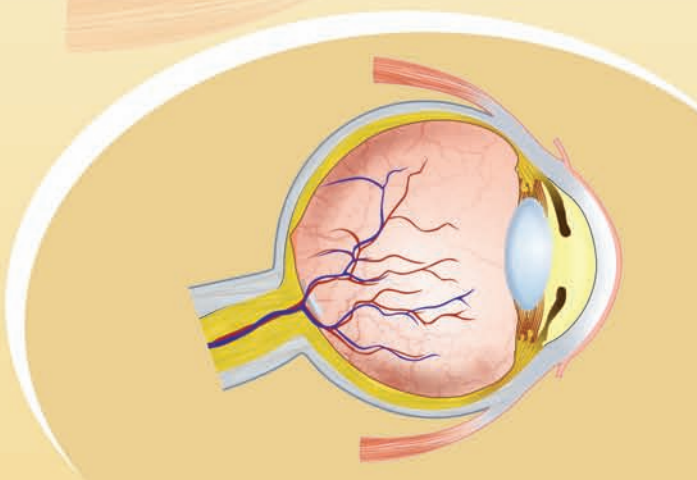
Section 11

Special Senses



Structure of the Eye

Chapter 165

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SPECIAL SENSES

Special senses or special sensations are the complex sensations which involve specialized sense organs. These sensations are different from somatic sensations that arise from skin, muscles, tendons and joints (Chapter 144).

Special senses are:

1. Sensation of vision
2. Sensation of hearing
3. Sensation of taste
4. Sensation of smell.

FUNCTIONAL ANATOMY OF THE EYEBALL

MORPHOLOGY

Human eyeball **(bulbus oculi)** is approximately globe shaped, with a diameter of about 24 mm. It is slightly flattened from above downwards. Eyeball is made up of two segments, an anterior part and a posterior part. Anterior part is small and forms one sixth of the eyeball. Posterior part is larger and forms five sixth of the eyeball. Radius of this part is about 8 mm. Posterior wall of this part is lined by the light-sensitive structure called **retina.**

Center of anterior curvature of the eyeball is called **anterior pole** and the center of posterior curvature is called **posterior pole.** Line joining both the poles is called **optic axis.** The line joining a point in cornea, little medial to anterior pole and fovea centralis, situated lateral to posterior pole is known as **visual axis.** Light rays pass through the visual axis of eyeball (Fig. 165.1). Optic nerve leaves the eye, little medial to posterior pole.

ORBITAL CAVITY

Except anterior one sixth, the eyeball is situated in a bony cavity known as **orbital cavity** or **eye socket.** A thick layer of areolar tissue is interposed between bone and eyeball. It serves as a cushion to protect the eyeball from external force. Eyeballs are attached to orbital cavity by the **ocular muscles.**

EYELIDS

Eyelids protect the eyeball from foreign particles coming in contact with its surface and cutoff the light during sleep. Eyelids are opened and closed voluntarily, as well as by reflex action.

Margins of eyelids have sensitive hair called the **cilia.** Each cilium arises from a follicle, which is surrounded by a sensory nerve plexus. When dust particle comes in contact with cilia, these sensory nerves are activated, resulting in rapid blinking of eyelids. It prevents the dust particles from reaching the eyeball. There are about 100 to 150 cilia in the upper eyelid and about 50 to 75 cilia in the lower eyelid. **Meibomian glands** and some **sebaceous glands** are also found in the eyelids. These glands open into the follicles of cilia. Infection of these glands leads to the development of common **eye sty.**

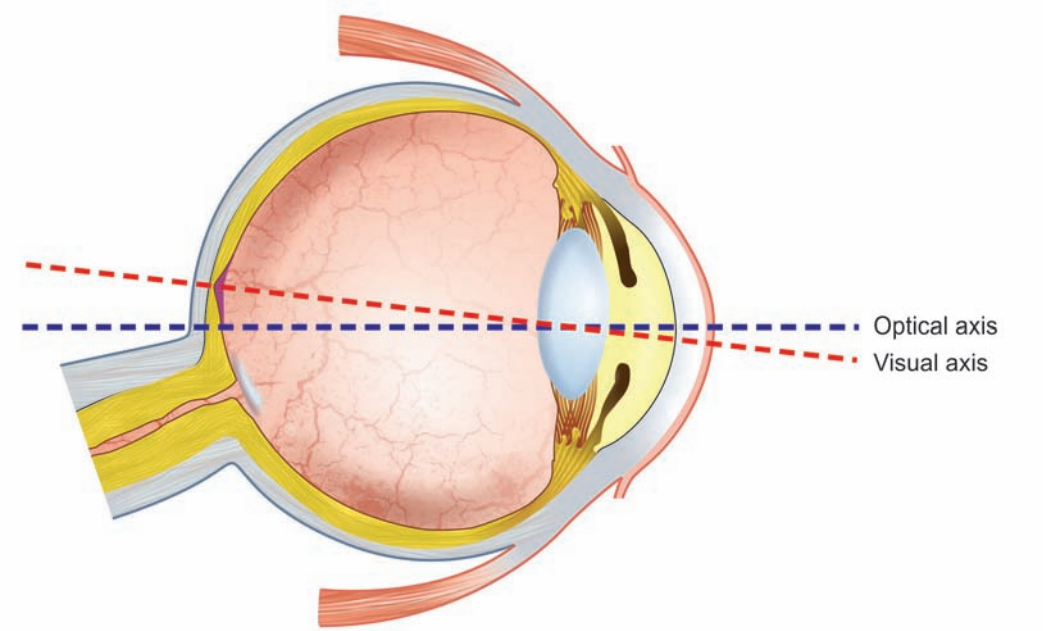

FIGURE 165.1: Optical and visual axis

Opening between the two eyelids is called **palpebral fissure.** In adults, it is about 25 mm long. Its width is about 12 mm to 15 mm, when opened.

CONJUNCTIVA

Conjunctiva is a thin mucus membrane, which covers the exposed part of eye. After covering the anterior surface, conjunctiva is reflected into the inner surfaces of eyelids. Part of conjunctiva covering the eyeball is called **bulbar portion.** Part covering the eyelid is called **palpebral portion.** During closure or opening of eyelids, the opposed portions of conjunctiva slide over each other. Surface of conjunctiva is lubricated by thin film of tear secreted by lacrimal gland.

LACRIMAL GLAND AND TEAR

Lacrimal gland is situated in the shelter of bone, forming upper and outer border of wall of the eye socket. From lacrimal gland, **tear** flows over the surface of conjunctiva and drains into nose via **lacrimal ducts, lacrimal sac** and **nasolacrimal duct.** Tear is a hypertonic fluid. Due to its continuous washing and lubrication, the conjunctiva is kept moist and is protected from infection. Tear also contains **lysozyme** that kills bacteria. Secretion of tears is controlled by the parasympathetic fibers of facial (VII cranial) nerve.

WALL OF THE EYEBALL

Wall of the eyeball is composed of three layers:

A. Outer layer, which includes cornea and sclera
B. Middle layer, which includes choroid, ciliary body and iris
C. Inner layer, the retina.

OUTER LAYER OR TUNICA EXTERNA OR TUNICA FIBROSA

Outer layer preserves the shape of the eyeball. Posterior five sixth of this coat is opaque and it is called the sclera. Anterior one sixth is transparent and is known as cornea.

1. *Sclera*

Sclera is the tough white fibrous outer layer of eyeball, that covers posterior five sixth of the eye. Anteriorly it is continuous with cornea (Fig. 165.2).

Sclera is formed by white fibrous tissues and elastic fibers. Posterior part of sclera, where it is pierced by the optic nerve is thin with perforations. It is named as **lamina cribrosa.**

2. *Cornea*

Cornea is the transparent convex anterior portion of the outer layer of eyeball, which covers the iris and pupil.

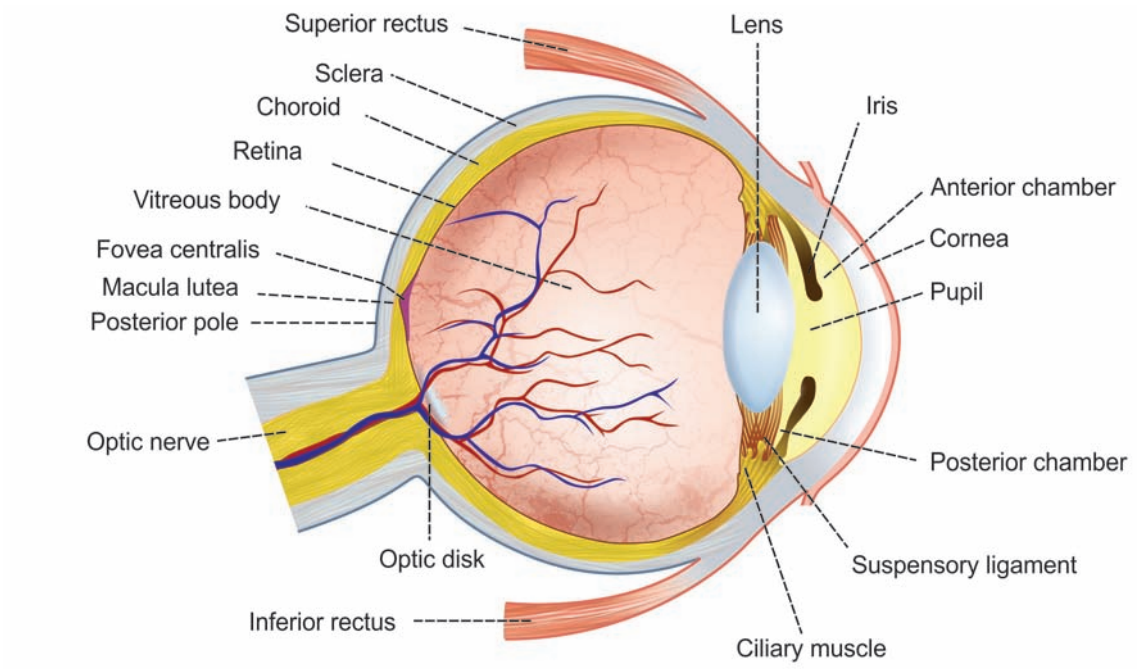

FIGURE 165.2: Structure of eyeball

It forms the anterior one sixth of outer layer and it is continuous with sclera.

Though cornea is transparent, it does not appear transparent. It appears in different colors such as blue, green, brown, grey and black. It is because of the color of iris, which is present just behind the cornea. Sclera overlaps cornea at its periphery and appears in front as white of the eye. Diameter of cornea is about 12 mm horizontally and 11 mm vertically.

Cornea is formed by five layers:

i. Layer of stratified epithelium
ii. Bowman membrane or anterior elastic lamina
iii. Substantia proper
iv. Descemet layer or posterior elastic lamina
v. Layer of endothelial cells.

Cornea has a refractory index of 1.376. It is very sensitive to pain, touch, pressure and cold. Center of cornea is more sensitive to pain because of rich supply of free nerve endings. Normally, cornea is not vascularized. Therefore, it derives its nourishment mainly from aqueous humor. However, in some pathological conditions, cornea becomes vascularized.

The transitional part of outer layer between sclera and cornea is called **limbus.** It is about 1 mm width. Blood vessels are seen only at the limbus. These blood vessels form **superficial marginal plexus** in limbus.

■ MIDDLE LAYER OR TUNICA MEDIA OR TUNICA VASCULOSA

Middle layer surrounds the eyeball completely, except for a small opening in front known as pupil.

This layer comprises of three structures:

1. Choroid
2. Ciliary body
3. Iris.

1. *Choroid*

Choroid is the thin vascular layer of eyeball situated between sclera and retina. It forms posterior five sixth of middle layer. Choroid is extended anteriorly up to the insertion of ciliary muscle (the level of **ora serrata**). Choroid is separated from sclera by perichoroidal space. Anteriorly, this space is limited by the insertion of ciliary muscle into sclera. Posteriorly, this space ends at a short distance from the optic nerve. Inner surface of choroid faces the pigment epithelium (innermost layer) of retina.

Choroid is composed of rich capillary plexus, numerous small arteries and veins.

2. *Ciliary Body*

Ciliary body is the thickened anterior part of middle layer of eye, situated between choroid and iris. It is situated in front of ora serrata. It is in the form of a ring. Its outer surface is separated from sclera by **perichoroidal space.** Inner surface of ciliary body faces the vitreous body and lens. **Suspensory ligaments** from the lens are attached to the ciliary body. Anterior surface of ciliary body faces towards the center of cornea. From the surface, the iris arises (Fig. 165.3).

Ciliary body has three parts:

i. **Orbiculus ciliaris:** It is continuous with choroid and it forms the posterior two third of ciliary body. It is about 4 mm broad.
ii. **Ciliary body proper:** It is made up of two sets of ciliary muscles, namely outer longitudinal and inner circular muscles. Ciliary muscles are innervated by the parasympathetic fibers of oculomotor nerve.
iii. **Ciliary processes:** Ciliary processes are the finger-like projections from inner surface of ciliary body. There are about 70 ciliary processes, projecting towards the central axis of eye to form radial fringes called **corona ciliaris.**

3. *Iris*

Iris is a thin colored curtain-like structure of eyeball, located in front of the lens. It forms a thin circular diaphragm with a circular opening in the center called **pupil.**

Iris is formed by muscles:

i. **Constrictor pupillae** or **iris sphincter** muscle or pupillary constrictor muscle: It is formed by circular muscle fibers. Contraction of this muscle causes constriction of pupil.
ii. **Dilator pupillae** or **pupillary dilator muscle:** It is formed by radial muscle fibers. Contraction of this muscle causes dilatation of pupil.

Activities of these muscles increase or decrease the diameter of pupil and regulate the amount of light entering the eye. Thus, iris acts like the diaphragm of a camera.

Iris separates the space between cornea and lens into two chambers, namely **anterior** and **posterior chambers.** Both the chambers communicate with each other through pupil. Lateral border of anterior chamber is angular in shape. It is called **iris angle** or **angle of anterior chamber.**

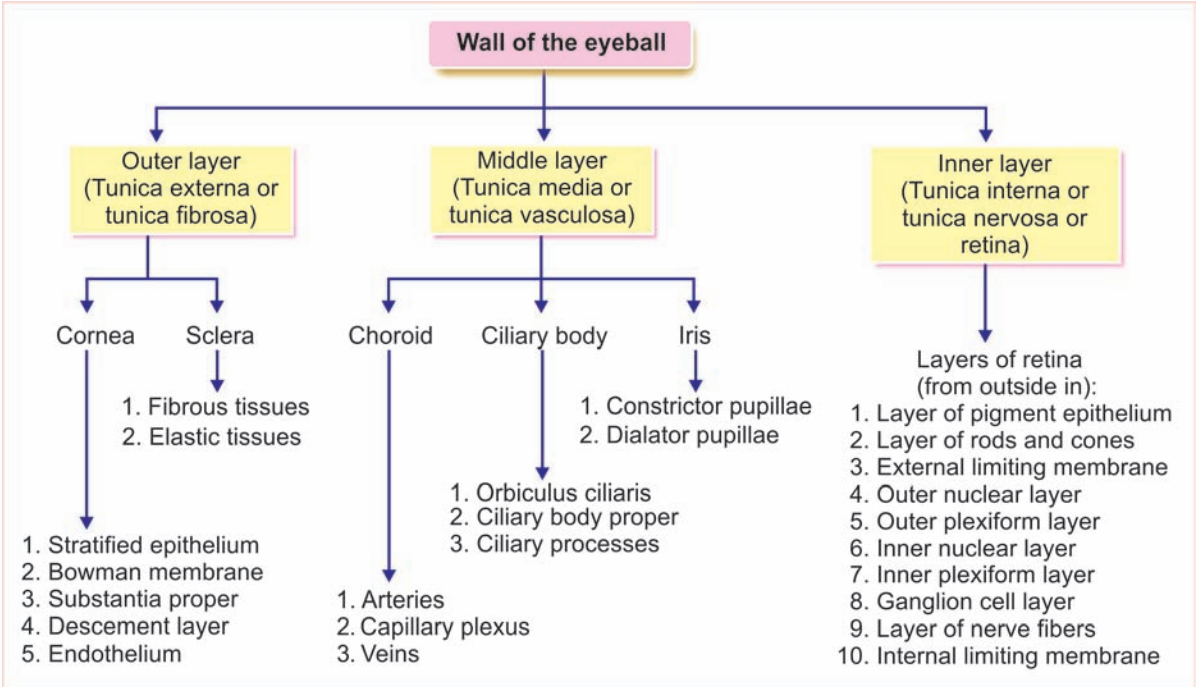

FIGURE 165.3: Wall of the eyeball

■ INNER LAYER OR TUNICA INTERNA OR TUNICA NERVOSA OR RETINA

Retina is a delicate light-sensitive membrane that forms the innermost layer of eyeball. It extends from the margin of **optic disk** to just behind **ciliary body.** Here, it ends abruptly as a dentated border known as ora serrata. Retina has the receptors of vision. Structurally, retina is made up of 10 layers (Fig. 165.4).

Layers of retina from outside in:

1. Layer of pigment epithelium
2. Layer of rods and cones
3. External limiting membrane
4. Outer nuclear layer
5. Outer plexiform layer
6. Inner nuclear layer
7. Inner plexiform layer
8. Ganglion cell layer
9. Layer of nerve fibers
10. Internal limiting membrane.

1. *Layer of Pigment Epithelium*

Layer of pigment epithelium is the outermost layer situated adjacent to choroid. It is a single layer of hexagonal epithelial cells. Outer portion of epithelial cells (towards choroid), contains nucleus and moderate number of round pigment granules. Inner portion has plenty of needle-shaped dark **pigment granules.** Many protoplasmic extensions arise from the inner surface of cells and pass between rods and cones. Cytoplasmic processes also contain dark pigment granules. The pigment present in this layer is a **melanin** called **fuscin.**

Pigment epithelial layer absorbs light and prevents reflection of light rays back from retina. If light rays are reflected back by retina, image becomes blurred. Epithelial cells store **vitamin A** (retinol) and remove the debris from rod cells and cone cells by phagocytic action.

2. *Layer of Rods and Cones*

Layer of rods and cones lies between pigment epithelial layer and external limiting membrane. Rods and cones are the light-sensitive portions of visual receptor cells, namely rod cells and cone cells. Receptor cells are arranged in a parallel fashion and are perpendicular to the inner surface of the eyeball. Structure of rod cell and cone cell is explained in the next chapter.

3. *External Limiting Membrane*

External limiting membrane is a thin layer, formed by the chief supporting elements of retina called the **Müller fibers.**

4. *Outer Nuclear Layer*

Outer nuclear layer is formed by the fibers and granules of rods and cones. **Granules** of rods and cones contain

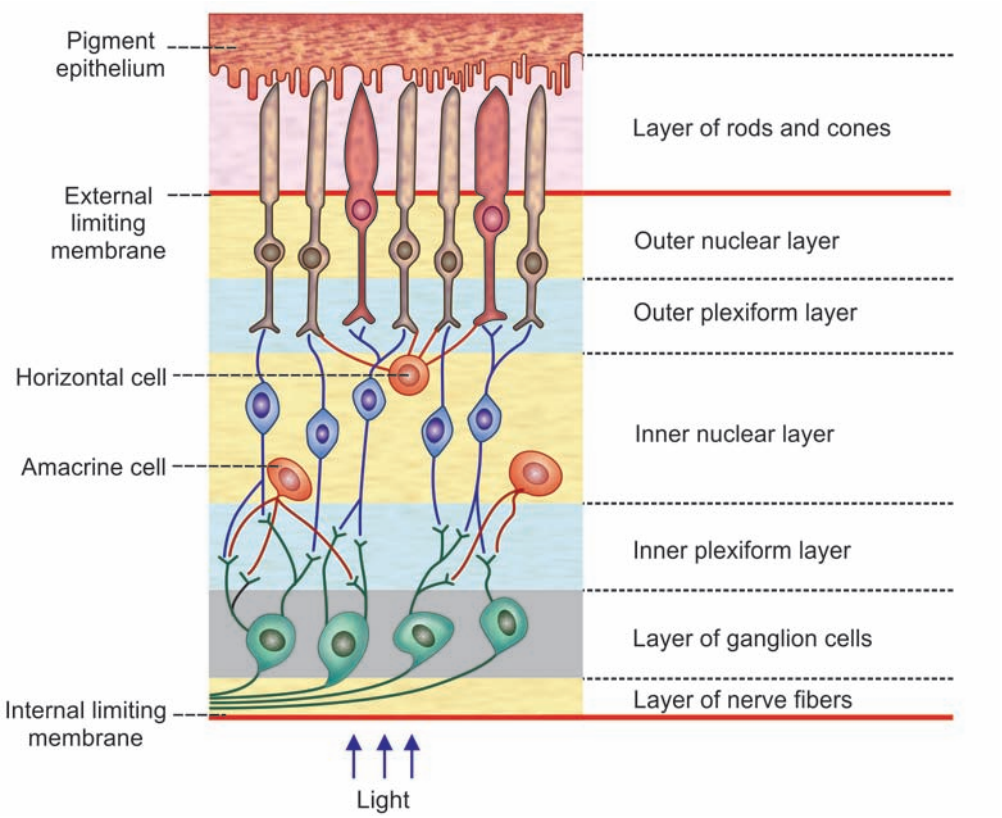

FIGURE 165.4: Layers of retina

nucleus. Nuclei of rods are smaller and round and the nuclei of cones are larger and oval in shape.

5. *Outer Plexiform Layer*

Outer plexiform layer contains reticular meshwork, formed by terminal fibers of rods and cones and dendrites from **bipolar cells,** situated in the inner nuclear layer.

6. *Inner Nuclear Layer*

Inner nuclear layer contains small oval-shaped, flattened **bipolar cells.** Axons of bipolar cells go inside and synapse with dendrites of **ganglionic cells** in the inner plexiform layer. Dendrites synapse with fibers of rods and cones in the **outer plexiform layer.** This layer also contains nuclei of **Müller supporting fibers** and some association neurons called **horizontal cells** and **amacrine** cells.

7. *Inner Plexiform Layer*

Inner plexiform layer of retina consists of synapses between dendrites of ganglionic cells and axons of bipolar cells. It also contains processes from amacrine cells.

8. *Ganglion Cell Layer*

Multipolar cells are present in this layer. Some cells are large and are called **giant ganglion cells.** Other cells are smaller called **midget ganglion cells.** Axons from ganglion cells are in the inner surface of the retina. These axons form the optic nerve. Dendrites of ganglion cells synapse with axons of bipolar cells in the inner plexiform layer. Retinal blood vessels are also present in this layer.

9. *Layer of Nerve Fibers*

Layer of nerve fibers is formed by non-myelinated axons of ganglionic cells. After taking origin, the axons run horizontally to a short distance. Afterwards, the fibers converge towards the **optic disk** and form the **optic nerve.** Neuroglial cells, **Müller cells** and retinal blood vessels are also present in this layer.

10. *Internal Limiting Membrane*

Internal limiting membrane is the innermost layer of retina and it separates retina from the vitreous body. It is a hyaline membrane, formed by the opposition of expanded ends of Müller fibers.

■ FUNDUS OCULI

Fundus oculi is the posterior part of interior eyeball. It is also called **fundus** (Fig. 165.5). In living subjects, fundus is examined by **ophthalmoscope.**

Fundus has two important structures:

1. Optic disk
2. Macula lutea with fovea centralis.

■ OPTIC DISK – BLIND SPOT

Optic disk is a pale disk, situated near the center of the posterior wall of eyeball. It is also called **optic papilla.** It is formed by the convergence of axons from ganglion cells, while forming the optic nerve. Optic disk contains all the layers of retina, except rods and cones. Therefore, it is insensitive to light, i.e. the object is not seen if the image falls upon this area. Because of this, the optic disk is known as blind spot.

■ MACULA LUTEA

Macula lutea is a small yellowish area, situated a little lateral to the optic disk in retina. It is also called **yellow spot.** Yellow color of macula lutea is due to the presence of a yellow pigment. Macula lutea has **fovea centralis** in its center.

Fovea Centralis

Fovea centralis is a minute depression in the center of macula lutea. Here, all the layers of retina are very thin. Diameter of fovea is only about 0.5 mm. Fovea is the region of most acute vision because it contains only cones.

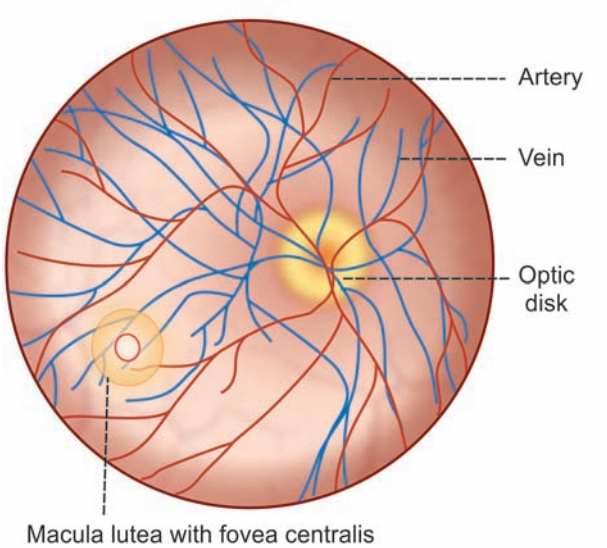

FIGURE 165.5: Fundus oculi

Foveal Vision and Extrafoveal vision

When one looks at an object, eyeballs are directed towards the object, so that, the image of that object falls on fovea of each eye and the person can see the object very clearly. It is known as **foveal vision.**

Vision in other parts of retina is called peripheral or **extrafoveal vision.** It is less sensitive and enables the subject to gain only a dim and an ill-defined impression of surroundings.

Degeneration of macula lutea leads to blindness.

■ INTRAOCULAR FLUID

Intraocular fluid (fluid in eyeball) is responsible for the maintenance of shape of the eyeball.

Intraocular fluid is of two types:

1. Vitreous humor
2. Aqueous humor.

■ VITREOUS HUMOR

Vitreous humor is a viscous fluid present behind lens, in the space between lens and retina. It is also known as vitreous body. It is a highly viscous and gelatinous substance that is formed by a fine fibrillar network of proteoglycan molecules. Major substances in vitreous humor are albumin and hyaluronic acid. These substances enter vitreous body from blood, by means of diffusion.

Vitreous humor helps to maintain the shape of eyeball.

■ AQUEOUS HUMOR

Aqueous humor is a thin fluid present in front of retina. It fills the space between lens and cornea. This space is divided into **anterior and posterior chambers** by iris. Both the chambers communicate with each other through **pupil.**

Properties of Aqueous Humor

Volume	:	0.13 mL
Reaction and pH	:	Alkaline with a pH of 7.5
Viscosity	:	1.029
Refractory index	:	1.34.

Composition of Aqueous Humor

Composition of aqueous humor is given in Figure 165.6.

Formation of Aqueous Humor

Aqueous humor is formed by **ciliary processes.** It is formed from plasma within capillary network of ciliary

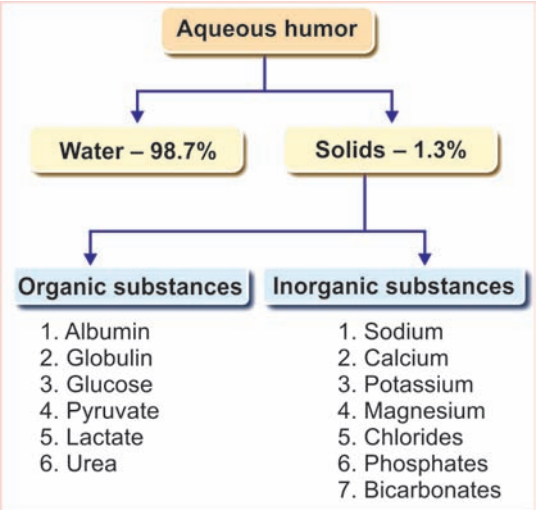

FIGURE 165.6: Composition of aqueous humor

process by diffusion, ultrafiltration and active transport through the epithelial cells lining the ciliary processes. After formation, aqueous humor reaches the posterior chamber by passing through the suspensory ligaments. From here, it reaches the anterior chamber via pupil.

Formation of aqueous humor is a continuous process. Rate of formation is about 2 to 3 µL per minute. Amount of aqueous humor in anterior chamber is about 230 µL to 250 µL and in posterior chamber it is about 50 µL to 60 µL.

Drainage of Aqueous Humor

From anterior chamber, the aqueous humor passes into the angle between cornea and iris called **limbus.** From here, it passes through meshwork of **trabeculae** situated near the junction of iris and cornea. Then it flows through **canal of Schlemm** and reaches the venous system via **anterior ciliary vein** (Fig. 165.7).

Functions of Aqueous Humor

Aqueous humor

i. Maintains the shape of eyeball
ii. Maintains the intraocular pressure
iii. Provides nutrients, oxygen and electrolytes to avascular structures such as lens and cornea
iv. Removes the metabolic end products from lens and cornea.

■ INTRAOCULAR PRESSURE

Intraocular pressure is the measure of fluid pressure in eye, exerted by aqueous humor. Normal intraocular pressure varies between 12 and 20 mm Hg.

Measurement of intraocular pressure is an important part of eye examination. It is measured by **tonometer.** When intraocular pressure increases to about 60 to 70 mm Hg, **glaucoma** occurs. Refer Applied Physiology in this chapter for details.

■ LENS

Lens of the eyeball is **crystalline** in nature. It is situated behind the pupil. It is a biconvex, transparent and elastic structure. It is avascular and receives its nutrition mainly from the aqueous humor.

Lens refracts light rays and helps to focus the image of the objects on retina. Focal length of human lens is 44 mm and its refractory power is 23 D.

Lens is supported by the **suspensory ligaments (zonular fibers),** which are attached with ciliary bodies.

■ STRUCTURE OF THE LENS

Lens is formed of three components:
1. Capsule
2. Anterior epithelium
3. Lens substance.

1. *Capsule*

Capsule is a highly elastic membrane that covers the lens.

2. *Anterior Epithelium*

Anterior epithelium is a single layer of cuboidal epithelial cells, situated beneath the capsule. At the margins, epithelial cells are elongated. Epithelial cells give rise to lens fibers present in the lens substance.

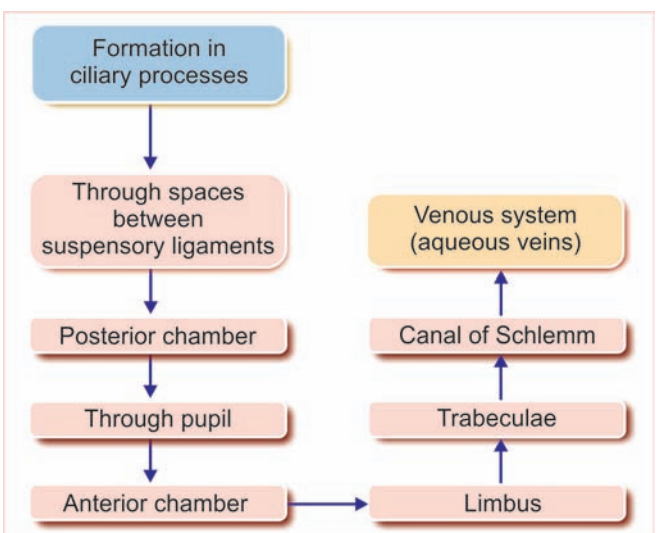

FIGURE 165.7: Circulation and drainage of aqueous humor

3. *Lens Substance*

Lens is formed by long lens fibers derived from anterior epithelium. Lens fibers are prismatic in nature and are arranged in concentric layers.

■ CHANGES IN THE LENS DURING OLD AGE

Elastic property of lens is decreased in old age due to the physical changes in lens and its capsule. It causes **presbyopia** (Chapter 171).

In old age, lens becomes opaque and this condition is called **cataract.** Refer Applied Physiology in this chapter for details.

■ OCULAR MUSCLES

■ MUSCLES OF THE EYEBALL

Muscles of the eyeball are of two types:

A. Intrinsic muscles
B. Extrinsic muscles.

Intrinsic Muscles

Intrinsic muscles are formed by smooth muscle fibers and are controlled by autonomic nerves.

Intrinsic muscles of eye are:

1. Constrictor papillae
2. Dilator papillae
3. Ciliary muscle.

Actions of constrictor pupillae and dilator pupillae are already explained along with iris. Contraction of ciliary muscle increases the anterior curvature of lens during accommodation (Chapter 169).

Extrinsic Muscles

In general, the term 'ocular muscles' refers to extrinsic muscles of the eyeball. Extrinsic muscles are formed by skeletal muscle fibers and are controlled by the somatic nerves. Eyeball moves within the orbit by six extrinsic skeletal muscles (Fig. 165.8). One end of each muscle is attached to the eyeball and the other end to the wall of orbital cavity. There are four straight muscles (rectus) and two oblique muscles.

Extrinsic muscles are:

1. Superior rectus
2. Inferior rectus
3. Medial or internal rectus
4. Lateral or external rectus
5. Superior oblique
6. Inferior oblique.

■ INNERVATION OF OCULAR MUSCLES

Innervation of Intrinsic Muscles

Intrinsic muscles of eyeball are innervated by both sympathetic and parasympathetic divisions of **autonomic nervous system.**

Parasympathetic nerve fibers

Parasympathetic preganglionic fibers arise from **Edinger-Westphal nucleus** of III cranial nerve. After passing through III cranial nerve, these fibers synapse with postganglionic neurons in **ciliary ganglion.** Postganglionic fibers arising from here pass through **ciliary nerves** and innervate the **ciliary muscle** and **constrictor pupillae.** Stimulation of parasympathetic nerve fibers causes contraction of ciliary muscle and constrictor pupillae.

Sympathetic nerve fibers

Sympathetic preganglionic nerve fibers arise from lateral horn of first thoracic segment of spinal cord, pass through **sympathetic chain** and synapse with neurons of **superior cervical sympathetic ganglion.** Postganglionic fibers arising from this ganglion, run along with carotid artery and its branches, to reach the intrinsic muscles of the eyeball. Stimulation of sympathetic nerve fibers causes relaxation of **ciliary muscle** and contraction of **dilator pupillae.**

Innervation of Extrinsic Muscles

Extrinsic muscles of eyeball are innervated by **somatic motor nerve fibers.** Somatic nerve fibers arise from cranial nerve nuclei in brainstem and reach the ocular muscles via three cranial nerves:

1. Oculomotor (third) nerve
2. Trochlear (fourth) nerve
3. Abducent (sixth) nerve.

1. *Oculomotor Nerve*

Oculomotor nerve supplies:

i. Superior rectus
ii. Inferior rectus.
iii. Medial rectus (internal rectus)
iv. Inferior oblique.

2. *Trochlear Nerve*

Trochlear nerve supplies the superior oblique.

3. *Abducent Nerve*

Abducent nerve supplies the lateral rectus (external rectus).

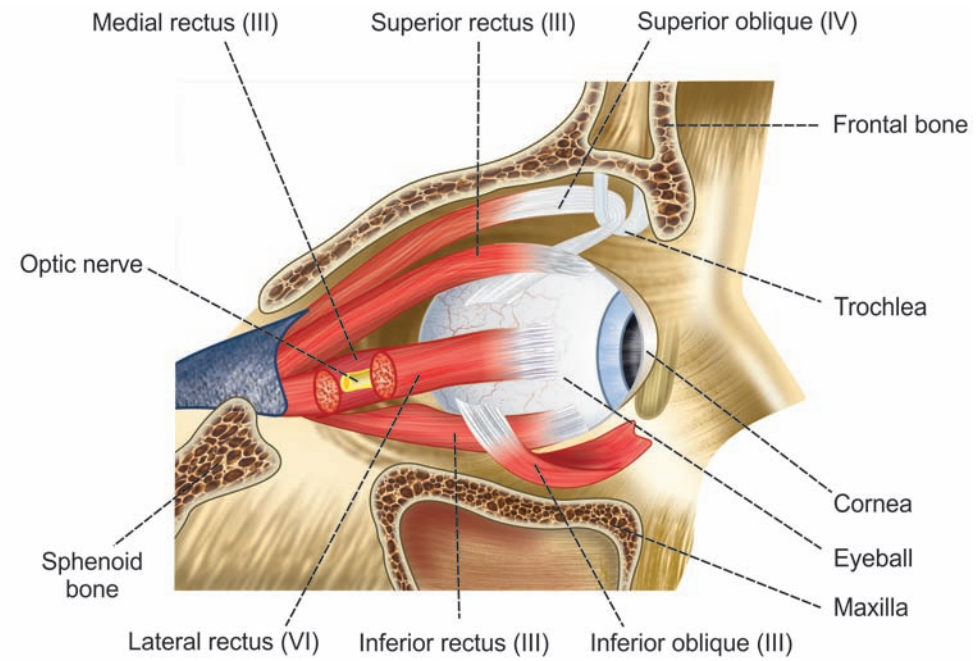

FIGURE 165.8: Extrinsic muscles of eyeball.
Numbers in parenthesis indicate the cranial nerve supplying the muscle.

■ OCULAR MOVEMENTS

Eyeball moves or rotates within the orbital socket in any of the three primary axes, namely vertical, transverse and anteroposterior axis (Fig. 165.9 and Table 165.1).

■ MOVEMENTS IN VERTICAL AXIS

Movements of eyeball in vertical axis or in horizontal plane are of two types:

1. *Abduction or Lateral Movement or Outward Movement*

Abduction of eyeball is due to the contraction of **lateral rectus** mainly. It is supported by the two oblique muscles.

2. *Adduction or Medial Movement or Inward Movement*

Adduction of the eyeball occurs because of the action of **medial or internal rectus,** along with action of superior rectus and inferior rectus.

■ MOVEMENTS IN TRANSVERSE AXIS

Movements of eyeball in transverse axis or in sagittal plane are of two types:

1. *Elevation or Upward Movement*

Elevation of eyeball occurs because of the contraction of **superior rectus** and **inferior oblique muscles.**

2. *Depression or Downward Movement*

Depression of eyeball is brought out by **inferior rectus** and **superior oblique.**

■ MOVEMENTS IN ANTEROPOSTERIOR AXIS

Movements of eyeball in anteroposterior axis or in the frontal plane are called **torsion** or **wheel movements.** Torsion movements are two types, namely extorsion and intorsion.

1. *Extorsion*

During extorsion, the eyeball is rotated in such a way that the cornea turns in upward and outward direction. This movement is due to contraction of **inferior oblique** and **inferior rectus.**

2. *Intorsion*

During intorsion, the eyeball is rotated so that, the cornea moves in downward and inward direction. It is produced by the contraction of **superior oblique** and **superior rectus muscles.**

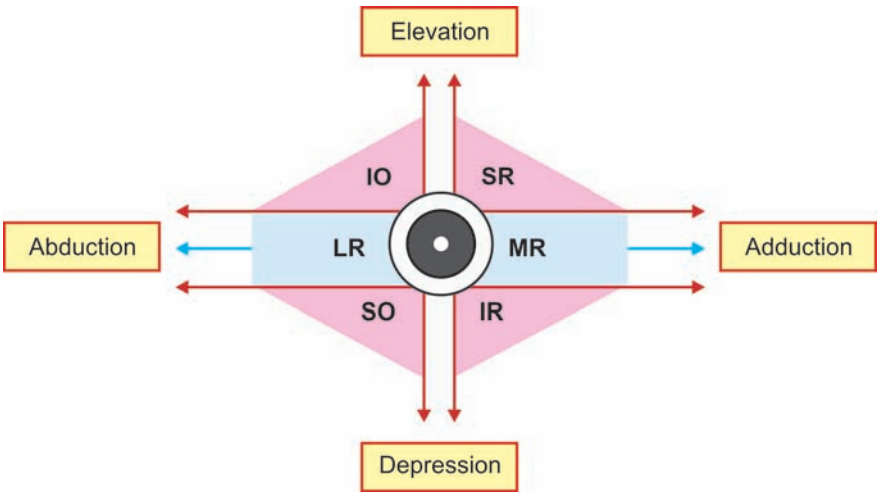

FIGURE 165.9: Diagram showing the movements of right eye. MR = Medial rectus, SO = Superior oblique, LR = Lateral rectus, IO = Inferior oblique, SR = Superior rectus, IR = Inferior rectus.

TABLE 165.1: Muscles taking part in ocular movements

Movement	Primary muscle	Secondary muscle
1. Abduction	Lateral rectus	Superior oblique Inferior oblique
2. Adduction	Medial rectus	Superior rectus Inferior rectus
3. Elevation	Superior rectus	Inferior oblique
4. Depression	Inferior rectus	Superior oblique
5. Extorsion	Inferior oblique	Inferior rectus
6. Intorsion	Superior oblique	Superior rectus

■ SIMULTANEOUS MOVEMENTS OF BOTH EYEBALLS

Simultaneous movements of both eyeballs are of four types:

1. *Conjugate Movement*

Conjugate movement is the movement of both eyeballs in the same direction. Visual axes of both eyes remain parallel. It is due to contraction of **medial rectus** of one eye and **lateral rectus** of the other eye.

2. *Disjugate Movement*

Disjugate movement is the movement of both eyeballs in opposite direction. There are two types of disjugate movement, namely convergence and divergence.

Convergence

Convergence is the movement of both eyeballs towards nose. It is due to simultaneous contraction of medial rectus and simultaneous relaxation of lateral rectus of both eyes. Visual axes move close to each other. Convergence of eyeballs occurs during accommodation.

Divergence

Divergence is the movement of both eyeballs towards temporal side. It is due to the simultaneous contraction of lateral rectus and simultaneous relaxation of medial rectus of both eyes. Visual axes of the eyes move away from each other.

3. *Pursuit Movement*

Pursuit movement is the movement of eyeballs along with object, when eyeballs follow a moving object.

4. *Saccadic Movement*

Saccadic movement is the quick jerky movement of both eyeballs when the fixation of eyes **(gaze)** is shifted from one object to another object. It is also called **optokinetic movement.**

■ APPLIED PHYSIOLOGY

■ GLAUCOMA

Glaucoma is a group of diseases characterized by increased intraocular pressure, which causes damage of optic nerve, resulting in blindness.

In glaucoma, the drainage of aqueous humor through trabeculae is blocked, resulting in increased intraocular pressure. When the intraocular pressure

rises above 60 mm Hg, the optic nerve fibers at the optic disk are compressed. Initially it decreases the visual field (loss of peripheral vision), which eventually leads to total blindness.

However, with early treatment, often the eyes may be protected against serious vision loss. Untreated glaucoma leads to permanent damage of the optic nerve and results in blindness. In old age, glaucoma occurs due to the obstruction of trabeculae by fibrous structures.

Types of Glaucoma

Elevation of intraocular pressure causing glaucoma can occur at any stage of life. Congenital glaucoma develops in babies born with increased intraocular pressure. Glaucoma in infants is called **infantile glaucoma.** When it occurs in childhood, it is known as **juvenile glaucoma.**

Generally glaucoma is divided into two types:

1. Primary open-angle glaucoma
2. Primary angle-closure glaucoma.

1. *Primary open-angle glaucoma (POAG)*

POAG is the most common type of glaucoma and it accounts for about 80% of all cases of glaucoma. The term open-angle refers to drainage system, which is responsible for draining the aqueous humor from the eye. Actually, in POAG there is no visible obstruction in the drainage system. Still intraocular pressure increases, causing damage to optic nerve. Exact cause of POAG is not known yet. It is suggested that a microscopic (minute) blockage in drainage system beyond limbus may obstruct the flow of aqueous humor. It causes a gradual increase in intraocular pressure.

2. *Primary angle-closure glaucoma (PACG)*

PACG is characterized by visible obstruction of drainage system for aqueous humor. Iris is pushed against cornea, preventing the drainage of aqueous humor. Intraocular pressure rises over the period of few hours.

Causes of Glaucoma

Major cause of glaucoma is the blockage in drainage system of aqueous humor in trabeculae, resulting in increased intraocular pressure. Glaucoma also develops secondary to other disorders, which affect the eyes. Common causes of secondary glaucoma are diabetes, inflammation or injury to eye and excess use of drugs such as corticosteroid.

Symptoms of Glaucoma

Primary open-angle glaucoma is a **silent chronic disease** without any early symptoms. Symptoms that develop in later stages include heaviness around eyeball, headache and rapid reduction in visual acuity and visual field.

Early symptoms of angle-closure glaucoma are severe pain in eye or eyebrow, headache, nausea, blurred vision and rainbow halo (colored rings) around bulb light. Immediate care should be taken if two or more of these symptoms appear together.

Treatment for Glaucoma

Treatment does not cure the disease but can prevent further damage of optic nerve. Treatment is aimed at lowering the intraocular pressure. It is achieved by using eye drops or medicines alone or in combination with laser treatment. If intraocular pressure cannot be controlled by these methods, surgery is required.

■ CATARACT

Cataract is the **opacity** or cloudiness in the natural lens of the eye. It is the major cause of blindness worldwide. When lens becomes cloudy, light rays cannot pass through it easily and vision is blurred. Cataract develops in old age after 55 to 60 years.

Lens is situated within the sealed capsule. Old cells die and accumulate within the capsule. Over years, the accumulation of cells is associated with accumulation of fluid and denaturation of proteins in lens fibers, causing cloudiness of lens and blurred image.

Causes of Cataract

In addition to age, cataract develops due to many other causes such as:

1. Eye injuries
2. Previous eye surgery
3. Diseases such as diabetes, Wilson disease and hypocalcemia
4. Long-term use of drugs such as steroids, diuretics and tranquilizers
5. Long-term unprotected exposure to sunlight
6. Alcoholism
7. Family history
8. Diet containing large quantity of salt.

Symptoms of Cataract

Common symptoms of cataract:

1. Glare
2. Painless blurred vision
3. Poor night vision
4. Diplopia in affected eye
5. Need for a bright light while reading
6. Fading of colors.

Treatment for Cataract

Surgery is the only treatment for cataract. During surgery, cloudy lens is removed from the eye through a surgical incision. The natural lens is replaced with a permanent, clear and plastic **intraocular lens** (IOL) implant. Different procedures are followed to remove the cloudy lens.

Common methods are:

1. *Extracapsular extraction*

Extracapsular extraction is rather an old technique. A 12 mm incision is made in the eye under an operating microscope, to remove the lens as a whole. Posterior capsule of lens is left in place to hold the IOL implant. Multiple sutures are required to seal the eye after surgery. Sutures must be perfect; otherwise astigmatism may develop.

2. *Phacoemulsification*

Phacoemulsification (Phaco) is the current technique. **Phaco** is the procedure in which cataract is broken into smaller fragments by **ultrasonic vibrations.** It is done through a small (3 mm) incision. An ultrasound (or laser) probe is used to break the lens material without damaging the capsule. Lens fragments are aspirated out of the eye. A foldable IOL is then introduced through the incision. After entering the eye, the lens unfolds to take position inside the capsule. No sutures are needed, as the incision is self-sealing.

Visual Process

Chapter 166

- INTRODUCTION
- IMAGE FORMING MECHANISM
- NEURAL BASIS OF VISUAL PROCESS
  - STRUCTURE OF ROD CELL
  - STRUCTURE OF CONE CELL
  - FUNCTIONS OF RODS AND CONES
- CHEMICAL BASIS OF VISUAL PROCESS
  - RHODOPSIN
  - PHOTOTRANSDUCTION
  - PHOTOSENSITIVE PIGMENTS IN CONES
  - DARK ADAPTATION
  - LIGHT ADAPTATION
  - NIGHT BLINDNESS
- ELECTRICAL BASIS OF VISUAL PROCESS – ELECTRORETINOGRAM
  - DEFINITION
  - METHOD OF RECORDING ERG
  - WAVES OF ERG
- ACUITY OF VISION
  - DEFINITION
  - TEST FOR VISUAL ACUITY

INTRODUCTION

Visual process is the series of actions that take place during visual perception. During visual process, image of an object seen by the eyes is focused on retina, resulting in production of visual perception of that object.

When the image of an object in environment is focused on retina, the energy in visual spectrum is converted into electrical potentials (impulses) by rods and cones of retina through some chemical reactions. Impulses from rods and cones reach the cerebral cortex through optic nerve and the sensation of vision is produced in cerebral cortex. Thus, process of visual sensation is explained on the basis of image formation and neural, chemical and electrical phenomena.

IMAGE FORMING MECHANISM

While looking at an object, light rays from that object are refracted and brought to a focus upon retina. Image of the object falls on the retina in an inverted position and reversed side to side. Inspite of this, the object is seen in an upright position. It is because of the role played by cerebral cortex.

Light rays are refracted by the lens and cornea. **Refractory power** is measured in **diopter** (D). A diopter is the reciprocal of focal length expressed in meters.

Focal length of cornea is 24 mm and refractory power is 42 D. Focal length of lens is 44 mm and refractory power is 23 D.

NEURAL BASIS OF VISUAL PROCESS

Retina contains the **visual receptors** (Fig. 166.1), which are also called light sensitive receptors, **photoreceptors** or **electromagnetic receptors.** Visual receptors are rods and cones. There are about 6 million cones and 12 million rods in the human eye. Distribution of the rods and cones varies in different areas of retina. Fovea has only cones and no rods. While proceeding from fovea towards the periphery of retina, the rods increase and the cones decrease in number. At the periphery of the retina, only rods are present and cones are absent.

STRUCTURE OF ROD CELL

Rod cells are cylindrical structures with a length of about 40 to 60 µ and a diameter of about 2 µ.

Each rod is composed of four structures:

1. Outer segment
2. Inner segment
3. Cell body
4. Synaptic terminal.

1. *Outer Segment*

Outer segment of rod cell is long and slender. So it gives the rod-like appearance. It is in close contact with the pigmented epithelial cells. Outer segment of rod cell is formed by the modified cilia and it contains a pile of freely floating flat **membranous disks.** There are about 1,000 disks in each rod. Disks in rod cells are closed structures and contain the photosensitive pigment, the **rhodopsin.**

Rhodopsin is synthesized in inner segments and inserted into newly formed membranous disks at the inner portion of outer segment. New disks push the older disks towards outer tip. Older disks are engulfed (by phagocytosis) from tip of the outer segment by cells of pigment epithelial layer. Thus, outer segment of rod cell is constantly renewed by the formation of new disks. Rate of formation of new disks is 3 or 4 per hour.

2. *Inner Segment*

Inner segment is connected to outer segment by means of modified **cilium.** Inner segment contains many types of organelles with large number of mitochondria.

3. *Cell Body*

A slender fiber called rod fiber arises from inner segment of the rod cell and passes to outer nuclear layer through external limiting membrane. In outer nuclear layer, the enlarged portion of this fiber forms the cell body or rod granule that contains the nucleus.

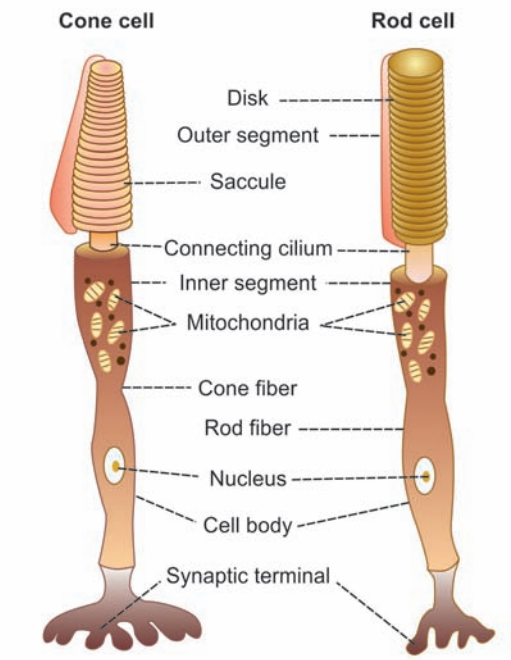

FIGURE 166.1: Structure of visual receptors

4. *Synaptic Terminal*

A thick fiber arising from the cell body passes to outer plexiform layer and ends in a small and enlarged synaptic terminal or body. Synaptic terminal of the rods synapses with dendrites of **bipolar cells** and **horizontal cells.** Synaptic vesicles present in the synaptic terminal contain neurotransmitter, **glutamate.**

STRUCTURE OF CONE CELL

Cone cell is the visual receptor with length of 35 µ to 40 µ and a diameter of about 5 µ. Generally, the cone cell is flask shaped. Shape and length of the cone vary in different parts of the retina. Cones in the fovea are long, narrow and almost similar to rods. Near the periphery of retina, cones are short and broad.

Like rods, cones are also formed by four parts:

1. Outer segment
2. Inner segment
3. Cell body
4. Synaptic terminal.

1. *Outer Segment*

Outer segment is small and conical. It does not contain separate membranous disks as in rods. In cone, the infoldings of cell membrane form **saccules,** which are the counterparts of rod disks.

Photopigment of cone is synthesized in the inner segment and incorporated into the folding of surface membrane forming **saccule.** Renewal of outer segment of cone is a slow process and it differs from that in rods. It occurs at many sites of the outer segment of cone.

2. *Inner Segment*

In cones also, the inner segment is connected to outer segment by a modified cilium as in the case of rods. Though various types of organelles are present in this segment, the number of mitochondria is more.

3. *Cell Body*

Cone fiber arising from inner segment is thick and it enters the inner nuclear layer through external limiting membrane. In the inner nuclear layer, cone fiber forms the cell body or cone granule that possesses nucleus.

4. *Synaptic Terminal*

Fiber from cell body of cone leaves the inner nuclear layer and enters outer flexiform layer. Here, it ends in the form of an enlarged synaptic terminal or body. Synaptic vesicle present in the synaptic terminal of cone cell also possesses the neurotransmitter, glutamate.

■ FUNCTIONS OF RODS AND CONES

Functions of Rods

Rods are very sensitive to light and have a **low threshold.** So, the rods are responsible for **dim light vision** or **night vision** or **scotopic vision.** But, rods do not take part in resolving the details and boundaries of objects (visual acuity) or the color of the objects (color vision). Vision by rod is black, white or in the combination of black and white namely, grey. Therefore, the colored objects appear faded or greyish in twilight.

Functions of Cones

Cones have high threshold for light stimulus. So, the cones are sensitive only to bright light. Therefore, cone cells are called receptors of **bright light vision** or **daylight vision** or **photopic vision.** Cones are also responsible for **acuity of vision** and the **color vision** (Table 166.1).

Achromatic Interval

When an object is placed in front of a person in a dark room, he cannot see any object. When there is slight illumination, the person can see the objects but without color. It is because, at this level, only rods are stimulated. When, the illumination is increased, the threshold for cones is reached. Now, the person can see the objects in finer details and in color. Interval between the threshold for rods and cones, i.e. interval from when an object is first seen and the time when that object is seen with color is called **achromatic interval.**

■ CHEMICAL BASIS OF VISUAL PROCESS

Photosensitive pigments present in rods and cones are concerned with chemical basis of visual process. Chemical reactions involved in these pigments lead to the development of electrical activity in retina and generation of impulses (action potentials), which are transmitted through optic nerve. Photochemical changes in the visual receptor cells are called **Wald visual cycle.**

■ RHODOPSIN

Rhodopsin or visual purple is the photosensitive pigment of rod cells. It is present in membranous disks located in outer segment of rod cells.

Chemistry of Rhodopsin

Rhodopsin is a conjugated protein with a molecular weight of 40,000. It is made up of a protein called **opsin** and a **chromophore.** Opsin present in rhodopsin is known as **scotopsin.** Chromophore is a chemical substance that develops color in the cell. Chromophore present in the rod cells is called **retinal.** Retinal is the aldehyde of **vitamin A** or retinol.

Retinal is derived from food sources and it is not synthesized in the body. It is derived from carotinoid substances like **β-carotene** present in carrots.

Retinal is present in the form of **11-*cis* retinal** known as **retinine 1.** Retinine 1 is present in human eyes. It is different from retinine 2 that is present in the eyes of some animals. Significance of 11-*cis* form of retinal is that, only in this form it combines with scotopsin to synthesize rhodopsin.

Photochemical Changes in Rhodopsin – Wald Visual Cycle

When retina is isolated and examined in dark, the rods appear in red because of rhodopsin. During exposure to light, rhodopsin is bleached and the color becomes yellow. When rhodopsin absorbs the light that falls on retina, it is split into retinine and the protein called **opsin** through various intermediate photochemical reactions (Fig. 166.2).

TABLE 166.1: Rods versus cones

Features	Rods	Cones
Number in each eye	12 million	6 million
Length	40 to 60 µ	35 to 40 µ
Diameter	2 µ	5 µ
Shape	Cylindrical	Flask shaped
Outer segment	Long and slender	Small and conical
Sensitivity to light	More sensitive	Sensitive only to bright light
Threshold	Low	High
Type of vision responsible for	Dim light vision or night vision or scotopic vision	Bright light vision or day light vision or photopic vision
Acuity of vision	Not responsible	Responsible
Color vision	Not responsible	Responsible
Photosensitive pigment	Rhodopsin	Porphyropsin or iodopsin or cyanopsin

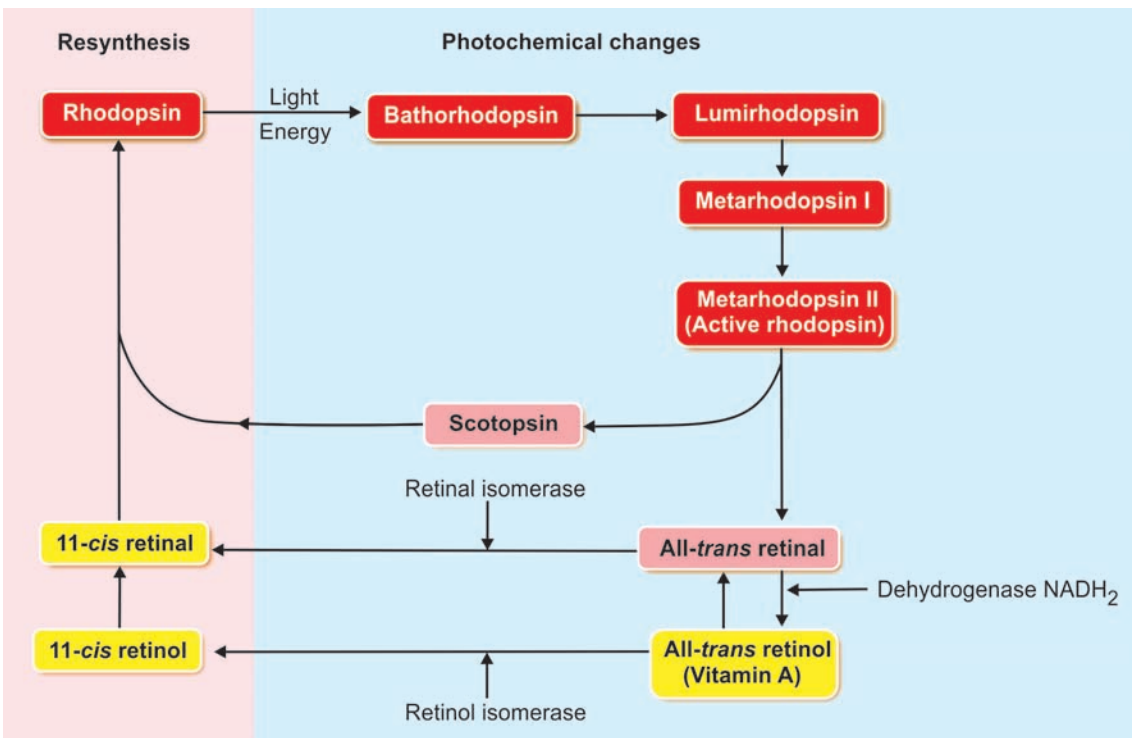

FIGURE 166.2: Photochemical changes and resynthesis of rhodopsin (Wald visual cycle). $NADH_2$ = Reduced nicotinamide adenine dinucleotide.

Following changes occur due to absorption of light energy by rhodopsin:

1. First, **rhodopsin** is decomposed into **bathorhodopsin** that is very unstable
2. Bathorhodopsin is converted into **lumirhodopsin**
3. Lumirhodopsin decays into **metarhodopsin I**
4. Metarhodopsin I is changed to **metarhodopsin II**
5. Metarhodopsin II is split into **scotopsin** and **all-*trans* retinal**
6. All-*trans* retinal is converted into **all-*trans* retinol (vitamin A)** by the enzyme dehydrogenase in the presence of reduced nicotinamide adenine dinucleotide ($NADH_2$).

Metarhodopsin is usually called **activated rhodopsin** since it is responsible for development of receptor potential in rod cells.

Resynthesis of Rhodopsin

First, the all-*trans* retinal derived from metarhodopsin II is converted into 11-*cis* retinal by the enzyme **retinal isomerase.** 11-*cis* retinal immediately combines with scotopsin to form rhodopsin.

All-*trans* retinol (vitamin A) also plays an important role in the resynthesis of rhodopsin. All-*trans* retinol is converted into 11-*cis* retinol by the activity of enzyme retinol isomerase. It is converted into 11-*cis* retinal, which combines with scotopsin to form rhodopsin. All-*trans* retinol is also reconverted into all-*trans* retinal.

Rhodopsin can be synthesized directly from all-*trans* retinol (vitamin A) in the presence of nicotinamide adenine dinucleotide ($NADH_2$). However, the synthesis of rhodopsin from 11-*cis* retinal (retinine) is faster than from 11-*cis* retinol (vitamin A).

■ PHOTOTRANSDUCTION

Visual or phototransduction is the process by which **light energy** is converted into **receptor potential** in visual receptors.

Resting membrane potential in other sensory receptor cells is usually between –70 and –90 mV. However, in the visual receptors during darkness, negativity is reduced and resting membrane potential is about –40 mV. It is because of influx of sodium ions. Normally in dark, sodium ions are pumped out of inner segments of rod cell to ECF. However, these sodium ions leak back into the rod cells through membrane of outer segment and reduce the **electronegativity** inside rod cell (Fig. 166.3). Thus, **sodium influx** maintains a decreased negative potential up to –40 mV. This potential is constant and it is also called **dark current.**

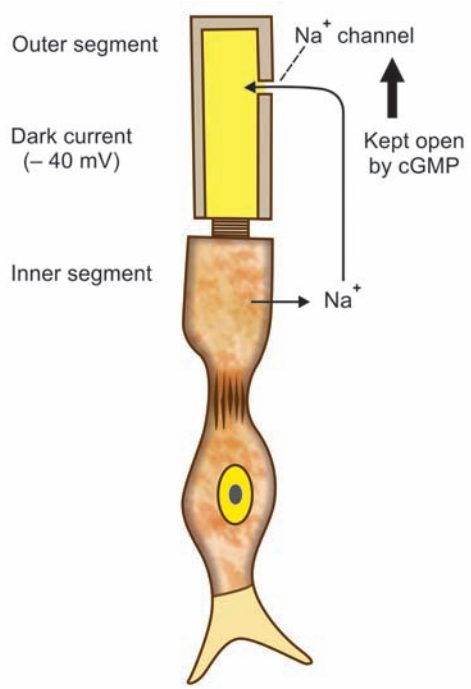

FIGURE 166.3: Maintenance of dark current (resting potential) in outer segment of rod cell

Influx of sodium ions into outer segment of rod cell occurs mainly because of cyclic guanosine monophosphate (cGMP) present in the cytoplasm of cell. The cGMP always keeps the sodium channels opened. Closure of sodium channels occurs due to reduction in cGMP. Concentration of sodium ions inside the rod cell is regulated by sodium potassium pump.

When light falls on retina, rhodopsin is excited leading to development of receptor potential in the rod cells.

Phototransduction Cascade of Receptor Potential

Following is the phototransduction cascade of receptor potential (Fig. 166.4):

1. When a **photon** (the minimum quantum of light energy) is absorbed by rhodopsin, the **11-*cis* retinal** is decomposed into **metarhodopsin** through few reactions mentioned earlier. Metarhodopsin II is considered as the active form of rhodopsin. It plays an important role in the development of receptor potential.
2. Metarhodopsin II activates a **G protein** called **transducin** that is present in rod disks

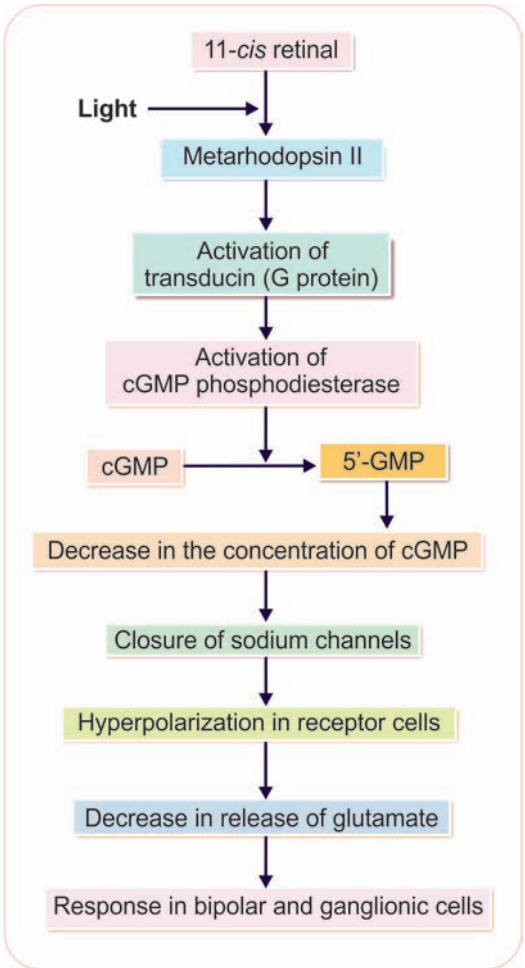

FIGURE 166.4: Phototransduction cascade.
cGMP = Cyclic guanosine monophosphate.

3. Activated transducin activates the enzyme called cyclic guanosine monophosphate phosphodiesterase **(cGMP phosphodiesterase),** which is also present in rod disks
4. Activated cGMP phosphodiesterase hydrolyzes cGMP to 5'-GMP
5. Now, the concentration of cGMP is reduced in rod cell
6. Reduction in concentration of cGMP immediately causes closure of sodium channels in the membrane of visual receptors
7. Sudden closure of sodium channels prevents entry of sodium ions leading to **hyperpolarization.** The potential reaches –70 to –80 mV. It is because of sodium-potassium pump.

Thus, the process of receptor potential in visual receptors is unique in nature. When other sensory receptors are excited, the electrical response is in the form of **depolarization (receptor potential).** But, in visual receptors, the response is in the form of **hyperpolarization.**

Significance of Hyperpolarization

Hyperpolarization in visual receptor cells reduces the release of synaptic transmitter glutamate. It leads to development of response in bipolar cells and ganglionic cells so that, the action potentials are transmitted to cerebral cortex via optic pathway.

■ PHOTOSENSITIVE PIGMENTS IN CONES

Photosensitive pigment in cone cells is of three types, namely **porphyropsin, iodopsin** and **cyanopsin.** Only one of these pigments is present in each cone. Photopigment in cone cell also is a conjugated protein made up of a protein and chromophore. Protein in cone pigment is called **photopsin,** which is different from scotopsin, the protein part of rhodopsin. However, chromophore of cone pigment is the retinal that is present in rhodopsin. Each type of cone pigment is sensitive to a particular light and the maximum response is shown at a particular light and wave-length. Details are given in the Table 166.2.

Various processes involved in phototransduction in cone cells are similar to those in rod cells.

■ DARK ADAPTATION

Definition

Dark adaption is the process by which the person is able to see the objects in dim light. If a person enters a dim-lighted room (darkroom) from a bright-lighted area, he is blind for some time, i.e. he cannot see any object. After sometime his eyes get adapted and he starts seeing the objects slowly. Maximum duration for dark adaptation is about 20 minutes.

Causes for Dark Adaptation

Dark adaptation is due to the following changes in eyeball:

1. *Increased sensitivity of rods as a result of resynthesis of rhodopsin*

Time required for dark adaptation is partly determined by the time for resynthesis of rhodopsin. In bright light, most of the pigment molecules are bleached (broken down). But in dim light, it requires some time for regeneration of certain amount of rhodopsin, which is necessary for optimal rod function.

Dark adaptation occurs in cones also.

2. *Dilatation of pupil*

Dilatation of pupil during dark adaptation allows more and more light to enter the eye.

TABLE 166.2: Sensitivity of cone pigments

Pigment	Sensitive to	Wavelength of maximum response
Porphyropsin	Red	665 nm
Iodopsin	Green	535 nm
Cyanopsin	Blue	445 nm

Radiologists, aircraft pilots and others, who need maximal visual sensitivity in dim light, wear **red glass** before entering dim-lighted area, because red light of spectrum stimulates the rods slightly while the cones are allowed to function well. Thus, the person wearing **red goggles** can see well in bright-lighted area and also can see the objects clearly, as soon as he enters the dim-lighted area.

Dark Adaptation Curve

Dart adaptation curve is the curve that demonstrates the relationship between threshold of light stimulus (illumination) and time spent in dark.

Procedure

Experiment to obtain dark adaptation curve is done in a completely dark room. First, the subject is exposed to a bright light in order to bleach (breakdown) most of the photopigment in retina. The subject looks directly at a bright flashing light with a wavelength of 420 nm against dark background for about 5 to 7 minutes.

Then the bright light is switched off and the subject is in dark. Now a small dim light (stimulus) is produced. Immediately, the absolute threshold (minimum strength of stimulus; minimum intensity of light stimulus) for detecting this dim light is determined by adjusting the intensity of light (illumination). Time interval between the switching off bright light and detection of dim light is noted. After a short time, absolute threshold is measured again and elapsed time is noted. This procedure is repeated for about 30 minutes.

When the experiment is completed, results are plotted and the dark adaptation curve is obtained.

Parts of dark adaptation curve

Dark adaptation curve is **biphasic.** First part of the curve represents threshold of photopic vision, which indicates the cone adaptation. Second part of the curve represents threshold of scotopic vision, which indicates the rod adaptation (Fig. 166.5).

Cone adaptation

This first phase is rapid and it is completed in 8 to 10 minutes. During this period the threshold decreases by 2 to 3 log units. That is the sensitivity of the eye in dark room increases by 1,000 times within 8 to 10 minutes. By this time, the cones get adapted.

Rod-cone break

After the first phase, there is a sudden change in slope of the curve and this point of curve is called rod-cone break. Rod-cone break represents the point where rod sensitivity begins to exceed cone sensitivity and the remaining part of the curve is determined by the continuing adaptation of rods.

During this phase the threshold decreases further by 5 to 6 log units. That is the sensitivity of eye in dark room increases by 100,000 to 1,000,000 times within 20 to 30 minutes. By this time, rods get adapted completely.

■ LIGHT ADAPTATION

Rod adaptation

Second phase of the curve is slow. During this phase, there is a gradual decrease in the threshold and it is completed in 20 to 30 minutes.

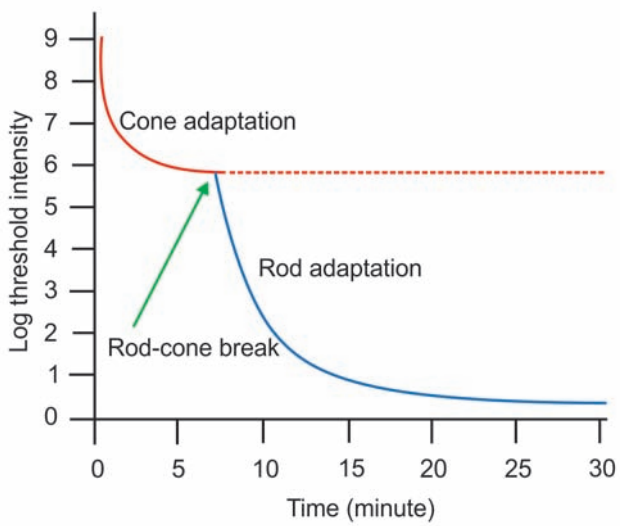

FIGURE 166.5: Dark adaptation curve. During cone adaptation, threshold decreases by 3 logs and sensitivity of the eyes increases 1,000 times. During rod adaptation, threshold decreases by 6 logs and sensitivity of the eyes increases by 1,000,000.

Definition

Light adaptation is the process in which eyes get adapted to increased illumination. When a person enters a bright-lighted area from a dim-lighted area, he feels discomfort due to the dazzling effect of bright light. After some time, when the eyes become adapted to light, he sees the objects around him without any discomfort. It is the mere disappearance of dark adaptation. Maximum period for light adaptation is about 5 minutes.

Causes of Light Adaptation

There are two causes of light adaptation:

1. *Reduced sensitivity of rods*

During light adaptation, the sensitivity of rods decreases. It is due to the breakdown of rhodopsin.

2. *Constriction of pupil*

Constriction of pupil reduces the quantity of light rays entering the eye.

■ NIGHT BLINDNESS

Definition

Night blindness is defined as the loss of vision when light in the environment becomes dim. It is otherwise called **nyctalopia** or **defective dim light** (scotopic) **vision.**

Causes of Night Blindness

Night blindness is due to the deficiency of **vitamin A,** which is essential for the function of rods.

Deficiency of vitamin A occurs because of following causes:

1. Diet containing less amount of vitamin A
2. Decreased absorption of vitamin A from intestine.

Vitamin A deficiency causes defective cone function. Prolonged deficiency leads to anatomical changes in rods and cones and finally the degeneration of other retinal layers occurs. So, retinal function can be restored, only if treatment is given with vitamin A before the visual receptors start degenerating.

■ ELECTRICAL BASIS OF VISUAL PROCESS – ELECTRORETINOGRAM

■ DEFINITION

Electroretinogram (ERG) is the record of electrical activity in retina. When light rays stimulate the retina, a characteristic sequence of potential changes occurs, which can be recorded in the form of ERG. This diagnostic procedure is useful in determining retinal disorders such as **cone dystrophy** (degeneration of cones) and **retinitis pigmentosa** (hyperactivity of the pigmented retinal epithelial cells, leading to damage of photoreceptors and blindness).

■ METHOD OF RECORDING ERG

Electroretinogram is recorded by using a **galvanometer** or a suitable recording device. Recording electrode is placed on the cornea of eye in its usual forward up looking position. Indifferent electrode is placed over any moist surface of body, like inside the mouth.

■ WAVES OF ELECTRORETINOGRAM

Electroretinogram has 4 waves namely 'A', 'B', 'C' and 'D' (Fig. 166.6). 'A' is the only negative wave and other three are positive waves. 'A', 'B' and 'C' waves occur when light stimulus falls on retina. 'D' wave occurs when light stimulus is stopped. 'A' and 'B' waves arise from rods and cones. 'C' wave arises from pigment epithelial layer and 'D' wave arises from inner nuclear layer.

■ ACUITY OF VISION

■ DEFINITION

Acuity of vision is the ability of eye to determine the precise shape and details of the object. It is also called **visual acuity.** Acuity of vision is also defined as the ability

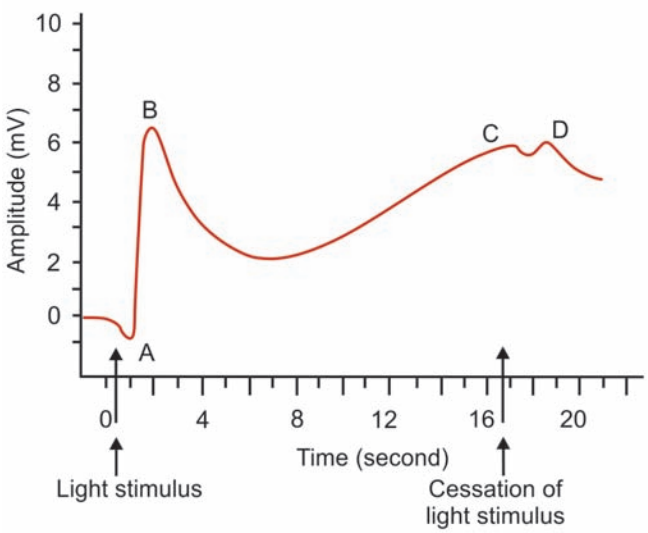

FIGURE 166.6: Electroretinogram

to recognize the separateness of two objects placed together. Cones of retina are responsible for acuity of vision. Visual acuity is highly exhibited in fovea centralis, which contains only cones. It is greatly reduced during the refractory errors.

■ TEST FOR VISUAL ACUITY

Acuity of vision is tested for distant vision as well as near vision. If there is any difficulty in seeing the distant object or the near object, the defect is known as **error of refraction.** Refractive errors are described separately in Chapter 171.

Distant Vision

Snellen chart is used to test the acuity of vision for distant vision in the diagnosis of refractive errors of the eye.

Near Vision

Jaeger chart is used to test the visual acuity for near vision.

Chapter 167

Field of Vision

DEFINITION

Part of the external world seen by one eye, when it is fixed in one direction is called field of vision or visual field of that eye. According to **Traquair**, the visual field is described as **'island of vision,** surrounded by a sea of blindness'.

BINOCULAR AND MONOCULAR VISION

BINOCULAR VISION

Binocular vision is the vision in which both the eyes are used together, so that a portion of external world is seen by the eyes together. In human and some animals, eyeballs are placed in front of the head. So, the visual fields of both the eyes overlap. Because of this, a portion of the external world is seen by both the eyes.

MONOCULAR VISION

Monocular vision is the vision in which each eye is used separately. In some animals like dog, rabbit and horse, the eyeballs are present at the sides of head. So, the visual fields of both eyes overlap to a very small extent. Because of this, different portion of the external world is seen by each eye.

DIVISIONS OF VISUAL FIELD

Visual field of the human eye has an angle of 160° in horizontal meridian and 135° in vertical meridian. Visual filed is divided into four parts:

1. Temporal field
2. Nasal field
3. Upper field
4. Lower field.

TEMPORAL AND NASAL FIELDS

Visual field of each eye is divided into two unequal parts, namely outer or temporal field and the inner or nasal field, by a vertical line passing through the fixation point (Fig. 167.1). The fixation point is the meeting point of visual axis with the object.

Temporal part of visual field extends up to about 100° but the nasal part extends only up to 60°, because it is restricted by nose.

UPPER AND LOWER FIELDS

Visual field of each eye is also divided into an upper field and a lower field by a horizontal line passing through the fixation point. Extent of the upper field is about 60°, as it is restricted by upper eyelid and orbital margin. Extent of lower field is about 75°. It is

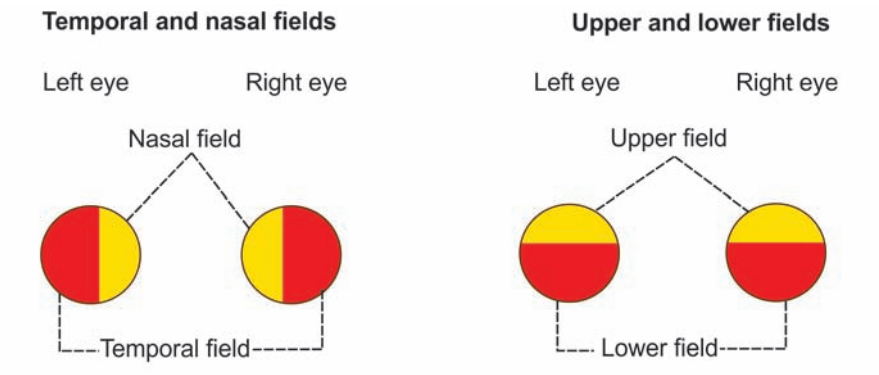

FIGURE 167.1: Divisions of visual field

restricted by cheek. Thus, the visual field is restricted in all the sides, except in the temporal part.

■ CORRESPONDING RETINAL POINTS

Corresponding retinal points are the area in retina of both eyes, on which the light rays from the object falls. It occurs in the binocular vision. The two images developed on retina of both eyes are fused into a single sensation. So, we see the objects with single image.

The single sensation is because of the ocular muscles, which direct the axes of the eyes in such a way that the light rays from the object fall upon the corresponding points of both retinas. If the light rays do not fall on the corresponding retinal points, diplopia occurs.

■ DIPLOPIA

Diplopia means **double vision.** While looking at an object, if the eyeballs are directed in such a way that the light rays from the object do not fall upon the corresponding point on the retina of both eyes, a double vision occurs, i.e. one single object is seen as two.

Causes of Diplopia

1. Permanent diplopia occurs during paralysis or weakness of ocular muscles. It occurs in myasthenia gravis also.
2. In alcoholic intoxication, the imbalanced actions of ocular muscles produce temporary diplopia
3. Lesions in III, IV and VI cranial nerves, oculomotor nucleus, red nucleus and cerebral peduncles also results in diplopia.

Experimental Diplopia

Diplopia can be produced experimentally, by the following methods:

1. Applying pressure from outer side of one eye and thus displacing the eye from its normal position
2. By holding an object like pen or pencil vertically in front of face, at about 5 cm from the root of nose. It is not possible for the convergence of the eyeballs sufficiently. The light rays from the object do not fall on the corresponding retinal points and diplopia occurs.

■ BLIND SPOT

Blind spot is the small area of retina where visual receptors are absent. The optic disk in the retina does not have any visual receptors and if the image of any object falls on the optic disk, the object cannot be seen. So this part of the retina is blind hence the name blind spot.

Normally, the darkness in the visual field due to the blind spot does not cause any inconvenience because, the fixation of each eye is at different angles. Even when one eye is closed or blind, the person is not aware of blind spot. However, one can recognize blind spot by some experimental procedures.

■ VISUAL FIELD AND RETINA

Light rays from different halves of each visual field do not fall on the same halves of the retina. Light rays from temporal part of visual field of an eye fall on the nasal half of retina of that eye. Similarly, the light rays from nasal part of visual field fall on the temporal half of retina of the same side.

■ MAPPING OF VISUAL FIELD

The shape and extent of visual field is mapped out by means of an instrument called **Goldmann perimeter** and this technique is called **perimetry.** Visual field is also determined by **Bjerrum (Tangent)** screen or by **confrontation test. Humphrey field analyzer** is also used to map visual field and it is more useful to test the central portion of visual fields.

Visual Pathway

Chapter 168

INTRODUCTION

Visual pathway or **optic pathway** is the nervous pathway that transmits impulses from retina visual center in cerebral cortex.

In binocular vision, the light rays from temporal (outer) half of visual field fall upon the nasal part of corresponding retina. The rays from nasal (inner) half of visual field fall upon the temporal part of retina.

VISUAL RECEPTORS

Rods and cones which are present in the retina of eye form the visual receptors. Fibers from the visual receptors synapse with dendrites of bipolar cells of inner nuclear layer of the retina.

FIRST ORDER NEURONS

First order neurons (primary neurons) are **bipolar cells** in the retina. Axons from the bipolar cells synapse with dendrites of ganglionic cells.

SECOND ORDER NEURONS

Second order neurons (secondary neurons) are the **ganglionic cells** in ganglionic cell layer of retina. Axons of the ganglionic cells form optic nerve. Optic nerve leaves the eye and terminates in lateral geniculate body.

THIRD ORDER NEURONS

Third order neurons are in the **lateral geniculate body.** Fibers arising from here, reach the visual cortex.

CONNECTIONS OF VISUAL RECEPTORS TO OPTIC NERVE

Two pathways exist between the visual receptors and optic nerve:

1. Private pathway
2. Diffuse pathway.

PRIVATE PATHWAY

The individual cones in fovea centralis are connected to separate bipolar cells. Each bipolar cell is connected to separate ganglionic cell, namely **midget ganglionic cell.** Thus, individual cone is connected to an individual optic nerve fiber. This type of private pathway is responsible for **visual acuity** and **intensity discrimination.**

DIFFUSE PATHWAY

A number of cones and rods are connected with a polysynaptic bipolar cell. The bipolar cells are connected to **diffused ganglionic cells.** So, there is great overlapping. This type of pathway is present outside the fovea.

COURSE OF VISUAL PATHWAY

Visual pathway consists of six components:

1. Optic nerve
2. Optic chiasma
3. Optic tract
4. Lateral geniculate body
5. Optic radiation
6. Visual cortex.

OPTIC NERVE

Optic nerve is formed by the axons of ganglionic cells (Fig. 168.1). Optic nerve leaves the eye through optic disk. The fibers from temporal part of retina are in lateral part of the nerve and carry the impulses from nasal half of visual field of same eye. The fibers from nasal part of retina are in medial part of the nerve and carry the impulses from temporal half of visual field of same eye.

OPTIC CHIASMA

Medial fibers of each optic nerve cross the midline and join the uncrossed lateral fibers of opposite side, to form

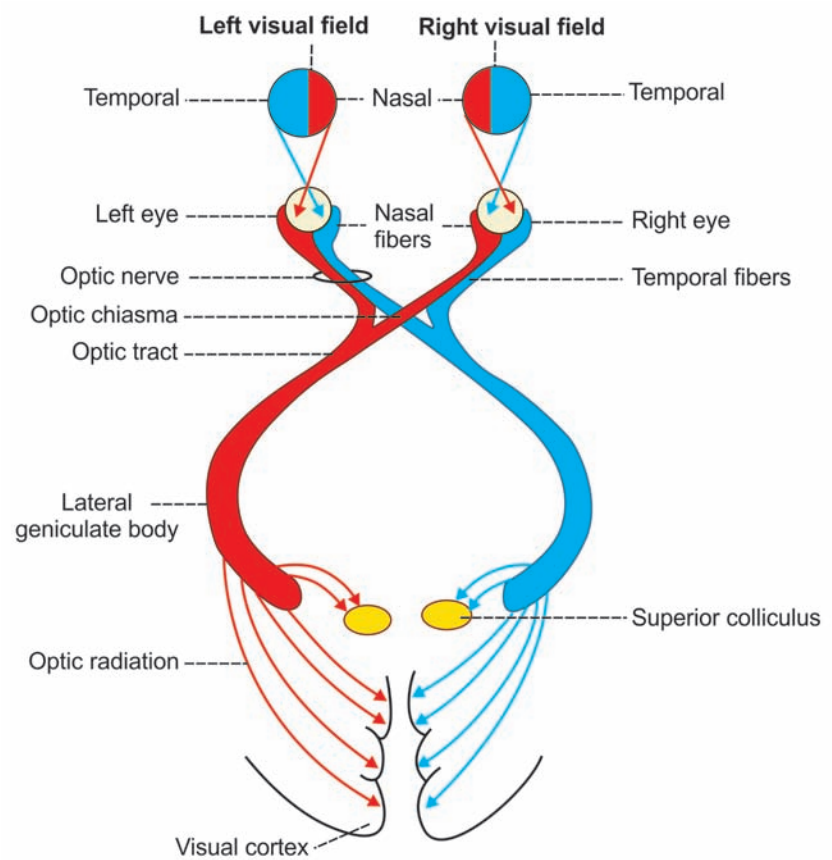

FIGURE 168.1: Visual pathway

the optic tract (Fig. 168.1). This area of crossing of the optic nerve fibers is called optic chiasma.

■ OPTIC TRACT

Optic tract is formed by uncrossed fibers of optic nerve on the same side and crossed fibers of optic nerve from the opposite side. All the fibers of optic tract run backward, outward and towards the **cerebral peduncle.** While reaching the peduncle, the fibers pass between **tuber cinereum** and **anterior perforated substance.** Then, the fibers turn around the peduncle to reach the **lateral geniculate body** in thalamus. Here, many fibers synapse while few fibers just pass through this and run towards superior colliculus in midbrain. Fibers from fovea do not enter **superior colliculus.**

Some fibers from fovea of each side pass through the optic tract of same side and others through the optic tract of opposite side. Due to crossing of medial fibers in optic chiasma, the left optic tract carries impulses from temporal part of left retina and nasal part of right retina, i.e. it is responsible for vision in nasal half of left visual field and temporal half of right visual field. The right optic tract contains fibers from nasal half of left retina and temporal half of right retina. It is responsible for vision in temporal half of left visual field and nasal half of right visual field.

■ LATERAL GENICULATE BODY

Majority of the fibers of optic tract terminate in lateral geniculate body, which forms the **subcortical center** for visual sensation. From here, the **geniculocalcarine tract** or **optic radiation** arises. This tract is the last relay of visual pathway.

Some of the fibers from optic tract do not synapse in lateral geniculate body, but pass through it and terminate in one of the following centers:

i. *Superior colliculus:* It is concerned with reflex movements of eyeballs and head, in response to optic stimulus
ii. *Pretectal nucleus:* It is concerned with light reflexes
iii. *Supraoptic nucleus of hypothalamus:* It is concerned with the retinal control of pituitary in animals. But in human, it does not play any important role.

■ OPTIC RADIATION

Fibers from lateral geniculate body pass through **internal capsule** and form optic radiation. The fibers between lateral geniculate body and visual cortex are also called **geniculocalcarine fibers.** Optic radiation ends in visual cortex (Fig. 168.2).

■ VISUAL CORTEX

Primary **cortical center** for vision is called visual cortex, which is located on the medial surface of occipital lobe. It forms the walls and lips of calcarine fissure in medial surface of occipital lobe.

There is a definite localization of retinal projections upon visual cortex. In fact, the point to point projection of retina upon visual cortex is well established. The peripheral retinal representation occupies the anterior part of visual cortex. **Macular representation** occupies the posterior part of visual cortex near occipital pole.

Areas of Visual Cortex and their Function

Three areas are present in visual cortex:

i. Primary visual area (area 17), which is concerned with the perception of visual impulses
ii. Secondary visual area or visual association area (area 18), which is concerned with the interpretation of visual impulses
iii. Occipital eye field (area 19), which is concerned with the movement of eyes (Chapter 152).

■ APPLIED PHYSIOLOGY – EFFECTS OF LESION AT DIFFERENT LEVELS OF VISUAL PATHWAY

Injury to any part of optic pathway causes visual defect and the nature of defect depends upon the location and extent of injury. Loss of vision in one visual field is known as **anopia.** Loss of vision in one half of visual field is called **hemianopia** (Figs. 168.3 to 168.5).

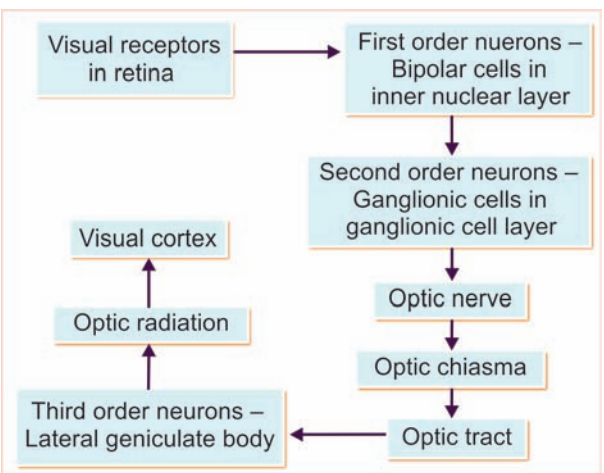

FIGURE 168.2: Representation of visual pathway

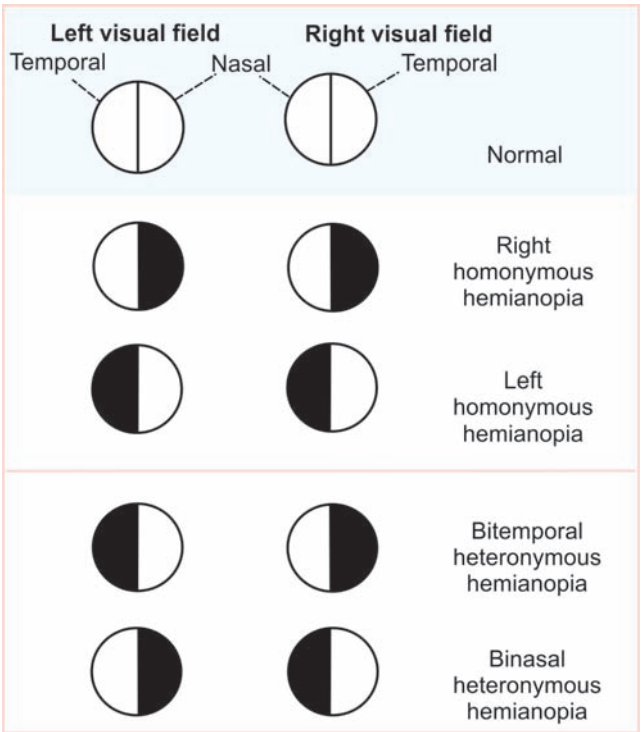

FIGURE 168.3: Types of hemianopia

Hemianopia is classified into two types:

1. Homonymous hemianopia
2. Heteronymous hemianopia.

1. *Homonymous hemianopia*

Homonymous hemianopia means loss of vision in the same halves of both the visual fields. Loss of vision in right half of visual field of both eyes is known as right homonymous hemianopia. Similarly, left homonymous hemianopia means loss of vision in left half of visual field of both eyes.

2. *Heteronymous hemianopia*

Heteronymous hemianopia means loss of vision in opposite halves of visual field. For example, binasal heteronymous hemianopia means loss of vision in right half of left visual field and left half of right visual field (nasal half of both visual fields). Bitemporal heteronymous hemianopia is the loss of sight in left side of left visual field and right side of right visual field (temporal half of both visual fields).

Effects of Lesion of Optic Nerve

Lesion in one optic nerve will cause **total blindness** or anopia in the corresponding visual field. Lesion occurs due to increased **intracranial pressure.**

Effects of Lesion of Optic Chiasma

Nature of defect depends upon the fibers involved:

i. Pressure on uncrossed lateral fibers by aneurysmal dilatation of carotid artery causes blindness in the temporal part of retina of same side, i.e. the retina cannot receive light stimulus from the objects in nasal half of same visual field. So, the hemianopia developed is called **left or right nasal hemianopia.**

ii. If lateral fibers of both sides are affected, the vision is lost in nasal half of both visual fields, causing **binasal hemianopia.** It occurs due to dilated third ventricle, which forces the angle of chiasma against carotid arteries. It also occurs due to dilatation of carotid artery on both sides.

iii. Compression of nasal fibers, i.e. crossed fibers by pituitary tumor causes **bitemporal hemianopia.**

Effects of Lesion of Optic Tract, Lateral Geniculate Body and Optic Radiation

Lesion of optic tract or lateral geniculate body or optic radiation causes **homonymous hemianopia.** In the right-sided lesion, there is loss of vision in right side of both retina, i.e. in left side of both visual fields – left homonymous hemianopia. In the left-sided lesion, there is loss of vision in left half of retina of both eyes and loss of sight on right half of both visual fields – right homonymous hemianopia.

Effects of Lesion of Visual Cortex

Lesion of upper or lower part of visual cortex leads to inferior or superior homonymous hemianopia.

Macular sparing

In all the conditions mentioned above, total blindness does not occur because, the macular vision is not lost. This phenomenon in which the macular vision is retained (unaffected) in conditions of hemianopia is called macular sparing.

Macular sparing occurs because of the following reasons:

i. Fibers from macula project into the visual cortex of both sides

ii. Fibers from macular region are projected into both anterior and posterior parts of each visual cortex.

Only the bilateral lesion of visual cortex causes total blindness.

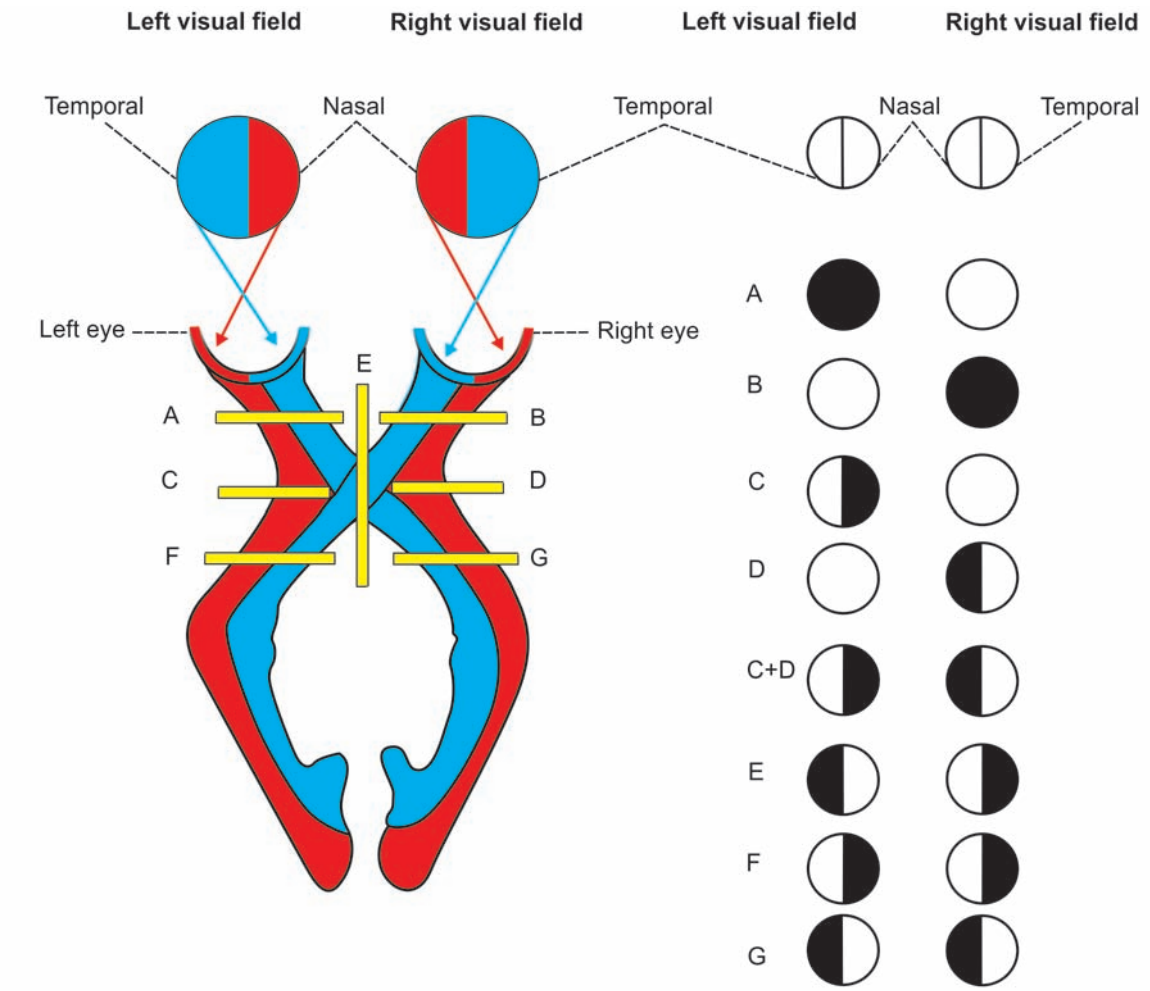

FIGURE 168.4: Effects of lesions of optic pathway. Dark shade in circles indicates blindness.
A. Lesion of left optic nerve: Total blindness of left eye
B. Lesion of right optic nerve: Total blindness of right eye
C. Lesion of lateral fibers in left side of optic chiasma: Left nasal hemianopia
D. Lesion of lateral fibers in right side of optic chiasma: Right nasal hemianopia
C + D. Lesion of lateral fibers in both sides of optic chiasma: Binasal hemianopia
E. Lesion of medial fibers in optic chiasma: Bitemporal hemianopia
F. Lesion of left optic radiation: Right homonymous hemianopia
G. Lesion of right optic radiation: Left homonymous hemianopia.

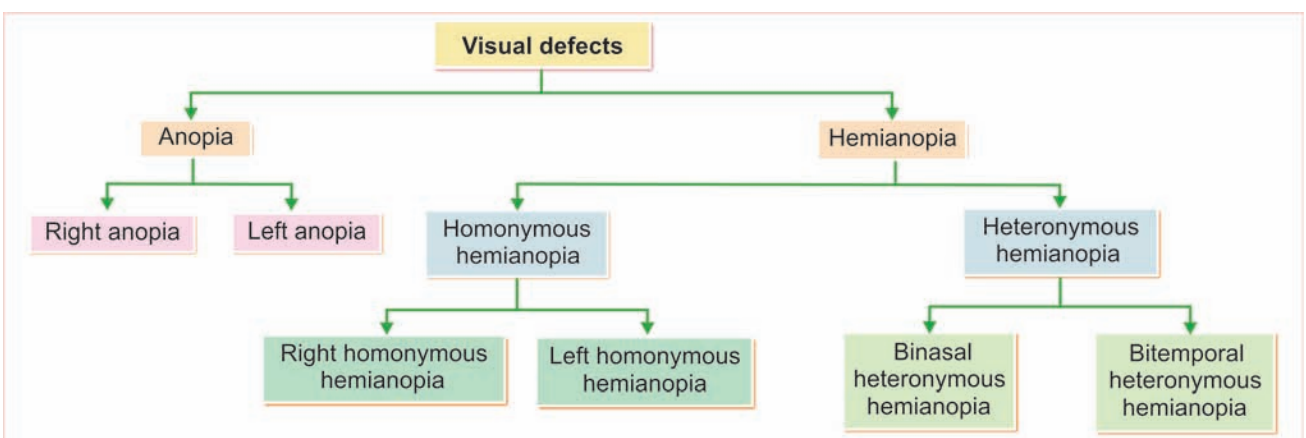

FIGURE 168.5: Visual defects

Pupillary Reflexes

Chapter 169

INTRODUCTION

Pupillary reflexes are the **visceral reflexes,** which alter the size of pupil. Pupillary reflexes are classified into three types:

1. Light reflex
2. Ciliospinal reflex
3. Accommodation reflex.

LIGHT REFLEX

Light reflex is the reflex in which pupil constricts when light is flashed into the eyes. It is also called **pupillary light reflex.** Light reflex is of two types:

1. Direct light reflex
2. Indirect light reflex.

DIRECT LIGHT REFLEX

Direct light reflex is the reflex in which there is constriction of pupil in an eye when light is thrown into that eye. It is also called the **direct pupillary light reflex** or the direct reaction to light.

INDIRECT LIGHT REFLEX

Indirect light reflex is the reflex that involves constriction of pupil in both eyes when light is thrown into one eye. If light is flashed into one eye, the constriction of pupil occurs in the opposite eye, even though no light rays falls on that eye. It is otherwise called **consensual light reflex.**

PATHWAY FOR LIGHT REFLEX

Pathway for light reflex is slightly deviated from visual pathway. Fibers of light reflex pathway and the fibers of visual pathway are the same up to optic tract. Beyond that, these two sets of fibers are separated.

When light falls on the eye, the **visual receptors** are stimulated. Afferent (sensory) impulses from the receptors pass through the **optic nerve, optic chiasma** and **optic tract.** At the midbrain level, few fibers get

separated from optic tract and synapse on the neurons of **pretectal nucleus,** which lies close to the superior colliculus. Pretectal nucleus of midbrain forms the **center for light reflexes.** Efferent (motor) impulses from this nucleus are carried by short fibers to **Edinger-Westphal nucleus** (parasympathetic nucleus) of **oculomotor nerve** (third cranial nerve). From Edinger-Westphal nucleus, preganglionic fibers pass through oculomotor nerve and reach the **ciliary ganglion.** Postganglionic fibers arising from ciliary ganglion pass through short ciliary nerves and reach the eyeball. These fibers cause contraction of constrictor pupillae muscle of iris (Fig. 169.1).

Reason for consensual light reflex is that, some of the fibers from pretectal nucleus of one side cross to the opposite side and end on opposite Edinger-Westphal nucleus.

■ CILIOSPINAL REFLEX

Ciliospinal reflex is the dilatation of pupil in eyes caused by painful stimulation of skin over the neck. It is due to the contraction of dilator pupillae muscle. Sensory impulses pass through cutaneous afferent nerve. The center is in first thoracic spinal segment. Efferent impulses pass through sympathetic fibers and reach dilator pupillae.

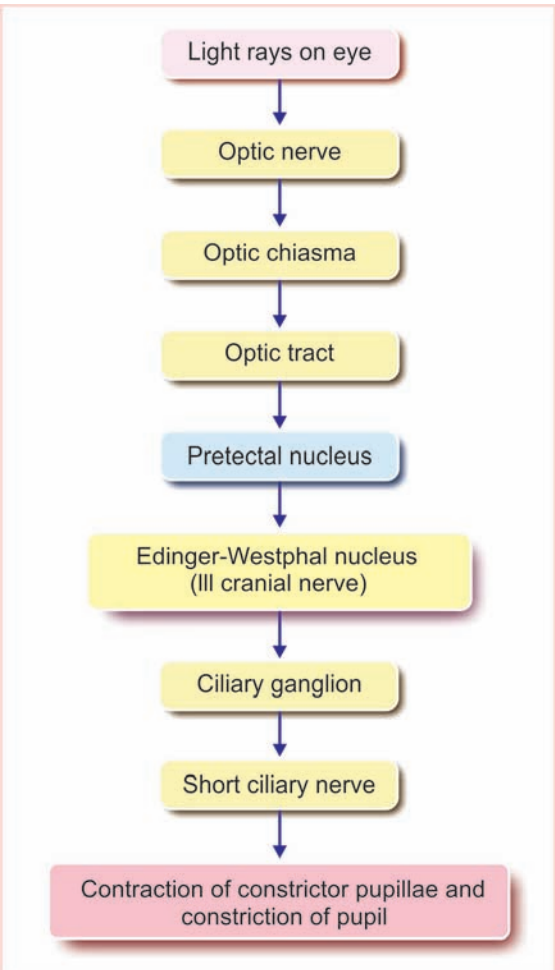

FIGURE 169.1: Pathway for light reflex

■ ACCOMMODATION

■ DEFINITION

Accommodation is the adjustment of eye to see either near or distant objects clearly. It is the process by which light rays from near objects or distant objects are brought to a focus on sensitive part of retina. It is achieved by various adjustments made in the eyeball.

■ MECHANISM OF ACCOMMODATION

Light rays from distant objects are approximately **parallel** and are less refracted while getting focused on retina. But, the light rays from near objects are divergent. So, to be focused on retina, these light rays should be **refracted (converged)** to a greater extent. There are three possible ways by which, accommodation occurs:

1. Retina must be moved towards or away from the lens. It is done by shortening or elongation of eyeball. So, the divergent, parallel or convergent rays are focused accurately. This mechanism is present only in some molluscs and not in human beings.
2. Lens must be moved towards or away from the retina. It is done in photography. This mechanism exists in some fishes.
3. Convexity of lens must be altered, so that the refractory power of lens is altered according to the need. This mechanism is present in human eye and it was first suggested by **Young** and later supported by **Helmholtz** (Fig. 169.2).

Young-Helmholtz Theory

Young-Helmholtz theory describes how the curvature of lens increases and thereby, the refractive power of lens is enhanced. When the eyes are fixed on a distant object (distant vision) lens is flat due to the traction of suspensory ligaments, which extend from the capsule of lens and are attached to ciliary processes. Ciliary processes are attached to choroid through the ciliary muscle (Refer Fig. 169.1).

When vision is shifted from the distant object to a near object (near vision), ciliary muscle contracts and draws the choroid forward. Ciliary processes are brought closer to lens, i.e. these processes form a small circle. Suspensory ligaments are slackened. Now, the tension on lens is released. Lens, due to its elastic property, bulges forward. **Anterior curvature** (convexity) of lens

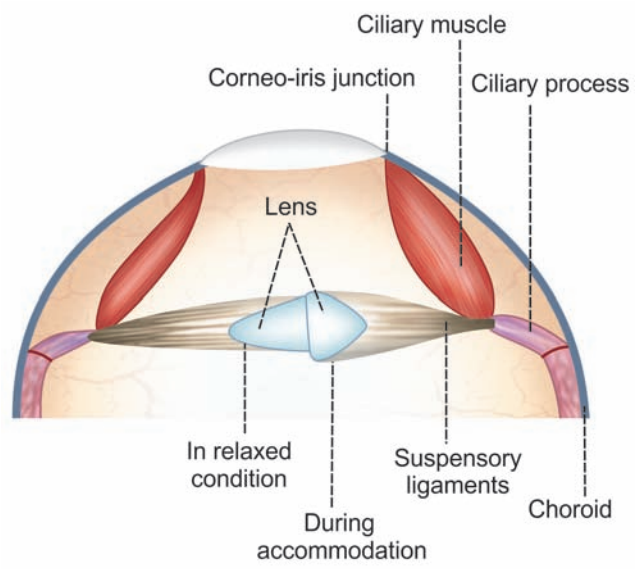

FIGURE 169.2: Accommodation

increases greatly. A very little change occurs in posterior curvature. This can be demonstrated by using Purkinje-Sanson images.

In resting eye, the intraocular pressure sets up tension in choroids and pulls the ciliary processes backward and outward. Suspensory ligaments are tensed up and the lens becomes flat.

Purkinje-Sanson Images

Purkinje-Sanson images are used to demonstrate the change in convexity of lens during accommodation for near vision. A subject is made to sit in a darkroom. A lighted candle is held in front. One eye is opened and the other eye is closed. Three images of the flame are seen in the opened eye (Fig. 169.3).

First image is upright and bright. It shines from the surface of cornea, which acts as a mirror. Second image is upright, but dim. It is reflected from the anterior convex surface of the lens. Third image is inverted and small. It is formed by posterior surface of the lens, which acts as a concave mirror.

When the person looks at a distant object, the second image reflected from anterior surface of lens is near the third image from posterior surface. During accommodation for near vision, no change occurs either in first image or the third image. But, the second image becomes smaller and moves towards the first image.

Thus, the increased convexity of the anterior surface of lens during accommodation for near vision is evident by the change in the size and the position of second image.

Other Adjustments in Eyeball during Accommodation

In addition to increase in anterior curvature of the lens, two more adjustments are made in the eyeball during accommodation for near vision.

1. Convergence of both eyeballs: It is necessary to bring the retinal images on to the corresponding points
2. Constriction of pupil: It is necessary to:
   i. Increase the visual acuity by reducing lateral chromatic and spherical aberrations
   ii. Reduce the quantity of light entering eye
   iii. Increase the depth of focus through more central part of lens as its convexity is increased.

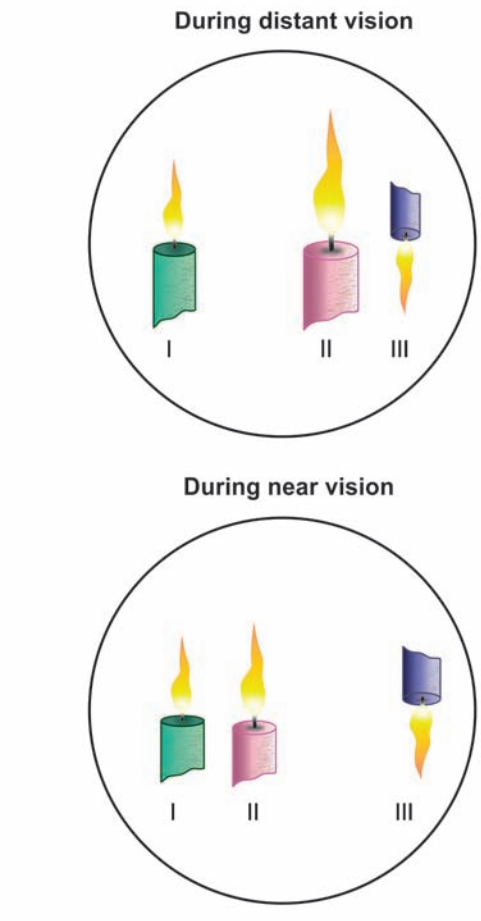

FIGURE 169.3: Purkinje-Sanson images

■ ACCOMMODATION REFLEX

Accommodation is a reflex action. When a person looks at a near object after seeing a far object, three adjustments are made in the eyeballs:

1. Convergence of the eyeballs due to contraction of the medial recti
2. Constriction of the pupil due to the contraction of constrictor pupillae of iris
3. Increase in the anterior curvature of the lens due to contraction of the ciliary muscle.

Thus, the accommodation reflex involves both skeletal muscle (medial recti) and smooth muscle (ciliary muscle and sphincter pupillae).

During accommodation, all the adjustments are carried out simultaneously. Although accommodation is a reflex action, it can be controlled by willpower to a certain extent.

■ PATHWAY FOR ACCOMMODATION REFLEX

Afferent Pathway

Visual impulses from retina pass through the optic nerve, optic chiasma, optic tract, lateral geniculate body and optic radiation to visual cortex (area 17) of occipital lobe. From here, the association fibers carry the impulses to frontal lobe (Fig. 169.4).

Center

The center for accommodation lies in frontal eye field (area 8) that is situated in the frontal lobe of cerebral cortex.

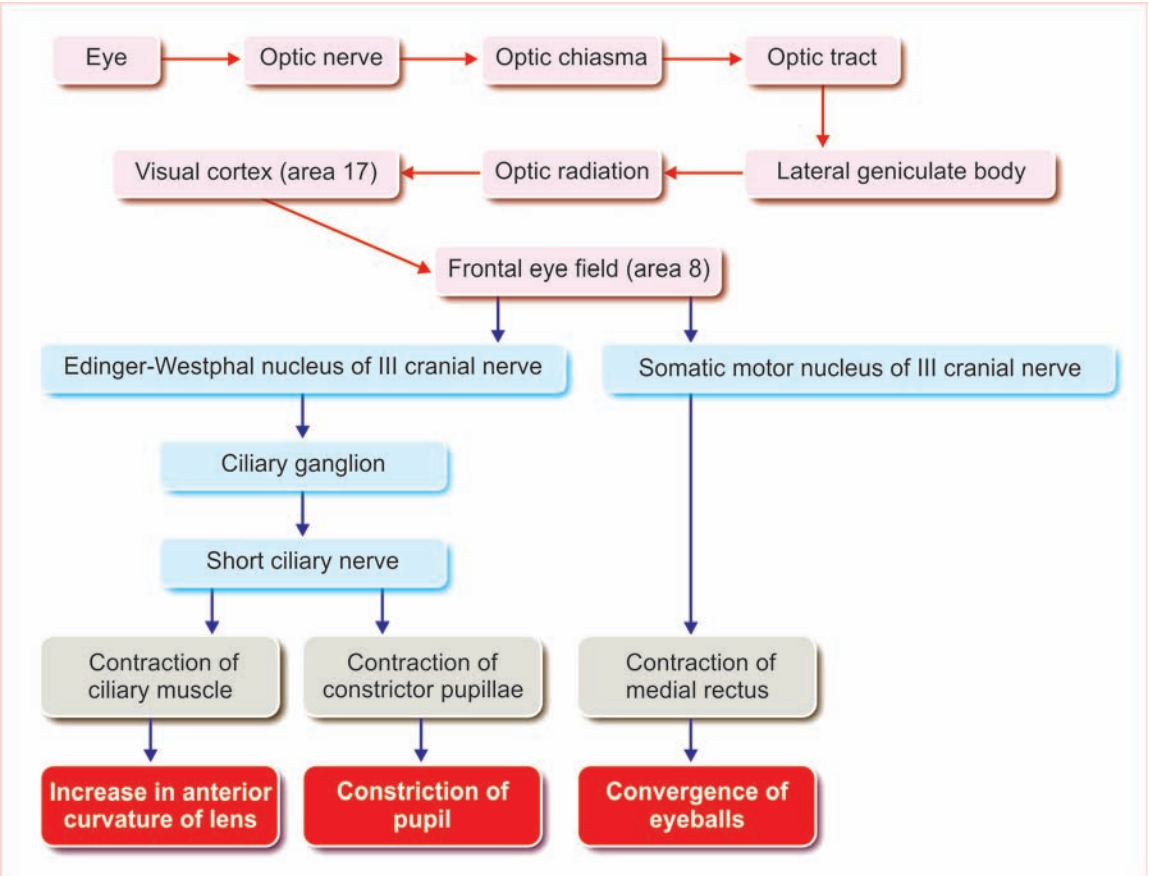

FIGURE 169.4: Pathway for accommodation reflex

Efferent Pathway

1. *Efferent fibers to ciliary muscle and sphincter pupillae*

From area 8, the corticonuclear fibers pass via internal capsule to the Edinger-Westphal nucleus of third cranial nerve. From here, the preganglionic fibers pass through the third cranial nerve to ciliary ganglion. Postganglionic fibers from ciliary ganglion pass via the short ciliary nerves and supply the ciliary muscle and the constrictor pupillae.

2. Efferent fibers to medial rectus

Some of the fibers from frontal eye field terminate in the somatic motor nucleus of oculomotor nerve. The fibers from motor nucleus supply medial rectus.

■ RANGE AND AMPLITUDE OF ACCOMMODATION

The farthest point from the eye at which the object can be seen is called **far point** or **punctum remotum.** In the normal eye, it is infinite, i.e. at a distance beyond 6 meters or 20 feet. It is limited only by the size of object, clearness of the atmosphere and the curvature of earth.

The nearest point from eye at which the object is seen clearly is called **near point** or **punctum proximum.** It is about 7 to 40 cm, depending upon the age. Distance between far point and near point is called **range of accommodation.**

Since, the focal length of eye is different in near vision and far vision, the refractive power of eye is also altered. The refractive power during far vision is called **static refraction** (R) and that during near vision is called **dynamic refraction** (P). The difference between these two refractive powers (P – R) is called **amplitude of accommodation,** which is expressed in diopter.

The **refractive power** is reciprocal of focal length and the unit for focal length is 1 meter or 100 cm. The refractory power is expressed as diopter (D).

For example, in a normal eye, if the near point is 10 cm, the dynamic refraction is:

$$P = \frac{1 \text{ meter}}{10 \text{ cm}} = \frac{100 \text{ cm}}{10 \text{ cm}} = 10 \text{ D}$$

In **emmetropic** (normal) **eye,** since the far point is at infinite distance, the static refraction is taken as zero.

Now,

Amplitude of accommodation = P – R
= 10 – 0
= 10 D

Amplitude of Accommodation at Different Ages

Amplitude of accommodation varies with age. Amplitude of accommodation at different age groups is:

10 years = 11.0 D
20 years = 9.5 D
30 years = 7.5 D
40 years = 5.5 D
50 years = 2.0 D
60 years = 1.2 D
70 years = 1.0 D

■ APPLIED PHYSIOLOGY

■ ARGYLL ROBERTSON PUPIL

Argyll Robertson pupil is a clinical condition in which the light reflex is lost but the accommodation reflex is present. It is common in tertiary syphilis. It also occurs because of lesion in Edinger-Westphal nucleus, diabetes and alcoholic neuropathy.

■ HORNER SYNDROME

Horner syndrome is an eye disorder caused by damage to cervical sympathetic nerve. It is also called **Bernard-Horner syndrome,** Claude Bernard-Horner syndrome or **oculosympathetic palsy.**

Symptoms of Horner syndrome appear on the affected side. The symptoms are:

1. **Ptosis** (drooping of upper eyelid)
2. Swelling of lower eyelid
3. **Miosis** (abnormal constriction of pupil)
4. **Enophthalmos** (sinking of eyeball into its cavity)
5. Absence of sweating on affected side of the face.

■ PRESBYOPIA

In old age, the amplitude of accommodation is decreased and the near point is away from the eye. This condition is called presbyopia. Details are given in Chapter 171.

Color Vision

Chapter 170

INTRODUCTION

Human eye can recognize about 150 different colors in the visible spectrum. Discrimination and appreciation of colors depend upon the ability of receptors in retina.

VISIBLE SPECTRUM

SPECTRAL COLORS

When sunlight or white light is passed through a glass **prism,** it is separated into different colors. Series of colored light produced by the prism is called the visible spectrum. Colors that form the spectrum are called spectral colors. Spectral colors are red, orange, yellow, green, blue, indigo and violet (**ROYGBIV** or **VIBGYOR**). In the spectrum, colors occupy the position according to their wavelengths. **Wavelength** is the distance between two identical points in the wave of light energy. Accordingly, red has got the maximum wavelength of about 8,000 Å and the violet has got the minimum wavelength of about 3,000 Å.

Light rays longer than red are called **infrared rays.** Rays shorter than violet are called **ultraviolet rays.** But, these two extraordinary types of rays do not evoke the sensation of vision. Refraction of spectral colors by the

prism also depends on wavelengths. Red is refracted less and violet is refracted more. So, longer the light rays, lesser is the refraction by the prism.

Purkinje Phenomenon

Purkinje phenomenon is the shift of brightest part of spectrum, when the intensity of illumination is changed.

When white light is passed through a prism, it splits into spectral colors from red to violet and if the colors are viewed at high illumination, the brightest part of the spectrum is the yellow, i.e. the brightest part of the spectrum is shifted to left. But when the light intensity is reduced to that of twilight, the color of the spectrum fades. Now the brightest part of spectrum is green, i.e. the brightest part of spectrum is shifted to right. It is called **Purkinje shift** or effect.

According to Purkinje, this effect is due to the maximal stimulation of cones by yellow and the maximal stimulation of rods by green.

■ EXTRASPECTRAL COLORS

Extraspectral colors are the colors other than those present in visible spectrum. These colors are formed by the combination of two or more spectral colors. For example, purple is the combination of violet and red. Pink is the combination of red and white.

■ PRIMARY COLORS

Primary colors are those, which when combined together produce the white. Primary colors are red, green and blue. These three colors in equal proportion give white.

■ COMPLEMENTARY COLORS

Complementary colors are the pair of two colors, which produce white when mixed or combined in proper proportion. Examples of complementary colors are red and greenish blue; orange and cyan blue; yellow and indigo blue; violet and greenish yellow; purple and green.

■ THEORIES OF COLOR VISION

Many theories are available to explain the mechanism of perception of color by eyes. However, most of the theories are not accepted universally. Following are the five theories, which are recognized:

■ 1. THOMAS YOUNG TRICHROMATIC THEORY

According to Thomas Young, retina has three types of cones. Each one possesses its own photosensitive substance. Each cone gives response to one of the primary colors – red, green and blue. Different color sensations are produced by the stimulation of various combinations of these three types of cones. For sensation of white light, all the three types of cones are stimulated equally.

■ 2. HELMHOLTZ TRICHROMATIC THEORY

Helmholtz substituted the **sensitive filaments** of optic nerve for cones. The sensitive filaments of nerves give response selectively to one or other of the three primary colors. It is also called Young-Helmholtz theory.

■ 3. GRANIT DOMINATOR-MODULATOR THEORY

Granit observed that the ganglionic cells of retina are stimulated by the whole of the visual spectrum. He studied the action potentials in ganglionic cells, which are stimulated by light and obtained some sensitivity curves. Sensitivity curves were recorded by using different wavelengths of light both in light-adapted and dark-adapted eyes. On the basis of these sensitivity curves, Granit classified the ganglionic cells into two groups namely, dominators and modulators.

Dominators

Dominators are responsible for brightness of light. Dominators are further divided into two types:

i. Dominators for cones, which respond in light-adapted eye and a broad sensitivity curve is produced with the maximum response around the wavelengths 55 Å
ii. Dominators for rods, which respond in dark-adapted eye and in the sensitivity curve the maximum response is given at the wavelengths of 500 Å.

Modulators

Modulators are responsible for different color sensations. Modulators are of three types:

i. Modulators of blue, which are stimulated by light with wavelengths of 450 to 470 Å
ii. Modulators of green, which are stimulated by light with wavelengths of 520 to 540 Å
iii. Modulators of red-yellow, stimulated by light with wavelengths of 580 to 600 Å.

If green light falls on retina, modulators of green are stimulated and other two are less affected. Thus, according to Granit, the dominators are responsible for brightness or intensity of light, both in dark-adapted

(rods) and light-adapted (cones) eyes. The modulators are responsible for color vision in light-adapted eyes.

4. HARTRIDGE POLYCHROMATIC THEORY

According to this theory, human retina has seven types of receptors. All the seven receptors are divided into three units:

First Unit

First unit is a tricolor unit consisting of receptors for orange, green and blue.

Second Unit

Second unit is a dicolor unit with receptors for yellow and blue colors. Receptors for yellow and blue are complementary to each other.

Third Unit

Third unit is another dicolor unit with red and blue-green receptors.

5. HERING'S THEORY OF OPPOSITE COLORS

According to Hering, retina has three photochemical substances. Each substance produces the sensation of a particular color by its breakdown or resynthesis.

First Substance

First substance is white-black substance. Its breakdown causes sensation of white and resynthesis causes sensation of black.

Second Substance

Second substance is yellow-blue substance. Its breakdown causes sensation of yellow and resynthesis causes sensation of blue.

Third Substance

Third substance is red-green substance. Its breakdown causes sensation of red and resynthesis causes sensation of green.

This theory explains the successive contrast and afterimages, but not the simultaneous sensation of antagonistic colors.

COLOR SENSITIVE AREAS IN RETINA

Peripheral part of retina is devoid of cones and is insensitive to color and gives sensation of white, black and grey only. Central portion of retina, fovea centralis has more cones so, it is more sensitive to color. In extrafoveal regions, cones are mingled with rods.

Retinal area sensitive to blue is largest and to green is smallest. Red comes next to blue and then comes yellow. All the color areas of retina are mapped out by using perimeter.

CONTRAST EFFECTS

SIMULTANEOUS CONTRAST

Simultaneous contrast is the effect that intensifies the contrast (difference) between two colors, which are placed against each other. When black is placed against white or white against black, these two colors set one another off, i.e. the black looks blacker and the white looks whiter. Similarly, the green is enhanced by red and red by green.

Maximum effect of simultaneous contrast is obtained when the complementary colors are paired. Reason for simultaneous contrast is that, the stimulation of an area of retina by one color modifies the response in the surrounding or neighboring areas. It increases the sensitivity to other colors in the surrounding receptors. This action is probably due to horizontal cells.

SUCCESSIVE CONTRAST

Successive contrast is the effect of previously viewed color field on the appearance of currently viewed color field. When a person looks at a green object after looking at a bright red, the green object appears to be more greenish. There is an increase in the sensitivity to the complementary color.

Reason for successive contrast is that, the stimulation of an area of retina modifies its sensitivity to the successive stimuli. Thus, there is an increase in the sensitivity to the second color.

AFTERIMAGE

Afterimage is the phenomenon in which retention of image occurs even after cessation of light stimulus. After looking at a bright object, if the eyes are closed, the image remains more distinct for some time and then fades away gradually. Afterimage is of two types:

1. POSITIVE AFTERIMAGE

Positive afterimage is an afterimage persisting after closure of eyes or turning towards a dark background. After looking at a bright object, if the eyes are closed or fixed on a black surface, the afterimage appears to be bright and with same color of the object.

■ 2. NEGATIVE AFTERIMAGE

Negative afterimage is an afterimage that persists while turning towards a bright background. After looking at bright object, if the eyes are fixed on white surface (instead of closing or fixing on a black surface), the afterimage appears in complementary color. Reason for negative afterimage is the persistence of activity in retina, even after the particular stimulus ceases to act.

■ APPLIED PHYSIOLOGY – COLOR BLINDNESS

Color blindness is the failure to appreciate one or more colors. It is common in 8% of males and only in 0.4% of females, as mostly the color blindness is an inherited sex-linked recessive character. In addition to hereditary conditions, color blindness occurs due to acquired conditions also such as ocular diseases or injury or disease of retina.

The term **'color blind'** does not mean that objects are seen only in black and white. Total color blindness is very rare. There are many types and degrees of color blindness. The most appropriate term for color blindness is deficiency of color vision.

■ CAUSES FOR ACQUIRED COLOR BLINDNESS

1. *Trauma*

Injury to eye due to accidents or strokes results in color blindness.

2. *Chronic Diseases*

Color blindness is caused by chronic diseases such as:

i. Glaucoma
ii. Degeneration of macula of eye
iii. Retinitis
iv. Sickle cell anemia
v. Leukemia
vi. Diabetes
vii. Liver diseases
viii. Parkinson disease
ix. Alzheimer disease
x. Multiple sclerosis.

3. *Drugs*

Frequent use of some drugs leads to color blindness:

i. Antibiotics
ii. Antihypertensive drugs
iii. Antituberculosis drugs
iv. Barbiturates
v. Drugs used to treat psychological problems and neural disorders.

4. *Toxins*

Industrial toxins or strong chemicals cause color blindness. Common substances causing color blindness are:

i. Fertilizers
ii. Carbon monoxide
iii. Carbon disulfide
iv. Chemicals with high lead content.

5. *Alcoholism*

Chronic alcoholism results in color blindness.

6. *Aging*

Color blindness can occur after 60 years of age due to various changes in eye.

■ CLASSIFICATION OF COLOR BLINDNESS

Based on Young-Helmholtz trichromatic theory, color blindness is classified into three types (Fig. 170.1):

1. *Monochromatism*

Monochromatism is the condition characterized by total inability to perceive color. It is also called **total color blindness** or **achromatopsia.** Monochromatism is very rare. Persons with monochromatism are called **monochromats.** Retina of monochromats is totally insensitive to color and they see the whole spectrum in only black, white and different shades of grey. So, their vision is similar to black and white photography. Monochromatism is divided into two types:

i. *Rod monochromatism*

Rod monochromatism is the condition in which cones are functionless and the vision depends purely on rods. So, **rod monochramats** are totally color blind. They are dazzled by light but definitely are not blind during daylight. Their visual acuity is lowered and foveal vision is absent which results in **central scotoma.** Central scotoma is the formation of big blind spot in fovea centralis due to the non-functioning of cones. Rods are also absent in fovea.

ii. *Cone monochromatism*

Cone monochromatism is the condition in which vision depends upon one single type of cone. Central scotoma does not occur in this condition.

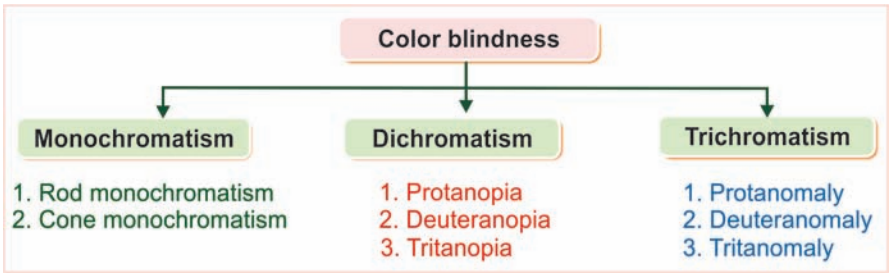

FIGURE 170.1: Color blindness

2. *Dichromatism*

Dichromatism is the color blindness in which the subject can appreciate only two colors. Persons with this defect are called **dichromats.** They can match entire spectrum of colors by only two primary colors because the receptors for third color are defective. The defects are classified into three groups:

i. *Protanopia*

Protanopia is the type of dichromatism caused by the defect in receptor of first primary color, i.e. red. So, the red color cannot be appreciated. Persons having protanopia are called **protanopes.** They use blue and green to match the colors. Thus, they confuse red with green.

ii. *Deuteranopia*

Deuteranopia is the dichromatism caused by the defect in receptor of second primary color, i.e. green. **Deuteranopes** use blue and red colors and they cannot appreciate green color.

iii. *Tritanopia*

Tritanopia is the dichromatism caused by the defect in receptor of third primary color, i.e. blue. **Tritanopes** use red and green colors and they cannot appreciate blue color.

3. *Trichromatism*

Trichromatism is the color blindness in which intensity of one of the primary colors cannot be appreciated correctly though the affected persons are able to perceive all the three colors. Persons with this defect are called **trichromats.** Even the dark shades of one particular color look dull for them. Trichromatism is classified into three types:

i. *Protanomaly*

Protanomaly is the type of trichromatism in which the perception for red is weak. So to appreciate red color, the person requires more intensity of red than a normal person.

ii. *Deuteranomaly*

Deuteranomaly is the trichromatism in which the perception for green is weak.

iii. *Tritanomaly*

Tritanomaly is the trichromatism with weak perception for blue.

■ TESTS FOR COLOR BLINDNESS

Three methods are available to determine the color blindness:

1. By using **Ishihara color charts**
2. By using **Holmgren colored wool**
3. By using **Edridge-Green lantern.**

Errors of Refraction

Chapter 171

- AMETROPIA
  - MYOPIA OR SHORT SIGHTEDNESS
  - HYPERMETROPIA OR LONG SIGHTEDNESS
- ANISOMETROPIA
- ASTIGMATISM
  - CAUSE
  - TYPES
  - CORRECTION
- PRESBYOPIA
  - CAUSES
  - CORRECTION

AMETROPIA

The eye with normal refractive power is called **emmetropic eye** and the condition is called **emmetropia.** Any deviation in the refractive power from normal condition, resulting in inadequate focusing on retina is called **ametropia** and the eye is called **ametropic eye.** The defect is due to the change in shape of the eyeball.

Ametropia is of two types:

1. Myopia
2. Hypermetropia.

MYOPIA OR SHORT SIGHTEDNESS

Myopia is the eye defect characterized by the inability to see the distant object. It is otherwise called short sightedness because the person can see near objects clearly but not the distant objects. In emmetropia, the far point is infinite. In myopia, the near vision is normal but the far point is not infinite, i.e. it is at a definite distance (Fig. 171.1 and Table 171.1). In extreme conditions, it may be only a few centimeter away from the eye (myo = half closed; ops = eye).

Cause

In myopia, the refractive power of lens is usually normal. But, the **anteroposterior diameter** of the eyeball is abnormally long. Therefore, the image is brought to focus a little in front of retina. Light rays, after coming to a focus, disperse again so, a blurred image is formed upon retina.

Correction

In myopic eye, in order to form a clear image on the retina, the light rays entering the eye must be divergent and not parallel. Thus, the myopic eye is corrected by using a **biconcave lens.** Light rays are diverged by the concave lens before entering the eye (Fig. 171.1).

HYPERMETROPIA OR LONG SIGHTEDNESS

Hypermetropia is the eye defect characterized by the inability to see near object. It is otherwise known as long sightedness because the person can see the distant objects clearly but not the near objects. It is also called **hyperopia.** In this defect, distant vision is normal but, near vision is affected (metras = measure).

Cause

Hypermetropia is due to **decreased anteroposterior diameter** of the eyeball. So, even though the refractive power of lens is normal, the light rays are not converged enough to form a clear image on retina, i.e. the light rays are brought to a focus behind retina. It causes a blurred image of near objects. Hypermetropia occurs in childhood, if the eyeballs fail to develop the correct size. It is common in old age also.

Correction

Hypermetropia is corrected by using **biconvex lens.** Light rays are converged by convex lens before entering the eye (Fig. 171.1).

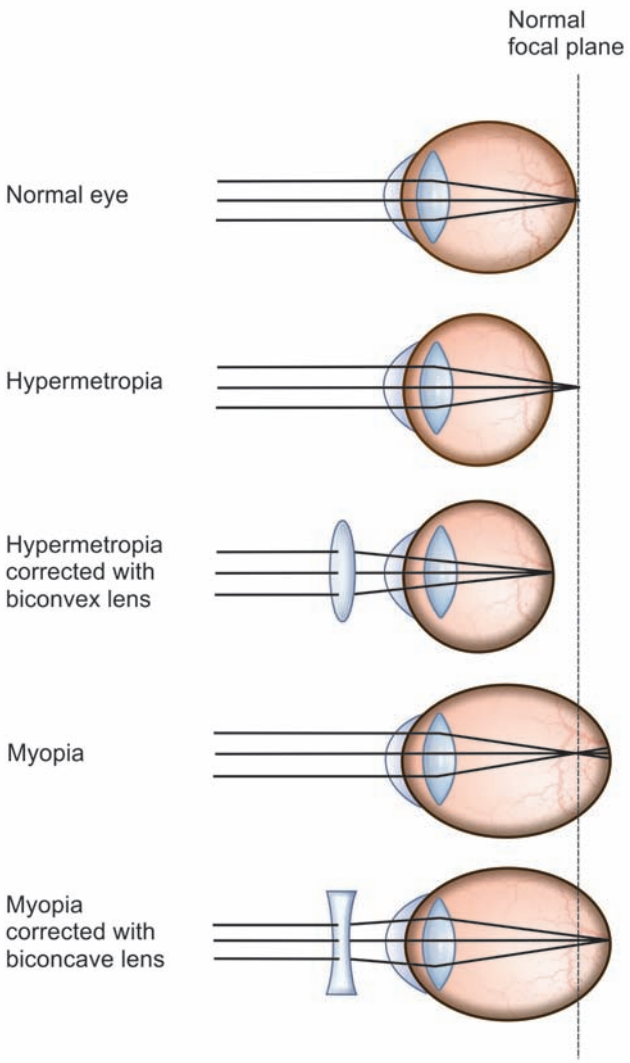

FIGURE 171.1: Errors of refraction

■ ANISOMETROPIA

Anisometropia is the condition in which the two eyes have **unequal refractive power.** It is corrected by using different appropriate lens for each eye (Table 171.1).

■ ASTIGMATISM

Astigmatism is the condition in which light rays are not brought to a sharp point upon retina. It is the common optical defect. This defect is present in all eyes. When it is moderate, it is known as **physiological astigmatism.** When it is well marked, it is considered abnormal. For example, the stars appear as small dots of light to a person with normal eye. But in astigmatism, the stars appear as radiating short lines of light (A = not; stigma = point).

■ CAUSE OF ASTIGMATISM

Light rays pass through all meridians of a lens. In a normal eye, lens has approximately same curvature in all meridians. So, the light rays are refracted almost equally in all meridians and brought to a focus.

If the curvature is different in different meridians, vertical, horizontal and oblique, the refractive power is also different in different meridians. The meridian with greater curvature refracts the light rays more strongly than the other meridians. So, these light rays are brought to a focus in front of the light rays, which pass through other meridians. Such irregularity of curvature of lens causes astigmatism.

■ TYPES OF ASTIGMATISM

Astigmatism is of two types:

1. Regular astigmatism
2. Irregular astigmatism.

1. *Regular Astigmatism*

In regular type of astigmatism, the refractive power is unequal in different meridians because of alteration of curvature in one meridian. But, it is uniform in all points throughout the affected meridian.

2. *Irregular Astigmatism*

In irregular type of astigmatism, the refractive power is unequal not only in different meridians, but it is also unequal in different points of same meridian.

TABLE 171.1: Errors of refraction

Type of error	Cause	Correction
Myopia	Increase in anteroposterior diameter of the eyeball	Biconcave lens
Hypermetropia	Decrease in anteroposterior diameter of the eyeball	Biconvex lens
Anisometropia	Difference in refractive power of both eyes	Separate lens (biconcave or biconvex) for each eye as required
Astigmatism	Refractory power of lens is different in different meridians	Cylindrical lens
Regular astigmatism	Refractory power of lens is unequal in different meridians but uniform in one single meridian	
Irregular astigmatism	Refractory power of lens is unequal in different meridians as well as in different points in same meridian	
Presbyopia	Loss of elasticity in lens and weakness of ocular muscles due to old age	Biconvex lens

■ CORRECTION OF ASTIGMATISM

Astigmatism is corrected by using **cylindrical glass** lens having the convexity in the meridians, corresponding to that of lens of eye having a lesser curvature, i.e. if the horizontal curvature of lens is less, the person should use cylindrical glass lens with the convexity in horizontal meridian.

■ PRESBYOPIA

Presbyopia is the condition characterized by progressive diminished ability of eyes to focus on near objects with age. It is due to the gradual reduction in the amplitude of accommodation. It progresses as the age advances (presbyos = old; ops = eye). Presbyopia starts developing after middle age. In presbyopia, the distant vision is unaffected. Only the near vision is affected. The near point is away from eye. In presbyopia, the anterior curvature of lens does not increase during near vision. So, the light rays from near objects are not brought to focus on retina.

■ CAUSES OF PRESBYOPIA

1. Decreased elasticity of lens is because of the physical changes in lens and its capsule during old age. So, the anterior curvature is not increased during near vision.
2. Decreased convergence of eyeballs due to the concomitant weakness of ocular muscles in old age.

■ CORRECTION OF PRESBYOPIA

Presbyopia is corrected by using **biconvex lens.**

Chapter 172

Structure of Ear

- EXTERNAL EAR
  - AURICLE OR PINNA
  - EXTERNAL AUDITORY MEATUS
- MIDDLE EAR
  - AUDITORY OSSICLES
  - AUDITORY MUSCLES
  - EUSTACHIAN TUBE
- INTERNAL EAR
  - COCHLEA
  - COMPARTMENTS OF COCHLEA
  - ORGAN OF CORTI

EXTERNAL EAR

Ear consists of three parts, namely external ear, middle ear and internal ear (Fig. 172.1).

External ear is formed by two parts:

1. Auricle or pinna
2. External auditory meatus.

AURICLE OR PINNA

Auricle or pinna of the external ear consists of **fibrocartilaginous plate** covered by connective tissue and skin. This plate is characteristically folded and ridged. Skin covering this plate is thin and contains many fine hairs and sebaceous glands. On the posterior surface of auricles, many sweat glands are present.

In many animals, auricle can be moved and turned to locate the source of sound or the auricle can be folded to avoid unwanted sound. But in man, extrinsic and intrinsic muscles of auricles are rudimentary and the movement is not possible. The depression of auricle, which forms the orifice of external auditory meatus, is called **concha.**

EXTERNAL AUDITORY MEATUS

External auditory meatus starts from the concha and extends inside as a slightly curved canal, with a length of about 55 mm.

Meatus consists of two parts:

i. Outer cartilaginous part
ii. Inner bony part.

i. *Outer Cartilaginous Part*

Cartilaginous part is the initial part of external auditory meatus and is made up of cartilage. It is covered by thick skin, which contains stiff hairs. These hairs prevent the entry of foreign particles.

Large **sebaceous glands** and **ceruminous glands** are also present in the skin covering this portion. These glands are coiled and tubular in nature and open on the surface of the skin. Columnar epithelial cells of the glands contain brown pigment granules and fat droplets. Secretions of ceruminous glands, sebaceous glands and desquamated epithelial cells form the earwax.

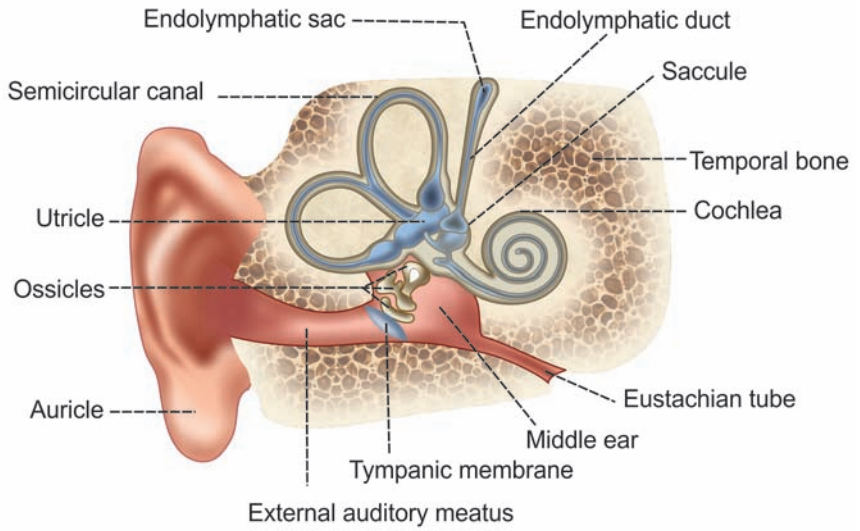

FIGURE 172.1: Diagram showing the structure of ear

ii. *Inner Bony Part*

Inner part of the external auditory meatus is also covered by skin, which adheres closely to periosteum. Only sebaceous glands are present here. Small fine hairs are present on the superior wall of the canal. Skin covering this portion is continuous with cuticular layer of tympanic membrane.

■ MIDDLE EAR

Middle ear or tympanic cavity is a small, narrow, irregular, laterally compressed chamber, situated within the temporal bone. It is also known as **tympanum.** It is separated from external auditory meatus by **tympanic membrane.**

Middle ear consists of the following structures:

1. Auditory ossicles
2. Auditory muscles
3. Eustachian tube.

Tympanic Membrane

Tympanic membrane is a thin, semitransparent membrane, which separates the middle ear from external auditory meatus. Periphery of the membrane is fixed to tympanic sulcus in the surrounding bony ring, by means of fibrocartilage (Fig. 172.2).

Structure of Tympanic Membrane

Tympanic membrane is formed by three layers:

1. Lateral cutaneous layer, which is the continuation of the skin of auditory meatus
2. Intermediate fibrous layer, which contains collagenous fibers
3. Medial mucus layer or tympanic mucosa, which is composed of single layer of cuboidal epithelial cells.

■ AUDITORY OSSICLES

Auditory ossicles are the three miniature bones, which are arranged in the form of a chain, extending across the middle ear from the tympanic membrane to oval window (Fig. 172.2).

Auditory ossicles are:

i. Malleus
ii. Incus
iii. Stapes.

i. *Malleus*

Malleus is otherwise called **hammer.** It has a handle, head and neck. **Hand** is called **manubrium** and it is attached to tympanic membrane. Neck extends from handle to the head. **Head** or **capitulum** articulates with the body of incus.

ii. *Incus*

Incus is also known as **anvil.** It looks like a premolar tooth. Incus has a body, one long process and one short process. Anterior surface of the body articulates with the head of malleus. The **short process** is attached to a ligament. The **long process** runs parallel to handle of malleus. Tip of the long process is like a knob called **lenticular process** and it articulates with the next bone, stapes.

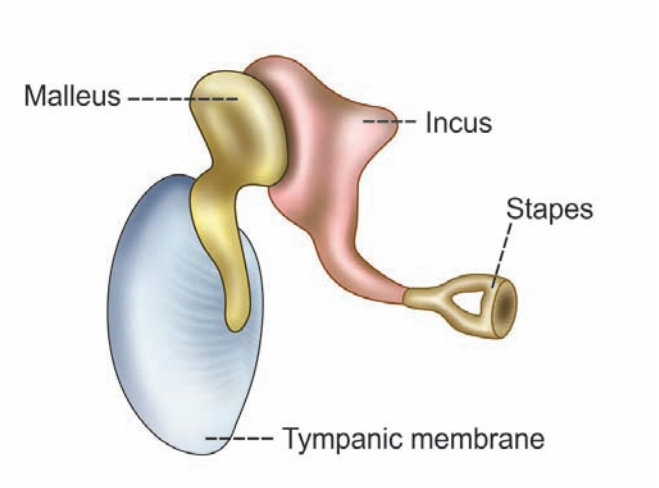

FIGURE 172.2: Tympanic membrane and auditory ossicles

iii. *Stapes*

Stapes is also called **stirrup.** It is the smallest bone in the body. It has a head, neck, anterior crus, posterior crus and a footplate. **Head** articulates with incus. **Footplate** fits into oval window.

■ AUDITORY MUSCLES

Two skeletal muscles are attached to ossicles:

i. Tensor tympani
ii. Stapedius.

i. *Tensor Tympani*

Tensor tympani is larger of the two muscles of tympanic cavity.

Origin, insertion and nerve supply

Tensor tympani arises from cartilaginous portion of eustachian tube (see below), adjacent to great wing of sphenoid bone and osseous canal. Its tendon is inserted on manubrium of malleus, which is in turn attached to tympanic membrane. Thus, the tensor tympani is attached to tympanic membrane through malleus. It is supplied by mandibular division trigeminal nerve.

Function

Tensor tympani muscle pulls and keeps the tympanic membrane stretched or tensed constantly. This constant stretching of tympanic membrane is essential for the transmission of sound waves, which may reach any part of the tympanic membrane. Paralysis of tensor tympani causes hearing impairment.

ii. *Stapedius*

Stapedius is the **smallest skeletal muscle** in human body with a length of just over 1 mm. It lies in a conical bony cavity, on the posterior wall of the tympanic cavity.

Origin, insertion and nerve supply

Stapedius arises from interior pyramid of tympanic cavity. Its tendon is inserted into the posterior surface of neck of stapes. It is supplied by branch of facial nerve.

Function

Stapedius prevents excess movements of stapes. When it contracts, it pulls the neck of stapes backwards and reduces the movement of footplate against the fluid in cochlea. Paralysis of stapedius allows wider range of oscillation of stapes, leading to **hyperreaction** of auditory ossicles to sound vibrations. This condition is called **hyperacusis.** Paralysis of stapedius occurs in the lesion of facial nerve.

Tympanic Reflex

Tympanic reflex is an **attenuation reflex** characterized by involuntary contraction of tensor tympani and stapedius muscles, in response to a loud noise. It has a latent period of 40 to 80 millisecond.

When both the muscles contract, manubrium of malleus moves inward and stapes is pulled outward. These two actions result in stiffness of auditory ossicles, so that the transmission of sound is decreased.

Significance of tympanic reflex

i. Tympanic reflex protects the tympanic membrane from being ruptured by loud sound
ii. It also prevents fixation of footplate of stapes, against oval window, during exposure to loud sound
iii. It helps to protect the cochlea from damaging effects of loud sounds. Contraction of tensor tympani and stapedius during exposure to loud sound develops stiffness of the auditory ossicles so that, the transmission of sound into cochlea is decreased.

■ EUSTACHIAN TUBE

Eustachian tube or the **auditory tube** is the flattened canal extending from the anterior wall of middle ear to nasopharynx. Its upper part is surrounded by the bony wall and the lower part is surrounded by fibrocartilaginous plate.

Eustachian tube connects middle ear with posterior part of nose and forms the passage of air between middle ear and atmosphere. So, the pressure on both sides of tympanic membrane is equalized.

■ INTERNAL EAR

Internal ear or labyrinth is a membranous structure, enclosed by a bony labyrinth in petrous part of temporal bone. It consists the sense organs of hearing and equilibrium. Sense organ for hearing is the cochlea and the sense organ for equilibrium is the vestibular apparatus. Vestibular apparatus is already explained in Chapter 158.

■ COCHLEA

Cochlea is a coiled structure like a snail's shell (cochlea = snail's shell). It consists of two structures:

1. Central conical axis formed by spongy bone called **modiolus**
2. Bony canal or tube, which winds around the modiolus.

In man, the **bony canal** makes two and a half turns, starting from the base of the cochlea and ends at the top (apex) of cochlea. End of the canal is called **cupula.** Base of modiolus forms the bottom of internal auditory meatus, through which cochlear nerve fibers pass and enter the modiolus. Thus, a section through the axis of cochlea reveals the central **bony pillar,** modiolus and **periotic or osseous canal,** which coils around the modiolus.

From modiolus, a bony ridge called **osseous spiral lamina** projects into the canal, winding around modiolus like the thread of a screw. Spiral lamina follows the spiral turns of cochlea and ends at the cupula in a hook-shaped process called **hamulus.**

■ COMPARTMENTS OF COCHLEA

Two membranous partitions extend between the osseous spiral lamina and outer wall of the **spiral canal.** Both the membranes divide the spiral canal of cochlea into three compartments.

Membranes of cochlea:

1. Basilar membrane
2. Vestibular membrane.

1. *Basilar Membrane*

Basilar membrane is a connective tissue membrane. It stretches from the tip of the osseous spiral lamina to tough dense fibrous band called **spiral ligament,** which lines the outer wall of the canal. Basilar membrane is also called **membranous spiral lamina.** Along the basilar membrane are twenty thousand to thirty thousand tiny fibers that are called **basilar fibers.** Each fiber has different size and shape. Fibers near the oval window are short and stiff. While approaching towards **helicotrema** (see below) the basilar fibers gradually become longer and soft.

2. *Vestibular Membrane*

Vestibular membrane is also known as **Reissner membrane** and it is a thin membrane. It is placed obliquely between the upper surface of osseous spiral lamina and upper part of spiral ligament.

Basilar membrane and vestibular membrane divide the spiral canal of cochlea into three compartments called scalae (Fig. 172.3).

Compartments of spiral canal of cochlea:

i. Scala vestibuli
ii. Scala tympani
iii. Scala media.

All the three compartments are filled with fluid. Scala vestibuli and scala tympani contain **perilymph.** Scala media is filled with **endolymph.**

i. *Scala vestibuli*

Scala vestibuli lies above scala media. It arises from **oval window (fenestra vestibuli),** which is closed by the

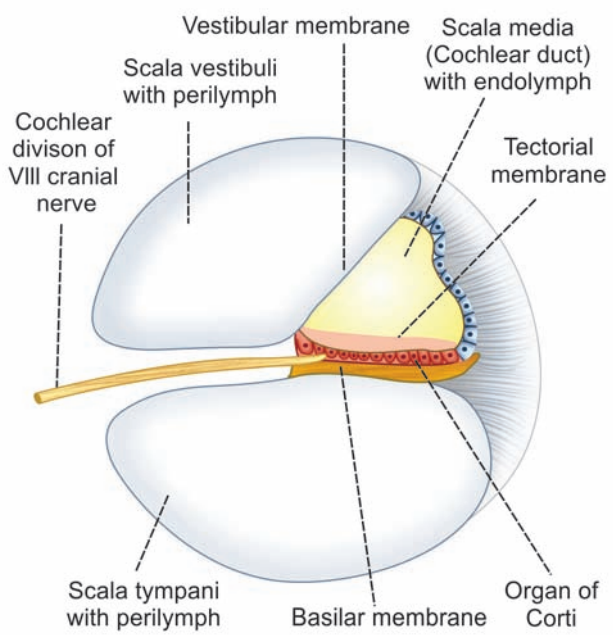

FIGURE 172.3: Cross-section of spiral canal of cochlea

footplate of stapes. It follows the bony canal up to its apex. At the apex, it communicates with the scala tympani through a small canal called **helicotrema.**

ii. *Scala tympani*

Scala tympani lies below scala media. It is parallel to scala vestibuli and ends at the round window. **Round window** is closed by a strong thin membrane known as **secondary tympanic membrane.**

iii. *Scala media*

Scala media is otherwise called **cochlear duct, membranous cochlea** or **otic cochlea.** It is a triangular compartment enclosed by basilar and vestibular membranes. It ends blindly at the apex and at the base of cochlea. A slender **ductus reuniens** arises from the basal end and connects scala media with the saccule of otolith organ.

Scala media is formed by upper, outer and lower walls. Upper wall or vestibular wall is formed by **vestibular membrane.** Outer wall is formed by spiral ligament, which is the thickening of periosteum. Lower wall is called **tympanic wall.** It is formed by **basilar membrane** (membranous spiral lamina) and a part of osseous spiral lamina. Scala media stretches between the tip of osseous spiral lamina and spiral ligament.

Basilar membrane consists of straight unbranched connective tissue fibers, which are called basilar fibers or the auditory fibers. On the upper surface of the basilar membrane, epithelial cells are arranged in the form a special structure called the organ of Corti. It is the sensory part of cochlea.

■ ORGAN OF CORTI

Organ of Corti is the **receptor organ** for hearing. It is the neuroepithelial structure in cochlea (Fig. 172.4).

Situation and Extent

Organ of corti rests upon the lip of osseous spiral lamina and basilar membrane. It extends throughout the cochlear duct, except for a short distance on either end. Roof of the organ of Corti is formed by **gelatinous tectorial membrane.**

Structure

Organ of Corti is made up of sensory elements called **hair cells** and various supporting cells. All the cells of organ of Corti are arranged in order from center towards the periphery of the cochlea.

Cells of organ of Corti:

1. Border cells
2. Inner hair cells
3. Inner phalangeal cells
4. Inner pillar cells
5. Outer pillar cells
6. Outer phalangeal cells
7. Outer hair cells
8. Cells of Hensen
9. Cells of Claudius
10. Tectorial membrane and lamina reticularis.

1. *Border Cells*

Border cells are the slender columnar cells, arranged in a single layer on the tympanic lip along the inner side of inner hair cells. Surfaces of the border cells have **cuticle.**

2. *Inner Hair Cells*

Inner hair cells are flask-shaped cells and are broader than the outer hair cells. Inner hair cells are arranged in a single row and occupy only the upper part of epithelial layer. Rounded base of each cell rests on the adjacent supporting cells called the inner phalangeal cell. Surface of the inner hair cell bears a **cuticular plate** and a number of short stiff hairs, which are called **stereocilia.** Each hair cell has about 100 sterocilia. One of the sterocilia is larger and it is called **kinocilium.** Stereocilia are in contact with the **tectorial membrane.** Inner hair cells and outer hair cells together form the receptor cells. Sensory nerve fibers are distributed around the hair cells. Both inner hair cells and outer hair cells have afferent and efferent nerve fibers (Chapter 173).

3. *Inner Phalangeal Cells*

Inner phalangeal cells are the supporting cells of inner hair cells and are arranged in a row along the inner surface of inner pillar cells. Their bases rest on the basilar membrane. Cuticular plate of cells (formed by the lower portion of cells) look like the finger bones, phalanges.

4 and 5. *Inner and Outer Pillar Cells – Rods of Corti*

Inner and outer pillar cells are called rods of Corti. Each pillar cell has a broader base, an elongated body

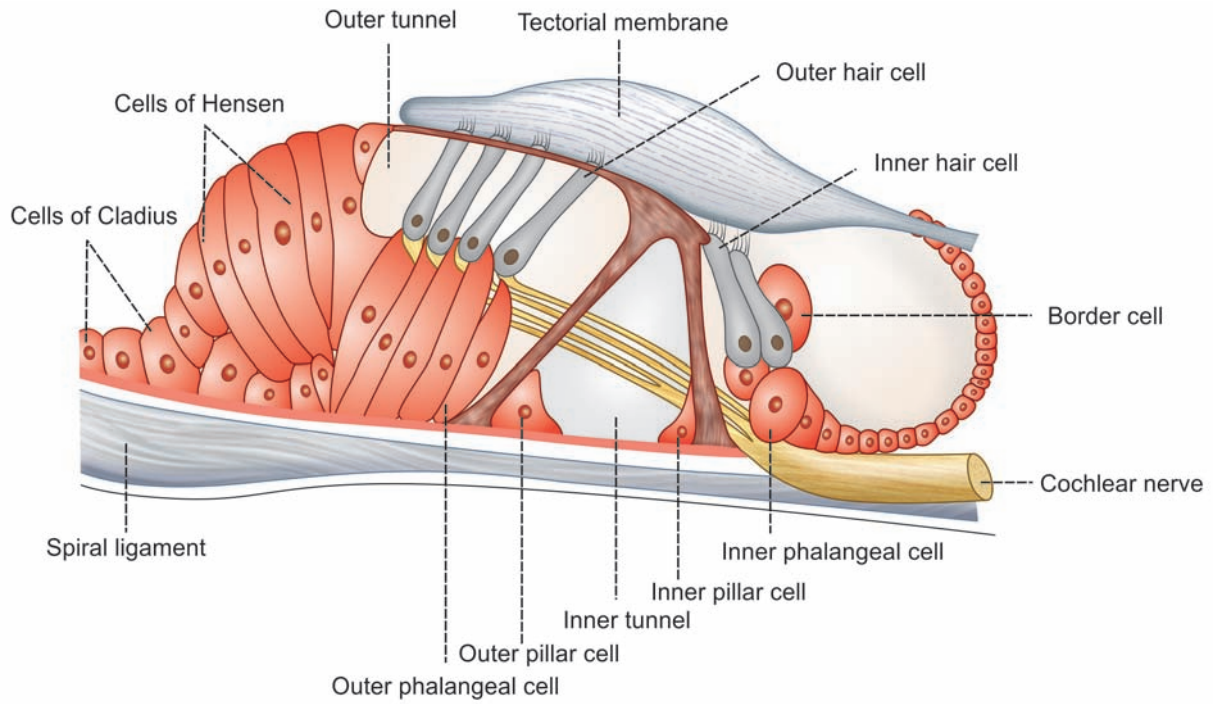

FIGURE 172.4: Organ of Corti

or pillar and a head at the tip of pillar. Bases of inner pillar cells are close to the lip of osseous spiral lamina (tympanic lip), whereas the bases of outer pillar cells are close to basilar membrane. Pillars of inner and outer pillar cells slope towards each other and their heads articulate. Thus, the pillars of cells form series of arches, which enclose a triangular tunnel called **inner tunnel** or **tunnel of Corti.**

6. *Outer Phalangeal Cells*

Outer phalangeal cells or the cells of Deiters are the supporting cells of outer hair cells. Outer phalangeal cell is the tall columnar cell. It sends stiff phalangeal processes upward between the hair cells, to form the part of **lamina reticularis.**

Rows of outer phalangeal cells vary in different regions of cochlear duct like the outer hair cells, i.e. from three to five rows. Between the inner most outer phalangeal cells and outer pillar cells, is a fluid space known as the **space of Nuel.**

7. *Outer Hair Cells*

Outer hair cells are the columnar cells occupying the superficial part of epithelium of organ of Corti. Their bases are supported by outer phalangeal cells. Structure of outer hair cells is similar to that of inner hair cells (see above).

8. *Cells of Hensen*

Cells of Hensen are tall columnar cells forming the outer border cells of organ of Corti. These cells are arranged in several rows on basilar membrane, lateral to outer phalangeal cells. The space between outer phalangeal cells and cells of Hensen is called outer tunnel.

9. *Cells of Claudius*

Cells of Claudius are cuboidal in nature and line the lower surface of external spiral sulcus. In certain areas, some groups of cells are present between the cells of Claudius and basilar membrane. These cells are called Boettcher cells.

10. *Tectorial Membrane and Lamina Reticularis*

Tectorial membrane extends from vestibular lip to the level of cells of Hensen. It forms the roof of organ of Corti. It is in contact with the processes of hair cells. It is assumed that the processes of hair cells are stimulated by the movements of tectorial membrane, in relation to vibrations in endolymph.

Cuticular plates of all the supporting cells collectively form a reticular membrane, which is known as lamina reticularis. It covers the organ of Corti. It looks like a mosaic and has rows of holes, through which the heads of hair cells are inserted.

Auditory Pathway

Chapter 173

- INTRODUCTION
- RECEPTORS
- FIRST ORDER NEURONS
- SECOND ORDER NEURONS
- THIRD ORDER NEURONS
- CORTICAL AUDITORY CENTERS
- APPLIED PHYSIOLOGY – EFFECT OF LESION

INTRODUCTION

Fibers of auditory pathway pass through **cochlear division** of **vestibulocochlear nerve** (VIII cranial nerve). It is also known as **auditory nerve.** Major part of the auditory pathway lies in medulla oblongata, midbrain and thalamic region.

Higher center for hearing is in temporal lobe of cerebral cortex, where the fibers of auditory pathway finally terminate. Fibers are both crossed and uncrossed, so that each cochlea is represented in the cortex on both sides.

RECEPTORS

Hair cells in organ of Corti are the receptors of the auditory sensation. Hair cells are of two types, outer hair cell and inner hair cell. All the hair cells are innervated by afferent and efferent nerve fibers. Afferent nerve fibers from the hair cells form auditory nerve (see below).

FIRST ORDER NEURONS

First order neurons of auditory pathway are the **bipolar cells of spiral ganglion,** situated in modiolus of cochlea (Fig. 173.1).

Peripheral short processes (dendrites) of the bipolar cells are distributed around hair cells of organ of Corti as afferent nerve fibers. Their long processes (axons) leave the ear as cochlear nerve fibers and enter medulla oblongata. In medulla oblongata, these fibers divide into two groups, which end on ventral cochlear nucleus and dorsal cochlear nucleus of the same side in medulla oblongata.

Efferent Nerve Fibers to Hair Cells

Efferent nerve fibers of hair cells arise from **superior olivary nucleus.** Fibers from this nucleus reach the hair cells by passing through the ventral and dorsal cochlear nuclei and cochlear nerve of the same side.

Efferent nerve fiber to outer hair cell terminates directly on the cell body and controls the motility of this cell (Chapter 174). Efferent nerve fiber to inner hair cell terminates on the auditory (afferent) nerve fiber, where it leaves the inner hair cell. It controls the impulse output from this hair cell.

SECOND ORDER NEURONS

Neurons of **dorsal** and **ventral cochlear nuclei** in the medulla oblongata form the second order neurons of auditory pathway. Axons of the second order neurons run in four different groups:

1. First group of fibers cross the midline and run to the opposite side, to form **trapezoid body.** Fibers from trapezoid body go to the **superior olivary nucleus.**

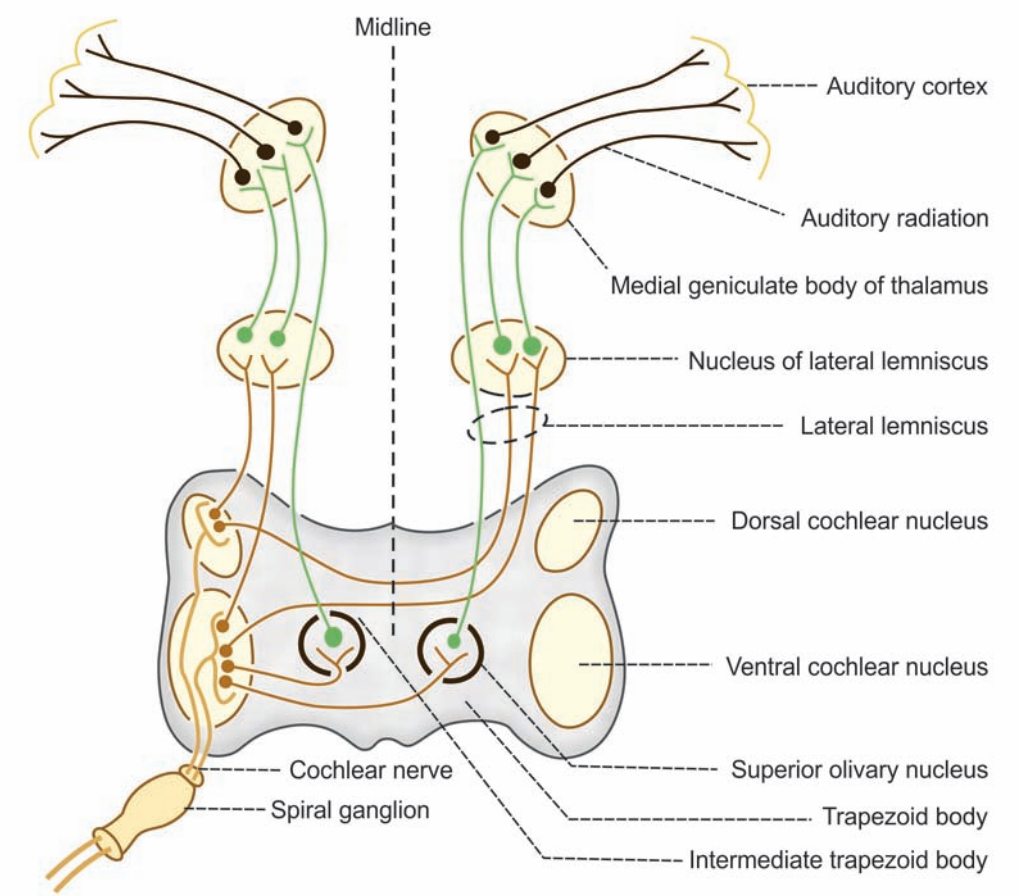

FIGURE 173.1: Auditory pathway. Blue = First order neuron, Red = Second order neuron, Green =Third order neuron, Black = Auditory radiation.

2. Second group of fibers terminate at superior olivary nucleus of same side via trapezoid body of the same side
3. Third group of fibers run in lateral lemniscus of the same side and terminate in **nucleus of lateral lemniscus** of same side
4. Fourth group of fibers run into reticular formation, cross the midline as **intermediate trapezoid fibers** and finally join the nucleus of lateral lemniscus of opposite side.

■ THIRD ORDER NEURONS

Third order neurons are in the **superior olivary nuclei** and **nucleus of lateral lemniscus.** Fibers of the third order neurons end in medial **geniculate body,** which forms the **subcortical auditory center.**

Fibers from medial geniculate body go to the temporal cortex, via internal capsule as **auditory radiation.** Some fibers from medial geniculate body go to **inferior colliculus** of tectum in midbrain. The fibers of auditory radiation are involved in reflex movement of head, in response to auditory stimuli.

■ CORTICAL AUDITORY CENTERS

Cortical auditory centers are in the temporal lobe of cerebral cortex (Chapter 152).

Auditory areas are:

1. Primary auditory area, which includes area 41, area 42 and Wernicke area
2. Secondary auditory area or auditopsychic area, which includes area 22.

Areas 41 and 42 are the primary auditory areas situated in the anterior transverse gyrus and lateral surface of superior temporal gyrus. Wernicke area is in upper part of superior temporal gyrus, posterior to areas 41 and 42. Area 22 occupies the superior temporal gyrus.

FUNCTIONS OF CORTICAL AUDITORY CENTERS

Cortical auditory centers are concerned with the perception of auditory impulses, analysis of pitch and intensity of sound and determination of source of sound.

Areas 41 and 42 are concerned with the perception of auditory impulses only. However, analysis and interpretation of sound are carried out by Wernicke area, with the help of area 22.

APPLIED PHYSIOLOGY – EFFECT OF LESION

1. Lesion of cochlear nerve causes **deafness** of the ear
2. Unilateral lesion of auditory pathway, above the level of cochlear nuclei causes **diminished hearing**
3. Degeneration of hair cells in the organ of Corti leads to **presbycusis.** Presbycusis is the gradual loss of hearing. It is common in old age.
4. Lesion in superior olivary nucleus results in **poor localization of sound.**

Mechanism of Hearing

Chapter 174

INTRODUCTION

Sound waves travel through external auditory meatus and produce vibrations in the tympanic membrane. Vibrations from tympanic membrane travel through malleus and incus and reach the stapes resulting in the movement of stapes. Movements of stapes produce vibrations in the fluids of cochlea. These vibrations stimulate the hair cells in organ of Corti. This, in turn, causes generation of action potential (auditory impulses) in the auditory nerve fibers. When auditory impulses reach the cerebral cortex, the perception of hearing occurs.

Thus, during the process of hearing, ear converts energy of sound waves into action potentials in auditory nerve fibers. This process is called **sound transduction.**

ROLE OF EXTERNAL EAR

External ear directs the sound waves towards tympanic membrane. Sound waves produce pressure changes over the surface of tympanic membrane. Accumulation of wax prevents conduction of sound. In many animals, the auricle (pinna) can be turned to locate the source of sound. Auricle can be folded to avoid unwanted sound. But in man, the extrinsic and intrinsic muscles of auricle are rudimentary and the movement is not possible.

ROLE OF MIDDLE EAR

ROLE OF TYMPANIC MEMBRANE

Due to the pressure changes produced by sound waves, tympanic membrane vibrates, i.e. it moves in and out of middle ear. Thus, tympanic membrane acts as a **resonator** that reproduces the vibration of sound.

ROLE OF AUDITORY OSSICLES

Vibrations set up in tympanic membrane are transmitted through the malleus and incus and reach the stapes, causing to and fro movement of stapes against oval window and against perilymph present in scala vestibuli of cochlea.

Impedance Matching

Impedance matching is the process by which tympanic membrane and auditory ossicles convert the sound energy into mechanical vibrations in cochlear fluid with minimum loss of energy by matching the impedance offered by fluid.

Impedance means obstruction or opposition to the passage of sound waves. When sound waves reach inner ear, the fluid (perilymph) in cochlea offers impedance, i.e. the fluid resists the transmission of sound due to its own inertia. Tympanic membrane and auditory ossicles effectively reduce the sound impedance.

Sound waves are conducted from external ear to inner ear, with an impedance of only 40%. Remaining 60% of sound energy developed in tympanic membrane is transmitted to cochlear fluid by the ossicles. Thus, along with the help of tympanic membrane, ossicles match the impedance offered by fluid to a great extent. It is because, the ossicles act like a **lever system** so that stapes exerts a greater force (pressure) against the cochlear fluid. This results in generation of vibrations in the cochlear fluid. The increased force is very essential to set up the vibrations in cochlear fluid because of higher inertia of the fluid.

Force exerted by footplate of stapes on cochlear fluid is 17 to 22 times greater than the force exerted by sound waves at the tympanic membrane. It is because of two structural features of ossicles:

1. Head of malleus is longer than long process of incus so that a higher force is generated in small structure
2. Surface area of tympanic membrane (55 sq mm) is larger compared to that of footplate of stapes (3.2 sq mm). So the pressure increases when force is applied to small area.

Thus, the tympanic membrane and the auditory ossicles are capable of converting the sound energy into mechanical vibrations in cochlear fluid with minimum loss of energy.

Significance of impedance matching

Impedance matching is the most important function of middle ear. Because of impedance matching the sound waves (stimuli) are transmitted to cochlea with minimum loss of intensity. Without impedance matching conductive deafness occurs.

Types of Conduction

Conduction of sound from external ear to internal ear through middle ear occurs by three routes:

1. Ossicular conduction
2. Air conduction
3. Bone conduction.

1. *Ossicular conduction*

Ossicular conduction is the conduction of sound waves through middle ear by auditory ossicles. In normal conditions, the sound waves are conducted through auditory ossicles.

2. *Air conduction*

Air conduction is the conduction of sound waves through air in middle ear. If the ossicular chain is broken, conduction occurs in an alternate route of air conduction. Air conduction is common in otosclerosis. **Otosclerosis** is the disease associated with fixation of stapes to oval window.

3. *Bone conduction*

Bone conduction is the conduction of sound waves through middle ear by bones. It occurs when middle ear is affected. In this type of conduction, sound waves are transmitted to cochlear fluid by the vibrations set up in skull bones. Bone conduction is tested by placing vibrating tuning forks or other vibrating bodies directly on the skull. This route plays a role in transmission of extremely loud sounds.

ROLE OF EUSTACHIAN TUBE

Eustachian tube is not concerned with hearing directly. However, it is responsible for **equalizing the pressure** on either side of tympanic membrane.

ROLE OF INNER EAR

TRAVELING WAVE

Movement of footplate of stapes against oval window causes movement of perilymph in scala vestibuli. This fluid does not move all the way from oval window to round window through helicotrema. It immediately hits the vestibular membrane near oval window. This causes movement of fluid in scala media, since the vestibular membrane is flexible.

Movement of fluid in scala media causes bulging of basal portion of basilar membrane towards scala tympani (Fig. 174.1).

Bulging of basilar membrane increases the elastic tension in basilar fibers in that portion of basilar membrane. Elastic tension in basilar fibers initiates a wave, which travels along basilar membrane towards the helicotrema like that of arterial pulse wave. It is called **traveling wave.**

Resonance Point

Resonance point is the part of basilar membrane, which is activated by traveling wave. In the beginning, each traveling wave is weak (Fig. 174.2). While traveling through basilar membrane from base towards apex (helicotrema), the wave becomes stronger and stronger and at one point (resonance point) of basilar membrane, it becomes very strong and activates the basilar membrane. This resonance point of basilar membrane immediately vibrates back and forth. The traveling wave stops here and does not travel further.

Distance between stapes and resonance point is inversely proportional to frequency of sound waves reaching the ear. Traveling wave generated by high-pitched sound disappears near the base of the cochlea. Wave generated by medium-pitched sound reaches half of the way and the wave generated by low-pitched sound travels the entire distance of basilar membrane.

EXCITATION OF HAIR CELLS

Stereocilia of hair cells in organ of Corti are embedded in tectorial membrane. Hair cells are tightly fixed by cuticular lamina reticularis and the pillar cells or rods of Corti.

When traveling wave causes vibration of basilar membrane at the resonance point, the basilar fiber, rods of Corti, hair cells and lamina reticularis move as a single unit. It causes movements of stereocilia leading to excitement of hair cells and generation of receptor potential.

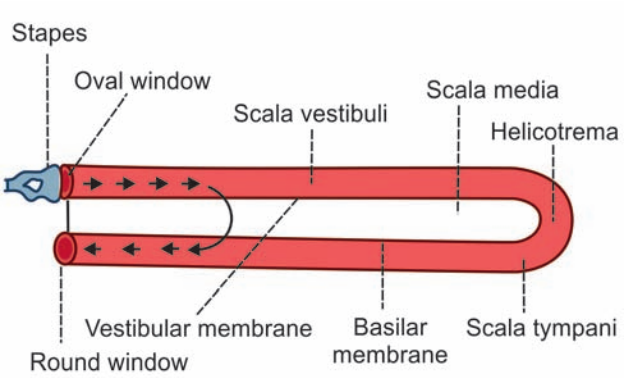

FIGURE 174.1: Diagrammatic representation of cochlea. Arrows show displacement of fluid

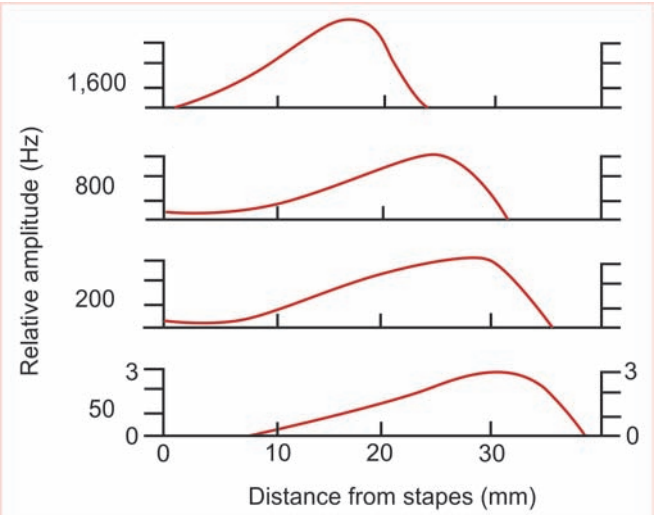

FIGURE 174.2: Traveling waves for different frequencies of sound

ELECTRICAL EVENTS DURING PROCESS OF HEARING

SOUND TRANSDUCTION

Sound transduction is a type of sensory transduction (Chapter 139) in the hair cell (receptor cells) in organ of Corti by which the sound energy is converted into action potentials in the auditory nerve fiber.

Electrical Events of Sound Transduction

Three types of electrical events that occur during sound transduction are:

1. Receptor potential or the cochlear microphonic potential
2. Endocochlear potential or endolymphatic potential
3. Action potential in auditory nerve fiber.

■ RECEPTOR POTENTIAL OR COCHLEAR MICROPHONIC POTENTIAL

Receptor potential or cochlear microphonic potential is the **mild depolarization** that is developed in the hair cells of cochlea when sound waves are transmitted to internal ear. Resting membrane potential in hair cells is –60 mV. Sensory transduction mechanism in cochlear receptor cells is different from the mechanism in other sensory receptors.

When sound waves reach internal ear traveling wave is produced. It causes vibration of basilar membrane, which moves stereocilia of hair cells away from modiolus (towards kinocilium). It causes opening of mechanically gated potassium channels (Chapter 3) and influx of potassium ions from endolymph, which contains large amount of potassium ions. Influx of potassium ions causes development of **mild depolarization** (receptor potential) in hair cells up to –50 mV.

Cochlear microphonic potential is non-propagative. But, it causes generation of action potential in auditory nerve fiber. Due to depolarization hair cells release a neurotransmitter, which generates action potential in the auditory nerve fibers. Probable neurotransmitter may be **gulatamate.**

Movement of stereocilia away from modiolus (towards kinocilium) causes **depolarization** in hair cells. Movement of stereocilia in the opposite direction (away from kinocilium) causes **hyperpolarization.** Ionic basis of hyperpolarization is not clearly known. It is suggested that **calcium** plays an important role in this process. Hyperpolarization in hair cells stops generation of action potential in auditory nerve fiber.

■ ROLE OF HAIR CELLS

Inner hair cells and outer hair cells have different roles during sound transduction.

Role of Inner Hair Cells

Inner hair cells are responsible for sound transduction, i.e. these receptor cells are the primary sensory cells, which cause the generation of action potential in auditory nerve fibers.

Role of Outer Hair Cells

Outer hair cells have a different action. These hair cells are shortened during depolarization and elongated during hyperpolarization. This process is called electromotility or mechanoelectrical transduction. This action of outer hair cells facilitates the movement of basilar membrane and increases the amplitude and sharpness of sound. Hence, the outer hair cells are collectively called **cochlear amplifier.** The electromotility of hair cell is due to the presence of a contractile protein, **prestin** (named after a musical notation **presto**).

Role of Efferent Nerve Fibers of Hair Cells

Efferent nerve fibers (Chapter 173) of hair cells also play important role during sound transduction by releasing acetylcholine.

Efferent nerve fiber to inner hair cell terminates on the auditory (afferent) nerve fiber where it leaves the inner hair cell. It controls the generation of action potential in auditory nerve fiber by inhibiting the release of glutamate from inner hair cells.

Efferent nerve fiber to outer hair cell terminates directly on the cell body. It inhibits the electromotility of this cell.

■ ENDOCOCHLEAR POTENTIAL OR ENDOLYMPHATIC POTENTIAL

Endocochlear or endolymphatic potential is the electrical potential developed in fluids outside the hair cells.

Cochlear Fluids

Cochlear fluids are the extracellular fluids in the inner ear. These fluids are perilymph and endolymph, which have different composition.

Perilymph

Scala vestibuli and scala tympani are filled with perilymph, which is similar to ECF in composition with high concentration of sodium ions.

Endolymph

Scala media is filled with endolymph, which contains high concentration of potassium and low concentration of sodium. It is due to continuous secretion of potassium ions by stria vascularis into scala media.

Electrical Potential

Difference in potassium concentration is responsible for the development of an electrical potential difference between endolymph and perilymph. Potential in endolymph is positive up to +80 mV.

Significance of Endocochlear Potential

Lower portion of the hair cells is bathed by perilymph. Head portion of hair cells penetrates the lamina reticularis and it is bathed by endolymph (Fig. 172.3). Endolymph has a positive potential (+80 mV).

So inside the hair cells, the electrical potential is –60 mV in comparison to that of perilymph and –140 mV in comparison to that of endolymph. High potential difference sensitizes the hair cells so that, the excitability of hair cells increases. It also increases the response of cells even to slight movement of stereocilia.

■ ACTION POTENTIAL IN AUDITORY NERVE FIBER

Action potential in auditory nerve fiber is generated by cochlear microphonic potential. It obeys **all-or-none law** and has a definite threshold and refractory period.

Action potential to a click sound with moderate intensity level consists of three successive spike potentials called N_1 N_2 N_3 representing synchronous repetitive firing in many fibers.

At high frequency, the synchronization of action potential disappears and single spike occurs. Action potential appears 0.5 to 1 millisecond after the development of cochlear microphonic potential.

■ PROPERTIES OF SOUND

Sound has two basic properties:

1. **Pitch,** which depends upon the frequency of sound waves. Frequency of sound is expressed in **hertz.** Frequency of sound audible to human ear lies between 20 and 20,000 Hz or cycles/second. The range of greatest sensitivity lies between 2,000 and 3,000 Hz (cycles/second).
2. **Loudness** or **intensity,** which depends upon the amplitude of sound waves. It is expressed in **decibel** (dB). The threshold intensity of sound wave is not constant. It varies in accordance to the frequency of sound.

■ APPRECIATION OF PITCH OF THE SOUND – THEORIES OF HEARING

Many theories are postulated to explain the mechanism by which the pitch of the sound is appreciated or the frequency is analyzed. These theories are generally classified into two groups. According to the first group, the analysis of sound frequency is the function of cerebral cortex and the cochlea merely transmits the sound.

According to the second group of theories, the frequency analysis is done by cochlea, which later sends the information to cerebral cortex.

■ THEORIES OF FIRST GROUP

1. *Telephone Theory of Rutherford*

Telephone theory was postulated by **Rutherford** in 1880. It is also called **frequency theory.** According to this theory, the cochlea plays a simple role of a **telephone transmitter.**

In telephone, sound vibrations are converted into electrical impulses, which are transmitted by cables to the receiving end. There the receiver instrument converts the electrical impulses back into sound waves. Similarly, cochlea just converts the sound waves into electrical impulses of same frequency. Impulses are transmitted by auditory nerve fibers to cerebral cortex, where perception and analysis of sound occur.

It is believed that, the nerve fibers can transmit maximum of 1,000 impulses per second. Thus, the telephone theory fails to explain the transmission of sound waves with frequency above 1,000 cycles per second. So, a second theory was postulated.

2. *Volley Theory*

In 1949, **Wever** postulated this theory. **Volley** means groups. According to this theory, the impulses of sound waves with frequency above 1,000 cycles per second are transmitted by different groups of nerve fibers. However, this theory has no evidence to prove it. Thus, these two theories were not accepted by many physiologists.

■ THEORIES OF SECOND GROUP

1. *Resonance Theory of Helmholtz*

Resonance theory was the first theory of hearing to emerge in 1863. According to **Helmholtz,** analysis of sound frequency is the function of cochlea. Basilar membrane contains many basilar fibers. Helmholtz named these basilar fibers resonators and compared them with the resonators of piano.

When a string in piano is struck, sound with a particular note is produced. Similarly, when the sound with a particular frequency is applied, the basilar fibers in a particular portion of basilar membrane are stimulated.

Resonance theory was not accepted because the individual resonators could not be identified in cochlea. Gradually, this theory was modified into another theory called the place theory, which is more widely accepted.

2. *Place Theory*

According to this theory, nerve fibers from different portions (places) of organ of Corti on basilar membrane give response to sounds of different frequency. Accordingly, corresponding nerve fiber from organ of Corti gives information to the brain regarding the portion of organ of Corti that is stimulated. Many experimental evidences are available to support place theory.

Experimental evidences supporting place theory

i. If a person is exposed to a loud noise of a particular frequency for a long period, he becomes deaf for that frequency. It is found that the specific portion of organ of Corti is destroyed as in the case of **boilermaker's disease.**
ii. In experimental animals, destruction of a portion of organ of Corti occurs by exposing the animal to loud noise of a particular frequency
iii. In human **high-tone deafness,** there is degeneration of organ of Corti near the base of cochlea or degeneration of nerve supplying the cochlea near the base
iv. During exposure to high-frequency sound, cochlear microphonic potentials show greater voltage in hair cells near base of the cochlea. Also, during the exposure to low-frequency sound, cochlear microphonic potentials show greater voltage in hair cells near apex of the cochlea.
v. There is point-to-point representation of basilar membrane in auditory cortex.

3. *Traveling Wave Theory*

From place theory, emerged yet another theory called the traveling wave theory. This theory explains how the traveling wave is generated in the basilar membrane. Development, generation, movement and disappearance of traveling wave are already described earlier in this chapter.

■ APPRECIATION OF LOUDNESS OF SOUND

Appreciation of loudness of sound depends upon the activities of auditory nerve fibers. Intensity or loudness of sound correlates with two factors:

1. Rate of discharge from the individual fibers of auditory nerve
2. Total number of nerve fibers discharging.

When loudness of sound increases, it produces large vibrations, which spread over longer area of basilar membrane. This activates large number of hair cells and recruits more number of auditory nerve fibers. So, the frequency of action potential is also increased.

■ LOCALIZATION OF SOUND

Sound localization is the ability to detect the source from where sound is produced or the direction through which sound wave is traveling. It is important for survival and it helps to protect us from moving objects such as vehicles.

Cerebral cortex and medial geniculate body are responsible for localization of sound.

Auditory Defects

Chapter 175

- TYPES AND CAUSES OF AUDITORY DEFECTS
  - CONDUCTION DEAFNESS
  - NERVE DEAFNESS
- TESTS FOR HEARING
  - RINNE TEST
  - WEBER TEST
  - AUDIOMETRY

TYPES AND CAUSES OF AUDITORY DEFECTS

Auditory defects may be either partial or complete. Auditory defects are of two types:

1. Conduction deafness
2. Nerve deafness.

CONDUCTION DEAFNESS

Conduction deafness is the type of deafness that occurs due to impairment in transmission of sound waves in the external ear or middle ear.

Causes for Conduction Deafness

i. Obstruction of external auditory meatus with dry wax or foreign bodies
ii. Thickening of tympanic membrane due to repeated middle ear infection
iii. Perforation of tympanic membrane due to inequality of pressure on either side
iv. **Otitis media** (inflammation of middle ear)
v. **Otosclerosis** (fixation of footplate of stapes against oval window) due to ankylosis. **Ankylosis** means the abnormal immobility and consolidation of a joint.

NERVE DEAFNESS

Nerve deafness is the deafness caused by damage of any structure in cochlea, such as hair cell, organ of Corti, basilar membrane or cochlear duct or the lesion in the auditory pathway.

Causes for Nerve Deafness

i. Degeneration of hair cells due to some antibiotics like streptomycin and gentamicin
ii. Damage of cochlea by prolonged exposure to loud noise
iii. Tumor affecting VIII cranial nerve.

TESTS FOR HEARING

There are various tests to assess the sensation of hearing. However, some simple tests called bedside tests are usually carried before doing conventional types of hearing tests. Such simple tests are useful to know whether the hearing is normal or less.

Bedside tests:

1. Whispering test
2. Tickling of watch test.

Whispering Test

The examiner stands about 60 cm away from the subject at his side and whispers some words. If the subject is not able to hear the whisper, then hearing deficit is suspected.

Tickling of Watch Test

Wrist watch with tickling sound is kept near the ear of the subject. The subject suffering from hearing defects cannot hear the tickling sound of watch.

Routine Tests for Hearing

Routine tests for hearing are of three types:

1. Rinne test
2. Weber test
3. Audiometry.

First two tests are done by using a tuning fork with high frequency. Mostly, a tuning fork with 512 cycles per second is used. By turning fork tests, only the nature of auditory defect is determined. By audiometry, both nature and severity of auditory defects can be determined.

■ RINNE TEST

Base of a vibrating tuning fork is placed on mastoid process, until the subject cannot feel the vibration and cannot hear the sound. When the subject does not hear the sound any more, the tuning fork is held in air in front of the ear of same side. Normal person hears vibration in air even after the bone conduction ceases because, in normal conditions, air conduction via ossicles is better than bone conduction. But in conduction deafness, the vibrations in air are not heard after cessation of bone conduction. Thus in conduction deafness, the bone conduction is better than air conduction.

In nerve deafness, both air conduction and bone conduction are diminished or lost.

■ WEBER TEST

Base of a vibrating tuning fork is placed on the vertex of skull or the middle of forehead. Normal person hears the sound equally on both sides. In unilateral conduction deafness (deafness in one ear), the sound is heard louder in diseased ear. In unaffected ear, there is a masking effect of environmental noise. So, the sound through bone conduction is not heard as clearly as on the affected side. In affected side, the sound is louder due to the absence of masking effect of environmental noise.

During unilateral nerve deafness, sound is heard louder in the normal ear.

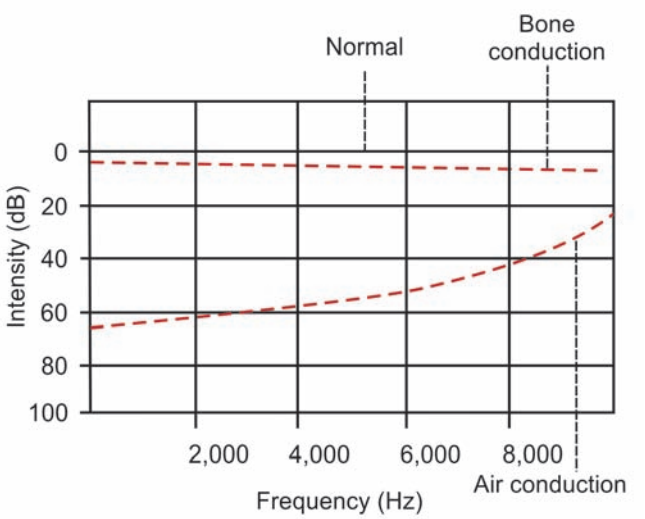

FIGURE 175.1: Audiogram in a patient with conductive deafness

■ AUDIOMETRY

Audiometry is the technique used to determine the nature and the severity of auditory defect. An instrument called **audiometer** is used. This instrument is an electronic **function generator** or **oscillator,** connected to an ear phone. This instrument is capable of generating sound waves of different frequencies from lowest to highest.

Intensity (loudness or volume) of sound at each frequency is adjusted on the basis of previous studies in normal persons.

Thus, before calibrating the instrument, minimum (threshold) volume or intensity or loudness, for each frequency of sound heard by normal persons is determined. Minimum intensity is set in the instrument as zero. Now, while testing the patient, the loudness is increased above zero level (Fig. 175.1). Intensity of sound is expressed in decibel (dB).

At a particular frequency, if the patient hears the sound with loudness of 30 dB above zero level, the person is said to have hearing loss of 30 dB for that particular frequency. During the tests by audiometer, the subject's ability to hear the sounds with 8 to 10 different frequencies is observed and the hearing loss is determined for each frequency. By using these values, the audiogram is plotted.

Audiometer has an **electronic vibrator** also. It is used to test the bone conduction from mastoid process into the cochlea.

Chapter 176

Sensation of Taste

- TASTE BUDS
- PATHWAY FOR TASTE
- PRIMARY TASTE SENSATIONS
- DISCRIMINATION OF DIFFERENT TASTE SENSATIONS
- TASTE SENSATIONS AND CHEMICAL CONSTITUTIONS
- TASTE TRANSDUCTION
- APPLIED PHYSIOLOGY – ABNORMALITIES OF TASTE SENSATION

TASTE BUDS

Sense organs for taste or **gustatory sensation** are the taste buds. Taste buds are ovoid bodies with a diameter of 50 µ to 70 µ. In adults, about 10,000 taste buds are present and the number is more in children. In old age, many taste buds degenerate and the taste sensitivity decreases.

SITUATION OF TASTE BUDS

Most of the taste buds are present on the papillae of tongue. Taste buds are also situated in the mucosa of epiglottis, palate, pharynx and the proximal part of esophagus.

Types of papillae located on tongue:

1. Filiform papillae
2. Fungiform papillae
3. Circumvallate papillae.

1. *Filiform Papillae*

Filiform papillae are small and conical-shaped papillae, situated over the dorsum of tongue. These papillae contain less number of taste buds (only a few).

2. *Fungiform Papillae*

Fungiform papillae are round in shape and are situated over the anterior surface of tongue near the tip. Numerous fungiform papillae are present. Each papilla contains moderate number of taste buds (up to 10).

3. *Circumvallate Papillae*

Circumvallate papillae are large structures present on the posterior part of tongue and are many in number. These papillae are arranged in the shape of 'V'. Each papilla contains many taste buds (up to 100).

STRUCTURE OF TASTE BUD

Taste bud is a bundle of taste receptor cells, with supporting cells embedded in the epithelial covering of the papillae (Fig. 176.1). Each taste bud contains about 40 cells, which are the modified epithelial cells. Cells of taste bud are divided into four groups:

Type of Cells in Taste Bud

1. Type I cells or sustentacular cells
2. Type II cells
3. Type III cells
4. Type IV cells or basal cells.

Type I cells and type IV cells are supporting cells. Type III cells are the **taste receptor cells.** Function of type II cell is unknown. Type I, II and III cells have microvilli, which project into an opening in epithelium covering the tongue. This opening is called **taste pore.** Neck of each cell is attached to the neck of other. All the cells of taste bud are surrounded by epithelial cells. There are tight junctions between epithelial cells and the neck portion of the type I, II and III cells, so that only the tip of these cells are exposed to fluid in oral cavity.

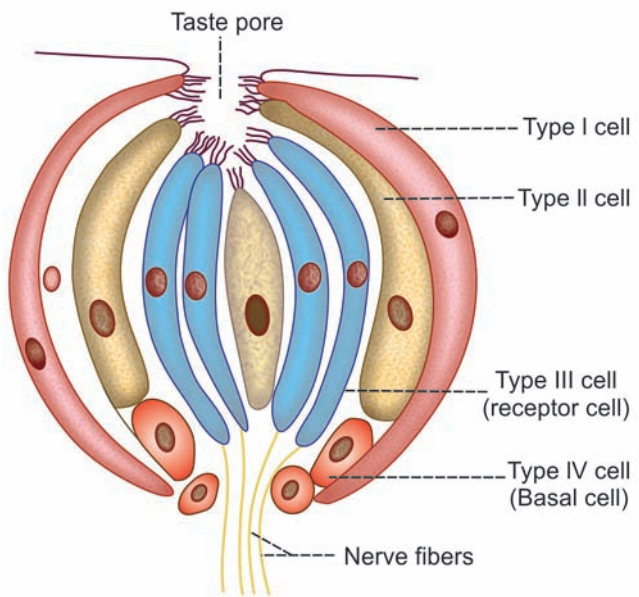

FIGURE 176.1: Taste bud

Cells of taste buds undergo constant cycle of growth, apoptosis and regeneration.

PATHWAY FOR TASTE

RECEPTORS

Receptors for taste sensation are the type III cells of taste buds. Each taste bud is innervated by about 50 sensory nerve fibers and each nerve fiber supplies at least five taste buds through its terminals.

FIRST ORDER NEURON

First order neurons of taste pathway are in the nuclei of three different cranial nerves, situated in medulla oblongata. Dendrites of the neurons are distributed to the taste buds. After arising from taste buds, the fibers reach the cranial nerve nuclei by running along the following nerves (Fig. 176.2):

1. **Chorda tympani fibers** of facial nerve, which run from anterior two third of tongue
2. **Glossopharyngeal nerve fibers,** which run from posterior one third of the tongue
3. **Vagal fibers,** which run from taste buds in other regions.

Axons from first order neurons in the nuclei of these nerves run together in medulla oblongata and terminate in the nucleus of **tractus solitarius.**

SECOND ORDER NEURON

Second order neurons are in the nucleus of tractus solitarius. Axons of second order neurons run through **medial lemniscus** and terminate in **posteroventral nucleus** of thalamus.

THIRD ORDER NEURON

Third order neurons are in the posteroventral nucleus of thalamus. Axons from third order neurons project into **parietal lobe** of the cerebral cortex.

TASTE CENTER

Center for taste sensation is in opercular insular cortex, i.e. in the lower part of postcentral gyrus, which receives cutaneous sensations from face. Thus, the taste fibers do not have an independent cortical projection.

PRIMARY TASTE SENSATIONS

Primary or fundamental taste sensations are divided into five types:

1. Sweet
2. Salt

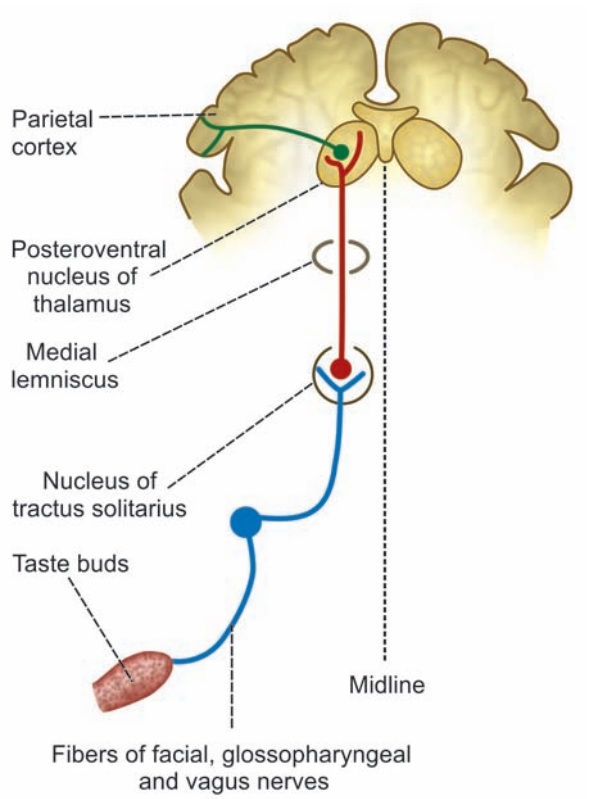

FIGURE 176.2: Pathway for taste sensation

3. Sour
4. Bitter
5. Umami.

Man can perceive more than 100 different tastes. Other taste sensations are just the combination of two or more primary taste sensations.

Combination of Taste Sensation with Other Sensations

Sometimes, taste sensation combines with other sensations to give rise to a different sensation. For example, combination of taste, smell and touch senses, gives rise to **sensation of flavor.** Combination of taste with pain gives rise to **sensation of ginger.**

■ DISCRIMINATION OF DIFFERENT TASTE SENSATIONS

Earlier, it was believed that different areas of tongue were specialized for different taste sensation. Now it is clear that all areas of tongue give response to all types of taste sensations. Usually, in low concentration of taste substance, each taste bud gives response to one primary taste stimulus. However, in high concentration, the taste buds give response to more than one type of taste stimuli.

It is also clear now that each afferent nerve fiber from the taste buds carry impulses of one taste sensation.

■ TASTE SENSATIONS AND CHEMICAL CONSTITUTIONS

Substances causing sour or salt tastes are mostly electrolytes. Bitter and sweet tastes are caused by electrolytes or non-electrolytes.

■ SWEET TASTE

Sweet taste is produced mainly by organic substances like monosaccharides, polysaccharides, glycerol, alcohol, aldehydes, ketones and chloroform. Inorganic substances, which produce sweet taste are lead and beryllium.

■ SALT TASTE

Salt taste is produced by chlorides of sodium, potassium and ammonium, nitrates of sodium and potassium. Some sulfates, bromides and iodides also produce salt taste.

■ SOUR TASTE

Sour taste is produced because of hydrogen ions in acids and acid salts.

■ BITTER TASTE

Bitter taste is produced by organic substances like quinine, strychnine, morphine, glucosides, picric acid and bile salts and inorganic substances like salts of calcium, magnesium and ammonium. Bitterness of the salts is mainly due to cations.

■ UMAMI

Umami is the recently recognized taste sensation. Umami is a Japanese word, meaning 'delicious'. Receptors of this taste sensation respond to glutamate, particularly **monosodium glutamate (MSG),** which is a common ingredient in Asian food. However, excess MSG consumption is proved to produce Chinese restaurant syndrome in some people taking Chinese food regularly. Common symptoms are headache, flushing, sweating, perioral numbness, chest pain. In severe conditions, airway swelling and obstruction and cardiac arrhythmia occur.

Threshold for Taste Sensations

Sweet taste	Sugar	: 1 in 200 dilution
Salt taste	Sodium chloride	: 1 in 400 dilution
Sour taste	Hydrochloric acid	: 1 in 15,000 dilution
Bitter taste	Quinine	: 1 in 2,000,000 dilution.

Bitter taste has very low threshold and sweet taste has a high threshold. Threshold for umani is not known.

■ TASTE TRANSDUCTION

Taste transduction is the process by which taste receptor converts chemical energy into action potentials in the taste nerve fiber. Receptors of taste sensation are chemoreceptors, which are stimulated by substances dissolved in mouth by saliva. The dissolved substances act on microvilli of taste receptors exposed in the taste pore. It causes the development of receptor potential in the receptor cells. This in turn, is responsible for the generation of action potential in the sensory neurons.

Taste Receptor

Generally, taste receptor is a **G-protein coupled receptor** (GPCR). It is also called **G protein gustducin.**

However, several other receptors are also involved in taste sensation. Transduction mechanism is different in each taste receptor cells.

SWEET RECEPTOR

Receptor for sweet taste is **GPCR.** The sweet substances bind to receptor and cause depolarization via cyclic AMP.

SALT RECEPTOR

Receptor for salt taste is called **epithelial sodium channel (ENaC).** It acts like ENaC receptors in other parts of the body. When sodium enters, this receptor releases glutamate, which causes depolarization.

SOUR RECEPTOR

Sour sensation also has the same ENaC receptor. The proton (hydrogen) enters the receptor and causes depolarization. It is believed that besides ENaC, other receptors such as **hyperpolarization-activated cyclic nucleotide-gated cation channel (HCN)** also are involved in sour sensation.

BITTER RECEPTOR

Bitter receptor is a GPCR. In bitter receptor, the sour substances activate phospholipase C through G proteins. It causes production of inositol triphosphate (IP_3), which initiates depolarization by releasing calcium ions.

UMAMI RECEPTOR

Umami receptor is called **metabotropic glutamate receptor (mGluR4).** Glutamate causes depolarization of this receptor. Exact mechanism of depolarization is not clearly understood. Activation of umami taste receptor is intensified by the presence of guanosine monophosphate (GMP) and inosine monophosphate (IMP).

APPLIED PHYSIOLOGY – ABNORMALITIES OF TASTE SENSATION

AGEUSIA

Loss of taste sensation is called ageusia. Taste buds in anterior two thirds of the tongue are innervated by the chorda tympani branch of facial nerve. Chorda tympani nerve receives taste fibers from tongue via lingual branch of mandibular division of trigeminal nerve. So, the lesion in facial nerve, chorda tympani or mandibular division of trigeminal nerve causes loss of taste sensation in the anterior two third of the tongue. Lesion in glossopharyngeal nerve leads to loss of taste in the posterior one third of the tongue.

Temporary loss of taste sensation occurs due to the drugs like captopril and penicillamine, which contain sulfhydryl group of substances.

HYPOGEUSIA

Hypoguesia is the decrease in taste sensation. It is due to increase in threshold for different taste sensations. However, the taste sensation is not completely lost.

TASTE BLINDNESS

Taste blindness is a rare genetic disorder in which the ability to recognize substances by taste is lost.

DYSGEUSIA

Disturbance in the taste sensation is called dysgeusia. It is found in temporal lobe syndrome, particularly when the anterior region of temporal lobe is affected. In this condition, the paroxysmal hallucinations of taste and smell occur, which are usually unpleasant.

Sensation of Smell

Chapter 177

OLFACTORY RECEPTORS

Olfactory receptors are situated in **olfactory mucus membrane,** which is the modified mucus membrane that lines upper part of nostril. Olfactory mucus membrane consists of 10 to 20 millions of olfactory receptor cells supported by the sustentacular cells. Mucosa also contains mucus-secreting **Bowman glands** (Fig. 177.1).

Olfactory receptor cell is a bipolar neuron. Dendrite of this neuron is short and it has an expanded end called olfactory rod. From olfactory rod, about 10 to 12 cilia arise. Cilia are non-myelinated, with a length of 2 μ and a diameter of 0.1 μ. These cilia project to the surface of olfactory mucus membrane.

Mucus secreted by Bowman glands continuously lines the olfactory mucosa. Mucus contains some proteins, which increase the actions of odoriferous substances on receptor cells.

VOMERONASAL ORGAN

Vomeronasal organ is an **accessory olfactory organ** found in many animals including mammals. This organ was discovered in 1813, by a Danish physician **Ludwig Jacobson,** hence it is also called **Jacobson organ.** It is enclosed in a cartilaginous capsule, which opens into the base of nasal cavity. Olfactory receptors of this organ are sensitive to non-volatile substances such as scents and pheromones. Vomeronasal organ helps the animals to detect even the trace quantities of chemicals. Impulses from this organ are sent to amygdala and hypothalamus via accessory olfactory bulb.

VOMERONASAL ORGAN IN HUMAN BEINGS

In human beings, the vomeronasal organ was considered as vestigial or non-functional. Recently, it is found that this organ is present in the form of vomeronasal pits on the anterior part of nasal septum. Receptors of vomeronasal pit detect odorless human **pheromones** or **vomeropherins,** at a very low concentration in air. Refer Chapter 62 for details of pheromones. The subconscious detection of odorless chemical messengers in air is considered as the **sixth sense** in human beings.

OLFACTORY PATHWAY

Axons of **bipolar olfactory receptors** pierce the **cribriform plate** of ethmoid bone and reach the

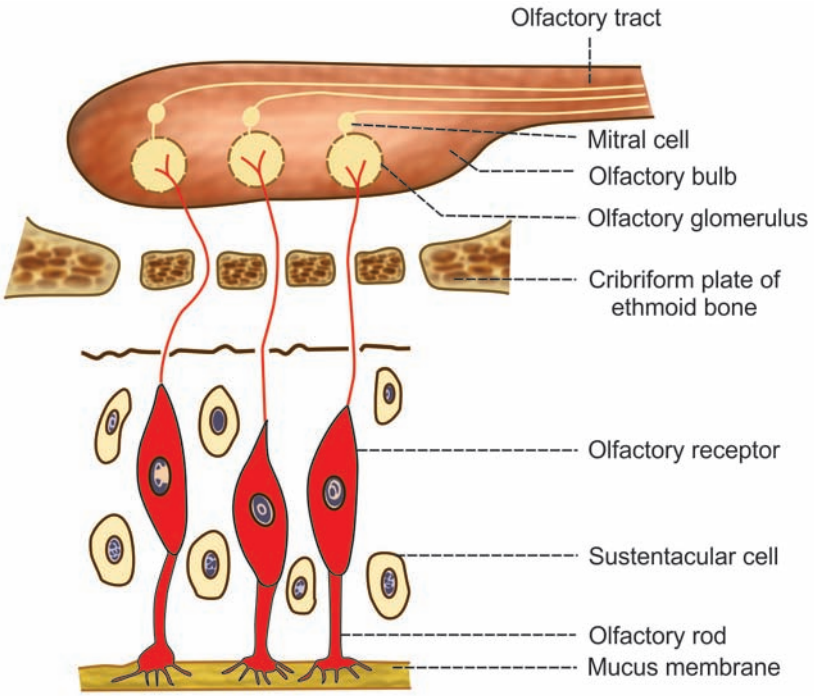

FIGURE 177.1: Olfactory mucus membrane and pathway for olfactory sensation

olfactory bulb. Here, the axons synapse with dendrites of **mitral cells.** Different groups of these synapses form globular structures called **olfactory glomeruli.**

Axons of mitral cells leave the olfactory bulb and form **olfactory tract.** Olfactory tract runs backward and ends in **olfactory cortex,** through the intermediate and lateral **olfactory stria.**

Olfactory cortex includes the structures, which form a part of limbic system. These structures are anterior olfactory nucleus, prepyriform cortex, olfactory tubercle and amygdala.

■ OLFACTORY TRANSDUCTION

Olfactory transduction is the process by which olfactory receptor converts chemical energy into action potentials in olfactory nerve fiber. The odoriferous substance stimulates the olfactory receptors, only if it dissolves in mucus, covering the olfactory mucus membrane. Molecules of dissolved substance, bind with receptor proteins in the cilia and form substance-receptor complex. Substance-receptor complex activates adenyl cyclase that causes the formation of cyclic AMP. Cyclic AMP in turn, causes opening of sodium channels, leading to influx of sodium and generation of receptor potential.

Receptor potential causes generation of action potential in the axon of bipolar neuron.

■ CLASSIFICATION OF ODOR

Odor is classified into various types. Substances producing different types of odor are:

1. Aromatic or resinous odor: Camphor, lavender, clove and bitter almonds
2. Ambrosial odor: Musk
3. Burning odor: Burning feathers, tobacco, roasted coffee and meat
4. Ethereal odor: Fruits, ethers and beeswax
5. Fragrant or balsamic odor: Flowers and perfumes
6. Garlic odor: Garlic, onion and sulfur
7. Goat odor: Caproic acid and sweet cheese
8. Nauseating odor: Decayed vegetables and feces
9. Repulsive odor: Bed bug.

■ THRESHOLD FOR OLFACTORY SENSATION

Ethyl ether	:	5.8 mg/L of air
Chloroform	:	3.3 mg/L of air
Peppermint oil	:	0.02 mg/L of air
Butyric acid	:	0.009 mg/L of air
Artificial musk	:	0.00004 mg/L of air
Methyl mercaptan	:	0.0000004 mg/L of air.

Thus, **methyl mercaptan** produces olfactory sensation even at a low concentration of 0.0000004 mg/L of air.

■ ADAPTATION

Olfactory receptors are phasic receptors and adapt very rapidly. Within one second, the adaptation occurs up to 50%.

■ APPLIED PHYSIOLOGY – ABNORMALITIES OF OLFACTORY SENSATION

■ ANOSMIA

Anosmia refers to total loss of sensation of smell, i.e. inability to recognize or detect any odor. It may be temporary or permanent. **Temporary anosmia** is due to obstruction of nose, which occurs during common cold, nasal sinus and allergic conditions. **Permanent anosmia** occurs during lesion in olfactory tract, meningitis and degenerative conditions such as Parkinson disease and Alzheimer disease.

■ HYPOSMIA

Hyposmia is the reduced ability to recognize and to detect any odor. The odors can be detected only at higher concentrations. It is the most common disorder of smell. Hyposmia also may be temporary or permanent. It occurs due to same causes of anosmia.

■ HYPEROSMIA

Hyperosmia is the increased or exaggerated olfactory sensation. It is also called **olfactory hyperesthesia.** It occurs in brain injury, epilepsy and neurotic conditions.

QUESTIONS IN SPECIAL SENSES

LONG QUESTIONS

1. Draw a diagram of visual pathway and explain it. Indicate the effects of lesions at different levels of optic pathway.
2. Give an account of accommodation. Add a note on presbyopia.
3. Explain the auditory pathway with suitable diagram. Add a note on auditory defects.
4. Explain the mechanism of hearing.
5. What is sound transduction? Explain the electrical events, which occur during sound transduction
6. Describe how the pitch of the sound is analyzed in human ear (theories of hearing).

SHORT QUESTIONS

1. Retina.
2. Ocular muscles.
3. Ocular movements.
4. Visual receptors.
5. Aqueous humor.
6. Intraocular pressure.
7. Glaucoma.
8. Fundus oculi.
9. Lens of eye.
10. Cataract.
11. Lacrimal glands.
12. Rhodopsin.
13. Phototransduction.
14. Dark adaptation.
15. Light adaptation.
16. Nyctalopia.
17. ERG.
18. Diplopia.
19. Hemianopia.
20. Effects of lesion in optic pathway.
21. Accommodation reflex.
22. Presbyopia.
23. Theories of color vision.
24. Color blindness.
25. Errors of refraction.
26. Auditory ossicles.
27. Tympanic reflex.
28. Cochlea.
29. Organ of Corti.
30. Role of middle ear in hearing (Functions of middle ear).
31. Role of inner ear in hearing (Functions of internal ear).
32. Impedance matching.
33. Traveling wave.
34. Electrical potentials in cochlea.
35. Theories of hearing.
36. Auditory defects.
37. Tests for hearing.
38. Audiometry.
39. Taste buds.
40. Taste transduction.
41. Taste pathway.
42. Olfactory transduction.
43. Olfactory pathway.