per second or above, then it produces fatigue more intensely than that of frequency below.

Noise. It is described in psychological terms as a sound which causes disturbance or annoyance. If the auditory apparatus is exposed to continued noise, the mechanism of hearing is seriously affected and this may lead to loss of hearing. The degree of loss of hearing depends upon the

frequency value of the noise. After continued exposure to industrial noises the sensitivity of hearing is found to be diminished or lost.

Masking. In a railway train journey unless the voice is louder one cannot hear it. The loudness of the voice can be revealed when the train stops. Weaker sounds may be completely inaudible in presence of louder sounds. This phenomenon is called *masking*. The degree of masking to which one component is masked by the other is measured by finding the threshold. The sounds of almost same frequency cause fluctuations in loudness. Békésy observed that about whole length of the basilar membrane is affected by *low* tones, whereas a limited region is affected by *high* tones and that is why the low tones are more effective in masking

Deafness

The primary **acoustic centre** is in the temporal lobe of the cerebrum. Removal of both temporal lobe is followed by complete deafness and of one temporal lobe is followed by impairment of hearing. This holds that some fibres from each ear cross at some point in their afferent pathways and terminate in the opposite cortex. This is similar to the partial decussation of visual fibres occurring in the optic chiasma.

Deafness may be of two types, viz., (1) **conductive deafness,** and (2) **nerve deafness.**

1. In **conductive deafness** there is interference with the passage of sound waves through the external ear and middle ear. (1) *External ear obstruction*—The conductive deafness occurs due to entrance of foreign bodies, or due to hard or dry wax in the external ear. The damage or perforation of tympanic membrane may be the cause of failure of conduction. (2) *Middle ear disease*—Any condition which prevents the normal functioning of the ossicles. This condition is frequently observed in nasal catarrh, otosclerosis, etc.

2. The **nerve deafness** is due to loss of function of organ of Corti and also due to interference of transmission of impulses by the auditory nerve. The temporary nerve deafness occurs after exposure to a very loud sound. The main causes are: (1) Due to bacterial or viral infection as in Meningitis in children. (2) Due to acoustic trauma as in boiler-makers. (3) Due to toxic action of the drugs, viz., streptomycin, quinine, measles, etc. (4) Due to pressure of a tumour at the junction of cerebellum and pons. The nerve deafness is also found in Ménière's syndrome which occurs in adult and is accompanied by vertigo. This is due to increased hydrostatic pressure in the endolymph. There may be also hereditary nerve deafness.

Test for deafness. RINNE'S TEST—The base of a vibrating tuning fork is placed over the mastoid process of the subject. When the sound fades away (bone-conduction ceases) the prongs of the fork are brought towards the external auditory meatus. If there is no abnormality in the tympanic membrane and ear ossicles, the air-conducted sound is heard for a longer period by the subject and the test is said to be positive. If the sound is heard longer by bone-conduction the test is said to be *negative* and indicates *conductive deafness* (FIG.6.132C). In case of internal ear disease affecting the nervous conduction the test is said to be *positive* and indicates *perceptive deafness* (FIG.6.132B). WEBER'S TEST—The base of a vibrating tuning fork is placed over the midline of the vertex of the subject (FIG. 6.132A). The sound is heard equally in both ears. In unilateral middle

ear disease affecting the conductive system, the sound is better heard in the diseased ear and Weber's test is said to be positive. In case of



FIG. 6.132. A=The Weber test furnishes only a comparison of two ears. If a subject has nerve deafness which is worse in one ear than the other, the tone will be heard best in the better ear. The tone will be heard in the poorer ear if the subject has asymmetric conductive deafness. B=Tone heard longer by air conduction=Rinne positive. C=Tone heard longer by bone conduction=Rinne negative.

unilateral internal ear disease affecting the nervous conduction the sound is better heard in the normal ear.

Rinne's and Weber's tests help to differentiate middle ear or conductive deafness from internal ear or perceptive deafness.

TRANSMISSION OF SOUND WAVE

The ear apparatus conducts sound waves from air to bone and to fluid and ultimately transforms into action potentials in the auditory nerves.

Functions of the external ear and middle ear

The **external ear** in many animals due to its shape and mobility helps in the reception of sound. In human being although the pinna of the external ear is not mobile but it also receives the sound of shorter wave lengths and transmits it into the external auditory meatus. The length and shape of the external auditory meatus not only transmits the sound but also acts as a protective organ. Through the external auditory meatus sound waves fall on the tympanic membrane. Part of the sound energy is reflected by the tympanic membrane and part of the sound energy is transmitted from the tympanic membrane to the ear ossicles. In response to the sound waves on its external surface, the tympanic membrane moves in and out. The membrane seems to function as *resonater* and stops vibrating soon after the cessation of sound waves.

Békésy found that at all frequency of the sound up to 2400 cycles per second (CPS) the central conical part of the tympanic membrane and the handle of the malleus move as a single unit.

When the frequency of the sound is over 2400 cycles per second (CPS), the tympanic membrane vibrates in segments. The **malleus** and **incus** are closely bound together and suspended by elastic ligaments. They move as a single unit. The axis of rotation is through the anterior and lateral processes and transmits the vibrations to the head of the stapes. The foot plate of the stapes shows a rocking movement which is marked at its posterior margin. If the intensity of the low frequency tone rises above a critical level, the foot plate rotates in its long axis. The ossicles thus function as lever system converting mechanically the resonant

the posterior edge of the oval window. The vibrations are transmitted through the oval window to the perilymph in the scala vestibuli (Figs. 6.119 & 6.120).

The muscles of the **middle ear** also play an important role. The muscles contract reflexly to sound. Due to contraction of the tensor tympani the handle of the malleus along with the tympanic membrane is pulled inwards. Due to contraction of the stapedius the foot plate of the stapes is pulled outwards. The contraction of these muscles checks the movement of the ear ossicles and protects the ear against intense sounds. The total effective area of the tympanic membrane is about 13 times greater than the area of the foot plate of the oval window. So due to reduction in area the pressure increases at the oval window. Further the handle of the malleus is longer than the long process of the incus so the pressure exerted at the oval window by the foot plate increases further (17 times greater). Conduction of sound waves from the external ear and the middle ear to the cochlear fluid is regarded as **ossicular conduction.** Sound waves may be conducted in the cochlear fluid through the membrane of round window (secondary tympanic membrane). This type of conduction is known as **air conduction** and is not normally required. Third type of conduction into the fluid of the cochlea is possible through transmission of vibration of the bones of the skull. Tuning fork or vibrating reed applied directly to the skull, considerable conduction may occur.

The movements of the foot plate of the stapes produce series of travelling waves which bring the cochlear duct or partition into oscillation. There are several theories regarding the transmission of sound wave in the cochlea and the mechanism of hearing.

Functions of the cochlea

The *role of the cochlea* in the mechanism of hearing has been investigated by Helmholtz, Rutherford, etc.

Helmholtz by his **resonance theory** suggested that the rods of Corti act as resonators. Later he suggested that fibres of the basilar membrane act like strings of a piano, each one resonating to a particular tone. The longer fibres (apical) respond to lower tones, while the shorter ones (basal) to higher tones (Fig. 6.133). Sound waves transmitted to the internal ear set up vibrations of particular fibres of the basilar membrane and thus stimulate the overlying hair cells.

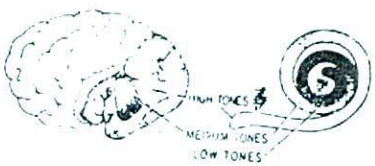


Fig. 6.133. It shows the relationship between the cochlea and acoustic area of the cortex and interpretations of sound in the brain.

Rutherford by his **telephone theory** suggested that cochlea acts like a telephone transmitter. None of these theories could explain the mechanism of frequency analysis of sound.

Later came the **place theory** of pitch discrimination which was widely accepted. According to this theory the cochlea is said to be a tuned structure which helps in the frequency analysis of sound. This theory is based on two facts: (*a*) mechanism of the basilar membrane, and (*b*) electrical potentials in the cochlea. The basilar membrane and the organ of Corti respond to different frequency of sound waves. A particular region of

the basilar membrane resonates to a particular frequency of sound waves. The longer (apical) fibres of the basilar membrane respond to lower tones, while the shorter ones (basal) to higher tones. The hair cells being situated over the basilar membrane, will consequently vibrate to different tones. The hair cells at the apex of the cochlea respond to lower tones while those at the base respond to higher tones. There is bending of the *hairs* due to a shearing motion between the reticular lamina (membrane) and membrana tectoria. When the hair cells are stimulated the nerve endings over their surface are also stimulated and carry the auditory impulse through the auditory nerve.

Wever (1949) proposed the **resonance-volley theory.** He had combined the **resonance theory of Helmholtz** and **telephone theory of Rutherford** so as to explain the response of the whole cochlea to low frequencies by the latter and to explain the cochlear analysis of higher frequencies by the former.

However both principles have got some solid basis as evident from the study of (*a*) mechanics of the cochlear partition, and (*b*) electrical potentials of the cochlea.

I. Mechanics of the cochlear duct (Cochlear partition)

Present-day knowledge of the ear physiology comes mostly from the work of von Békésy who obtained the Nobel prize in 1961. He has proved by studying the physical properties of the cochlear partition, the patterns of movements of the cochlear partition and the basilar membrane as well under microscope through stroboscopic illuminations that the **place theory** has got some scientific basis. The cochlea has been considered as **frequency analyser of sound.** It is a tuned structure and this tuning is partly due to

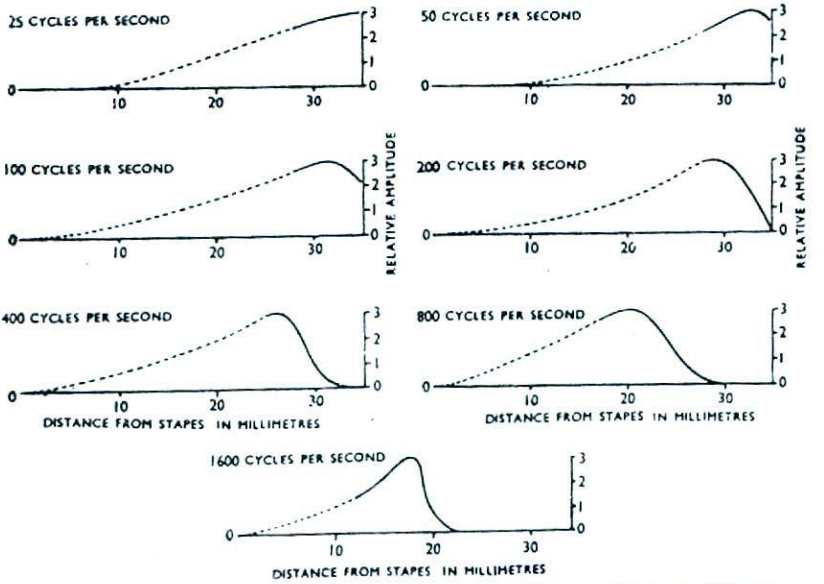


FIG. 6.134. Amplitude of displacement of the cochlear partition at different frequencies (*from Békésy*, 1949).

gradually increasing width of the basilar membrane from the basal end of the cochlea to the helicotrema and partly due to increase in mass of the organ of Corti in the same direction. Beside these, the basilar membrane has graded stiffness, increasing greatest towards the stapes and lowest towards the apex. Basilar membrane can be considered as a gelatinous mass covered by a homogeneous layer of fibres and forming a continuous structure with organ of Corti. The basilar membrane is not under tension. Due to gradation of stiffness of the cochlear partition, the resonant frequency is greatest towards the stapes than in the apical side.

von Békésy by measuring the amplitude and phase of vibration at a number of positions on the cochlear partition has found that each position shows a maximum displacement for a certain frequency of sound. He has observed that with increased frequency, maximum displacement is observed towards the stapes, but with decreased frequency, maximum displacement occurs towards the apex. Fig. 6.134 shows the amplitude of displacement (*envelope of vibration*) of the cochlear partition at different frequencies. High-pitched sounds create waves that reach maximum height near the base and the low-pitched sounds create waves that reach maximum height at the apex.

Travelling waves are akin to pulse wave and are generated because the cochlear partition near the stapes is the stiffest and the waves being transmitted rapidly towards the flexible portions. The cochlear partition near the stapes vibrates in response to all frequencies and the wave moves at increasing amplitude towards the apex reaching its maximum height where the natural frequency of the cochlear partition is the same as the driving frequency. The amplitude declines abruptly beyond this point.

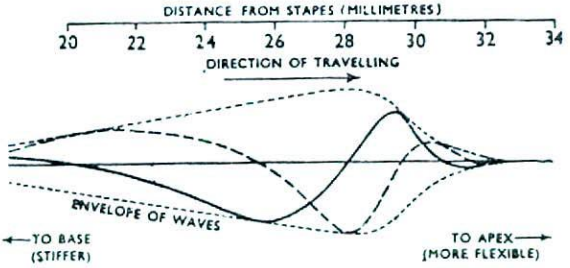


Fig. 6.135. Diagram shows the travelling wave patterns on the basilar membrane (*after Davis*).

Fig. 6.135 shows the travelling waves pattern on the basilar membrane with increasing amplitude as they proceed towards the apex but decline abruptly near the apex.

Thus the movements of the foot plate of the stapes set up a series of travelling waves in the perilymph and the cochlear partition. In the cochlear partition, the whole organ comprising the basilar membrane, organ of Corti, tectorial membrane and usually Reissner's membrane, all move in phase with one another.

As the basilar membrane bulges upward and downward, then the stiff reticular lamina tends to rock on the support of rods of Corti around an axis at the attachment of the basilar membrane to the bony modiolus. The tectorial membrane also begins to swing resulting shearing action

between the tectorial membrane and the reticular lamina. FIG. 6.136 represents that the shearing action between two structures—the tectorial membrane and the reticular lamina, bends the hairs of the hair cells (FIG.6.136B).

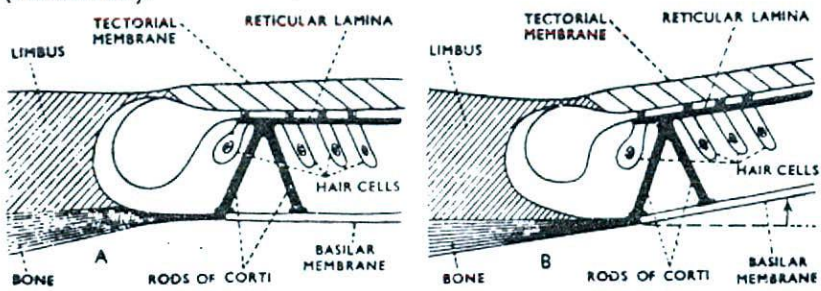


FIG.6.136. Diagram showing the mechanism of stimulation of the hairs of the hair cells through the shearing actions between two stiff structures, the reticular lamina and the tectorial membrane:
A = during rest, B = during activity (*from Davis*, 1957).

Under microscope, von Békésy has examined in detail the nature of movements in different tissues relative to the basilar membrane. FIG. 6.137 represents that on stapes side where the vibration is maximum, the movement of the ends of the hair cells under the tectorial membrane and of the cells of Hensen is in a radial direction. The inner and outer hair cells show vertical vibration at the region of maximum vibration but at the direction of the helicotrema the vibration becomes longitudinal.

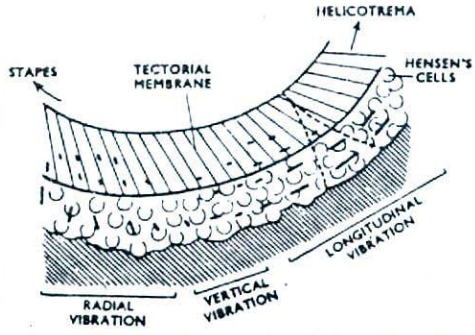


FIG. 6.137. Distribution of radial and longitudinal vibrations along organ of Corti as seen through Reissner's membrane (*from Békésy*).

But how these shearing forces on the hair cells lead to excitation of the fibres of the eighth cranial (VIII) nerve is not known and requires further investigations though electrophysiological studies of the cochlea may throw some light on it.

II. Electrical potentials of the cochlea

Wever and Bray in 1930 proved that the electrical potentials recorded from the auditory nerve reproduced the frequency and the amplitude of the sound waves reaching the ear. The observer's findings supported Rutherford's **telephone theory.** But several electrical potentials of the cochlea have been demonstrated in order to find out the actual mechanism of hearing. These are described below:

A. **Cochlear microphonic potential (CM).** Previously it was known that microphonic potential was the same as action potential in the cochlear nerve fibres. But after proper investigation it has been proved that they are not the same.

Microphonic potential originates in the cochlea and spreads into neighbouring structures. It is produced due to acoustic stimulation. CM is generated by deformation of the processes of the hair cells, and is linearly proportionate to the magnitude of the displacement of the basement membrane. The reproduction of the frequency and amplitude of sound is such that if music is played into an experimental animal's ear then a rendition of the song can be produced correctly by simply feeding the cochlear microphonic into an audio-amplifier. Recent idea is that when the tectorial membrane vibrates and the hair cells bend, there is alteration of the DC endolymphatic potential which helps in the development of the cochlear microphonic potential. It can be recorded by placing one electrode in the scala media and another in the scala tympani. The intensity of the stimulus is directly proportional to the amplitude of the response. It is developed by deformation of the processes of the hair cells and is linearly proportionate to the magnitude of the displacement of the basement membrane. It also increases linearly with increase in the pressure of the applied sound wave to a maximum of 2 mV, and decreases with further increase in sound pressure.

Tasaki (1954), by recording the cochlear microphonic potential at various points in the cochlea of a guinea-pig, has demonstrated (FIG. 6.138) that the basal turn of the cochlea responds mostly to all frequencies of sound stimuli but the third turn (apical turn) of the cochlea gives response only with low frequency and the response is gradually diminished with higher frequencies (FIG. 6.138). Present knowledge available in favour of the theory is that the increase in current flow associated with the cochlear microphonic potential stimulates the terminal non-medullated auditory nerve endings. Increased negativity in the scala media is associated with the current flow outwards across the membrane of the nerve fibres.

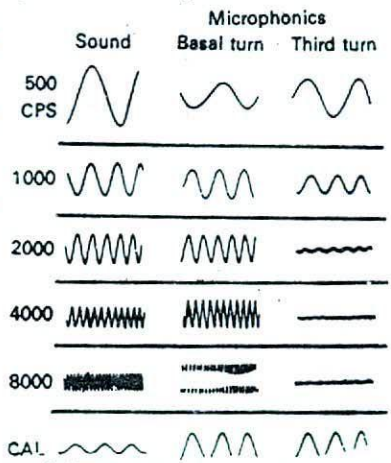


FIG. 6.138. Relation between intensity of sound (left column) and cochlear microphonic responses recorded from basal turn (middle column) and from third turn (right column). Three records at each frequency were photographed simultaneously. Basal turn responds to all frequencies as demanded by travelling wave theory and the amplitude of sound waves need not be altered greatly to give equal responses. There is absence of response to high frequencies at third turn. Calibration (CAL.) at bottom indicates sound pressure (*drawn from Tasaki's*).

Unlike nerve action potential, the CM is highly resistant to drugs, anaesthesia, cold and fatigue. It is not a neural response and can be recorded from a recently killed animal in which neural activity has ceased.

B. **Summating potential (SP).** Two additional potentials—the *negative* and the *positive* summating potentials have been recorded in response to acoustic stimulation. These potentials are the changes in the endolymphatic potentials in response to acoustic or mechanical stimulation. Summating potential is either an increase or a decrease of

endolymphatic potentials. These potentials appear where the cochlear microphonic potential is non-linear. The positive summating potential (SP+) has been thought to originate in the outer hair cells whereas the negative summating potential (SP−) has been regarded to originate from the inner hair cells.

C. **Endolymphatic potential (EP).** Positive electrical potential is recorded from the endolymph in the cochlear duct by introducing a micro-electrode into the cochlea. At rest there is a steady +80 mV potential in the *scala media* with respect to the scala tympani, scala vestibuli or the tissues of the head (Fig.6.139). The endolymphatic potential (endocochlear potential) is not only dependent upon ionic composition, but

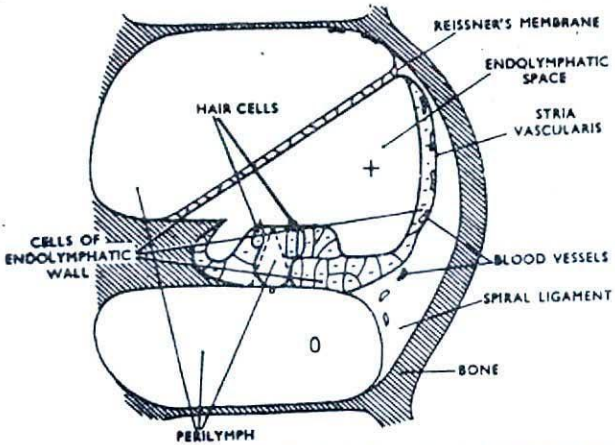


Fig.6.139. Diagram shows the distribution of positive endocochlear potential. It is positive + relative to the perilymph (*after Tasaki et al*).

also upon oxygen supply. In asphyxia it decreases. Metabolic activity of the stria vascularis probably forms the source of the endolymphatic potential. The potential is increased when the basilar membrane moves towards the scala tympani by the inward movement of the stapes. The outward movement of the stapes causes diminution of the potential.

D. **Cortilymphatic potential.** If the micro-electrode is introduced into the organ of Corti, then the −20 mV to −80 mV potentials are recorded in respect to perilymph. This negative potential is known as *cortilymphatic potential.*

E. **Action potential from the auditory nerve.** The cochlea, both mechanically and electrically, behaves like an acoustic analyser. The analysis is coded in terms of all-or-none activity in single auditory nerve fibres and is subsequently transmitted to the CNS. Tasaki (1954) has recorded response from single fibres of auditory nerve to tonal *pips* of different frequencies and intensities. He has shown that the frequency of the action potential, in single auditory nerve fibres is proportionate to the loudness of the sound stimuli. At threshold intensity the nerve endings give response only to a narrow range of frequencies. With the increase of intensities, the nerve endings respond to an increasingly wide range of sound frequencies which lie below that to which they maximally respond. But

there is little if any impulse discharge to frequencies above that of maximal responsiveness. FIG. 6.140 demonstrates the responses of a single auditory nerve fibre to tones of different frequencies.

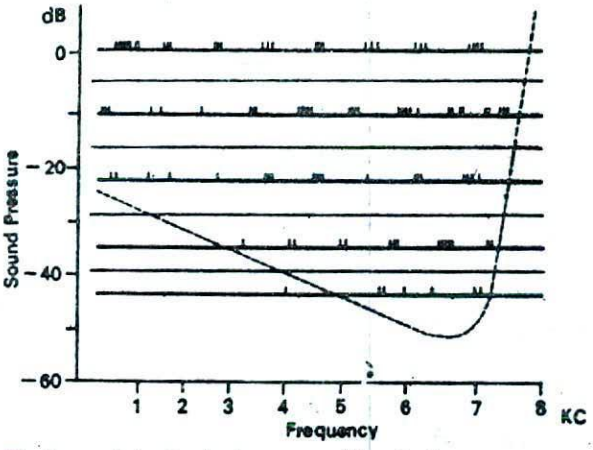


FIG. 6.140. Frequency response range of a single auditory unit becoming narrower as the sound intensity decreased. Sound intensity is expressed in decibels (dB) below an arbitrary standard. Responses (*vertical lines*) of a single auditory fibre to tonal *pips* of different frequencies and intensities. Each row of records shows a short burst of auditory discharge in response to a tone which is increased in frequency between the pips. Different rows of records represent responses obtained at different sound levels. The lowest intensity is demonstrated in the lowermost row. Dotted line shows boundary of 'response area' for this particular fibre (*drawn from Tasaki's*).

Path of auditory impulses

The cells of the *spiral ganglion constitute the first-order neurones* (FIGS. 6.141 & 6.142). Their short dendrites terminate round the hairs cells of the organ of Corti and carry the auditory impulses to the ganglion cells. The axons of these cells constitute the cochlear division of the eighth cranial nerve (*vide* p.5-153). At the lower border of the pons the nerve divides into two parts—one part ends in the **dorsal cochlear nucleus** and the other in the **ventral cochlear nucleus,** lying dorsal and ventral to the restiform body respectively. *From these nuclei the second-order neurones arise,*

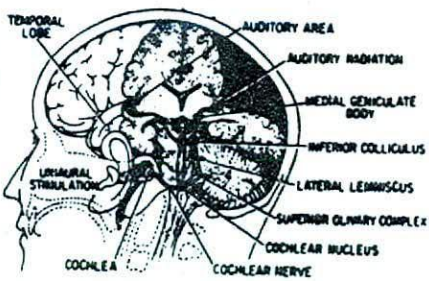


FIG. 6.141. An anatomical representation of auditory pathway. The impulses generated by displacement of the hair cells of the organ of Corti are carried along the corresponding nerve fibres to the auditory area of the cortex where they are resynthesised to produce sound (diagrammatic).

From the ventral nucleus a transverse band of fibres arises, called the **corpus trapezoideum.** Its fibres have three fates: (*a*) A few end round the **superior olivary nucleus** and **trapezoid nucleus** of the same side. (*b*) Most of the fibres cross and join the lateral fillet on the opposite side. (*c*) Some remain uncrossed and join the lateral fillet on the same side.

Fibres from the dorsal nucleus pass along the floor of the fourth ventricle towards the midline forming a conspicuous band of fibres called the **medullary striae** or **acoustic striae.** These fibres also have similar three fates: (*a*) Most of them cross the midline, sharply turn forwards and

join the lateral fillet on the opposite side. (*b*) Some fibres remain uncrossed and enter the lateral fillet on the same side. (*c*) A few fibres terminate in the superior olivary nucleus on the same side.

The **lateral fillet** on each side, therefore, is composed of auditory fibres of both sides. It ascends through the pons and midbrain and ends in many fibres: (1) In inferior colliculus. (2) In the medial geniculate body. [*Vide* page 5-171.] The two medial geniculate bodies remain united by commissural fibres, passing through the optic chiasma and forming *Gudden's commissure* (Fig. 6.101).

The cochlea receives efferent innervation from the olivocochlear bundle. They are often called the efferent inhibitory pathway to the cochlea and play an important role in the regulation of the sensory output from this organ.

From the medial geniculate body the third-order neurones arise and constitute **the auditory radiations.** The fibres pass through the posterior limb of internal capsule (*vide* pp.5-124,5-174) and end in the auditory centre in the

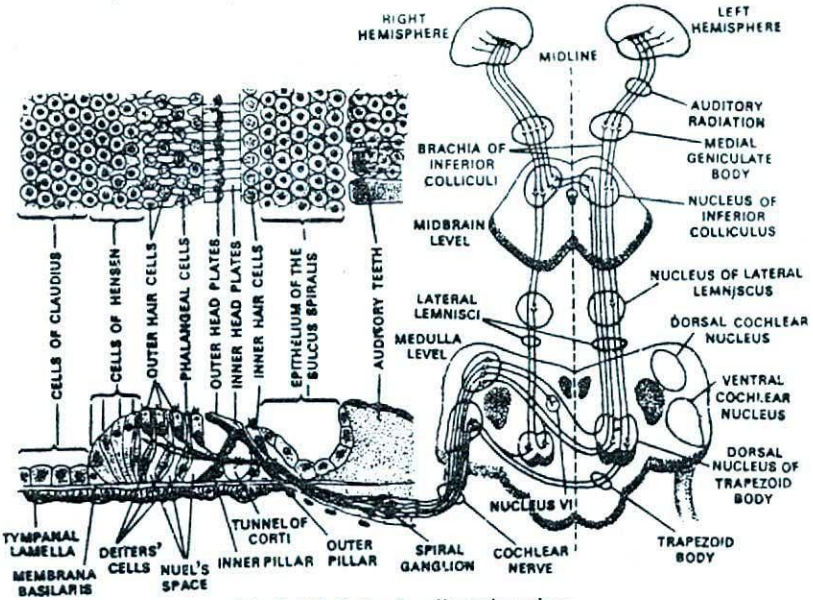


Fig. 6.142. Path of auditory impulses.

temporal lobe. The centre is situated in Hesch'l's gyrus and superior temporal gyrus (areas 41, 42, 22, *vide* pp.5-237-238). There is a regular, point-to-point representation of the basilar membrane, at first in the medial geniculate body and then in the auditory centre (same as with *vision*). The apex of the cochlea is represented in the anterior end; the basal part in the posterior end and the intermediate region in the middle of the centre. The audiopsychic centre remains close to it, occupying the superior, middle and inferior temporal gyri (areas 20, 21, 22) Here, the full meaning of a particular sound is interpreted. The auditory impulses from each ear project to the auditory centres of both cerebral hemispheres. So unilateral removal of one auditory cortex will not produce deafness.