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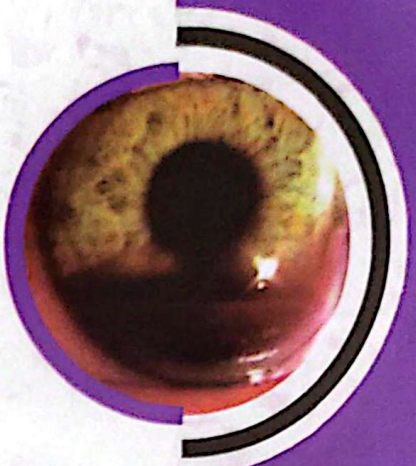
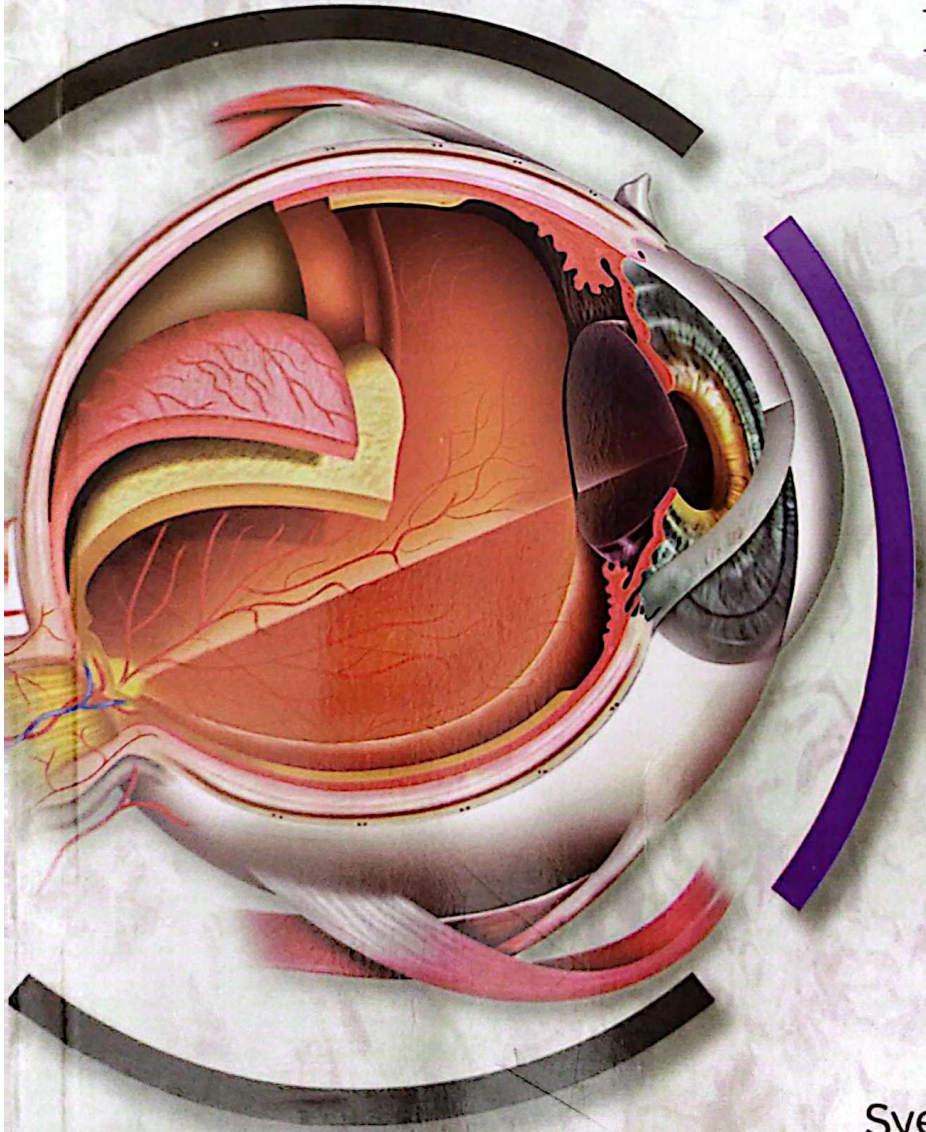


Concise Ophthalmology

Text & Atlas

Fifth Edition



Syed Imtiaz Ali Shah

Concise Ophthalmology Text & Atlas

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Fifth Edition B : 50

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Contents

Dedicated

to the great leader of the Muslims,

Syed Ali Khamenai

for his dedicated efforts in leading the world towards peace and progress

Chapter 01

Embryology of the Eye

Development of the eye in the human embryo can be traced back to, as early as the 21st day after fertilisation, when the optic sulcus appears in the lateral wall of the diencephalon. It grows to form a neuroectodermal diverticulum called the optic vesicle. The optic vesicle invaginates in such a way that it forms a double layered optic cup, while its proximal end constricts to form the optic stalk; simultaneously it induces the surface ectoderm to form the lens placode. (Fig. 1.1). The outer layer of the optic cup forms the retinal pigment epithelium while the inner layer forms the neural retina posteriorly. Anteriorly, the two layers fuse to form the epithelia of the iris, ciliary body and the muscles (sphincter and dilator pupillae) of the iris, while the stroma of both structures and the ciliary muscle are formed from mesoderm, which is continuous with the choroid posteriorly. The optic stalk has the choroidal fissure inferiorly continuous with the optic cup, to accommodate the hyaloid vessels and the axons of ganglion cells. (Fig: 1.2). The choroidal fissure closes approximately at the seventh week of gestation to convert the optic stalk into the optic nerve and the hyaloid vessels will form the central retinal artery and vein. The lens placode invaginates and forms the lens vesicle, which detaches from the surface ectoderm and settles at the edge of the optic cup and will form the future lens, meanwhile the surface ectoderm further forms the corneal and conjunctival epithelia, the lacrimal apparatus and lids. However, corneal stroma is mesodermal in origin and the endothelium is derived from neural crest cells. Vitreous is probably the combination of neuroectodermal and mesodermal elements. Finally the outer fibrous coat of the eye called sclera and the extra ocular muscles are derived from mesoderm. Development of the eye does not end at birth but continues far beyond the intrauterine period, and the eye attains its adult dimensions at approximately eight years of life.

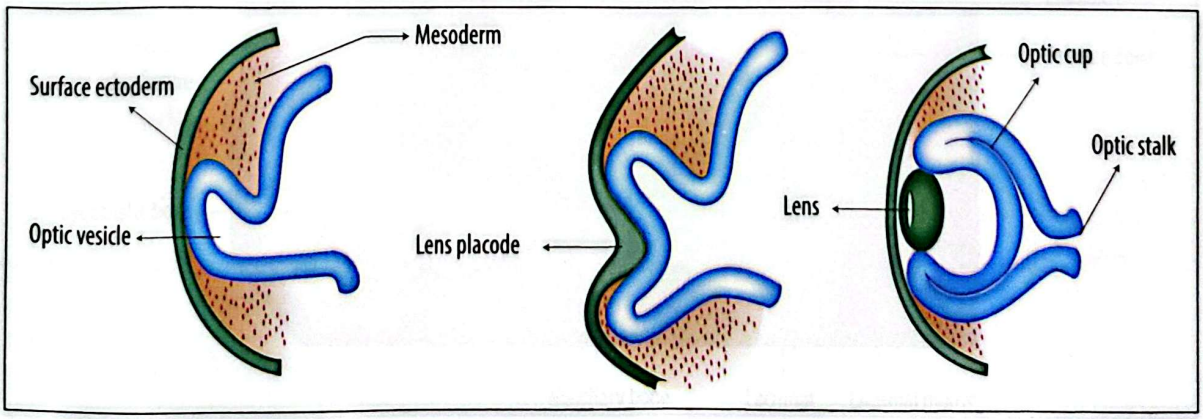


Fig. 1.1 Formation of the optic cup

Chapter 01

Embryology of the Eye

Development of the eye in the human embryo can be traced back to, as early as the 21st day after fertilisation, when the optic sulcus appears in the lateral wall of the diencephalon. It grows to form a neuroectodermal diverticulum called the optic vesicle. The optic vesicle invaginates in such a way that it forms a double layered optic cup, while its proximal end constricts to form the optic stalk; simultaneously it induces the surface ectoderm to form the lens placode. (Fig. 1.1). The outer layer of the optic cup forms the retinal pigment epithelium while the inner layer forms the neural retina posteriorly. Anteriorly, the two layers fuse to form the epithelia of the iris, ciliary body and the muscles (sphincter and dilator pupillae) of the iris, while the stroma of both structures and the ciliary muscle are formed from mesoderm, which is continuous with the choroid posteriorly. The optic stalk has the choroidal fissure inferiorly continuous with the optic cup, to accommodate the hyaloid vessels and the axons of ganglion cells. (Fig: 1.2). The choroidal fissure closes approximately at the seventh week of gestation to convert the optic stalk into the optic nerve and the hyaloid vessels will form the central retinal artery and vein. The lens placode invaginates and forms the lens vesicle, which detaches from the surface ectoderm and settles at the edge of the optic cup and will form the future lens, meanwhile the surface ectoderm further forms the corneal and conjunctival epithelia, the lacrimal apparatus and lids. However, corneal stroma is mesodermal in origin and the endothelium is derived from neural crest cells. Vitreous is probably the combination of neuroectodermal and mesodermal elements. Finally the outer fibrous coat of the eye called sclera and the extra ocular muscles are derived from mesoderm. Development of the eye does not end at birth but continues far beyond the intrauterine period, and the eye attains its adult dimensions at approximately eight years of life.

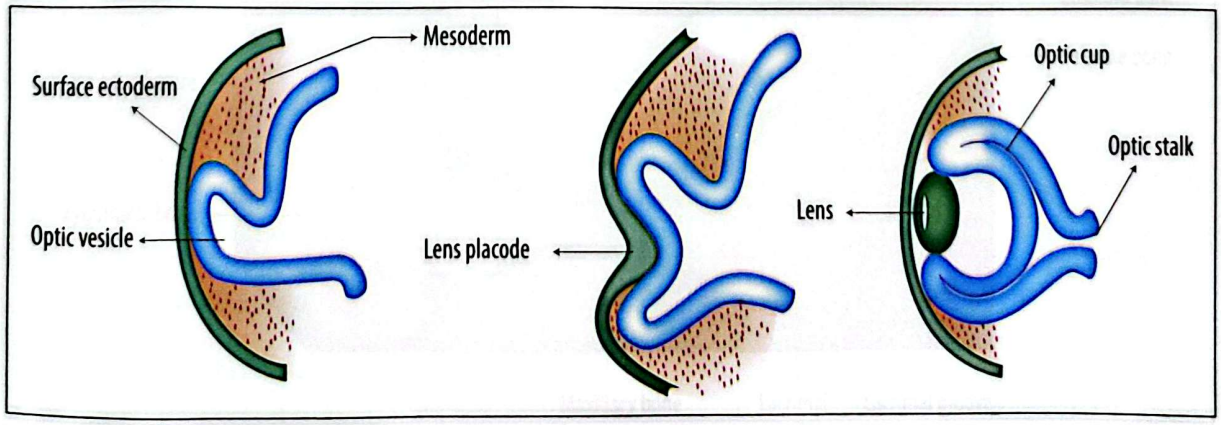


Fig. 1.1 Formation of the optic cup

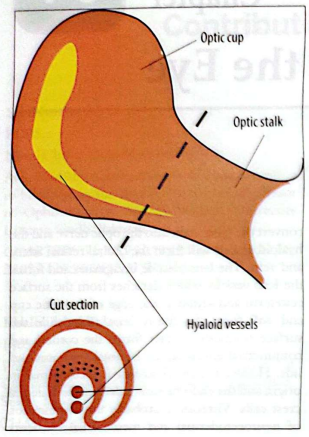


Fig. 1.2 The Choroidal fissure and hyaloid vessels

Ocular Structure	Embryonic Origin
Retinal Pigment epithelium	Neuroectoderm
Neural Retina	Neuroectoderm
Cornea	Surface ectoderm, Mesoderm, Neural crest cells
Sclera	Mesoderm
Choroid	Mesoderm
Ciliary body	Neuroectoderm, Mesoderm
Iris	Neuroectoderm, Mesoderm
Lens	Surface ectoderm
Vitreous	Neuroectoderm, Mesoderm
Extra ocular muscles	Mesoderm
Lacrimal apparatus	Surface ectoderm
Skin of the eyelids	Surface ectoderm

Chapter 02

Basic Anatomy of the Eye

Of all the infinite creations of Almighty Allah, the human eye is one of the most mesmerising. This chapter deals with the basic anatomy of the eye and its related structures.

Orbital Cavity

The human eye is housed in a pyramidal bony cavity called the orbit Fig. 2.1, whose apex is directed posteriomedially and its base faces anteriorly. The orbital margin is formed by the frontal, maxillary and zygomatic bones. The roof of the orbit is formed by the frontal bone and the lesser wing of the sphenoid. The floor is formed by the maxilla, zygomatic and palatine bones. The medial wall has contributions from the maxillary, lacrimal, ethmoid and sphenoid bones, while the lateral wall is formed by the zygomatic bone and the greater wing of the sphenoid bone. The orbit communicates with the neighbouring regions via certain openings, the main of which are described below:

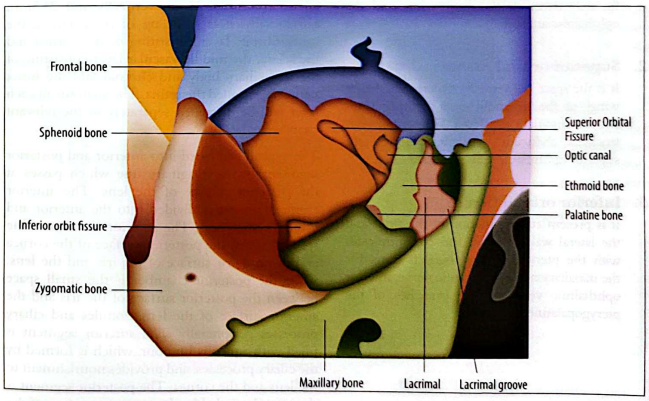


Fig. 2.1 The Choroidal fissure and hyaloid vessels

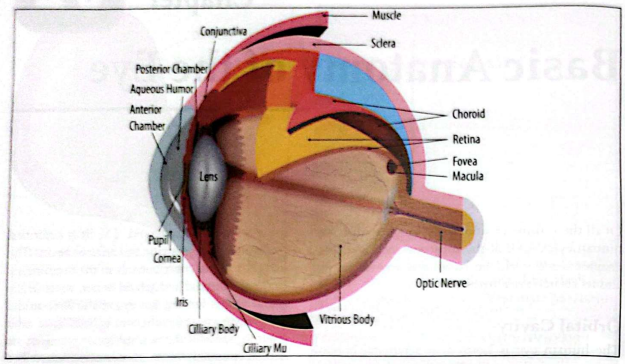


Fig. 2.2 The Eyeball

1. Optic canal

It is present at the apex of the orbit, and communicates with the middle cranial fossa. It transmits the optic nerve and the ophthalmic artery.

2. Superior orbital fissure

It is the space between the greater and lesser wings of the sphenoid, and communicates with the middle cranial fossa. The oculomotor, trochlear, abducent, ophthalmic nerves and superior ophthalmic vein pass through it.

Inferior orbital fissure

It is present at the junction of the floor and the lateral wall of orbit, and communicates with the pterygopalatine fossa. It transmits the maxillary nerve, zygomatic nerve, inferior ophthalmic vein and the branches of the pterygopalatine ganglion.

The Eyeball

It is a roughly spherical structure weighing around 28 grams, measuring about 24mm anteroposteriorly, 23mm vertically and 23.5mm horizontally. It is made up of three layers; the outer fibrous layer consisting of the cornea and the sclera, the middle vascular layer consisting of the iris, ciliary body and choroid and the inner neural layer called the retina. The anatomy of each layer will be discussed separately in the relevant chapters.

The eyeball is divided into anterior and posterior segments by an imaginary line which passes at the posterior surface of the lens. The anterior segment is further divided into the anterior and posterior chambers. The anterior chamber is the space between the posterior surface of the cornea and the anterior surface of the iris and the lens; while the posterior chamber is the small space between the posterior surface of the iris and the anterior surface of the lens, zonules and ciliary processes peripherally. The anterior segment is filled with aqueous humour, which is formed by the ciliary processes, and provides nourishment to the lens and the cornea. The posterior segment is the space bounded by the posterior surface of the lens, zonules and ciliary processes anteriorly and the retina posteriorly. It is filled with the vitreous humor, which is discussed in detail in the chapter on diseases of the vitreous (Fig. 2.2).

Ocular Appendages

These include the eyelids and associated glands, conjunctiva, the lacrimal gland, lacrimal passages and the extra ocular muscles. The anatomy of each structure will be discussed individually in subsequent chapters.

Nerve Supply of the Eye

This is sensory, motor and autonomic.

i. Sensory supply

a. General sensory supply is via the frontal, lacrimal and nasociliary nerves, which are the branches of the ophthalmic division of the trigeminal nerve.

b. Special sensory supply for vision is via the optic nerve. (for details of the optic nerve and visual pathway see chapters on diseases of the optic nerve and neuro-ophthalmology respectively)

ii. Motor supply

It is via the oculomotor, trochlear and abducent nerves.

a. The oculomotor nerve supplies the levator palpebrae superioris and the superior rectus muscles through its superior division, while the medial rectus, inferior rectus and the inferior oblique muscles through its inferior division; which also supplies preganglionic parasympathetic fibers to the ciliary ganglion.

b. The trochlear nerve supplies the superior oblique muscle.

c. The abducent nerve supplies the lateral rectus muscle.

iii. Autonomic supply

a. This is divided into the sympathetic and parasympathetic.

b. The sympathetic nerve supply: This includes distribution to the dilator pupillae, the orbitalis muscle (bridging the inferior orbital fissure), Muller's muscle in the upper lid and the lacrimal gland.

c. The parasympathetic nerve supply: It is distributed to the sphincter pupillae, the ciliary muscle and the lacrimal gland.

Arterial Supply of the Eye

This is from the ophthalmic artery, branch of the internal carotid artery, via its branches, the anterior ciliary, long posterior ciliary, short posterior ciliary and the central retinal artery. Numerous anterior ciliary and two long posterior ciliary arteries supply the structures anterior to the equator, while 12 to 20 short posterior ciliary arteries supply the structures posterior to the equator. The long and short posterior ciliary arteries anastomose with each other at the equator. The central retinal artery supplies the inner retinal layers up to the outer plexiform layer. Outer retinal layers are avascular and receive their nutrition from the choroid.

Venous Drainage of the Eye

The vortex veins, short ciliary veins and the anterior ciliary veins draining the eye join the superior and inferior ophthalmic veins, which eventually drain in the cavernous sinus. The central retinal vein also drains in the cavernous sinus either directly or via the superior ophthalmic vein.

Chapter 03

Biochemistry of the Eye

Recent advancements in the field of biochemistry have done wonders in helping us understand the molecular properties and functions of different structures of the body. This chapter deals with the biochemistry of various ocular structures.

Tear Film

It is the liquid wetting the eye. It is composed of three layers; chemically different from each other. The deepest layer, 0.5 micrometres thick, comprises of mucin, secreted by goblet cells, crypts of Henle and glands of Manz. The middle layer is 7.5 micrometres thick, aqueous in nature and is secreted by the glands of Krause and Wolfring. The superficial layer is lipid chemically, 0.5 micrometres thick, and is secreted by the tarsal glands, and the glands of Zeis and Moll.

Aqueous Humour

It is the liquid circulating in the posterior and anterior chambers and is formed by the ciliary processes. It maintains the intra ocular pressure and nourishes the cornea and the lens. The chemical constituents of aqueous are sodium, potassium, bicarbonate, chloride, ascorbic acid, lactic acid, pyruvic acid, glucose, oxygen, carbon dioxide, very sparse protein and plenty of water.

Vitreous Humour

It is a transparent gel, 4ml in volume and comprising of 99% water and 1% solids. Biochemical constituents of vitreous include collagen fibrils, hyaluronic acid molecules, electrolytes, glucose, amino acids, ascorbate and a few hyalocytes.

Visual Cycle

It is the cascade of reactions occurring in the retina, leading to the genesis of nerve impulse in the optic nerve fibres, which is to be perceived in the visual cortex. The cycle starts when the photon of light strikes the outer segment of the photoreceptors leading to isomerisation of 11-cis-retinal to all-trans-retinal. This change starts a series of reactions leading to the formation of metarhodopsin-II, the active product which produces the electrical changes in the photoreceptors and finally the action potential in the optic nerve fibres is generated. Metarhodopsin-II is deactivated by an enzyme rhodopsin kinase. (Fig. 3.1)

Generation of Receptor Potential

Metarhodopsin-II formed in the visual cycle activates transducin which in turn activates the enzyme phosphodiesterase, which decomposes the cyclic-GMP. The fall in cyclic-GMP levels in the photoreceptors causes decreased sodium conductance of the membrane and resultant hyperpolarisation. Retinal photoreceptors are unique from most of the other excitable cells of the body because they are excited when hyperpolarised unlike most which are excited when depolarised. Signals generated in the photoreceptors are not action potentials but are transmitted to the ganglion cells which generate action potentials and transmit them to the brain via the optic nerve.

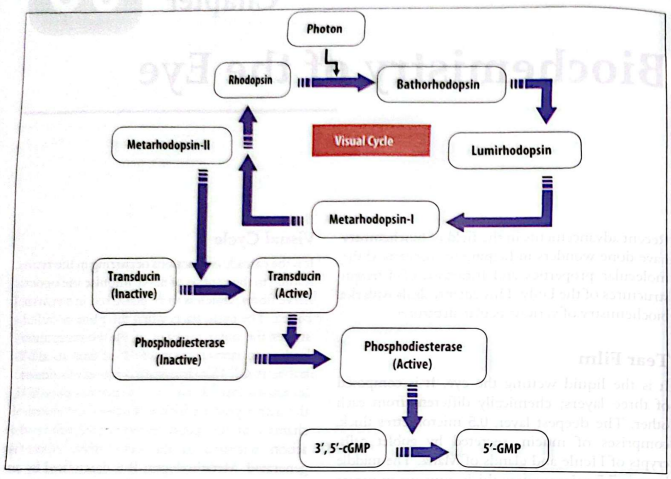


Fig. 3.1 The visual cycle

Chapter **04**

Physiology of the Eye

The function of the human eye is not just to see but it has multiple and complicated functions. Vision itself is a complex phenomenon, and to understand it, is a difficult task. In this chapter, we will discuss the various functions of the eye and will try to keep it as simple as we can.

BASIC TERMS USED IN OPTICS

Light

It is the form of electromagnetic energy, travelling in the air at a speed of 300,000 km/sec. Portion of it is visible to the human eye (370-730nm), and is called the visual spectrum. The visual spectrum contains colours famously known as VIBGYOR (violet, indigo, blue, green, yellow, orange and red), all with their specific wavelengths. Red has the longest and violet has the shortest wavelength.

Refractive Index

Refractive index of a substance is the ratio of velocity of light in the air to the velocity of light in that substance.

Optical Axis

It is an imaginary line which connects the anterior pole of the eye with its posterior pole.

Visual axis

It is an imaginary line which connects an object in view with fovea of both eyes in the normal state of vision.

VISUAL FUNCTIONS OF THE EYE

Visual Acuity

It is the ability of an eye to locate and distinguish the components of a target and identify it as a whole. It is the function of cones which are accumulated in the fovea of the macula. So, visual acuity is best at fovea.

Stereopsis

It is the ability to perceive the depth of an object and the relative distances between different objects. Stereopsis is the function of binocular vision.

Dark and Light Adaptation

When a person enters a dark room from a bright atmosphere, it takes some time for him to be able to see. The time interval required by the person to be able to see in the dark room is called the dark adaptation time, and this phenomenon is called the **dark adaptation**. This occurs due to the depletion of photo-chemicals in the retinal photoreceptors in the brighter atmosphere, which need some time to replenish. Vice versa occurs when a person moves to a brighter atmosphere from a dark room. The person is unable to see properly until some time has passed (**light adaptation**), because the photo-chemicals have replenished in the dark and the retina has become oversensitive to light.

Colour Vision

The trichromatic theory defines the mechanism of colour vision. It is based on the fact that the human eye has red, green and blue cones, which are stimulated in various combinations to produce different shades and grades of colours. For example, all the three cones when stimulated equally will give the perception of a white colour.

Color Blindness

Blue cone deficiency (Tritanopia) and weakness (Tritanomaly) are rare, but green cone deficiency (Deuteranopia) or weakness (Deutranomaly) and red cone deficiency (Protanopia) or weakness (Protanomaly) are more frequently seen. Red-green colour blindness is an X-linked disease seen in males and can be detected on testing with Ishihara charts, in which the affected person cannot distinguish the red colour from the green.

Contrast Vision

It is the ability to differentiate in luminance of adjacent regions, which are not separated by definite borders. It occurs due to the phenomenon of lateral inhibition caused by the horizontal cells.

The photochemistry of the visual cycle and the electrical changes occurring afterwards are discussed in the chapter of Biochemistry of the eye.

Fusion

It is the ability of the occipital cortex to integrate two similar images from both eyes into one image (**sensory fusion**) and ability of the two eyes to remain aligned (**motor fusion**).

Chapter 05

Clinical Approach to Ophthalmic Patients

Patients should be approached clinically on the basis of scientific foundations and the first scientific foundation is to follow appropriate steps in orderly manner such as the following steps.

- History
- Examination
- Clinical Tests
- Differential Diagnosis
- Provisional Diagnosis
- Investigations
- Provisional Treatment
- Result of Investigations
- Final Diagnosis
- Final Treatment
- Follow up
- Prognosis
- Research Oriented Aspects

After introducing yourself to the patient in a friendly atmosphere the first important step is to take the history.

History

Take the history of illness from the patient. The following points may be recorded.

Particulars of the patient

Including **name, age, gender, address, occupation** and **date.**

Presenting complaint or complaints with duration

It is to be noted that the duration of a complaint is a strong weapon for early diagnosis and timely treatment in patients suffering from eye disease, e.g., loss of vision of a half-hour's duration may prompt you to pick up a timely diagnosis and subsequent treatment of **central retinal artery occlusion** without wasting precious time in asking for other aspects of history.

History of the present illness

It is the basis of developing a clear list of diseases in the mind as one proceeds further.

Past history of illnesses

This supports us in the diagnosis of recurrent disorders, e.g., recurrent uveitis.

History of systemic diseases

It is crucial for saving time in managing associated ocular complications of systemic diseases like **diabetes mellitus, hypertension, AIDS, malignancies** etc. Diabetic retinopathy/ hypertensive retinopathy may be associated with diabetes mellitus and hypertension respectively. A secondary in or around the eye condition from a primary malignancy in another part of the body may simulate a primary eye condition (ocular masquerade syndrome), Fig 5.1 B&C shows a secondary malignant deposit in the lower eyelid simulating chalazion.

Fig. 5.1 B&C

Treatment history

History of already taken treatment of the present disorder and associated diseases, if any, helps us diagnose such situations as steroid induced cataract and glaucoma, drug induced mydriasis or miosis, radiation retinopathy etc. which otherwise becomes difficult to locate.

Occupational history

This helps us diagnose occupation related disorders like glass blower's cataract, ultraviolet keratopathy like keratopathy prevalent along the Makran coast etc.

Socio-economic history

This not only determines nutritional disorders like nyctalopia resulting from vitamin A deficiency but also guides us in future management planning of a particular patient e.g. a poor patient has to go to a general ward with limited treatment options (a poor show of society throughout the world).

Personal history

Although difficult to record, personal history definitely helps us reach a diagnosis in time and subsequent timely management of affected patients e.g. methyl alcohol poisoning.

After taking appropriate **history**, a clue towards diagnosis always enlightens our mind and thus we ut ***clinical examination.***

Examination

It starts by seating the patient at an appropriate distance from us which is usually one meter. General examination should be carried out including recording vital signs (pulse rate, respiratory rate, temperature and blood pressure), jugular venous pulsation, wasting, obesity, clubbing, koilonychia and splinter haemorrhages of the nails, anaemia, jaundice, dehydration, spider nevi, vitiligo (Fig.5.2), alopecia, pitting oedema, ecchymosis and palpation for lymphadenopathy. The head of the doctor and the patient should be at the same level and examination may commence after introducing oneself, taking consent from the patient and informing the patient about the methodology.

Although both eyes are to be examined and **functional**, **physical** and **instrumental** examinations are performed distinctly, but to save time, to avoid misery to the patient and to collect maximum information about disease process, one has to examine both the eyes simultaneously, and mix up all the three methods of examination as and when required, e.g., colour difference between the iris of two eyes may be missed if examination of two eyes is done separately.

Physical examination

This may be performed with the help of a torch with dim background illumination. To start with **inspection**, head position and facial symmetry is seen to detect extra ocular muscle palsies and facial palsy respectively (Fig. 5.4B). Palpebral fissures are compared to ascertain status of the eye lids. A

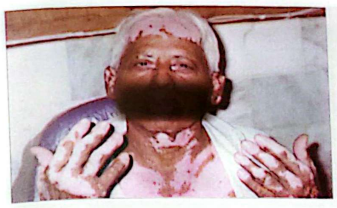


Fig. 5.2 A patient with vitiligo

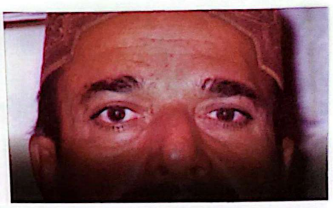


Fig. 5.4(A) Comparing palpebral fissures showing right wide palpebral fissure due to mild proptosis

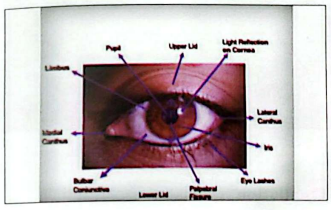


Fig. 5.3 Photograph showing normal parts of the eye as seen in a clinical setting

Fig. 5.4(B) Left sided facial palsy

narrow palpebral fissure may be due to ptosis and a wide palpebral fissure (Fig.5.4A) may be due to retraction of the lids or due to proptosis.

Lid margins should be seen for inflammation, depositions, swellings, misdirected eye lashes etc. Bulbar conjunctiva should be examined for congestion, foreign bodies, Imtiaz sign1 (Fig. 5.4C), Bitot's spots, mass, nevus (Fig. 5.5B) etc. Examination of palpebral conjunctiva requires lid eversion to look for papillae, follicles, concretions etc. **Palpation** starts with a regurgitation test by pressing over the lacrimal sac area to look for pus or mucous coming out from puncta in chronic dacryocystitis. Palpation of orbital margins gives information about bony irregularities which may be the first sign of hidden malignancy of orbital bones or periosteum. Palpation of orbit around the eyeball detects reachable orbital masses. Auscultation may be done in selected cases for bruit where arteriovenous fistula is suspected or pulsating mass is present.

Anterior segment (Cornea, Anterior Chamber, Iris, Pupil, and Lens) (Fig.5.3) **examination** requires a **slit lamp biomicroscope** (Fig.5.6). The following methods may be utilised on slitlamp biomicroscope for extracting maximum information about disease process in this region.

Direct illumination

With a broad beam, it is useful to perform a general survey of the structures in view.

With a narrow beam, it is useful in forming optical sections of the cornea and lens and in preliminary assessment of the angle of the anterior chamber and blood (Fig. 5.5A) or pus in the anterior chamber (Fig. 5.5C).

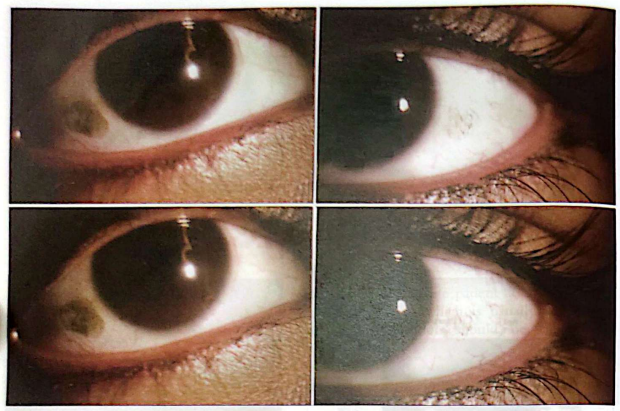


Fig. 5.4(C) Imtiaz Sign of Vitamin A Deficiency

With a spot light, it helps to see the flare with unilluminated surroundings.

Indirect illumination

It helps see iris stroma details not visible under direct illumination, e.g., a small bleed or nevus.

Retro-illumination

It is useful in detecting iris atrophy, lens vacuolation etc.

Specular reflection

This helps see the fine golden mosaic of corneal endothelium. This method requires experience therefore, a specular microscope is the better alternative.

Sclerotic scatter

It helps enlighten the focal area of the cornea to see the details of peripheral lesion.

Oscillation

It helps detect findings which have escaped during examination under sustained illumination.

Ocular motility examination

This needs to be performed in every patient. Ductions, versions and vergence are normal ocular movements. Persistent nystagmus is abnormal movement. Phorias are temporary deviations and Tropias are permanent deviations. Lateral, medial, superior and inferior unilateral eyeball movements with the other eye closed should be checked which are called abduction, adduction, hyperabduction and hypoduction respectively. Unilateral movement of an eyeball in a circle is called circumduction. Similarly, binocular movements should be checked on the right, left, superiorly, inferiorly and in a circle, which are called dextroversion, levoversion, hyperversion, hypoversion and circumversion respectively. When the patient is asked to look at a near target, the eyes converge and when asked to look at a distant target the eyes assume a straight

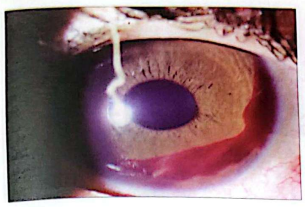


Fig. 5.5(A) Hyphaema (blood in anterior chamber) seen on Slitlamp examination

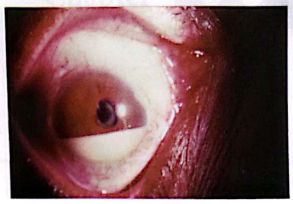


Fig. 5.5(C) Pus in the anterior chamber called hypopyon

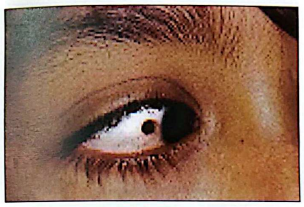


Fig. 5.5(B) Nevus on bulbar conjunctiva

Fig. 5.6 Slitlamp examination is being performed

position, i.e., diverge from a converged position. Pathological nystagmus can be observed by simple observation of the eyes of the patient as this is an involuntary rhythmic movement of the eyes and should be examined in all directions of gaze, i.e., right, left, up and down. Phorias and tropias need to be examined by covering one eye and looking at the behaviour of both eyes, i.e., covered and uncovered.

Functional examination

This comprises of performing Snellen's visual acuity test with and without pin hole to check the refractive status of the eye and macular function in adults and verbal children. In preverbal children, an IZZP device is used to pick up the VA acuity in the shortest possible time. Accommodation is checked with a near vision chart to see the status of the apparatus of accommodation and convergence which includes the lens, ciliary body, suspensory ligament, pupil and medial recti. Colour vision is checked by showing standard colour configurations to the patient like the Ishihara chart or 100 hue test to see the status of cone function. Visual field examination is performed initially by a confrontation test and later with the help of a perimeter to see the function of the peripheral retina. A dark adaptation test is performed to see Rod function. The Maddox rod test is performed for examination of muscle balance for distance. The Maddox wing test is performed to check the muscle balance for near. In patients with extremely low vision, finger counting, hand movements and finally light projection and light perception is recorded.

Instrumental examination

It is performed with instruments like the ophthalmoscope, tonometer etc. which are discussed in the next chapter.

Chapter 06

Clinical Tests for Ophthalmic Patients

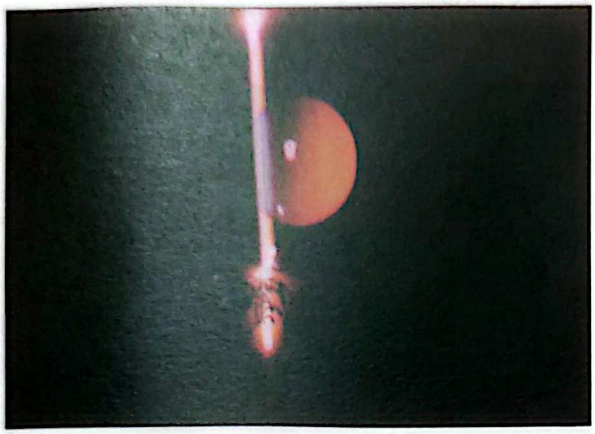


Fig. 6.1 Clear fundus reflex on slitlamp examination

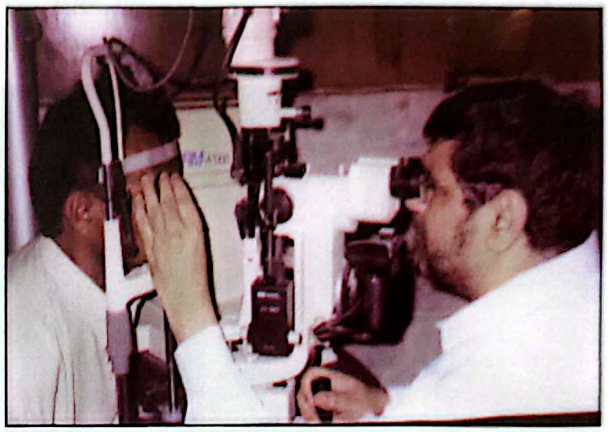


Fig. 6.2 Fundoscopy is being performed on a slitlamp with the help of a 90 D lens

Fundoscopy

This is the clinical examination of the retina which is performed with the help of direct and indirect ophthalmoscopes and with 90 D or 78 D lens on a slitlamp (Fig.6.2). Advantages with direct ophthalmoscopic examination are 15 times magnification and it is simple to use. Disadvantages of direct ophthalmoscopic examination are its uniocular use therefore lack of adequate depth perception, only 5 degrees of retina can be seen at a time and overall 30 degrees of retina can only be seen. Another disadvantage is its very close working distance. Advantages of indirect ophthalmoscopic examination are its binocularity, thus, excellent depth perception, 30 degree of retina can be seen at a time and full retinal examination can be performed up to ora serrata with the help of an indenter. Another advantage is its adequate working distance of one meter. Among its disadvantages is its less magnification of 2 to 3 times only. Fundus reflex is seen initially to detect clarity of media (Fig.6.1).

Fundoscopy determines the status of the optic disc including papilloedema, cupping, optic atrophy and retinal disorders like diabetic retinopathy, hypertensive retinopathy, other retinopathies, retinal vasculitis, retinal detachment etc.

Tonometry

It is performed to check the **intraocular pressure (IOP)** of the eyeball which normally ranges between 10 to 20 mmHg +/- 3. Any rise or fall beyond this level should be taken as abnormal until proved otherwise. Non-contact tonometry (pneumotonometry) (Fig. 6.5) is performed in special circumstances like infected cornea, irregular surfaced cornea, screening a population. Contact tonometry is performed by either the **indentation method,** e.g., Schiotz tonometer (Fig. 6.3), which measures the amount of corneal deformation produced by a given force, or by the **applanation method,** e.g., Goldmann applanation tonometry (Fig. 6.4) which measures

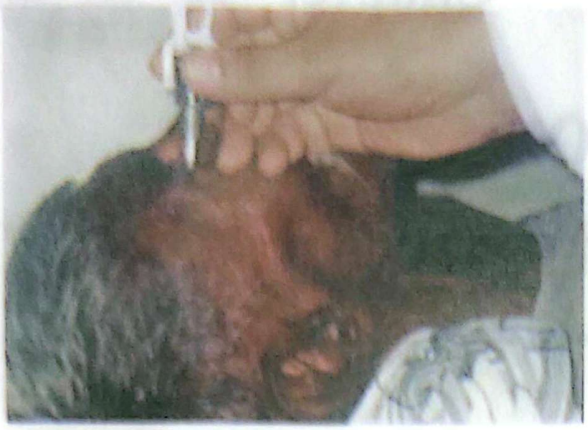


Fig. 6.3 Schiotz tonometry

Fig. 6.5 Non-contact applanation tonometry

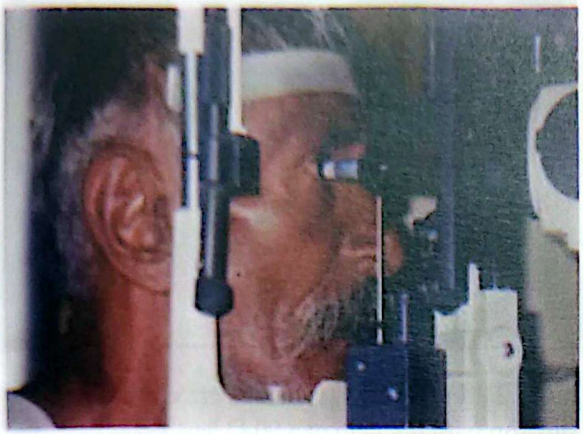


Fig. 6.4 Contact applanation

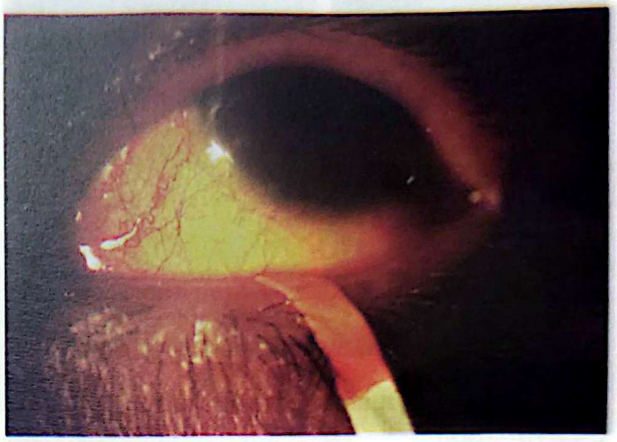


Fig. 6.6 Fluorescein staining of cornea is being performed with a fluorescein strip

the force necessary to flatten a given area (3.06 mm) of the cornea. Presently indentation tonometry is used infrequently and applanation tonometry has become the method of choice due to its better results.

Fluorescein Dye Tests

These are a great asset for ophthalmologists across the world since a long time in making readymade diagnosis of eye disorders.

Fluorescein staining of the cornea

It is performed with sterilized fluorescein tipped strips (Fig. 6.6), easily available in the market, to see the type of corneal ulceration under the blue filter of a slitlamp and thus helps in differential diagnosis of viral, bacterial, fungal, protozoal, atopic, neurotrophic, dystrophic and ischaemic corneal ulcers (Fig. 6.7).

The seidel test is performed with sterilised fluorescein tipped strips to diagnose wound leakage after intraocular surgery.

It is performed to see patency of lacrimal drainage system by checking the fluorescein staining of nasal swab after instilling fluorescein dye with a sterilised fluorescein tipped strip in the conjunctival sac.

Fundus Fluorescein Angiography (FFA)

It is an important diagnostic aid for detecting window defects in retinal pigment epithelium, leakage from retinal or choroidal circulations, non-perfused areas of retina and new vessels. 3 ml of 25% or 5ml of 10% sodium fluorescein solution is injected intravenously as a bolus. A test dose should be given first to avoid undesirable complications like anaphylactic reaction.

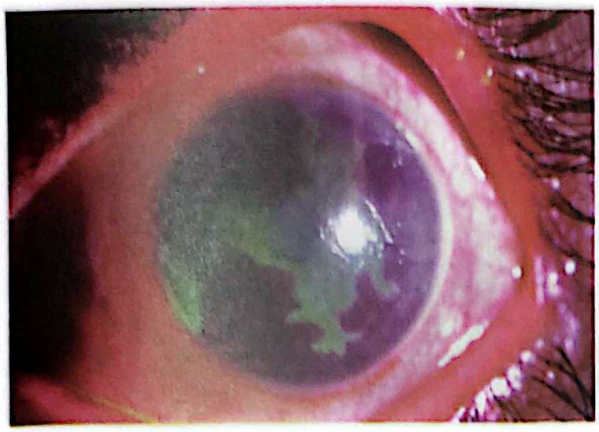


Fig. 6.7 Corneal ulceration stained with fluorescein

Fig. 6.8 Gonioscopic view of the anterior chamber angle

Indocyanine green angiography

This gives better results when looking for occult vessels in subretinal neovascular membrane.

Anterior segment fluorescein angiography

It is not done routinely. It helps differentiate iris melanoma from iris nevus.

Few surgeons use **fluorescein during anterior vitrectomy** to remove the vitreous completely from the anterior chamber as it stains transparent vitreous which stands out clearly.

Fluorescein is used in contact lens fitting.

Gonioscopy

This is performed with the help of a gonioscope to examine the angle of the anterior chamber. The following grading is simple and helpful in assessment of the angle of the anterior chamber which is limited anteriorly by peripheral cornea and posteriorly by the iris.(Fig. 6.8)

The structures seen in between are from anterior to posterior.
Schwalbe line
Trabecular meshwork
Scleral spur
Ciliary body
Grade 4 (wide angle) all structures visible
Grade 3 (open angle) ciliary body not visible
Grade 2 (moderately narrow angle) scleral
Spur not visible
Grade 1 (very narrow angle) trabecular
Meshwork not visible
Grade 0 (closed angle) schwalbe's line not visible

Direct gonioscopy is performed with direct gonioscope which utilises prisms. **Indirect gonioscopy** is performed with an indirect gonioscope which utilises mirrors.

Ultrasonography

It is performed routinely in two different modes available, either as an A-scan machine and B-scan machine, or both are available in one machine.

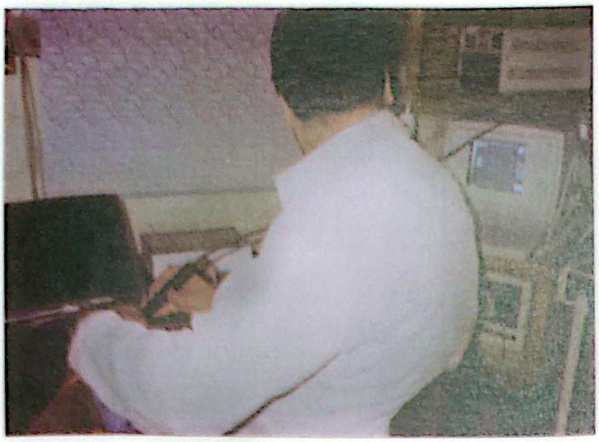


Fig. 6.9(A) B-scan ultrasonography is being performed on a child with a white pupil

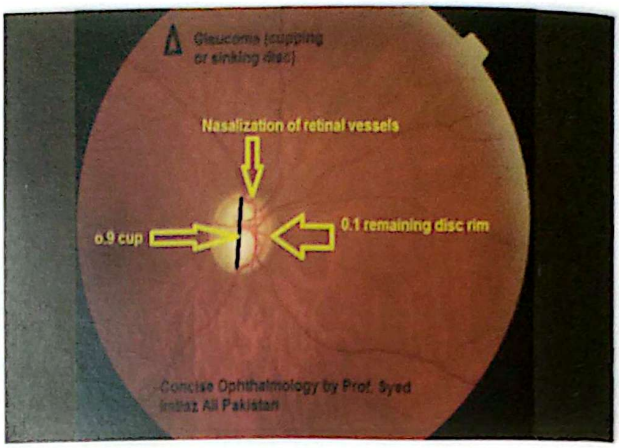


Fig. 6.9(B) Glaucoma (cupping or sinking disc)

A-scan mode

It is used to take the axial length of the eyeball which is coupled with keratometry in calculating Intraocular Lens (IOL) power with the help of formulas, e.g., SRK III.

B-scan mode

It is used to take two dimensional images of the eyeball, particularly the posterior segment to detect retinal detachment, retinal tears, vitreous haemorrhage, intraocular tumours, posterior vitreous detachment, etc., in patients with opaque media (Fig.6.9A).

Keratometry

It is used to measure corneal curvature in vertical and horizontal meridians mainly to calculate IOL power. Manual and automated keratometers are available and are simple to use.

Corneal Topography

It is done with a computer-assisted videokeratoscope to evaluate distortion of corneal contour, e.g., in keratoconus. It has replaced the old Placido disc and photokeratoscope.

Perimetry

It is the method of recording the visual fields of the patient with the help of a perimeter. The defects in visual fields (SCOTOMAS) are picked up easily with this method.

Fundus Photography

It is a useful method of keeping record of progressive disorders like diabetic retinopathy to detect progression of disease. It utilises the same fundus camera which is used for performing FFA.

Papillary Photography

It is taking a picture of the optic disc for future comparison to detect progressive changes. This is done with the fundus camera (Fig. 6.9 B).

Retinoscopy

It is the method of evaluating the refractive status of the eye to determine refractive errors and is performed with retinoscope or with an auto refractometer.

Lensometer

It is the device to verify the power and axis of the spectacle glasses and is a useful tool for assessing the patient's glasses (Fig.6.10).

Optical Coherence Tomogram

It is the device which offers high-resolution, cross-sectional images of the retina and quantitative measurement of retinal thickness.

Therefore OCT is a very useful tool in assessment of morphological changes in the retina Fig. 6.11.

Specular Microscope

It is the device used to count corneal endothelial cells (Fig 6.12).

Fig. 6.10 Dioptric power and axis of the cylinder of spectacle lenses is being verified with a lensometer

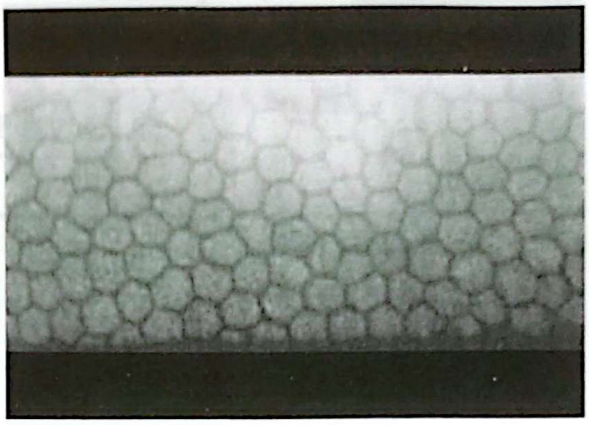


Fig. 6.12 Corneal endothelium seen on a specular microscope

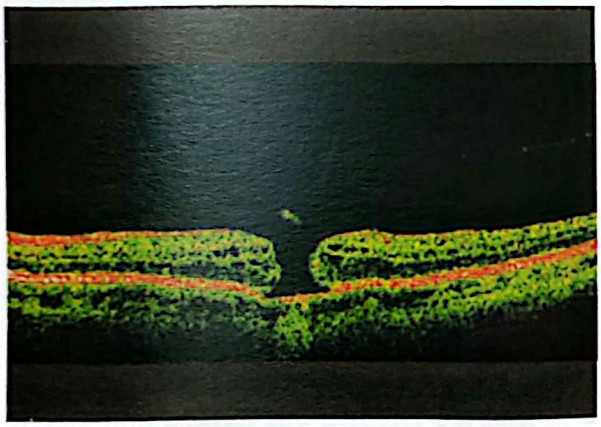


Fig. 6.11 Macular hole seen on OCT

Fig. 6.13 The Imtiaz zigzag pattern device (IZZP)

IZZP Device

The Imtiaz zigzag pattern device (IZZP device Fig. 6.13) is utilised for assessing vision in preverbal children, i.e., the infants and small children of up to 2 years of age who are not able to speak.

Chapter 07

Diseases of Eyelids

Eye lids are natural protective curtains over the eyes. Structurally they are made up of from the outside in,

1. Skin
2. Superficial fascia
3. Muscle layer made up with fibers of orbicularis oculi and levator palpebrae superioris
4. Tarsal plate containing Meibomian glands
5. Palpebral conjunctiva

When open, the space between the upper and lower lid margins is called the palpebral fissure which normally measures around 12 mm. Eye lid margin can be examined easily on lid eversion and is seen divided into anterior and posterior portions by a grey line which represents the anterior edge of the tarsal plate. Anterior to the grey line, the portion of lid margin is made up of skin and muscle layers and possesses eye lashes which receive sebaceous glands of Zeis and sweat glands of Moll into their hair follicles. Posterior to the grey line the portion of the lid margin is made up of conjunctiva and tarsus containing Meibomian gland openings. Skin of the eyelids is devoid of fat and is thinnest in the body therefore it is easily distensible and underlying blood vessels may become visible in fair-coloured people as dark blue lines. The upper lid is innervated by ophthalmic division of the trigeminal nerve and the lower lid is innervated by maxillary division of the trigeminal except in its small lateral portion which gets innervation from ophthalmic division. The medial one third of the upper lid and medial two thirds of the lower lid drain into the submandibular lymph nodes and lateral two thirds of the upper and lateral one third of the lower lid drain into the preauricular lymph nodes.

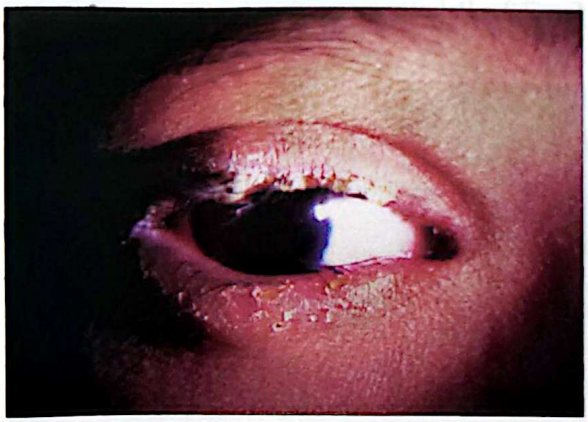


Fig. 7.1 Madarosis of eyelids due to blepharitis

COLOBOMA OF THE UPPER EYELID

When the upper eyelid fails to develop partially or completely (partial or total coloboma of the upper eyelid), the cornea is exposed and is in danger of becoming ulcerated and finally perforated leading to a loss of vision. This emergency situation is first treated by keeping the cornea wet with artificial tears and ointment or covered with a bandage contact lens. Finally The lid is reconstructed by surgery[3].

COMMON CLINICAL FEATURES OF EYELID DISEASE

Pain, oedema, crusting of lid margin, loss of eyelashes (madarosis Fig.7.1), whitening of eyelashes (poliosis Fig. 7.2), misdirection or loss of eyelashes (trichiasis Fig.7.6), narrowing or widening of palpebral fissure and eyelid drooping are few of the clinical presentations associated with eyelid disease.

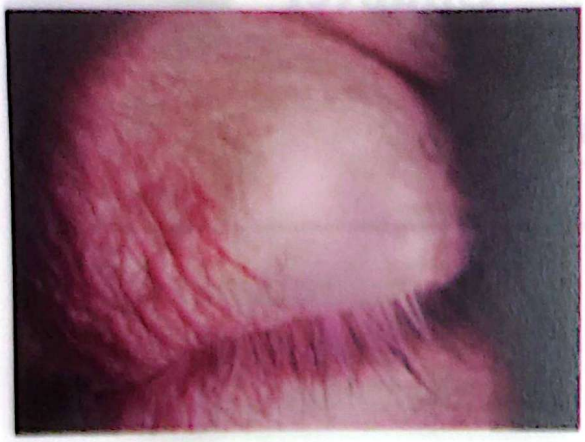


Fig. 7.2 Poliosis

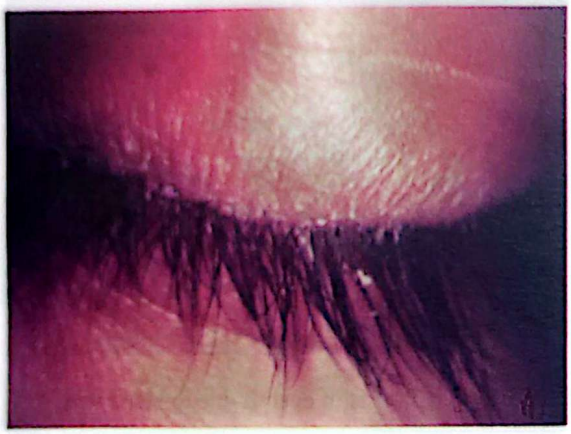


Fig. 7.3 Squamous blepharitis

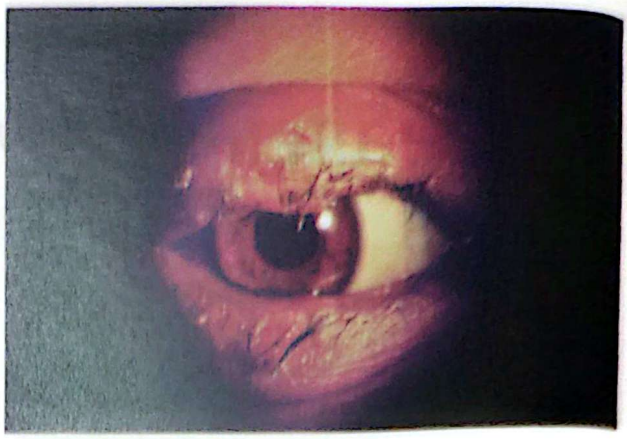


Fig. 7.4 Ulcerative blepharitis

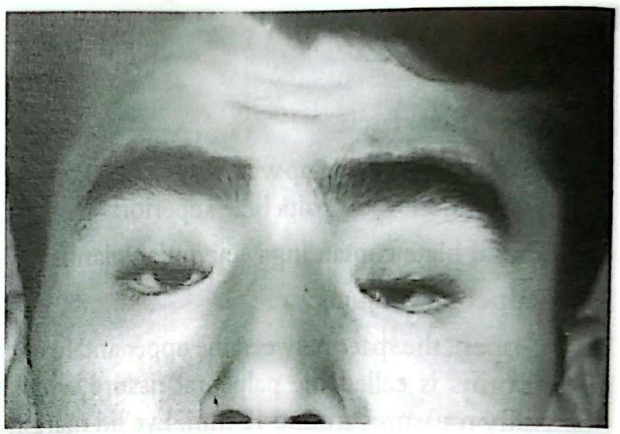


Fig. 7.5 Blepharophimosis

Blepharitis

This is a chronic inflammation which affects margins of eyelids and usually associated with chronic conjunctivitis. Staphylococcal infection and/or seborrhoeic dermatitis are the underlying causes. It usually starts somewhere during the first decade of life and almost the becomes patient's companion for life. It clinically presents in two forms, squamous blepharitis and ulcerative blepharitis.

Squamous blepharitis

(Fig 7.3) It is always associated with seborrhoea of the scalp and presents with hyperaemia of the lid margins, scales and flakes around the eyelashes. Patients complain of irritation and burning of the eyes. Treatment is removal of scales with moistened cotton tipped applicators and local application of suitable antibiotics with steroids and antihistamines in addition to treatment of seborrhoea of the scalp.

Ulcerative blepharitis

It is a more severe inflammation of lid margins. Clinically presents with suppurative lesions of follicles of eyelashes and crusts at lid margins, which on removal take away the affected eyelash leaving behind a raw ulcerated area. Treatment is with local application of suitable antibiotics like fucidic acid, neomycin, polymyxin and bacitracin. In severe cases, systemic antibiotics, anti-inflammatory agents and antihistamines may be added. Loss of eyelashes, whitening of eyelashes (poliosis) and distortion of lid margin leading to ectropion are among the complications of ulcerative blepharitis (Fig.7.4).

Epicanthus

It is a congenital condition seen in children produced by the presence of vertical skin folds on inner canthi. These folds are normal findings during foetal development but when present,

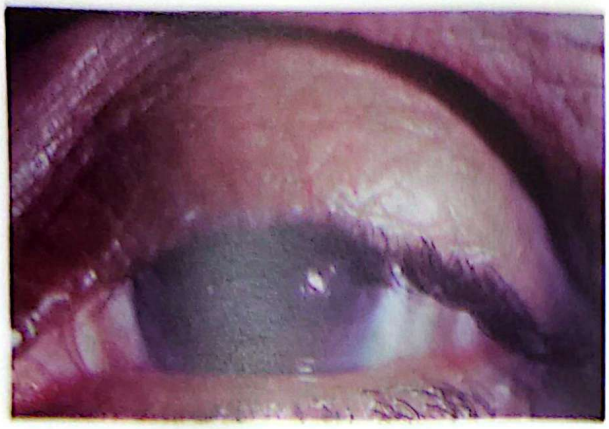


Fig. 7.6 Trichiasis

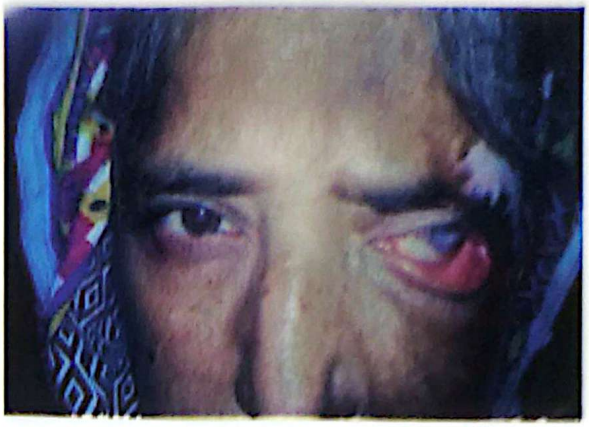


Fig. 7.8(A) Cicatricial ectropion of the left lower eyelid

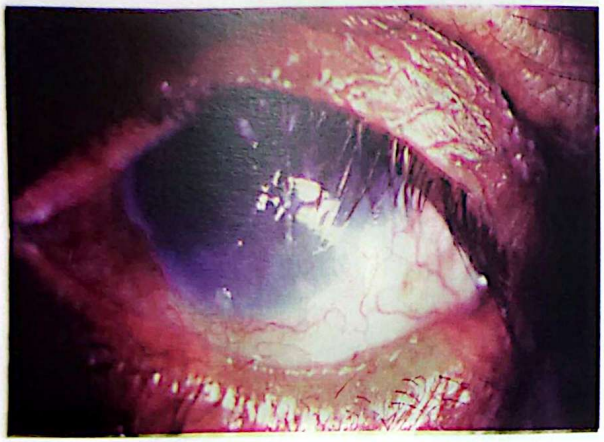


Fig. 7.7 Entropion

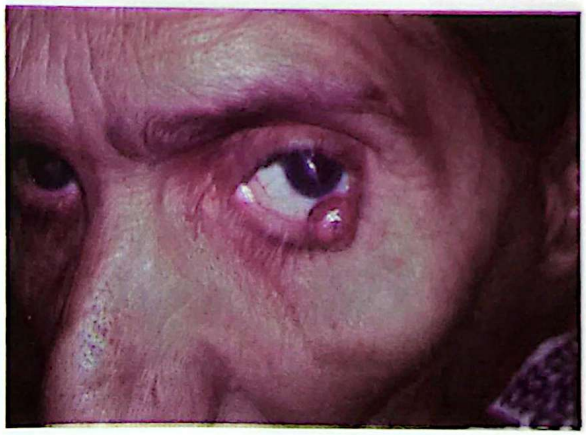


Fig. 7.8(B) Ectropion of the lower lid due to weight of the lower lid tumour

other than Mongolian races where it is normal finding, in infancy and childhood they may cause pseudo strabismus. The uneventful cover-uncover test and pinching up the loose skin over the bridge of nose to make the squint disappear are frequently adequate measures to satisfy the parents of the patient.

Blepharophimosis

It is a congenital narrowing of palpebral fissure in both horizontal and vertical meridians with autosomal dominant inheritance. Treatment is cosmetic surgery when required and genetic counselling (Fig. 7.5).

Entropion

This is a condition of inward turning of the upper or lower lid margin with lashes rubbing over the cornea. Four types of entropion seen clinically are congenital, senile, spastic and cicatricial. Immediate treatment is retraction of the lid margin with the help of adhesive tape or temporary everting sutures to keep the lashes away from the cornea. Treatment is surgical and a wide variety of surgical procedure, with or without modifications, are being carried out to treat entropion including procedures by Jones, Wies, Wobig, Quickert, Burrow, Reeh, Arlt, Bick, Imtiaz and others (Fig. 7.7).

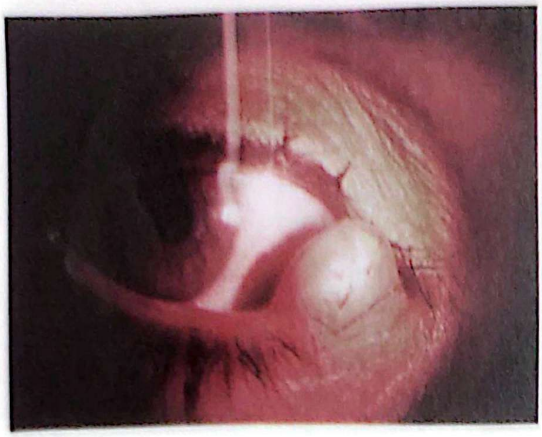


Fig. 7.9 Ectropion of the lower lid due to weight of the lower lid tumour

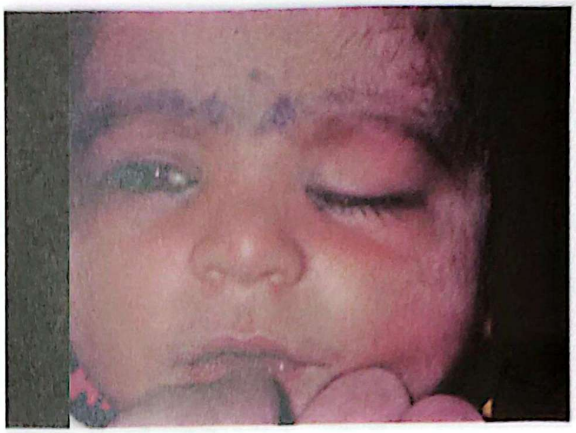


Fig. 7.10(B) Congenital ptosis of the left upper lid

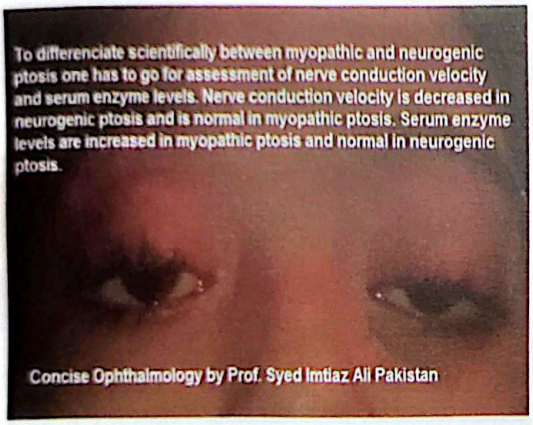


Fig. 7.10(A)

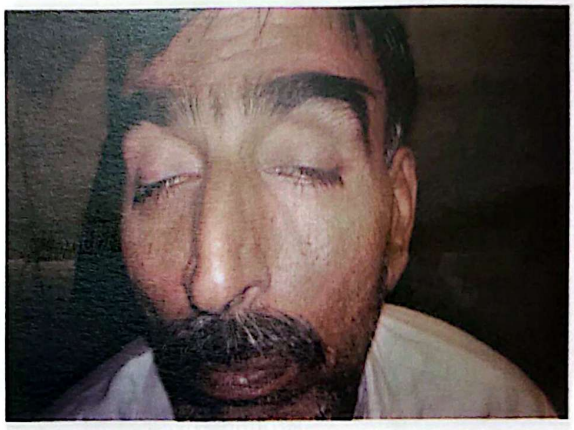


Fig. 7.11 Bilateral Ptosis due to Myopathy

Ectropion

This is a condition of eversion and/or falling away of the lower lid from the globe resulting in epiphora and excoriation of the affected area. Five types of ectropion seen clinically are congenital, senile, cicatricial (Fig. 7.8 A), mechanical (Fig. 7.8 B, 7.9) and paralytic. Mechanical ectropion (Fig. 7.8, 7.9) results from the weight of the mass on the lid margin. Immediate treatment is lubricating gel or a bandage contact lens to protect the globe from exposure, particularly in the absence of Bell's phenomena. Treatment is surgical, depending upon the type of ectropion. The Kuhnt-Szymanowski procedure, or its Byron Smith modification, Z-Plasty, skin grafting, full thickness pentagonal resection, three snip procedure and cautery punctures are few of the procedures available to treat ectropion (Fig. 7.8, 7.9).

Drooping of the eyelids

Called ptosis or blepharoptosis, this condition may be congenital or acquired, unilateral or bilateral, partial or complete, myogenic or neurogenic (Fig. 7.10 A). Congenital ptosis (Fig. 7.10 B) is due to maldevelopment of the levator of the upper lid with or without weakness of the superior rectus. Acquired ptosis is due to denervation (third nerve palsy), disinsertion of levator aponeurosis from tarsus (traumatic) or levator weakness (myasthenia gravis, myopathy (Fig. 7.11). In congenital ptosis, a history of eyelid drooping from birth is present.

An upper lid marginal mass can cause mechanical ptosis (Fig. 7.12 A). On examination congenital ptosis is recognised by the absence of superior palpebral sulcus (Fig. 7.10) which marks the insertion of levator aponeurosis into the skin of the upper eyelid. In acquired ptosis the palpebral fissure width on down gaze is usually lesser on

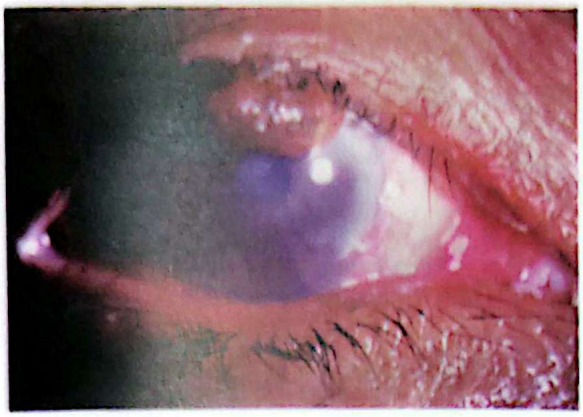


Fig. 7.12(A) Intermittent mechanical ptosis and corneal ulceration due to mass on the upper lid margin

the ptotic side than on the normal side, while the opposite is true for congenital ptosis. Treatment includes treatment of the cause and surgery. Type of surgical procedures employed include partial resection of the levator muscle, resection of Muller's muscle with conjunctiva, frontalis sling with fascia lata or Prolene suture4 (when the levator is non-functional), re-insertion of a detached or recessed levator muscle and levator aponeurosis advancement on the anterior surface of the tarsal plate[5]. When the patient is unfit for surgery, lid crutches (glasses with upper lid support) may be used (Fig. 7.12 B & C).

Hordeolum Externum (Stye)

It is an acute staphylococcal inflammation of eyelash follicle. Commonly presents as a painful swelling of the lid margin. To differentiate it from hordeolum internum, on examination of the lid margin, the point of discharging pus is anterior to the grey line while that of hordeolum internum is posterior to the grey line. Treatment is with local antibiotics and anti-inflammatory agents. Affected eyelashes the soft may need to be epilated.

Phthiriasis Palpebrarum

It is lice infestation of the eye lashes associated with severe itching and flakes on the lashes and lid margin. It results from poor hygiene and living in overcrowded places. Treatment is eliminating the cause and mechanical removal of lice with cotton tipped applicator and anti-lice shampoo followed by meticulous cleaning (Fig.7.13).

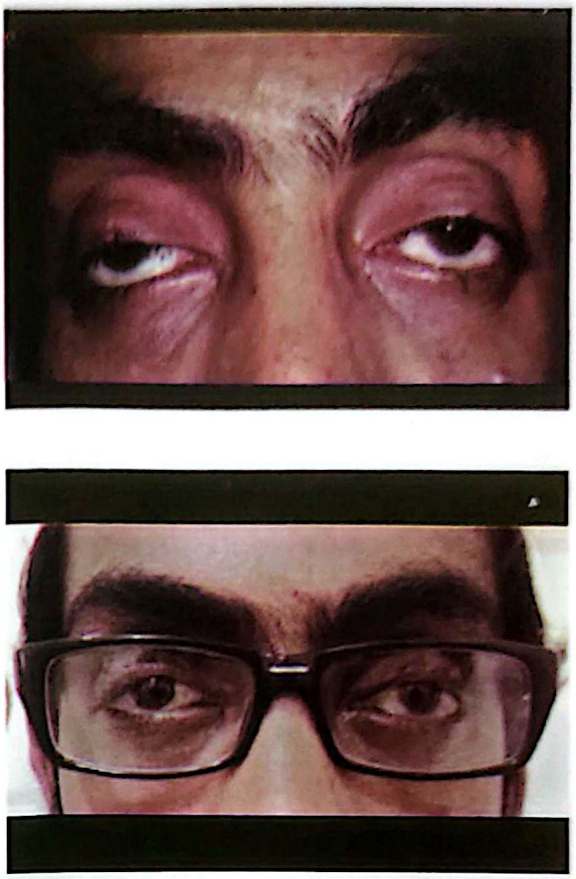


Fig. 7.12(B&C) Ptosis and correction after wearing lid crutches

Benign Masses of the Eyelids

These include chalazion (Fig. 7.15), hordeolum internum (Fig. 7.14), xanthelasma (Fig.7.16), cutaneous horn (Fig. 7.17). Raised Naevi (Fig. 7.17).

Chalazion

Chalazion is a chronic lipogranulomatous inflammation of the tarsal gland and is the most common eyelid mass. Clinically presents as a painless swelling in the lid with a history of slow enlargement. When secondarily infected it becomes painful and called "hordeolum internum" (Fig.7.14 A). It can rupture on the lid margin like a fungating lesion (Fig. 7.14 B)

Treatment of chalazion is incision and curettage from conjunctival side (fig.7.14). In case of recurrence, a biopsy should be sent for histopathology in an attempt to exclude adenocarcinoma at its early stage.

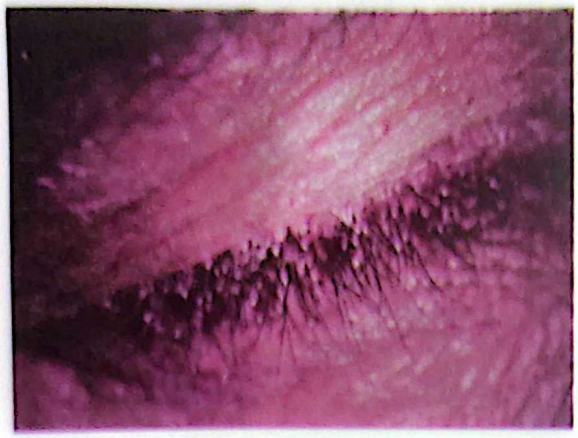


Fig. 7.13 Phthiriasis palpebrarum

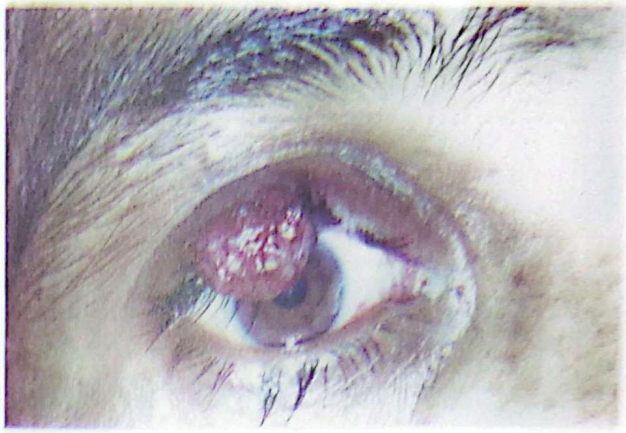


Fig. 7.14(B) Ruptured chalazion

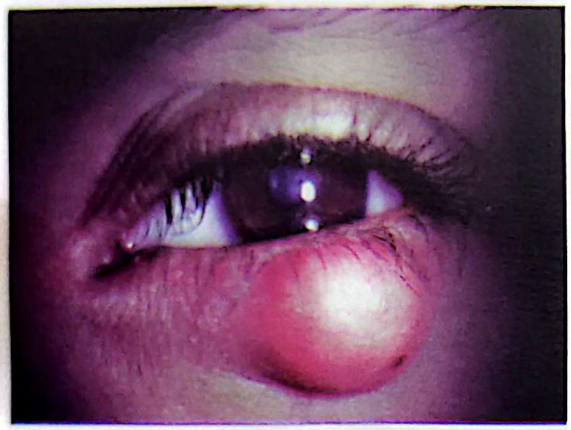


Fig. 7.14(A) A big hordeolum internum of the lower lid

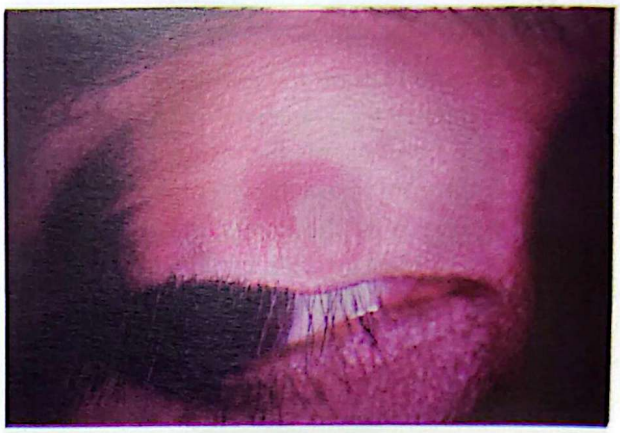


Fig. 7.15 Chalazion of the upper lid

Xanthelasma

Deposition of lipid material usually in the medial side of the skin of the lids in the form of soft plaques is called xanthelasma. Usually found in diabetic females and in hyperlipidaemia, it causes no symptoms except cosmetic (Fig.7.15).

Cutaneous horn

It is a cylindrical pigmented mass which may represent keratic growth of epidermal cells or a malignancy like malignant melanoma or squamous cell carcinoma. Treatment is excisional biopsy and further management in accordance with the biopsy report (Fig.7.16).

Tumours of the Eyelids

Although tumour can arise from any of the components of the eyelids, we will focus on tumours seen in routine clinical practice.

Benign tumours of the eyelids

Include capillary haemangioma, papilloma, neurofibroma and dermoid cyst (Fig.7.19 A).

Capillary Haemangioma (Fig. 42 A)

Capillary haemangioma is a congenital tumour, reducible with pressure. Clinically this bluish lesion may enlarge during early childhood giving rise to ptosis (when in the lid) or proptosis (when in the orbit) and regresses spontaneously or treated with intralesional steroid injections[6] or recently Beta-blockers, applied topically or used

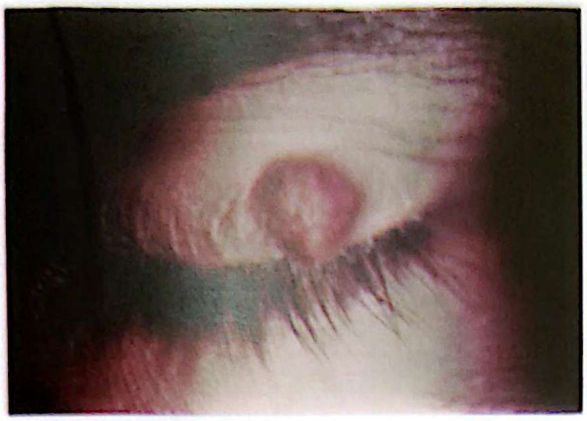


Fig. 7.16 Xanthelasma

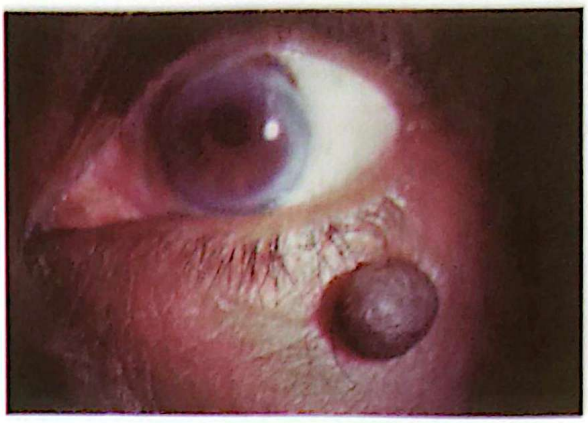


Fig. 7.18 Melanotic mass in the lower lid

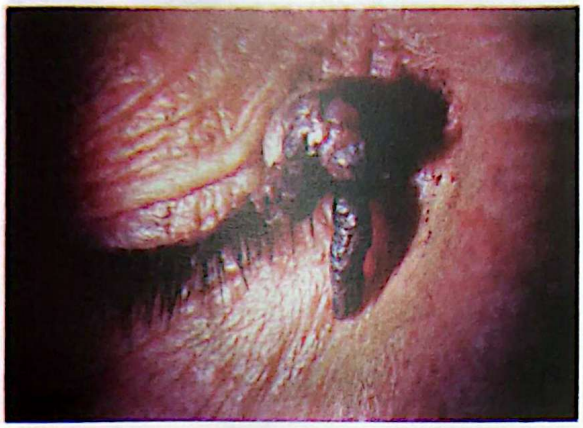


Fig. 7.17 Cutaneous horn arising from the upper lid

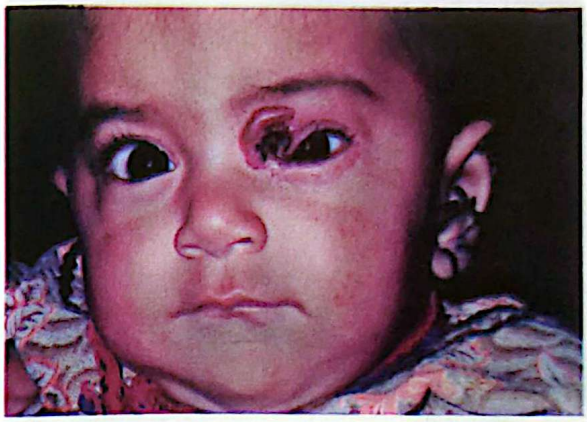


Fig. 7.19(A) Capillary haemangioma in a child being treated with sclerosing therapy

systemically, constitute a promising treatment option[7].

Papilloma

Solitary or multiple may appear on the lid margins at any age and are excisable. Rarely it may occur on tarsal conjunctiva (Fig. 7.19 B) as a hidden cause of bloody tears (Fig. 7.19 C).

Neurofibroma

Neurofibroma of the lid, a tumour arising from nerve sheaths, is usually part of neurofibromatosis (one of the phakomatoses) evident from multiple tumours over the body and presence of café au lait spots over the body.

Dermoid cyst

Dermoid cyst (is a slowly enlarging congenital benign tumour arising from aberrant ectodermal tissue and consisting of histologically normal cells but found at abnormal locations. More than half of them occur in the head neck region including the orbit. When left untreated, a dermoid cyst may enlarge enormously (Fig. 7.20 B) They are removed surgically for cosmetic purpose or when the cause complications like pressing nearby structures, or getting inflamed. When found on limbus (Fig. 7.20 A), treatment may consist of periodic removal of hairs, artificial tears, or excision if it is causing significant cosmetic disfigurement or interfering with vision. When surgery is indicated, a superficial sclerokeratectomy, cutting flush with the surface of the globe is done without attempting to remove it completely as it may have deeper extensions and heroic efforts of removal may end up in globe perforation.

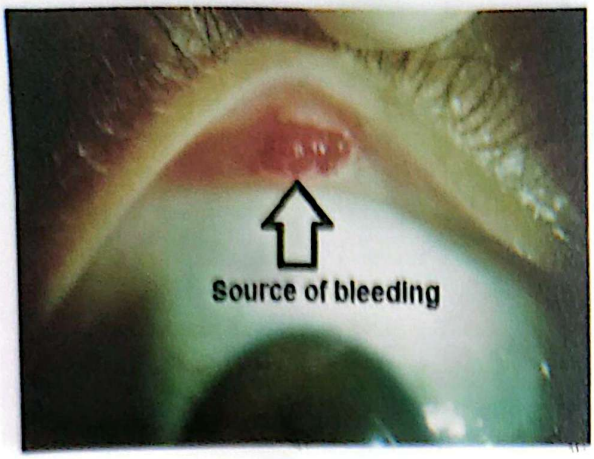


Fig. 7.19(B) Tarsal papilloma

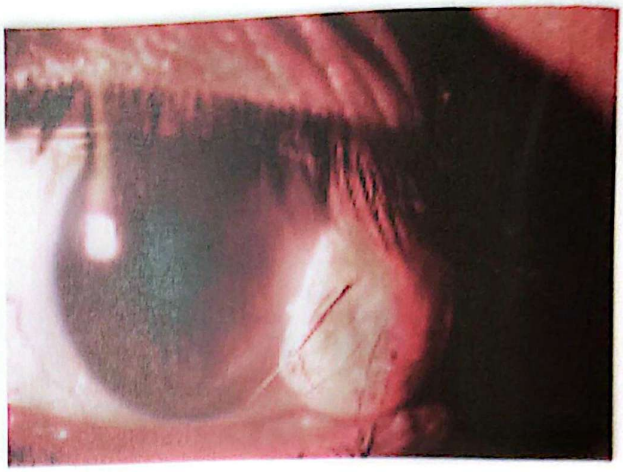


Fig. 7.20(A) Dermoid cyst

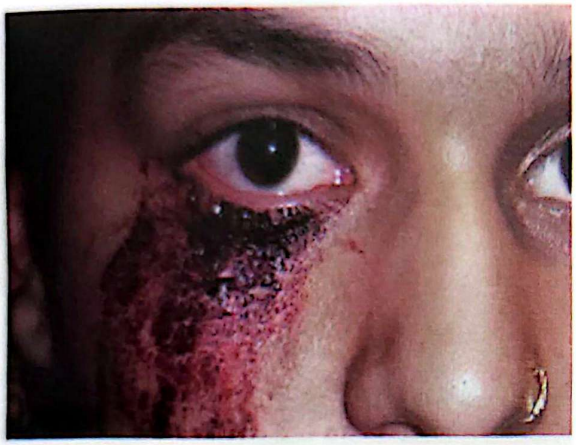


Fig. 7.19(C) Bloody tears

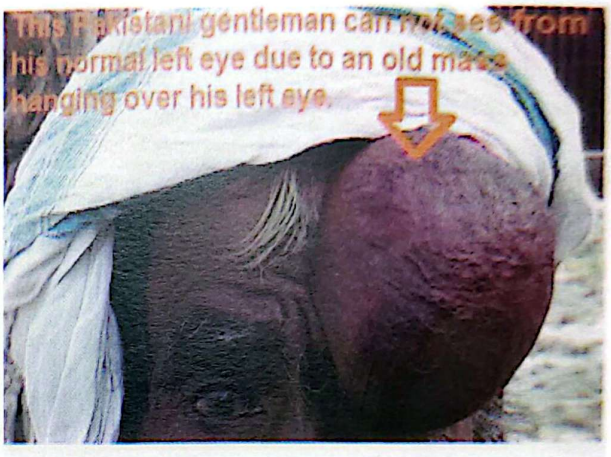


Fig. 7.20(B) Big dermoid cyst closing the upper eyelid

Malignant tumours

Arising from the eyelids are basal cell carcinoma (Fig.7.20 C), squamous cell carcinoma (Fig.7.21) and adenocarcinoma (sebaceous gland carcinoma). Rarely malignant melanoma may arise from the skin of the lids from a benign lesion (Fig.7.17).

Basal cell carcinoma

Also called rodent ulcer, the commonest lid tumour of the aged, is locally malignant and instead of metastasising it locally erodes the structures underway including bone, like a rat. Commonest site is the lower lid where it presents as a raised area with central ulcer and dilated vessels. When in involves the inner canthus, prognosis becomes guarded as it becomes difficult to eradicate the tumour without damaging important structures in this area. Fortunately, basal cell carcinoma is sensitive to radiotherapy.

Treatment is surgical excision[8] with one millimeter healthy margin and radiotherapy followed by skin grafting where indicated.

Squamous cell carcinoma

Squamous cell carcinoma of the eyelids (Fig.7.22), seen in older age groups, arises as a nodular mass or ulcer and grows faster than basal cell carcinoma in addition to metastasising. Treatment is surgical excision and radiotherapy.

Adenocarcinoma

Arises usually from the Meibomian gland (Fig.7.23 A) and is always a cause of concern to ophthalmologists as some times it may clinically present as a chalazion and a delay in diagnosis may end up in fatal metastasis. Therefore, histopathology of all recurrent chalazia may

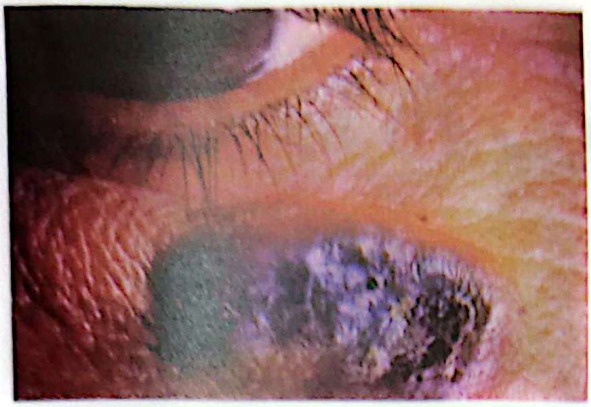


Fig. 7.20(C) Basal cell carcinoma of the lower lid arising as a pigmented lesion

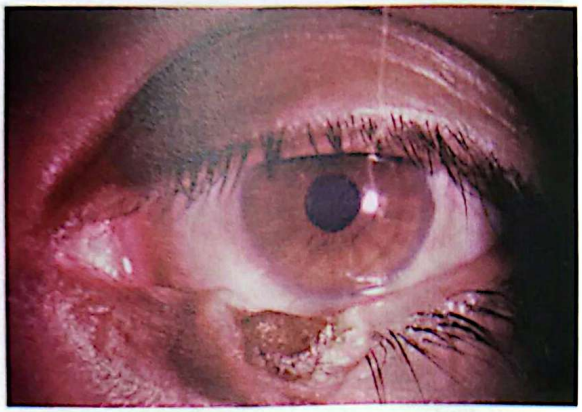


Fig. 7.21 Sqaumous cell carcinoma involving the lower lid

be performed to detect this malignancy at an earlier stage. Treatment is surgical removal and radiotherapy followed by plastic surgery.

Tarsorrhaphy

Creating an adhesion between the upper and lower eyelid margin to close the eye in order to protect the globe is called tarsorrhaphy. Tarsorrhaphy can be medial, lateral or central. Tarsorrhaphy (Fig.7.23 B) is performed in a desperate effort in threatened corneal perforation, indolent corneal ulcers, exposure keratitis and in cases of loss of corneal sensitivity. Temporary tarsorrhaphy[9] can be done with adhesives or temporary sutures. Permanent tarsorrhaphy can be opened (Fig.7.23 C) after having confirmed the healing.

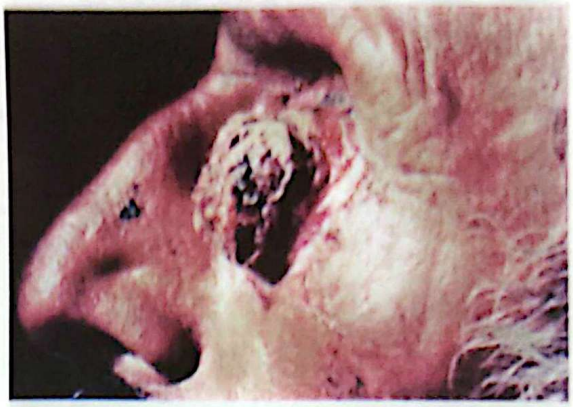


Fig. 7.22 Squamous cell carcinoma of the lid involving the globe

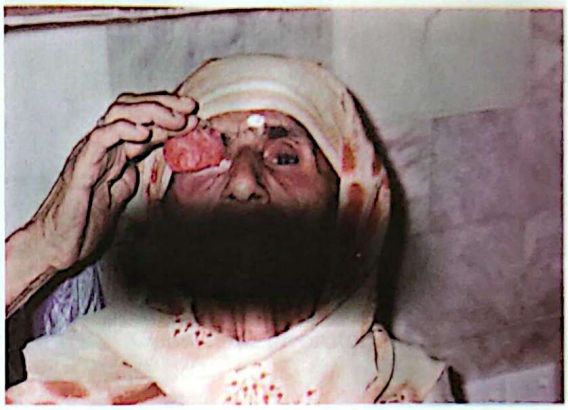


Fig. 7.23(A) Adenocarcinoma of the right upper lid.

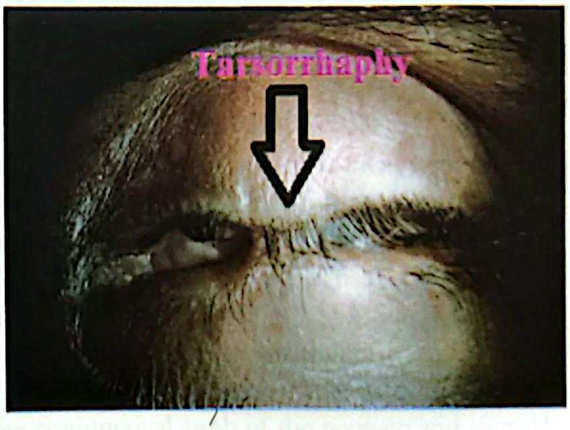


Fig. 7.23(B) Central tarsorrhaphy

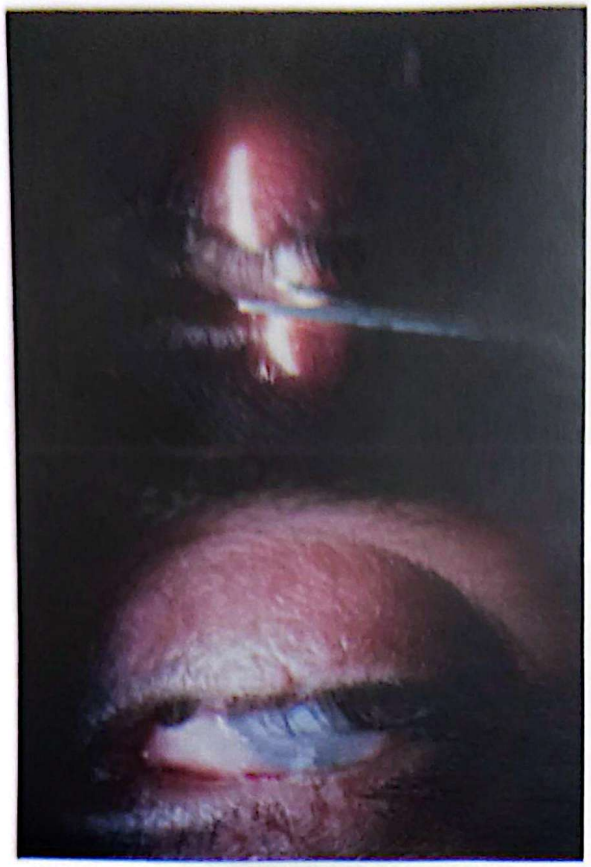


Fig. 7.23(C) Tarsorrhaphy is being opened.

Chapter 08

Diseases of Conjunctiva

Fig. 8.1(A) Subconjunctival Haemorrhage

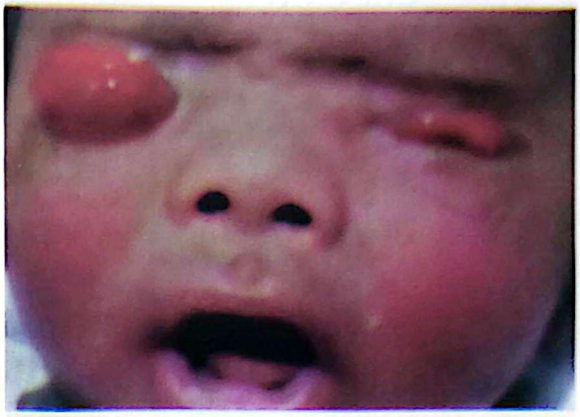


Fig: 8.1(B) Ophthalmia Neonatorum

Conjunctiva is a mucous membrane which covers the anterior surface of sclera (bulbar conjunctiva) and inner surface of the upper and lower eyelids (palpebral conjunctiva). Its normal secretions not only allow smooth movement of the eyeballs over the lids but also protects the corneal surface from drying.

COMMON CLINICAL FEATURES OF CONJUNCTIVAL DISEASE

Burning, itching, redness and matting of eye lids particularly after waking up from sleep are the usual features of conjunctival disease.

Subconjunctival Haemorrhage

Due to conjunctival translucency and its presence at a cosmetically crucial place, subconjunctival haemorrhage alarms the patient more than its clinical significance(Fig. 8.1A) In most of the cases it occurs due to trivial reasons like a blowing effort, cough, vomit etc, but sometimes it may be associated with serious condition like trauma to the eyeball, blood dyscrasias, systemic hypertension etc. It resolves spontaneously in two to three weeks.

Ophthalmia Neonatorum

It is infective conjunctivitis seen in neonates in the first 28 days of life. Infection is transmitted from the birth canal of the mother to the neonate. Causes are chlamydia trachomatis (Fig. 8.1 B), neisseria gonorrhoeae (Fig. 8.1 C), Staphylococcus aureus, Streptococcus pneumoniae and herpes simplex virus. Management starts with sending the conjunctival swab of the neonate and vaginal swab of the mother for microscopy, culture and sensitivity. Start broad spectrum antibiotic eye drops like ofloxacin 0.3% after cleaning the eyes with sterile saline. If gonococcal infection is suspected, systemic penicillin/cephalosporin should be given. If chlamydial infection is suspected, oral erythromycin should be given.

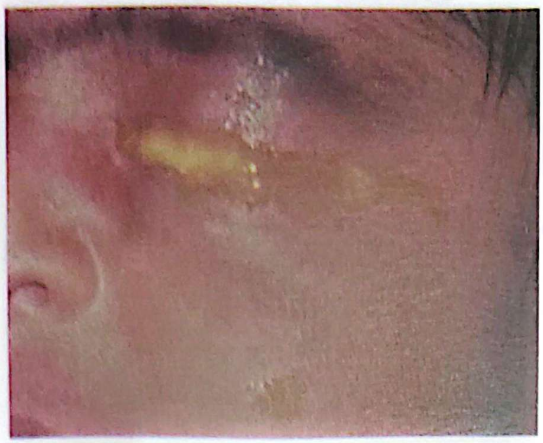


Fig. 8.1(C) Ophthalmia Neonatorum

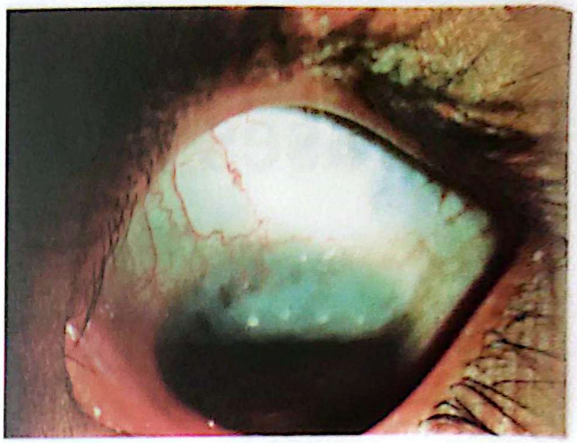


Fig. 8.3(A) Trachomatous pannus with limbal follicles

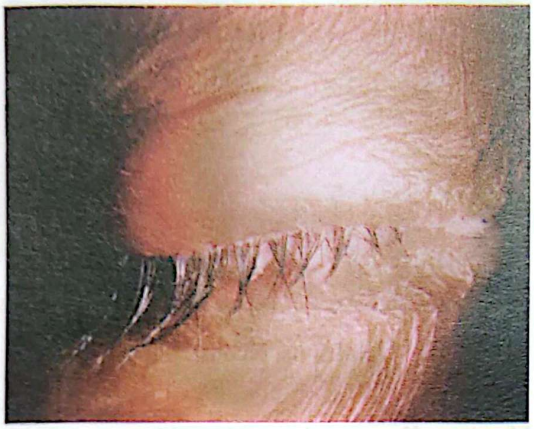


Fig. 8.2 Purulent conjunctivitis

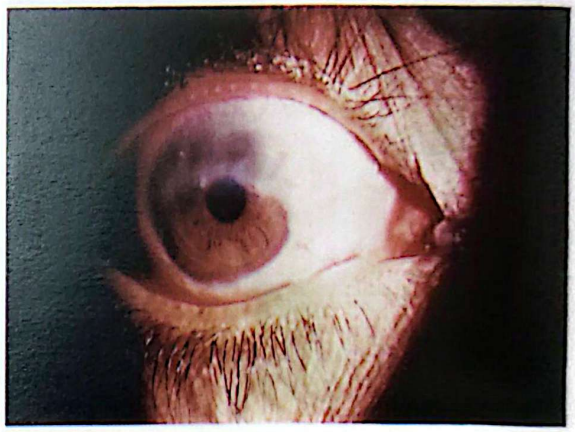


Fig. 8.3(B) Advanced trachomatous pannus

If staphylococcus aureus infection is suspected, fusidic acid eye drops should be added. If herpes simplex infection is suspected, acyclovir eye ointment should be instilled 6 hourly. The patient should be hospitalised under paediatric care. Preventive measures include child birth in aseptic conditions and the mother's infection control before delivery.

Bacterial Conjunctivitis

It presents as a mucopurulent conjunctivitis or purulent conjunctivitis.

Mucopurulent conjunctivitis

It may be caused by any of a wide variety of Gram positive or Gram negative bacteria and is associated with mucopurulent discharge. It usually responds to broad spectrum antibiotic eye drops used during the day time and ointments at night time, like gentamycin, tobramycin or ciprofloxacin, although the ideal treatment is to treat according to the culture and sensitivity report after having submitted the conjunctival swab to the pathology laboratory.

Purulent conjunctivitis

It is caused by gonococci. If not recognised and treated in time, it may cause serious complications including perforation of the cornea and spread to others. After sending the swab to the laboratory, treatment should commence immediately with intravenous injections of penicillin G 25000 units per kg body weight, twice a day for seven days. Frequent irrigation of the conjunctival sac with normal saline and topical use of tetracycline is advisable in addition (Fig. 8.2).

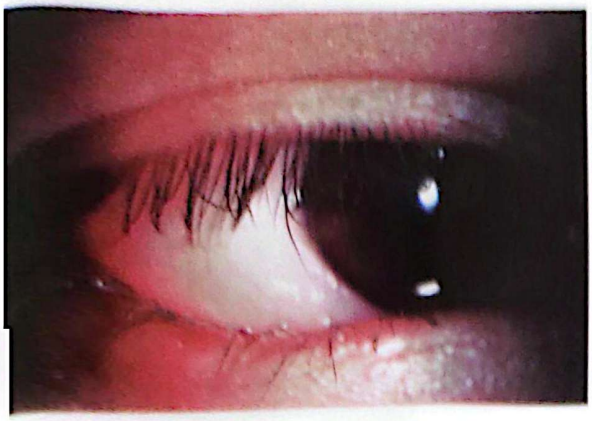


Fig. 8.4 Conjunctival Chemosis

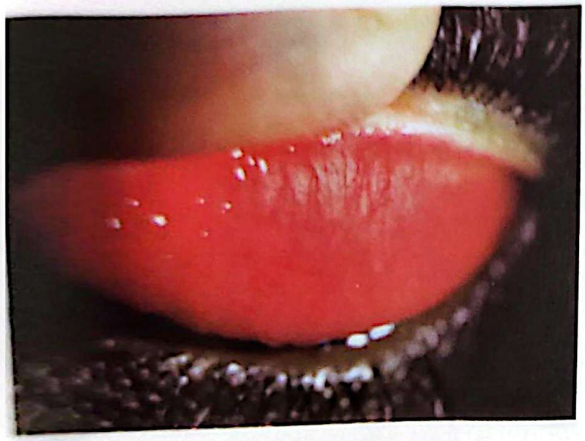


Fig. 8.5 Fine haemorrhages in upper tarsal conjunctiva

Trachoma

Caused by a separate group of small bacteria of genus chlamydia, this is a chronic cicatrising conjunctivitis affecting both eyes[27]. Specific causative organisms are A, B, Ba, and C strains of chlamydia trachomatis. Although the WHO has recently updated classification of trachoma, I have chosen MacCallan's classification as it also helps understand clinical progression of trachoma.

Stage	Description	Signs
Stage 1	Incipient Trachoma	Immature follicles, minimal papillary hypertrophy
Stage 2	Established Trachoma	Mature follicles on the upper tarsal plate
Stage 2A	With predominant follicular hypertrophy	Limbal follicles with pannus (Fig.49 A), Keratitis
Stage 2B	With predominant papillary hypertrophy	Intense activity with mature follicles buried in fulminant papillary hypertrophy
Stage 3	Cicatrising Trachoma	Scars on the upper tarsal conjunctiva, beginning of trichiasis and entropion
Stage 4	Healed Trachoma	Varying degrees of scarring with resolving pannus (Fig. 8.3 B), no papillary or follicular hypertrophy

Medical treatment of trachoma in adults is 250mg of tetracycline six hourly or doxycycline 100mg once daily for three weeks and/or topical sulphacetamide 10 to 30% four times a day for three weeks. Recently trachoma is being treated with a single dose of azithromycin which is easily available in Pakistan.

Surgical treatment is desirable for complications like trichiasis, entropion, corneal scarring etc.

Viral Conjunctivitis

A wide variety of viruses cause conjunctivitis, including the common offenders, the adenoviruses. Viral conjunctivitis is associated with watery discharge and conjunctival chemosis (Fig. 8.4) and sometimes with fine haemorrhages (Fig. 8.5). Treatment is symptomatic.

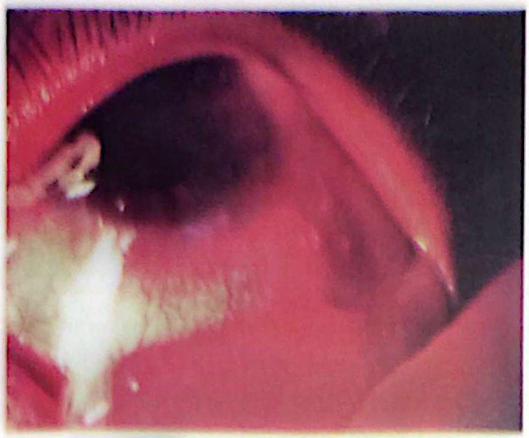


Fig: 8.6 Fungal conjunctivitis

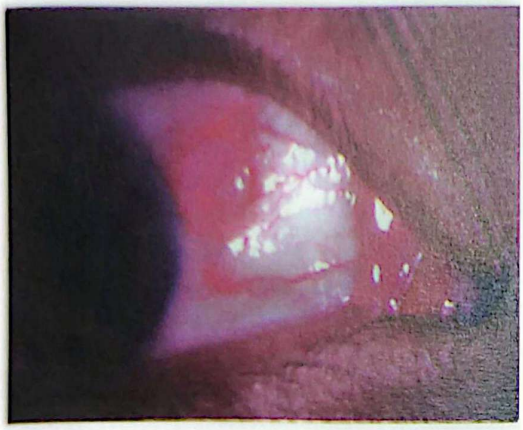


Fig. 8.7 Phlyctenular conjunctivitis

Fungal Conjunctivitis

It is common in the endemic areas of Pakistan like Larkana where rice crop is cultivated. The usual infection occurs by direct inoculation mostly together with corneal involvement. Treatment is effective with antifungal drugs, topical and sometimes systemic if presented late. Farmers should be advised to wear protective glasses during the sowing and harvesting season. (Fig.8.6)

Allergic Conjunctivitis

Various types of conjunctivitis under this group include atopic conjunctivitis, contact conjunctivitis, vernal conjunctivitis and phlyctenular conjunctivitis (Fig. 8.7).

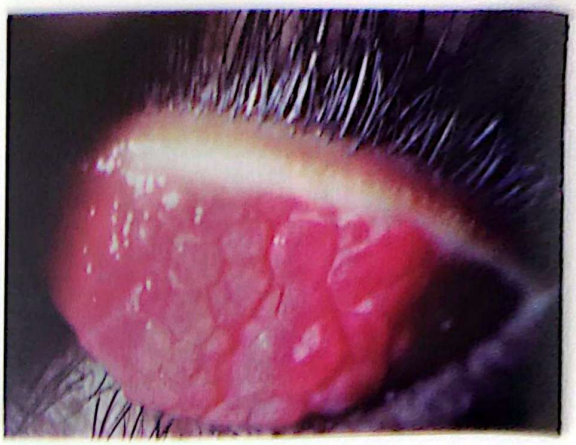


Fig: 8.8 Vernal conjunctivitis, palpebral form

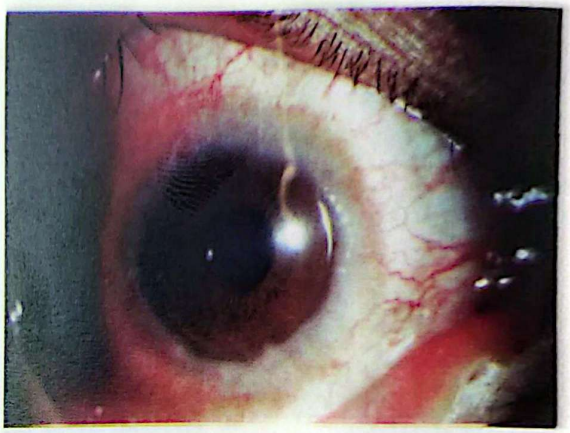


Fig: 8.9 Vernal conjunctivitis, bulbar form.

Atopic conjunctivitis

It is an acute conjunctivitis associated with lacrimation and chemosis, usually seasonal, in patients known to have allergic tendencies. Cause of allergy is usually not evident although pollen grains are known as one of the etiological factors. Treatment is topical and in severe cases systemic antihistamines.

Contact conjunctivitis

It Occurs due to instillation of antigen into the conjunctival sac including drugs prescribed for other ocular disorders. Treatment is withdrawal of offending agent.

Vernal conjunctivitis

Vernal conjunctivitis or vernal keratoconjunctivitis (VKC) is erroneously called as spring catarrh as it occurs in hot months and the better term should

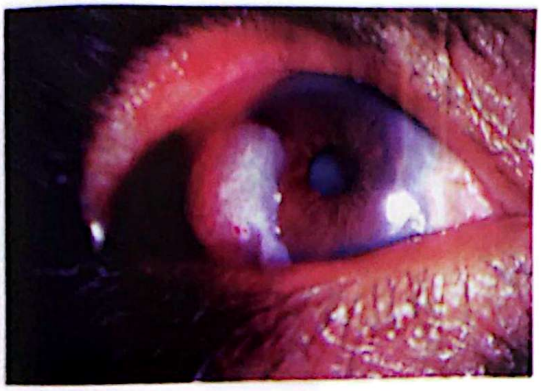


Fig. 8.10(A) Intraepithelial carcinoma of conjunctiva

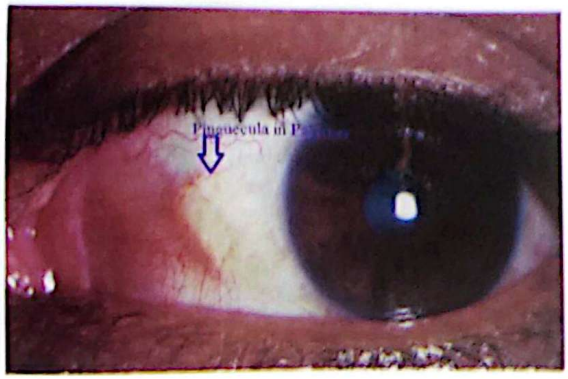


Fig. 8.10(B) Pinguecula

be a summer catarrh[22]. It occurs in three forms palpebral, bulbar and mixed. It is more common in boys and starts around five years of age and usually resolves at puberty. Severe itching, burning and foreign body sensation are the main symptoms of the disease and are almost intolerable. Palpebral form (Fig. 8.8) is associated with hyperaemia and giant papillary hypertrophy mostly in upper tarsal conjunctiva giving a typical appearance like cobble stones. The bulbar form (Fig. 8.9) is associated with a gelatinous elevation of corneoscleral limbus. Both changes are found in mixed form which is not common. Corneal involvement occurs in form of the punctate corneal erosions and plaque formation which results from deposition of desiccated mucus layers over a bare epithelial area provided by macro erosions on corneal surface. Treatment is mainly with decongestant drops and topical low potency steroids like fluorometholone. Supratarsal injections of long acting steroids have been tried with better results. Main danger to these patients remains steroid induced glaucoma as these patients, once exposed to high potency steroid drops, may keep on using them as self medication and may end up which blindness. Therefore routine intraocular pressure measurement in these patients is highly desirable.

Concretions

These are hard deposits in palpebral conjunctiva caused by accumulation of desiccated mucus and epithelial cell debris in micro clefts of conjunctiva. Although calcareous material is never deposited over them, they become hard and may cause foreign body sensation. They may be removed with the tip of a disposable needle when causing symptoms.

Bowen's Disease

It is intraepithelial carcinoma (Fig. 8.10 A) or carcinoma in situ which arise as a mass in the conjunctiva usually near the limbus in adults. Excisional biopsy with removal of one millimeter healthy margin around the lesion is always curative[10].

Pinguecula

A yellowish raised area usually on the nasal side of interpalpebral conjunctiva in adults called pinguecula (Fig. 8.10 B) is a hyaline degeneration and proliferation of elastic fibers due to exposure to dust, sun and wind. It requires no treatment.

Pterygium

Although this degenerative condition, which is found in people exposed to hot and dusty climate, is described as a fibrovascular proliferation of subconjunctival tissue arising as a triangular sheet usually over the nasal side (Fig.8.14) but some times over the temporal side of the limbus (Fig. 8.11) encroaching upon the cornea, it seems to be a disease of the cornea. It can be unilateral (Fig. 8.13 A) or bilateral (Fig. 8.13 B). Pathologically the process begins as a degeneration of Bowman's zone and superficial stromal lamellae at the nasal and sometimes temporal borders of the cornea.

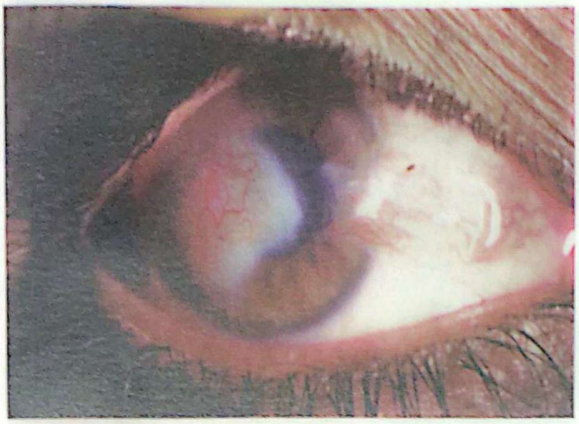


Fig: 8.11 Double pterygium

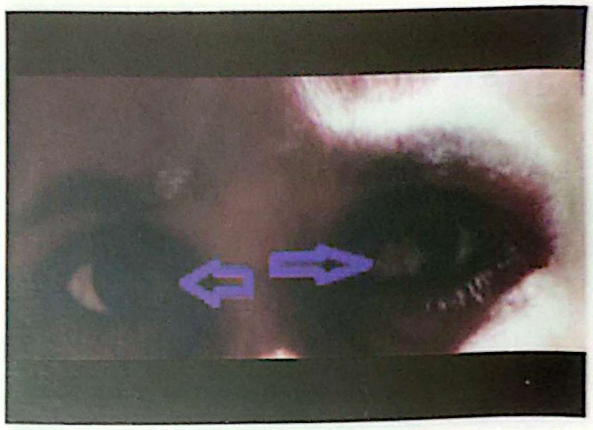


Fig. 8.13(B) Bilateral pterygium

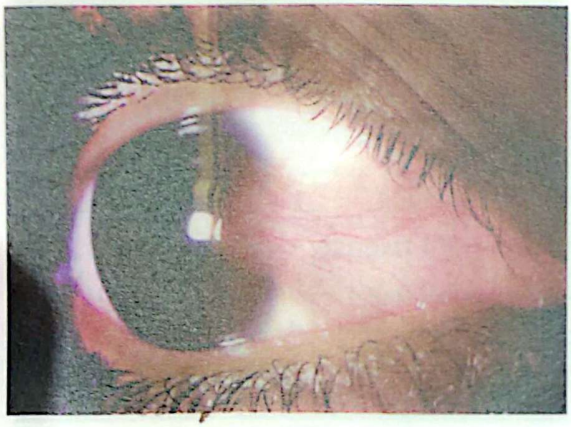


Fig: 8.12 Bleeding pterygium of Imtiaz

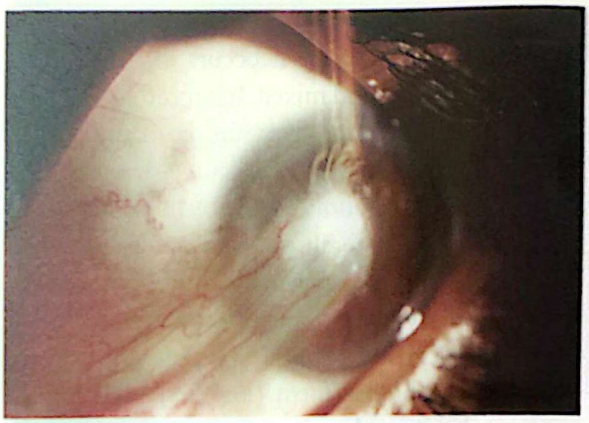


Fig. 8.13(C) Pterygium encroaching upon the visual axis

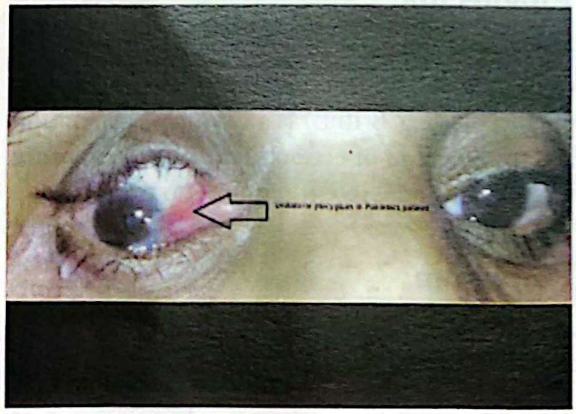


Fig. 8.13(A) Unilateral pterygium

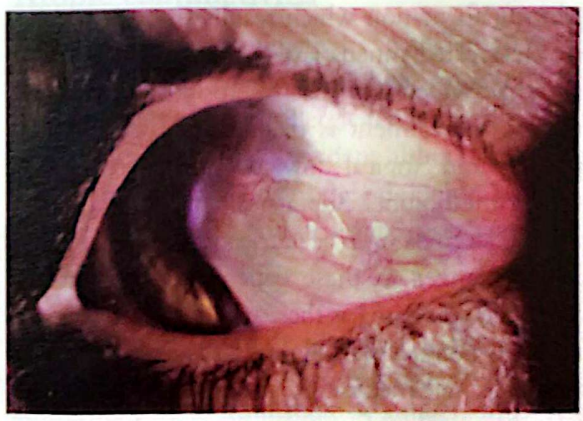


Fig. 8.14 Nasal Pterygium

taking with it a continuation of the conjunctival epithelium.

In early stages its main symptom is a cosmetic problem but as it progresses towards the center of the cornea, serious visual disturbance occurs due to corneal astigmatism or direct obscuration of vision (Fig. 8.13 C). Rarely bleeding may occur in pterygium, called bleeding pterygium of Imtiaz (Fig. 8.12) after its identification by the author. Another rare presentation is congenital pterygium of Imtiaz (Fig. 8.15), where pterygium is present

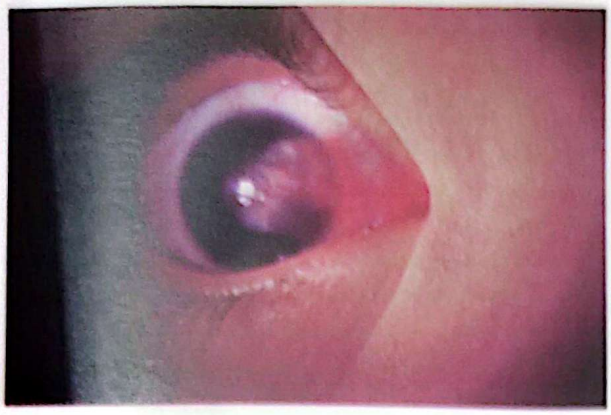


Fig. 8.15 Congenital pterygium of Imtiaz

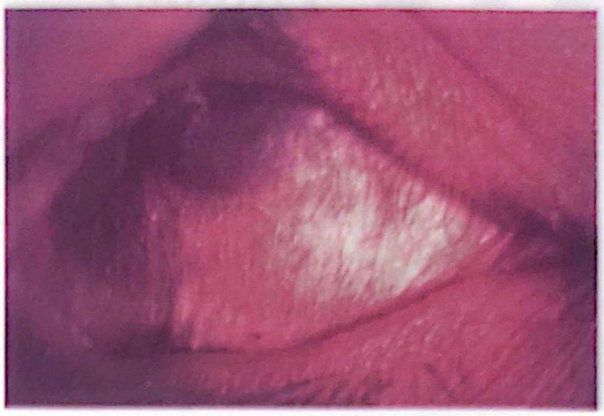


Fig. 8.17 Ocular pemphigoid

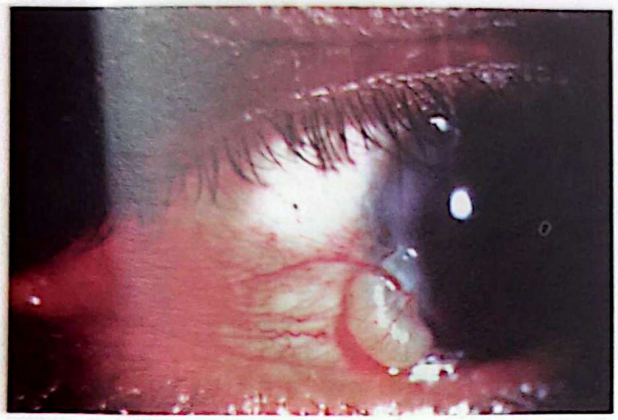


Fig. 8.16 Cystic pterygium of Imtiaz

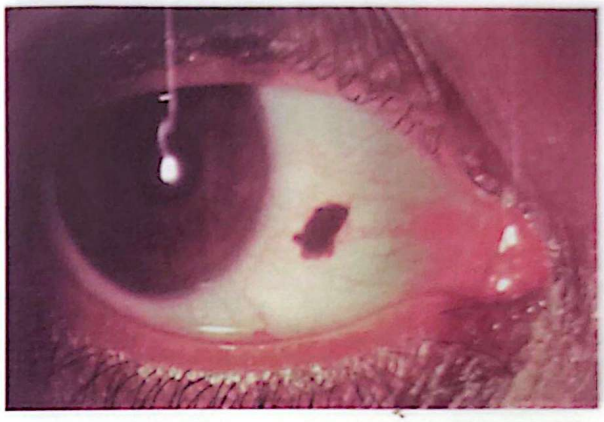


Fig. 8.18 Conjunctival nevus

at birth indicating multifactorial etiology of pterygium[11]. Cystic pterygium of Imtiaz is among the rare varieties containing single (Fig. 8.16) or multiple cysts. Treatment consists of surgical excision. Recurrence is common and can be prevented by use of diluted mitomycin C during surgery over the affected area or exposure of the bed of pterygium to B-radiations within 24 hours after surgery. Other treatments tried are subconjunctival bevacizumab (Avastin) injection[12], conjunctival autograft[13], amniotic membrane grafting[14] and stem cell transplantation[15].

Pseudopterygium

It is produced by attachment of a loose area of conjunctiva with an ulcerated area of cornea in an attempt to heal. After healing, this conjunctival attachment may be confused with pterygium. A probe can be passed after topical anaesthesia under a pseudopterygium as conjunctiva is only attached with cornea at its apical contact, whereas it can not pass under a pterygium.

Ocular Pemphigoid

Also known as benign mucous membrane pemphigoid is a bilateral idiopathic, chronic cicatrising conjunctivitis, seen in middle aged and older people, associated with oral lesions and in some cases skin lesions. Clinically presents with symptoms of dry eye and signs of symblepharon, trichiasis, entropion and epidermalisation of the cornea. It is a devastating condition and may end up in blindness if vigorous efforts of lubrication with artificial tears and lubricating gels are not taken. Surgery required is meticulous and is often a failure (Fig. 8.17).

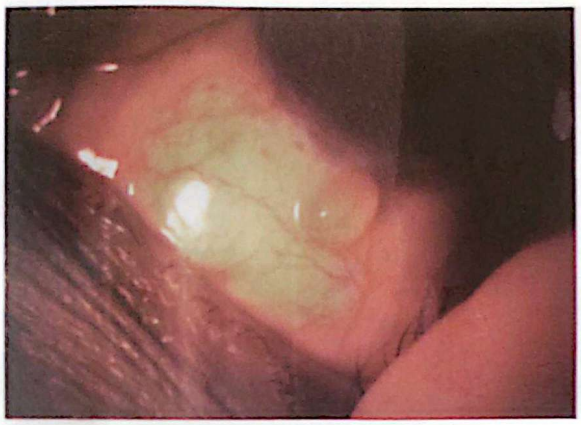


Fig. 8.19 Conjunctival cyst

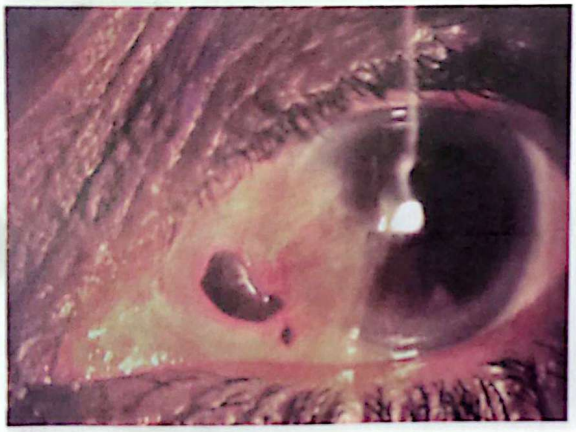
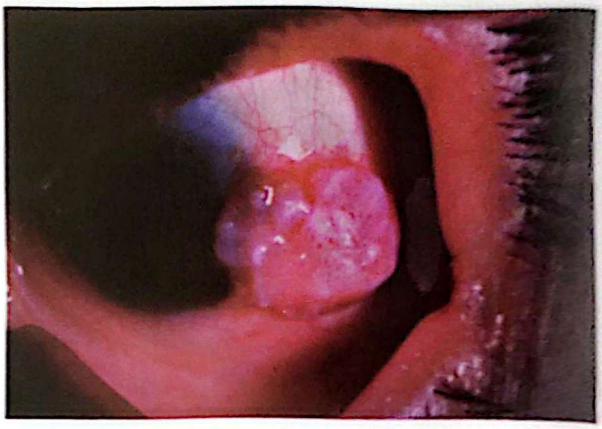


Fig. 8.20 Conjunctival malignant melanoma



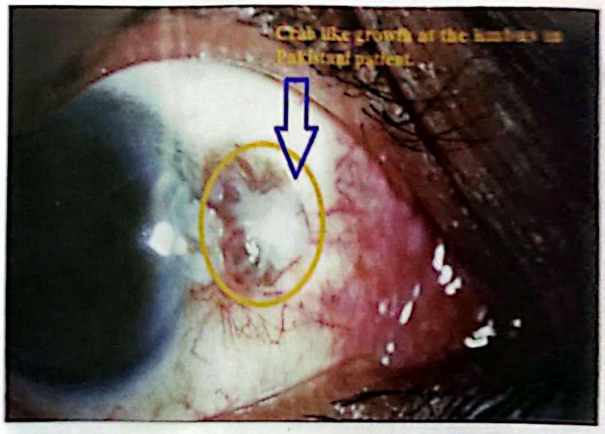


Fig. 8.21(A&B) Squamous cell carcinoma of conjuntiva

Conjunctival Nevus

It is a normal finding like nevi, present in other parts of the body. Their importance lies in cosmetic concern and when they start enlarging. Removal is indicated with biopsy to exclude malignant change when it increases in size (Fig. 8.18).

Benign Conjunctival Cysts

These are a normal finding and need reassurance as they disappear spontaneously. When persistent, they may be perforated with a disposable needle tip (Fig. 8.19).

Malignant Tumours

Malignant tumours of conjunctiva include malignant melanoma (Fig. 8.20), squamous cell carcinoma (Fig. 8.21 A & B) and lymphoma. Treatment is by surgical removal, chemotherapy and radiotherapy.

Chapter 09

Diseases of the Cornea

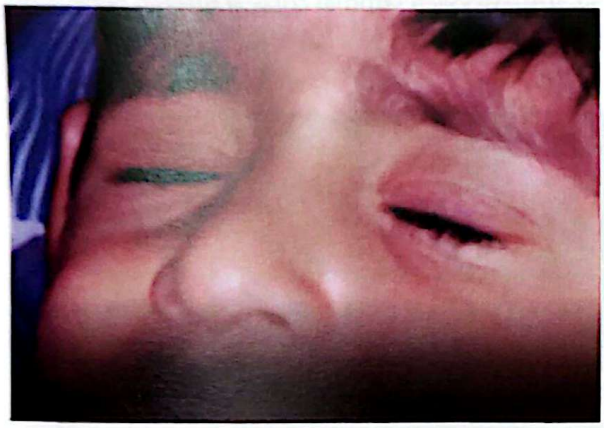


Fig. 9.1 Blepharospasm in a child with corneal abrasion

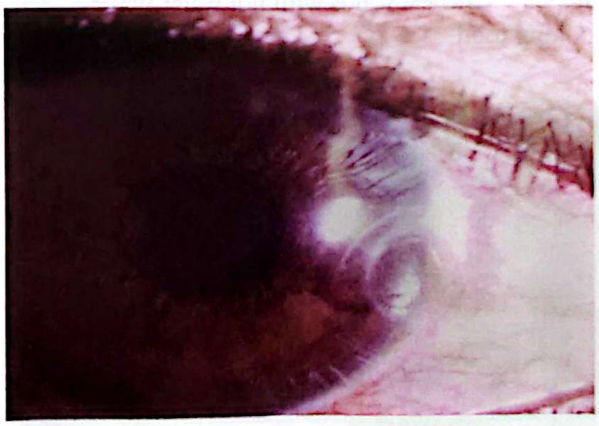


Fig. 9.2 Corneal dellen

The cornea forms the anterior one sixths of the outer most coat of the eyeball. It is elliptical anteriorly due to more limbal extension of tissue superiorly and inferiorly. Thus, if its horizontal diameter is 11.7 mm the vertical becomes 10.6 mm, however posteriorly it is spherical with a diameter of 11.7 mm horizontally and vertically. The central anterior radius of curvature of the cornea measures 7.8 mm but it flattens to an average of 6.5 mm in periphery. From the outside in cornea is made up of epithelium, Bowman zone (which is a condensed acellular layer of stroma), stroma, Descemet's membrane (which is the basement membrane of corneal endothelium and is the toughest basement membrane of the body) and endothelium. Epithelium is the most regular of all the squamous epithelia of the body. It is 5 to 6 layers thick, superficial cells being squamous, middle area contains polygonal cells and the basal area contains a single layer of columnar cells which are the source of new cells. Epithelium is made up of labile variety of cells thus it replaces itself normally every 7 days. Epithelial insults heal without scarring. Bowman zone is a rigid acellular structure which is composed of interdigitating collagen fibrils. Insults of Bowman zone heal with scarring. Corneal stroma is composed of approximately 225 lamellae of collagen fibril bundles separated by ground substance of glycosaminoglycans. Stroma makes up about 90 % of the cornea. Stromal cells, called keratocytes, transform into fibroblasts which eventually lead to formation of corneal scar during corneal wound healing. Corneal endothelium is a single layer of cells with the density of approximately 5500 cells/mm2 of cornea at birth. Several hundred endothelial cells are lost every year as part of the aging process, so that at 10 years of age cell count declines to approximately 3500 cells/mm2 of cornea. At seventy years of age approximately 2000 cells /mm2 of cornea are left. Remaining endothelial cells compensate by cell spreading and enlargement rather than by division. Endothelium keeps the cornea dehydrated by way of its barrier

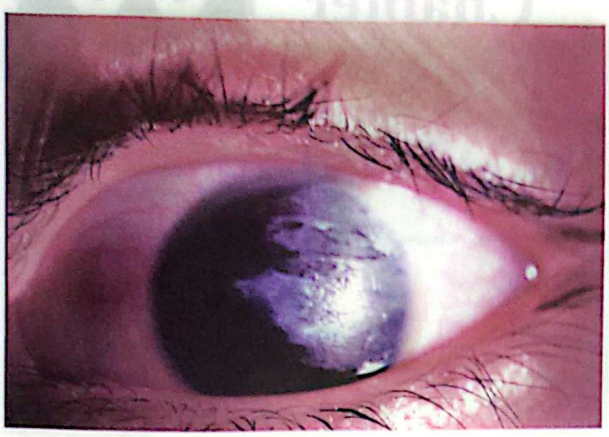


Fig. 9.3 Corneal scalds with hot water while cooking food

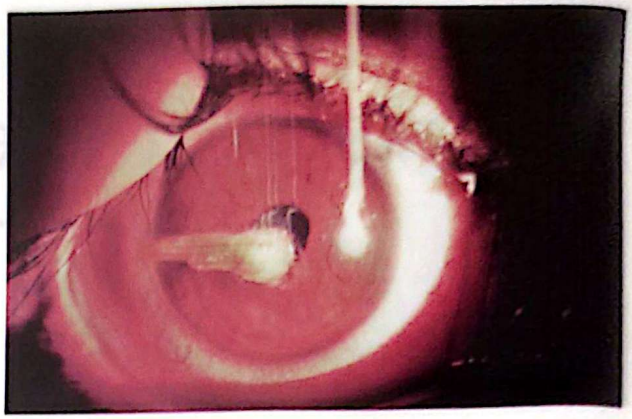


Fig. 9.5 Wooden stromal corneal foreign body

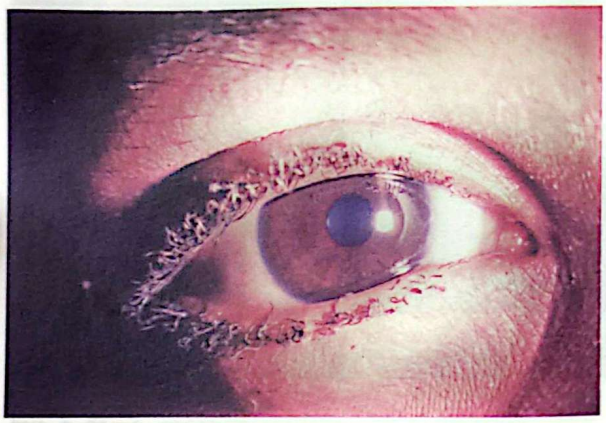


Fig. 9.4 Burns of eyelashes while cooking food

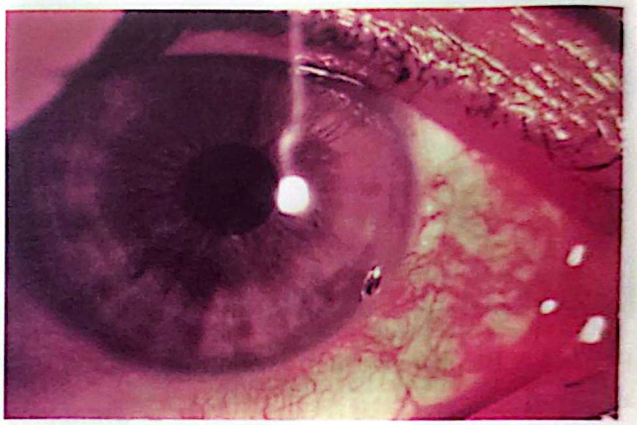


Fig. 9.6 Insect body embedded at the limbus

and pump functions. The cornea acts as the main refracting medium of the eye. It provides +40 to +45 dioptres of refracting power to the eye. Corneal transparency depends mainly upon its avascularity, dehydrated state, smooth surface epithelium and well organized stromal collagen fibers. The cornea does not depend on oxygen provided by the lungs through blood but takes oxygen from the air.

COMMON CLINICAL FEATURES OF CORNEAL DISEASE

Decreased vision, foreign body sensation, blepharospasm in children (Fig. 9.1), halos around lights, pain, and white opacity in the eye (cornea) and localised dimple in the cornea (dellen Fig. 9.2) are the clinical features of corneal disease.

Corneal Abrasion, Burn (Fig. 9.4) and Scald (Fig. 9.3)

Corneal abrasion is a focal loss of corneal epithelium due to a trivial day to day trauma. Corneal scald occurs usually due to a drop of hot water or cooking oil instilled during preparation of food. Both conditions are alarming for the patient due to pain, photophobia and profuse tearing. Abraded area of the cornea may be seen on a slit lamp after fluorescein staining. Corneal scald looks like a raised white lesion. Both conditions respond quickly to antibiotic eye drops and pad and bandage for 24 hours.

Corneal Foreign Body

(Fig.9.5) A wide variety of corneal foreign bodies (Fig.9.6) are seen in clinical ophthalmic practice, starting from an insect to a piece of metal. Iron foreign bodies are frequently found with rust

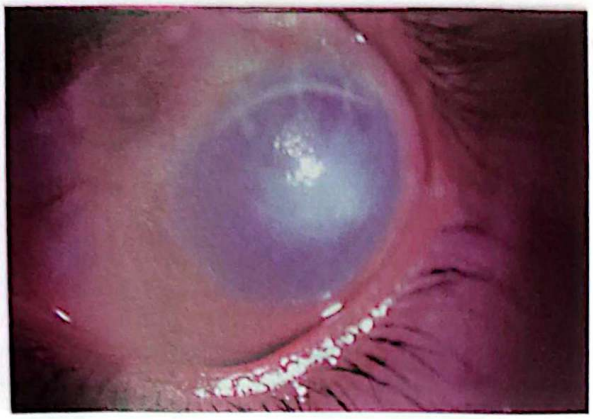


Fig. 9.7 Advanced stage of bullous keratopathy with corneal opacification

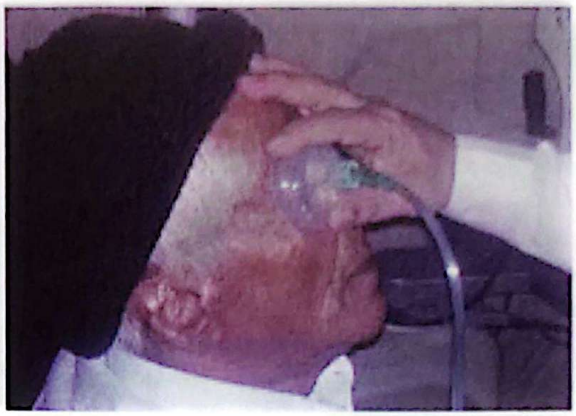


Fig. 9.9(A)

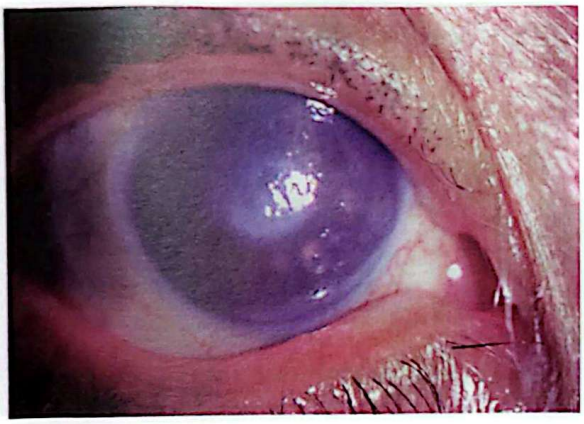


Fig. 9.8 Bullous keratopathy

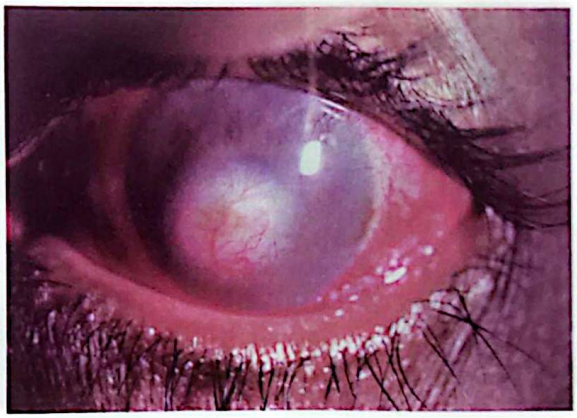


Fig. 9.9(B) Deep corneal vascularization

which is more difficult to remove than the foreign body itself. After instilling local anaesthetic eye drops, one drop to be instilled thrice with one minute interval, the foreign body is usually removed with the tip of a disposable 26-gauge syringe needle or with a sterilised foreign body spud after well placing the patient's head on a slit lamp. Antibiotic drops are instilled after removal of the foreign body and pad and bandage may be done for 24 hours.

Corneal Edema

Epithelial and subepithelial corneal oedema develops either as a result of rapid rise in IOP or due to corneal suffocation, as e.g., caused by a hard contact lens. Stromal oedema results from corneal endothelial dysfunction which finally ends up in bullous keratopathy (Fig. 9.7).

Bullous Keratopathy

Corneal endothelial decompensation/dysfunction leads to accumulation of fluid in the cornea which is seen in the form of bullae at the surface of the cornea (Fig. 9.8). Bullae rupture at the corneal surface exposing free nerve endings resulting in severe discomfort to the patient. Endothelial damage can occur during cataract surgery[16] and glaucoma surgery or may occur in Fuch's dystrophy or due to aging. Medical treatment of bullous keratopathy includes topical lubricants, hypertonic solution (5% sodium chloride), bandage contact lenses or multiple needle punctures to give symptomatic relief to the patient. Presently a quasi-experimental study[17] on ITOT (Imtiaz's Technique of Oxygen Therapy) is in progress in Pakistan aimed at improving vision in addition to symptomatic relief (Fig. 9.9 A). Surgical treatment is endothelial keratoplasty.

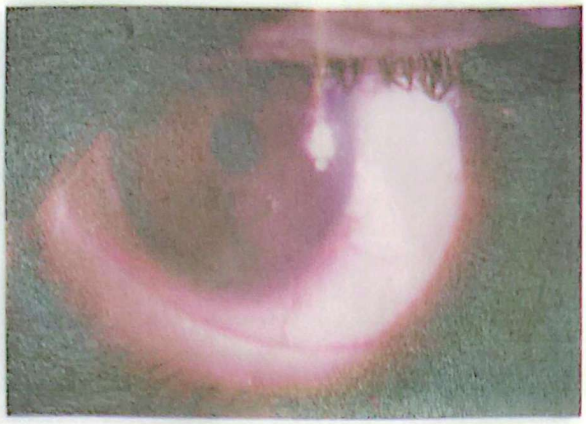


Fig. 9.10 Superficial corneal vascularisation due to corneal foreign body

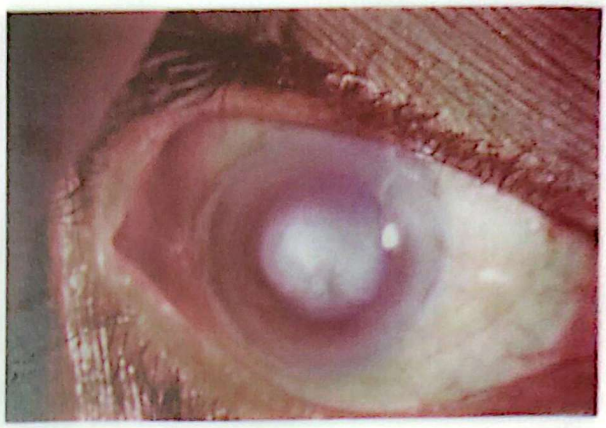


Fig. 9.12(A) Corneal leukoma

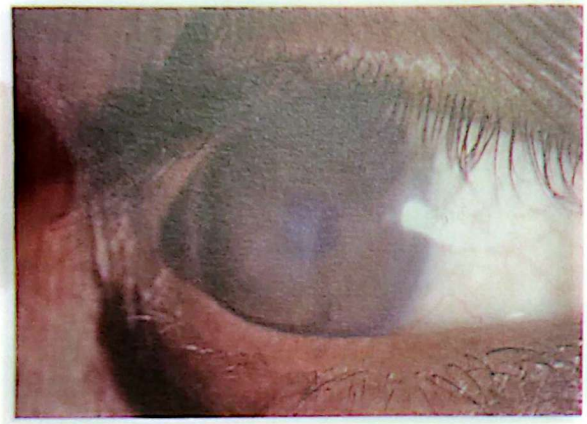
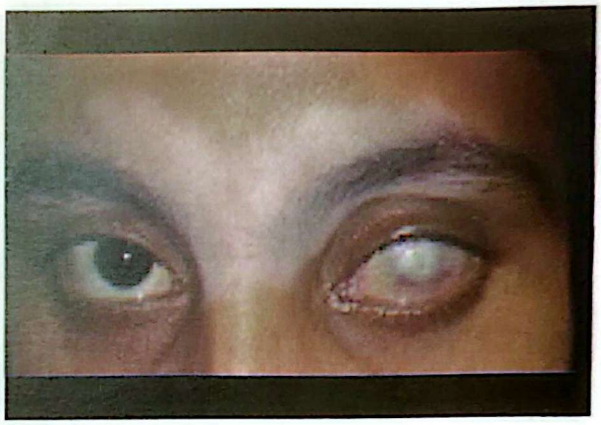


Fig. 9.11 Corneal macula



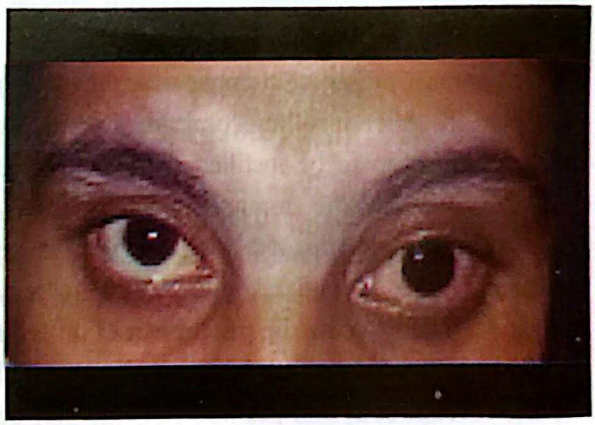


Fig. 9.12(B&C) Corneal prosthesis for cosmetic reason

Corneal Vascularisation

The cornea is normally avascular but vascularisation can occur as a part of an inflammatory process. Most serious results of vascularisation are its loss of transparency and loss of privilege of being a good recipient site for graft, due to biochemical modification of its tissues. Superficial corneal vascularisation occurs in the subepithelial area or over the epithelium as a fasciculus (Fig. 9.10). Deep vascularisation (Fig. 9.9 B) occurs in stromal area. When subepithelial vascularisation is combined with fibroblastic proliferation, it is called a pannus (Fig. 49). When vascularisation resolves, the left over blood less channels are called ghost vessels.

Corneal Opacities

Focal or generalised loss of transparency leads to corneal opacity. Mild loss of transparency can be seen with the help of a slit lamp and is called nebula. Moderate loss of transparency can be seen with the naked eye and is called macula (Fig. 9.11). Severe loss of corneal transparency is called leukoma (fig. 9.12 A). When the iris is attached with leukoma, the condition is called adherent leukoma. Central opacities in the cornea impair vision more than peripheral opacities.

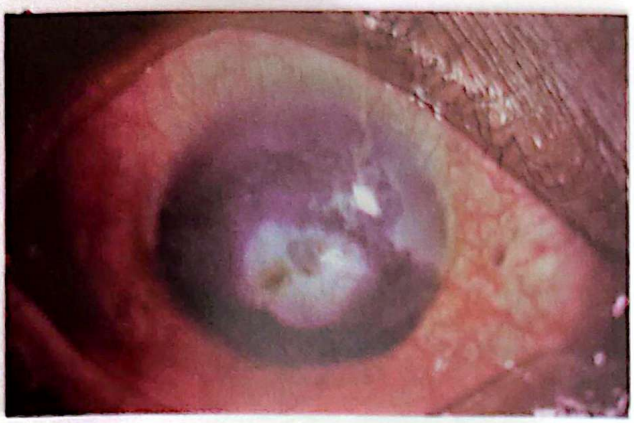


Fig. 9.13 Corneal tattooing done for a white cornea

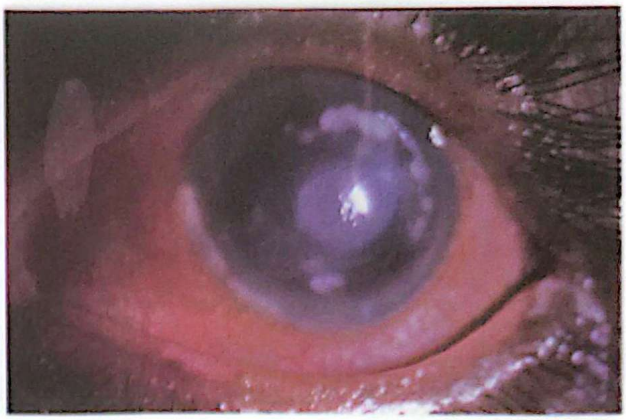


Fig. 9.15 Ring ulcer in amoebic keratitis

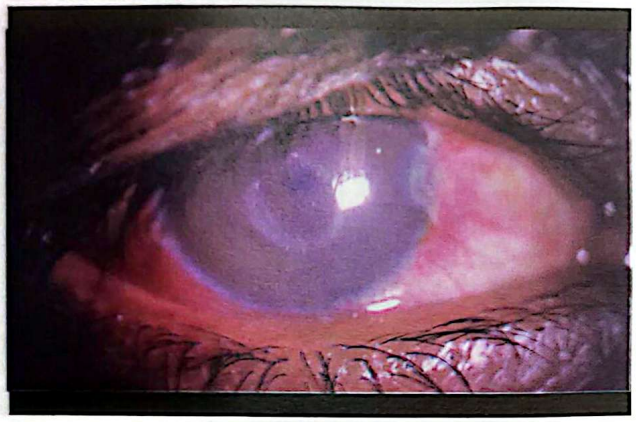


Fig. 9.14 Amoebic keratitis

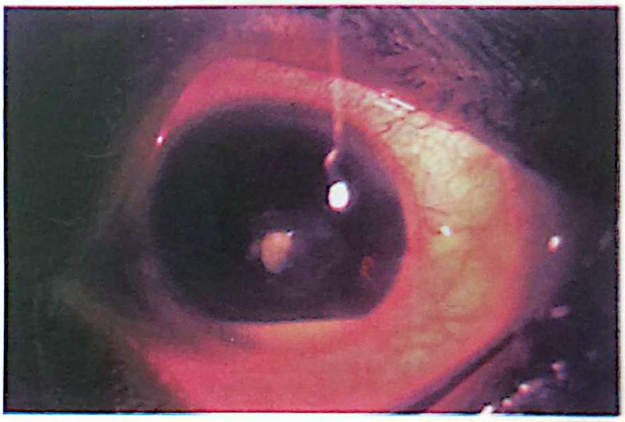


Fig. 9.16 Hypopyon ulcer caused by staph aureus

In visually capable eyes, an opaque cornea is replaced with a transparent donor cornea (keratoplasty) but in a blind eye with an opaque cornea, cosmetic prosthesis is advised, particularly in females (Fig. 9.12 B & C) as a temporary measure and tattooing of the cornea (Fig. 9.13) as a permanent measure.

Corneal Inflammations (Keratitis)

Keratitis may be classified into ulcerative and nonulcerative types.

Ulcerative keratitis may be central or peripheral.

Central ulcerative keratitis

It is caused by bacteria[18], viruses, fungi, protozoa (amoebic keratitis Fig. 9.14, 9.15), corneal anaesthesia (after some neurosurgical procedures) and nutritional deficiency (keratomalacia).

Acute hypopyon ulcer

An acute corneal ulcer with hypopyon (sterile pus in the anterior chamber) is usually caused by pneumococci (Fig. 9.17 A) and staphylococcus aureus (Fig. 9.16). Other bacteria like enteric bacteria, staphylococcus epidermidis and Moraxella lacunata may also cause hypopyon corneal ulcer.

Pneumococcal corneal ulcer usually follows trauma to the cornea. Bacterial invasion starts at the site of the trauma causing a dirty grey coloured ulcer with overhanging margins. Patients suffer from decreased vision, photophobia, pain and discharge. Corneal perforation occurs if left untreated (Fig. 9.17 B). After taking swab for culture and sensitivity, treatment should commence with appropriate broad spectrum antibiotics such as cephalosporins, ofloxacin or ciprofloxacin and fuesidic acid if staph aureus

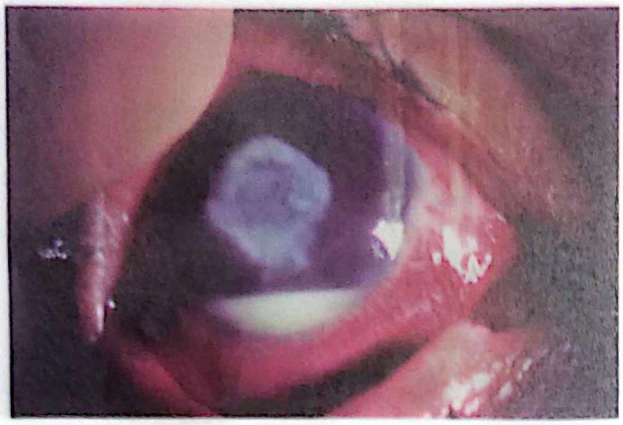


Fig. 9.17(A) Hypopyon ulcer caused by pneumococci

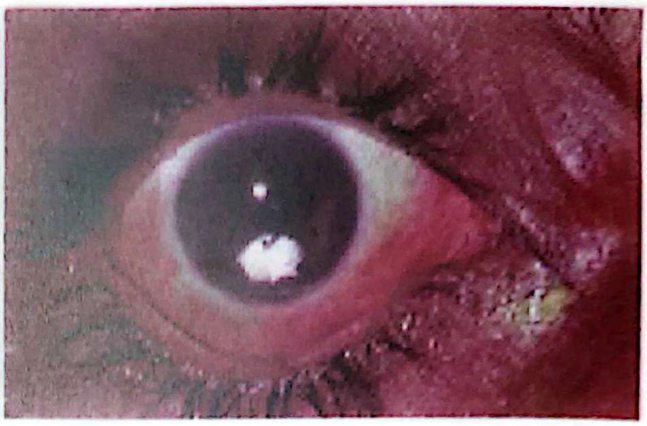


Fig. 9.17(C) Scleral autograft covered with BCL

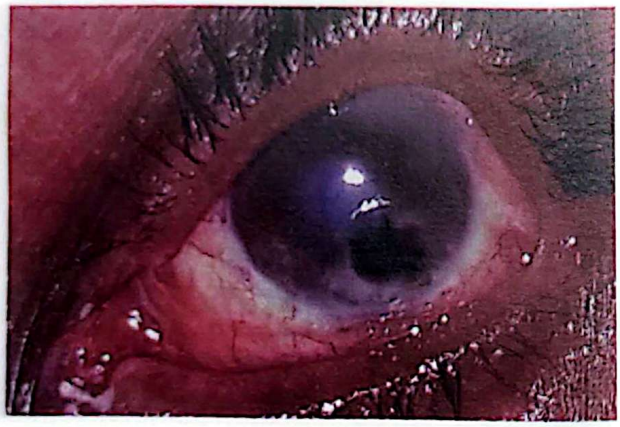


Fig. 9.17(B) Perforated corneal ulcer

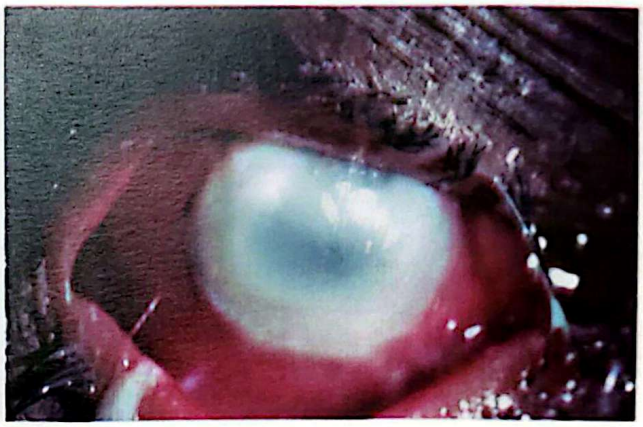


Fig. 9.18 Necrotising keratitis (corneal sloughing)

is suspected. Atropine eye drops are added to restrict ciliary body and iris movement to avoid pain. Systemic antibiotics and anti-inflammatory agents may be required in severe cases. In cases of perforated corneal ulcers, treatment options are bandage contact lenses (BCL), scleral autograft[19] (Fig. 9.17 C), conjunctival flapping or therapeutic keratoplasty.

Necrotising keratitis

Fulminating corneal ulcer usually begins centrally and rapidly broadens and deepens leading to corneal sloughing within 2 to 5 days (Fig. 9.18). Necrotising keratitis (Fig. 9.19) is caused by pseudomonas strains which produce proteases, lipases and exotoxin A, it usually follows a mild corneal trauma like finger nail abrasion with the use of contaminated cosmetics like mascara. This unhindered destruction of cornea occurs due to unavailability of vascular response as cornea is avascular and acts like a tied prisoner. Gentamycin or tobramycin are very effective in addition to other antibiotics and treatment should commenced without loss of precious time, preferably with fortified eye drops e.g., addition of 2cc of injectable tobramycin (40mg/cc) to 5cc tobramycin drops will give fortified tobramycin eye drops.

Herpes simplex keratitis

Herpes simplex keratitis (HSK) (Fig. 9.20) is usually caused by herpes virus hominis type 1 (oral) and rarely by type 2 (genital) and is among the common causes of corneal ulceration in Pakistan. HSK presents in three forms, i.e., congenital, primary and recurrent. Congenital ocular herpes occurs either by way of a transplacental or ascending route. Clinical manifestations vary from

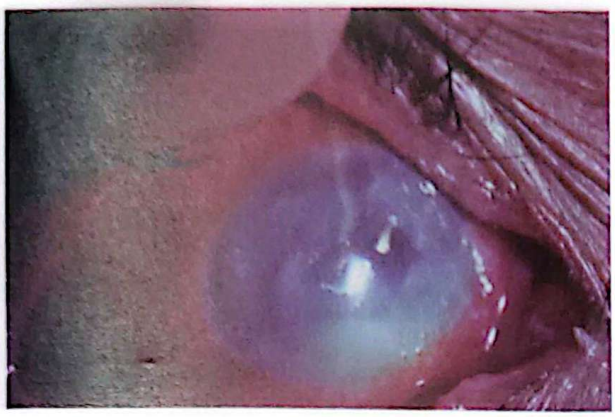


Fig. 9.19 Necrotising keratitis

Fig. 9.21 Dendritic ulcer of herpes simplex

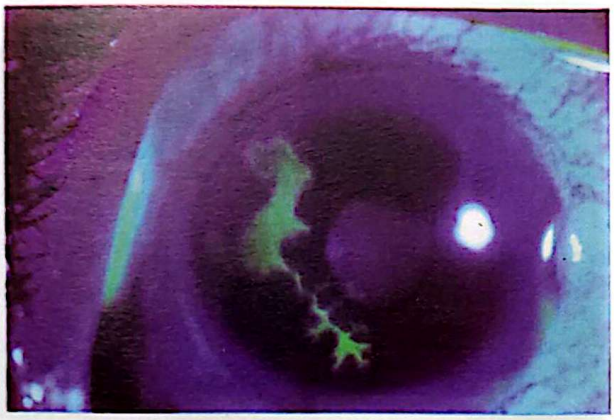


Fig. 9.20 Dendritic ulcer of herpes simplex

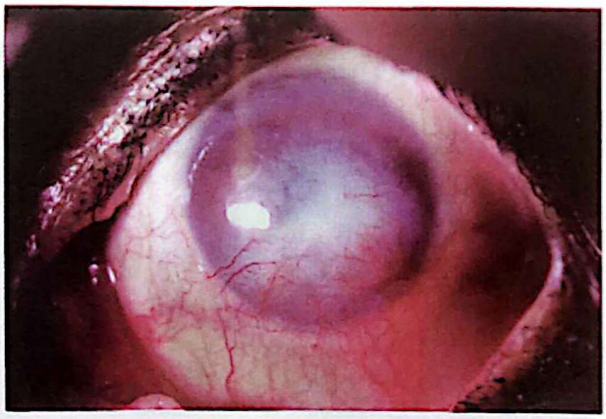


Fig. 9.22 Vascularisation of the cornea after the disciform stage of herpes simplex keratitis

conjunctivitis, epithelial keratitis, stromal keratitis, cataracts to chorioretinitis. Primary ocular herpes may present as acute keratoconjunctivitis with preauricular lymphadenopathy. Initially superficial punctate keratitis may evolve into scattered microdendritic ulcers limited to epithelial layer. Stromal involvement is not seen in primary HSK because the patient lacks immunity against this virus. Primary HSK does not occur in the first six months of life due to partial protection provided by maternal antibodies. Recurrent HSK may be classified into epithelial, stromal and disciform keratitis.

Epithelial HSK manifests as infectious ulceration and may progress to trophic (metaherpetic) ulceration. Infectious ulceration presents with characteristic dendritic (Fig. 9.21) or geographic ulcers. These patients complain of tearing, irritation, photophobia and blurring of vision. Treatment is debridement of the ulcerated area and topical antiviral drugs like acyclovir. In trophic ulceration basement membrane of corneal epithelium is affected which may take at least nine weeks to repair itself. Stromal HSK occurs due to immune complex hypersensitivity to viral capsid proteins and presents as interstitial keratitis or limbal vasculitis. Stromal HSK may present as viral necrotizing keratitis in AIDS. Disciform keratitis occurs due to delayed hypersensitivity and involves almost the entire corneal stroma (Fig. 9.22).

Treatment of stromal HSK is with mild steroids under cover of antiviral agents.

Adenoviruses may cause epithelial keratitis and stromal lesions (Fig. 9.23) may occur due to an antigenic reaction towards the virus capsid proteins. Treatment is the use of topical low potency steroids.

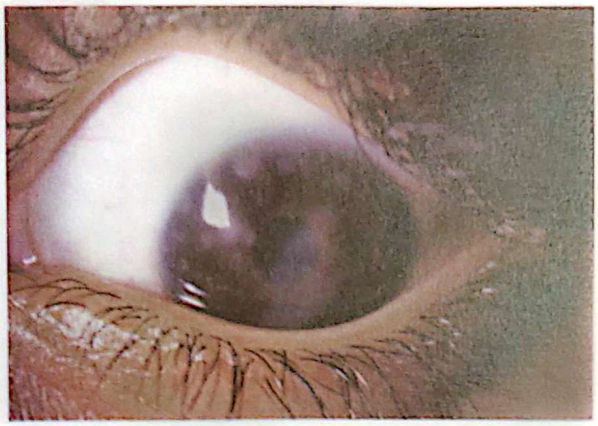


Fig. 9.23 Stromal reaction in adenovirus keratitis (Nummular keratitis)

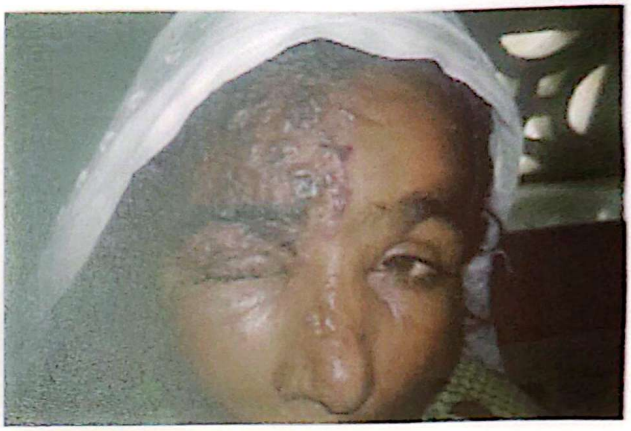


Fig. 9.24(B) Herpes zoster ophthalmicus involving theright ophthalmic nerve

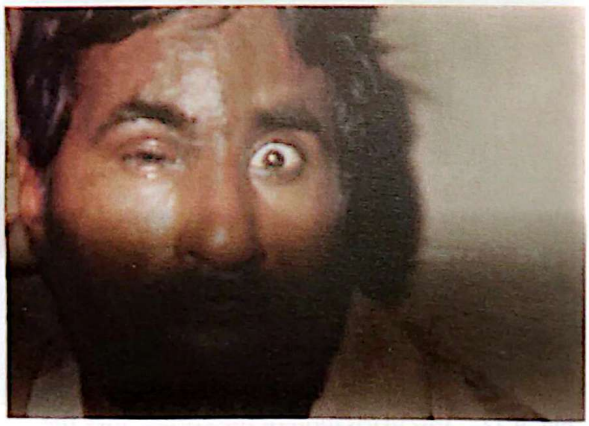


Fig. 9.24(A) Herpes zoster ophthalmicus involving the right ophthalmic nerve

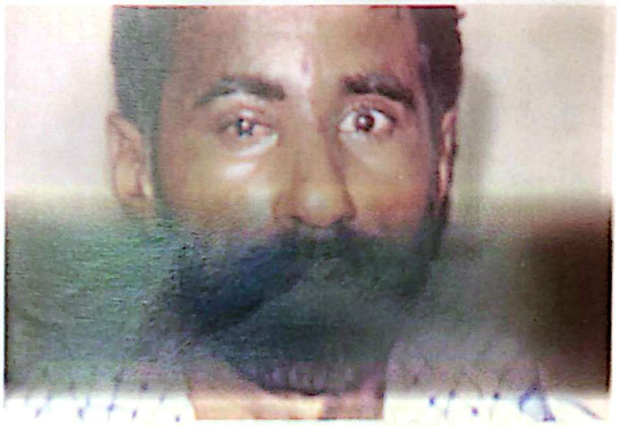


Fig. 9.25 Herpes zoster ophthalmicus involving the right ophthalmic nerve after treatment

Herpes zoster ophthalmicus (HZO)

It is an acute vesicular eruption of the skin involving the area supplied by the ophthalmic division of the trigeminal nerve (Fig. 9.24 A & B) in middle aged and the elderly. Rarely maxillary division may be involved (Fig. 9.26). HZO is caused by human herpes virus 3. Ocular involvement occurs in 50% of cases developing HZO[20] and is in the form of conjunctivitis, keratitis, episcleritis, scleritis, anterior uveitis or optic neuritis in various combinations. HZO, when seen in young people, may be a reflection of underlying immunodeficiency including AIDS. Treatment is with oral antiviral drugs in the acute stage, e.g. famciclovir 750 mg daily in divided doses for one week, in addition to symptomatic measures and antibiotics in the presence of secondary infection. These patients may suffer from post herpetic neuralgia after resolution of the herpes zoster eruption (fig. 9.25) which may be incapacitating and has been treated with cold packs applied over the affected area, analgesics, antidepressants, Transcutaneous Electrical Nerve Stimulation (TENS), anticonvulsants and rhizotomy (severing or damaging the affected nerve to relieve pain).

Fungal keratitis

Fungal keratitis is not uncommon, particularly in Larkana, Pakistan, where the author works, and mostly occurs as a result of direct inoculation of fungus typically in a farmer working in the fields. A few cases are due to prolonged use of steroids or antibiotics or in immunocompromised patients. Fusarium, Aspergillus, Cephalosporium, Curvularia, Alternaria and Candida albicans species are the causes of human fungal keratitis. Fortunately most of the time clinical presentation

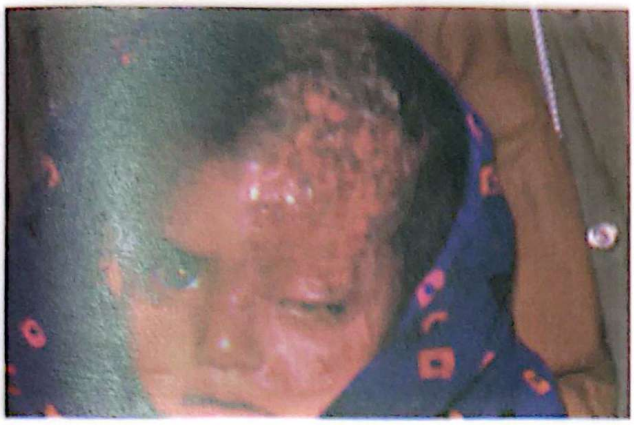


Fig. 9.26 Herpes zoster ophthalmicus involving the left ophthalmic and maxillary nerves

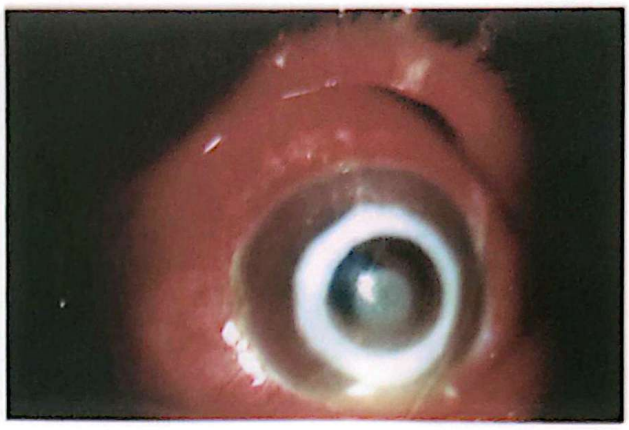


Fig. 9.28(A) Bull's eye fungal corneal ulcer with hypopyon

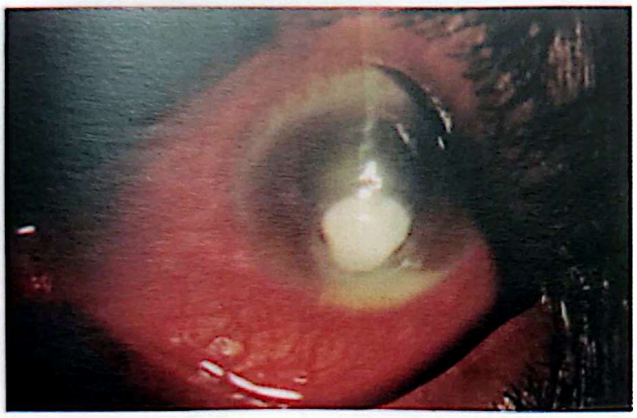


Fig. 9.27 Fungal keratitis with raised fluffy area

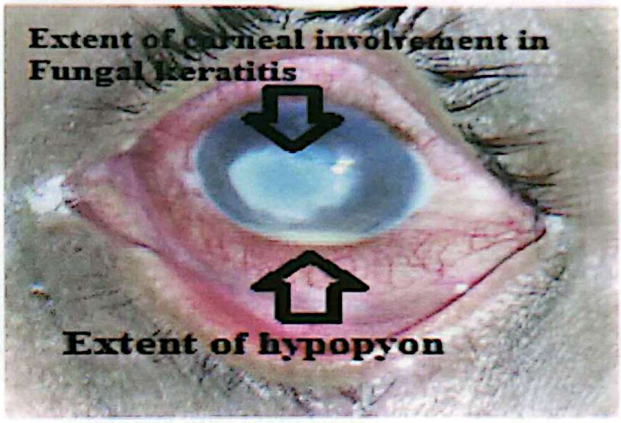


Fig. 9.28(B) Imtiaz's law of inversely proportional hypopyon

is typical with a history of trauma with organic material of 2 to 3 days duration with severe pain, photophobia and red eye.

On examination, a white, fluffy raised area (Fig. 9.27) surrounded by a grey halo (Fig. 9.28 A) is seen, with or without a tip of paddy, sometimes associated with scattered small lesions inside the grey area called satellite lesions (Fig. 9.29). In fungal keratitis, hypopyon is less than half of the size of the ulcer (Fig. 9.30) in contrast to a bacterial ulcer where hypopyon is usually more in size than the ulcer area known as Imtiaz's law of inversely proportional hypopyon[21], a very helpful diagnostic sign (Fig. 9.28 B). Sometimes fungal growth proceeds as a brown sheet over the top of the cornea without hypopyon, called brown growth (Fig. 9.31) by the author. Rarely fungal growth takes an annular shape (Fig. 9.32) with devastating sloughing of the cornea. Direct conjunctival involvement may be an associated feature (Fig. 9.33). Corneal scrapings of the affected area seen as unstained preparation in an allocated room beside the eye ward under a microscope shows the fungal hyphae.

Treatment should commence immediately without further loss of time with antifungal preparations, topical and, if necessary, systemic in addition to symptomatic measures. Brown growth must be removed with a blunt instrument like a spud. Natamycin 5% eye drops are easily available in Pakistan and when instilled one hourly are very effective. Delay in treatment may end up in complications including complete corneal necrosis (Fig. 9.34) and perforation of the cornea. After healing, a corneal opacity may remain which requires keratoplasty. Wearing sunglasses is a good preventive measure which should be adopted by the exposed population.

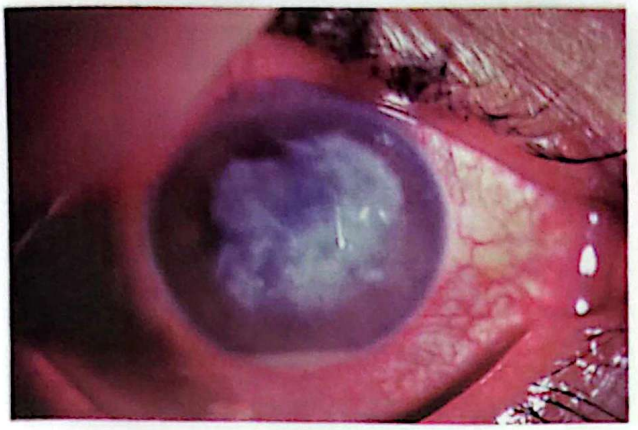


Fig. 3.33 Fungal keratitis with fungal conjunctivitis

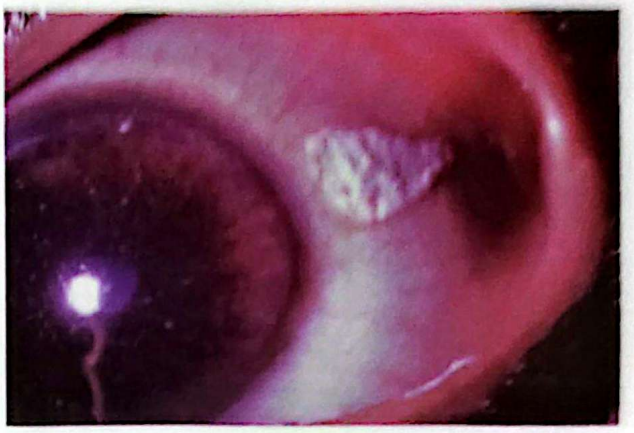


Fig. 9.35 Bito't spot with epithelial keratitis

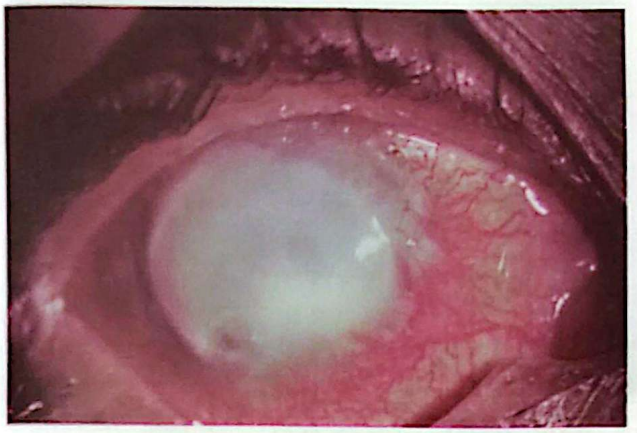


Fig. 9.34 Late stage of fungal keratitis with complete corneal destruction

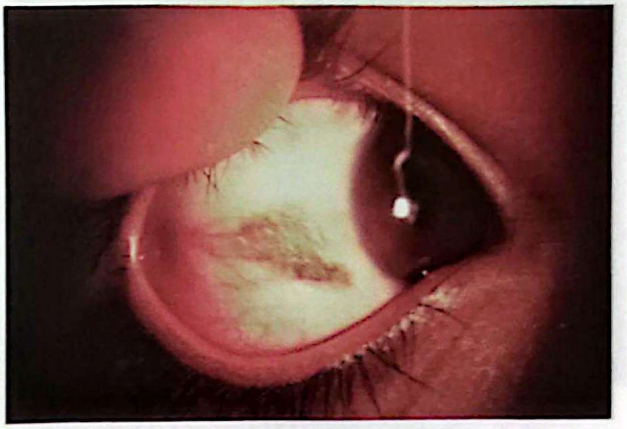


Fig. 9.36 Bitot's spot with pigmentation

rheumatoid arthritis (Fig. 9.39) and porphyria), phlyctenular keratoconjunctivitis, Terrien's marginal degeneration (Fig. 9.40) and Mooren's ulcer (fig. 9.41) (idiopathic) are classified under this heading.

Nonulcerative keratitis

Keratitis involving stroma without corneal ulceration is seen in sarcoidosis, tuberculosis, syphilis and disciform stage of herpes simplex keratitis.

Exposure keratitis

Inability to close the lids due to facial nerve palsy or proptosis leads to corneal drying and ulceration called exposure keratitis. Initial treatment includes frequent instillation of lubricating agents to keep the cornea wet or a bandage contact lens to protect the cornea during sleep followed by treatment of the cause, e.g., removal of a space occupying lesion in the orbit or orbital decompression (Fig.9.43).

Band keratopathy

Exposed part of the cornea may get deposition of calcium salts in the deeper layers of epithelium and Bowman's zone called band keratopathy in a wide variety of conditions. Removal is done with mechanical scraping or with the help of an excimer laser followed by application of a chelating agent like sodium versenate (Fig.9.42).

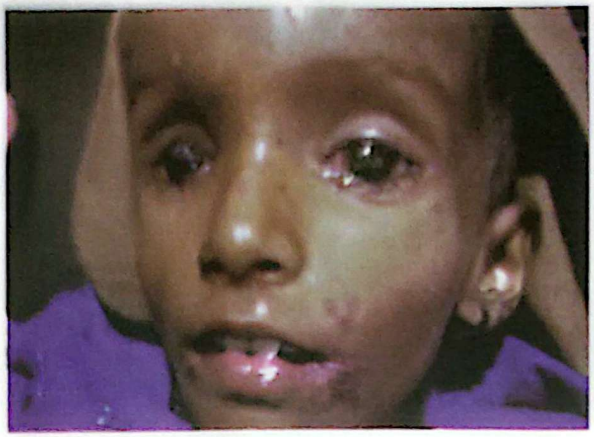


Fig.9.37. Keratomalacia

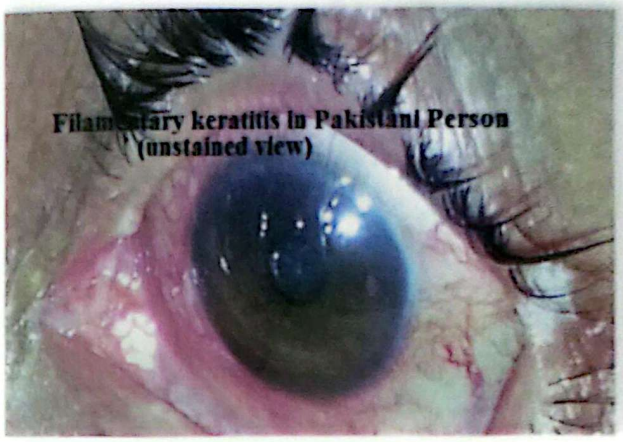


Fig. 9.38(B) Filamentary keratitis

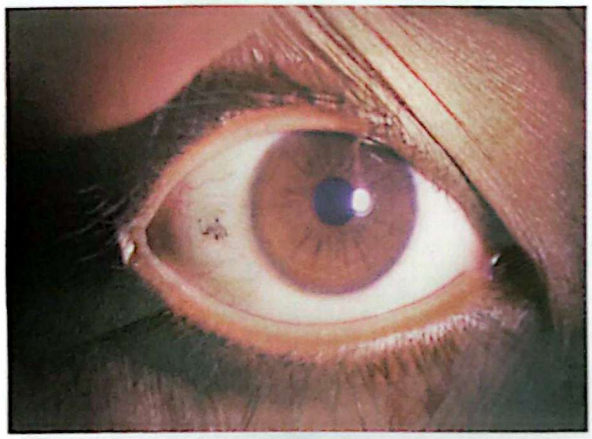


Fig. 9.38(A) Imtiaz's sign of Vitamin A Deficiency

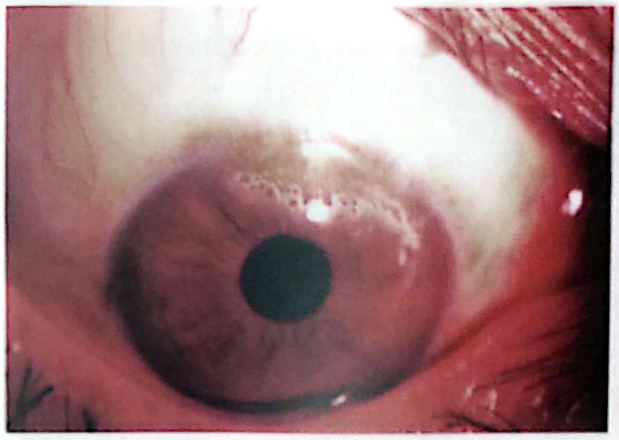


Fig. 9.39 Peripheral corneal ulceration in rheumatoid arthritis

Keratoconus

Ectatic dystrophy of the cornea characterised by central stromal thinning leading to central forward bulging of the cornea is called as keratoconus (Fig. 9.44). This condition usually manifests around puberty and progresses for around a decade. Keratoconus can be detected in its earliest stage by computer assisted videokeratoscopy, however other simpler tests can detect this condition, which includes bulging of the lower lid margin due to the corneal cone when a patient looks down (Munson's sign), oil droplet reflex on ophthalmoscopy, scissor reflex on retinoscopy and distortion of Placido disc reflex over the corneal surface. According to the presenting shape of keratoconus and involvement of the area of the corneal disc it is classified into nipple cone, oval cone and globus cone. History of allergy is present in more than two thirds of the patients suffering from keratoconus in addition to its association with a wide variety of disorders like vernal keratoconjunctivitis, retinitis pigmentosa, atopic dermatitis, Down syndrome etc. Treatment is the prescription of glasses in early stages to correct the refractive error followed by use of hard contact lenses when the condition does not improve with glasses. The second line of treatment of keratoconus is corneal collagen cross-linking (CXL) and insertion of intracorneal ring segments (Intacs). In the advanced stage, penetrating keratoplasty is recommended with good results.

Corneal Dystrophies

Mostly transmitted as autosomal dominant hereditary disorders, corneal dystrophies (Fig. 9.45 B, 9.46 A & B) are bilateral, slowly progressive and usually become evident during adolescence. Three types are seen clinically, i.e., epithelial, stromal or endothelial. When a corneal dystrophy seriously hampers vision, the treatment is keratoplasty.

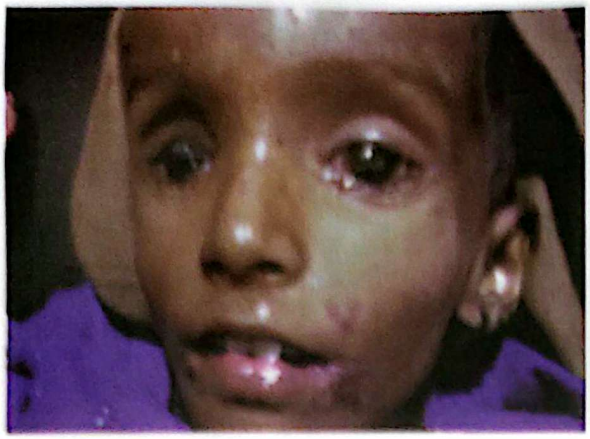


Fig.9.37. Keratomalacia

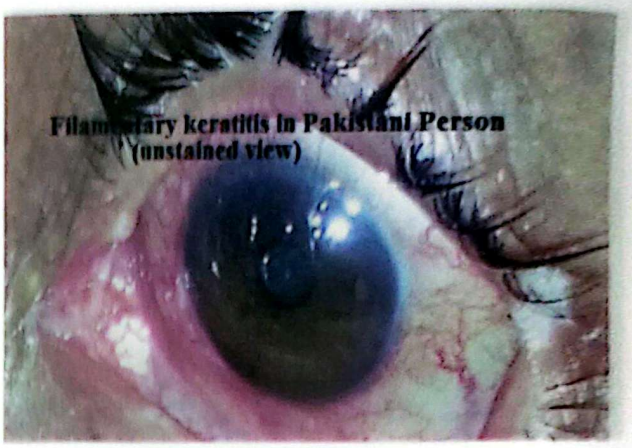
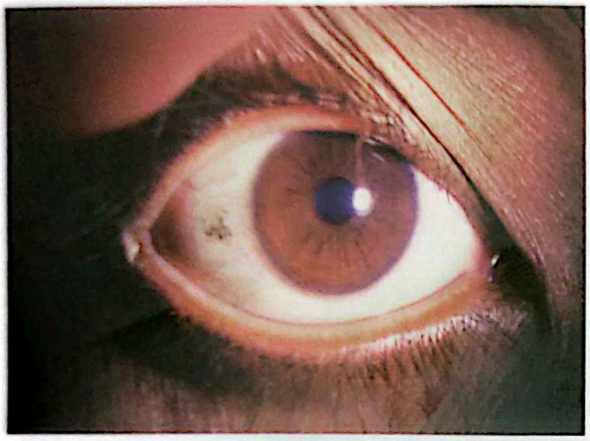


Fig. 9.38(B) Filamentary keratitis



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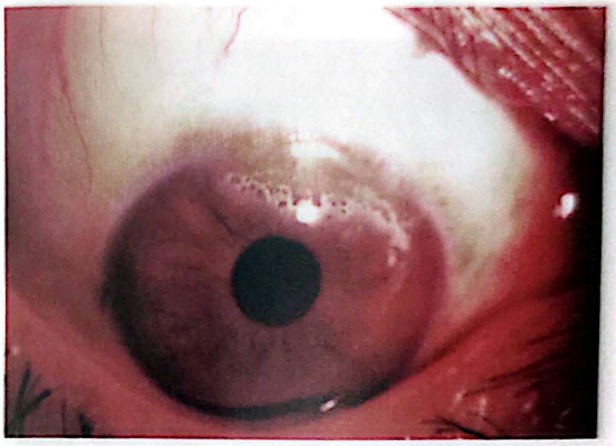

Fig. 9.39 Peripheral corneal ulceration in rheumatoid arthritis

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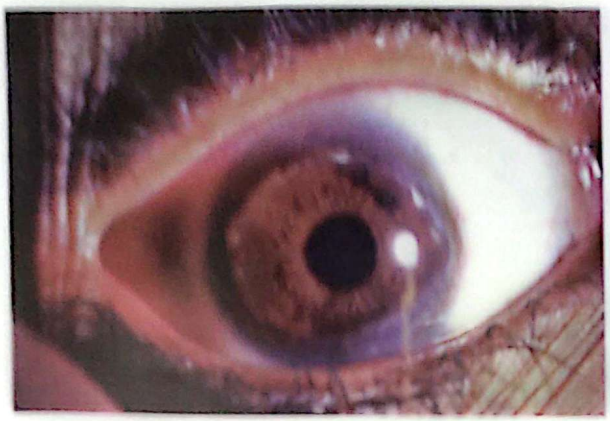


Fig. 9.40 Terrien's marginal degeneration

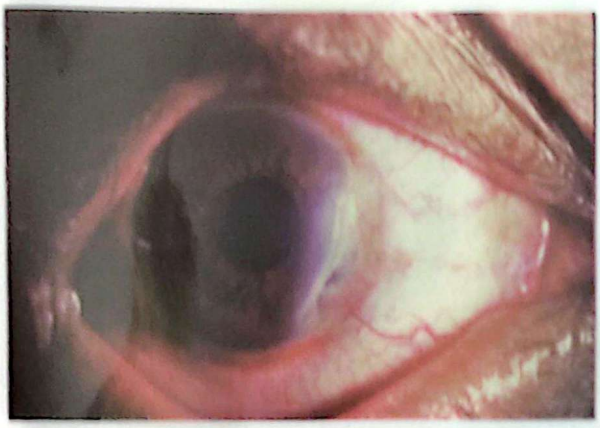


Fig. 9.41 Mooren's ulcer of the cornea

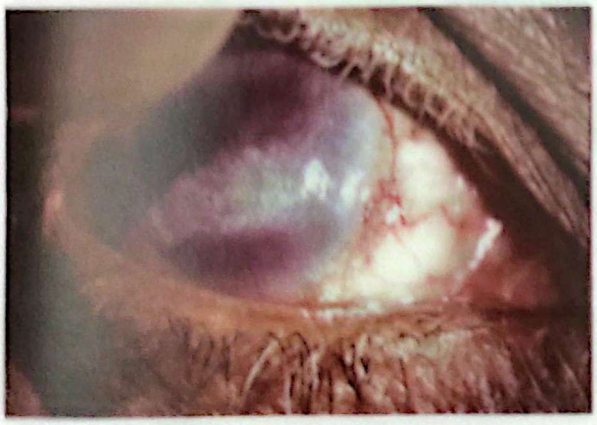


Fig. 9.42 Band keratopathy

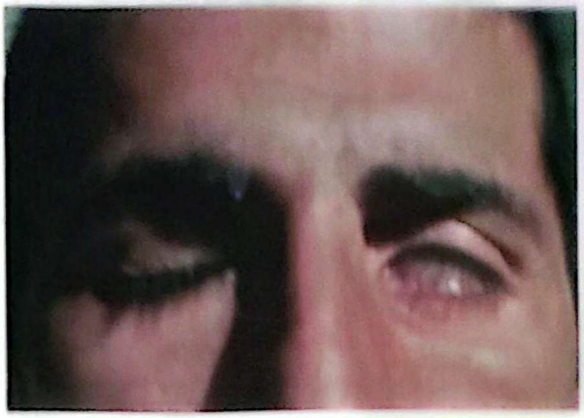


Fig. 9.43 Exposure keratitis

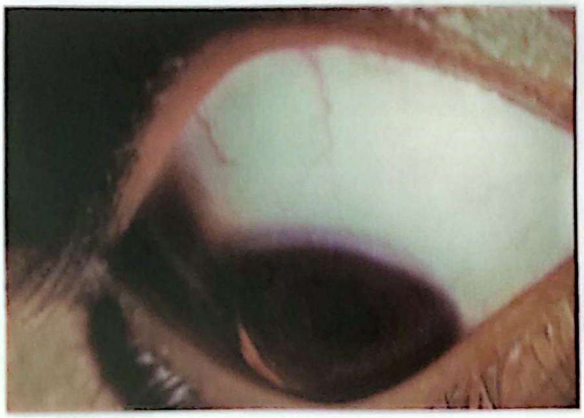


Fig. 9.44 Keratoconus

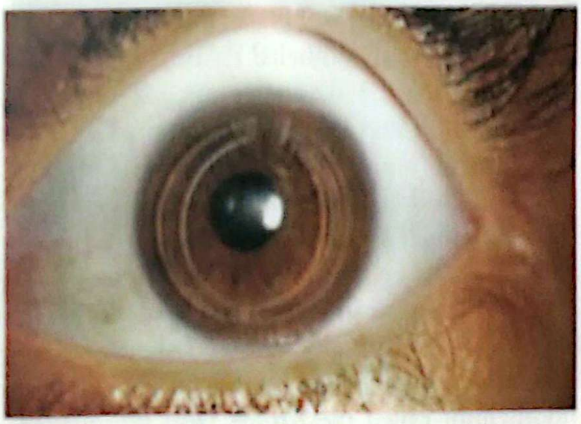


Fig. 9.45(A) Intracorneal ring segments

Keratoplasty (Corneal transplantation)

Removal of an opaque and/or diseased cornea and transplanting a clear human donor cornea is termed as keratoplasty (Fig. 9.47) or corneal grafting, which may be of partial thickness called lamellar keratoplasty or total thickness called penetrating keratoplasty. Success of penetrating keratoplasty depends upon multiple factors including healthy donor cornea with adequate endothelial reserve, condition of the recipient cornea surrounding graft, surgical technique, preoperative, intraoperative and postoperative

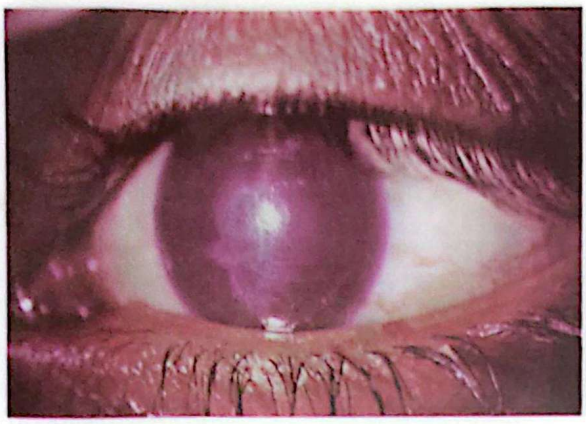


Fig. 9.45(B) Cogan microcystic corneal dystrophy

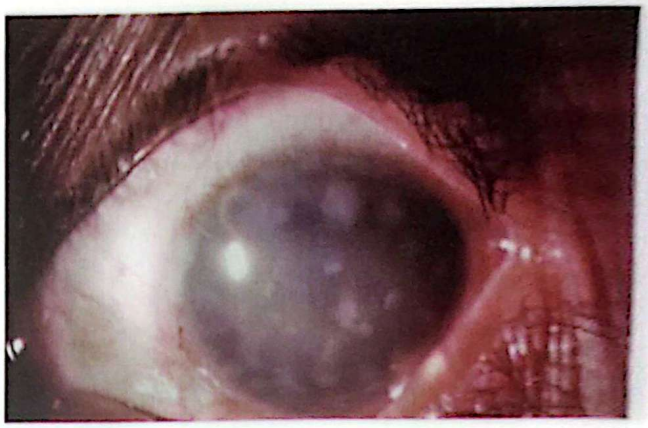


Fig. 9.46(B) Advanced stage of Reis-Bücklers corneal dystrophy

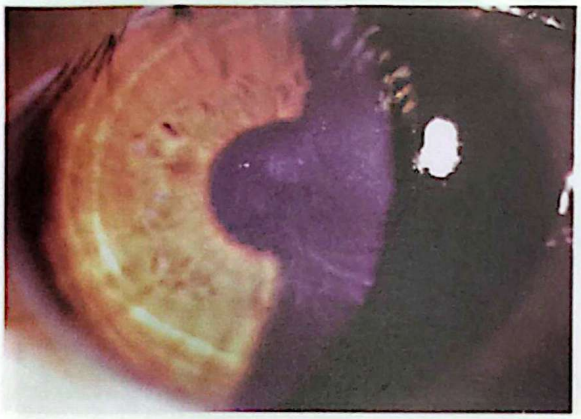


Fig. 9.46(A) Lattice dystrophy Type 1

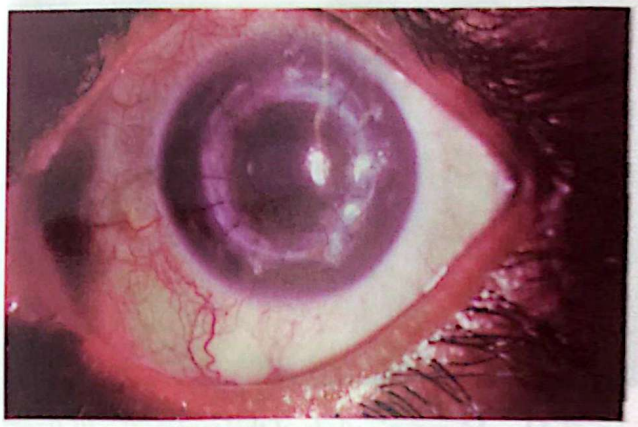


Fig. 9.47 Penetrating keratoplasty with wound vascularization

care of the graft. Primary graft failure occurs due to donor corneal endothelial dysfunction and is seen in the first few days after surgery, although it can occur at any time after keratoplasty. Graft rejection is an immunological reaction and may start any time after 10 days but is usually seen after a few months. Graft rejection may be epithelial (a linear defect in corneal epithelium progresses across the graft), stromal (peripheral full thickness haze progresses centrally) and endothelial (a line of keratic precipitates is found on the corneal endothelium called the Khuda Dost line named after an Iranian ophthalmologist who first described it). Treatment of graft rejection is with high potency steroids and cytotoxic drugs where necessary. Herpes simplex keratitis[23], corneal dystrophies[24], rabies and Creutzfeldt-Jakob disease[25] have been transmitted to the recipient from the donor, in some cases resulted in death of the recipient.

Chapter 10

Diseases of the Sclera

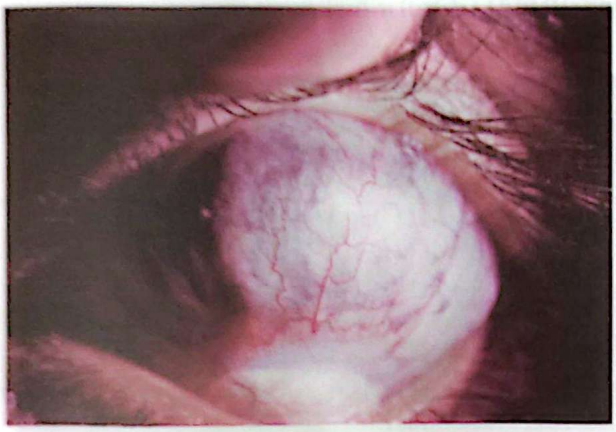


Fig. 10.1(A) Anterior staphyloma

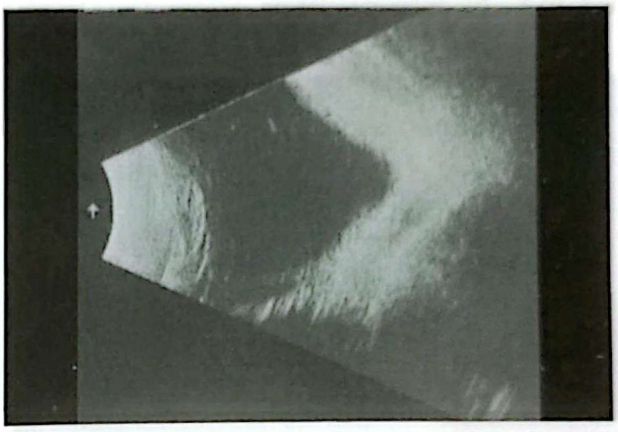


Fig. 10.1(B) Ultrasound B-scan showing posterior staphyloma

The sclera comprises five sixth of the globe and forms the white of the eye anteriorly although covered by a vascular connective tissue sheet called the episclera and conjunctiva. It receives the insertion of six extraocular muscles and is perforated by the optic nerve posteriorly and by the vortex veins, long and short ciliary arteries and long and short ciliary nerves.

COMMON CLINICAL FEATURES OF SCLERAL DISEASE

Pain and redness are the main features, although, in the advanced stage of scleral disease the involved sclera may bulge anteriorly or posteriorly giving rise to anterior (Fig. 10.1 A) or posterior staphyloma (Fig. 10.1 B).

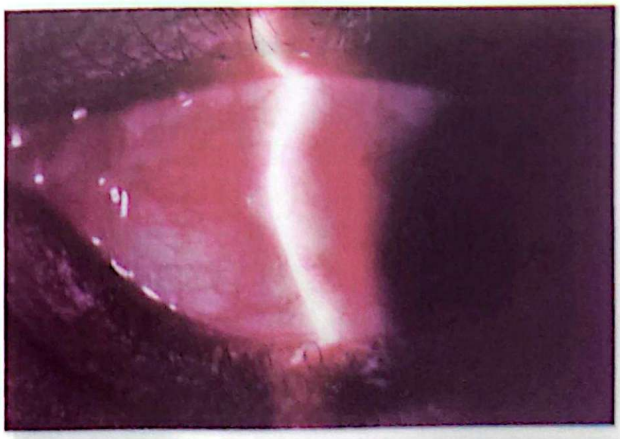


Fig. 10.2 Simple episcleritis

Episcleritis

Acute inflammation of episclera presents as a congested sectorial area over the white of the eye, called simple episcleritis (Fig. 10.2) or as a congested nodule over the white of the eye called nodular episcleritis (Fig. 10.3). It is associated with pain and tenderness. Underlying atopy or

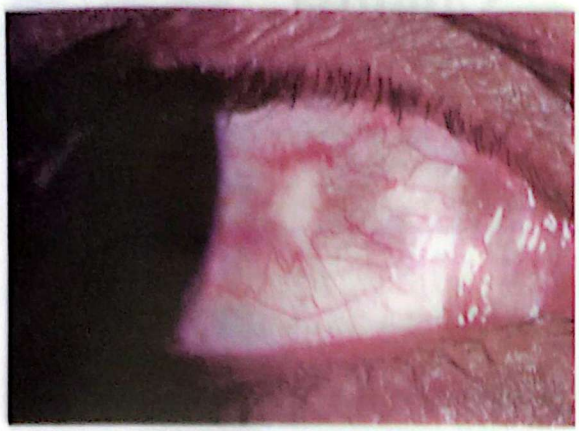


Fig. 10.3 Nodular episcleritis

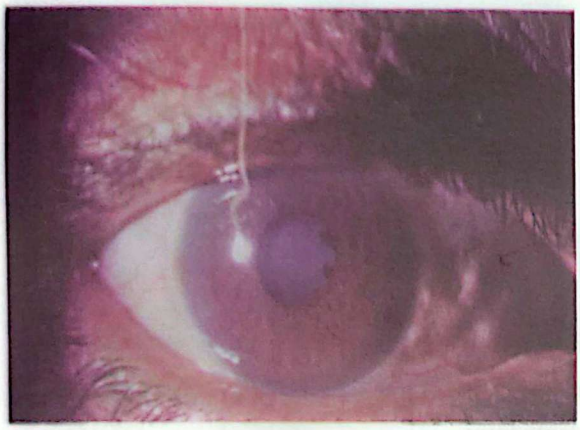


Fig. 10.4 Sclerouveitis

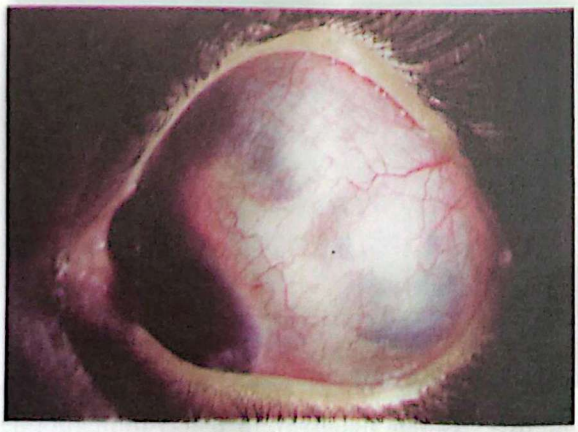


Fig. 10.5 Scleral necrosis after annular scleritis

immune reaction may is responsible for this self-limiting condition. Treatment is symptomatic with anti-inflammatory agents.

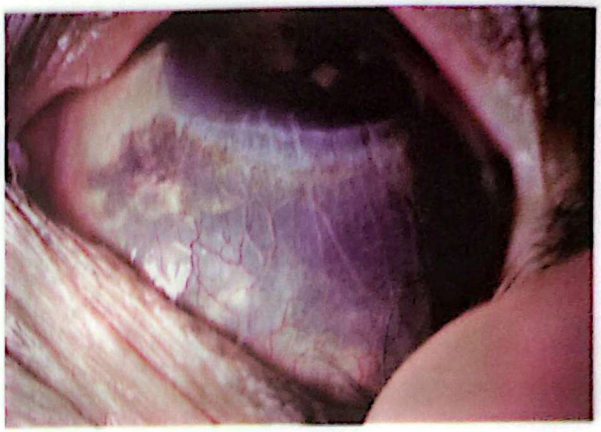


Fig. 10.6(A) Ocular melanosis

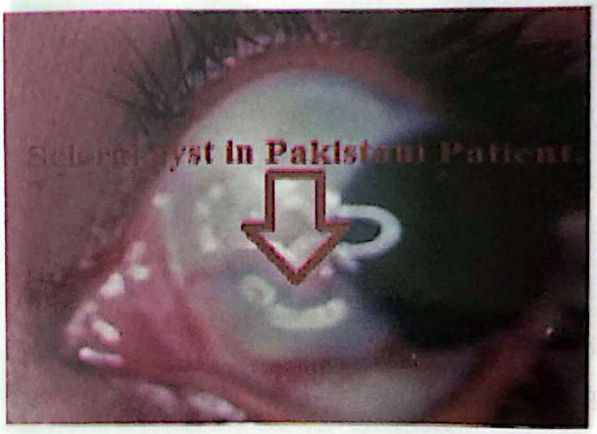


Fig. 10.6(B) Scleral cyst

Scleritis

Inflammation of the sclera, although not common, is a threatening condition. When uveal inflammation is associated with scleritis, it is termed sclerouveitis (Fig. 10.4). In more than half of the cases it is associated with collagen disorders, therefore, when present, it needs investigations to exclude polyarteritis nodosa, Wegener's granulomatosis, systemic lupus erythematosus, rheumatoid arthritis and others. Scleritis may occur in the anterior sclera or posterior sclera and may be diffused or nodular, necrotising or non-necrotising. When it extends around the cornea it is called annular scleritis (Fig. 10.5).

Clinical presentation is varied according to the type and site of inflammation and is painful, particularly with eye movements, but with retained vision in contrast to retrobulbar neuritis which is associated with visual loss in addition to pain on eye movements. Treatment is with steroids and treatment of associated disorders.

Scleral Discolourations

Blue sclera indicates scleral thinning. Ocular melanosis (Fig. 10.6 A) may lead to apparent discolouration of the sclera. Presence of jaundice apparently discolours sclera, although sclera is not stained and it is conjunctiva that is stained with bilirubin in jaundice.

Scleral cyst (Fig. 10.6 B)

Scleral cysts are rare, may be congenital or acquired. When acquired, the cause is trauma, postsurgical[33] or idiopathic. Treatment is deflation or surgical removal.

Chapter 11

Diseases of the Lens

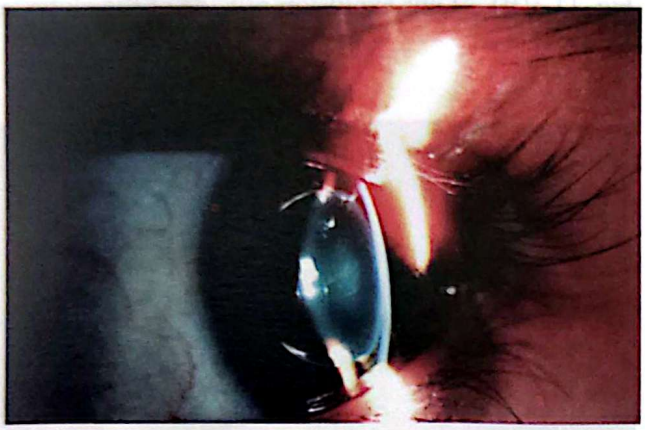


Fig. 11.1(A) Ectopia lentis

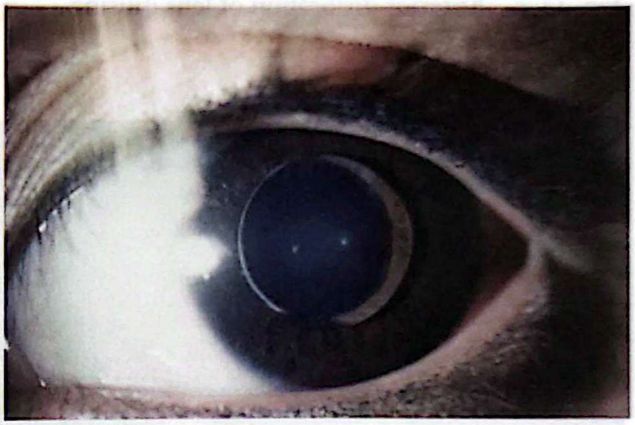


Fig. 11.1(B) Ectopia lentis

Embryologically a piece of skin, the elastic lens is a transparent biconvex structure with a relatively flatter anterior surface. In young adult's it has a diameter of 9 mm and a thickness of 4 mm. It is held in its place in the posterior chamber by suspensory ligament attached at its equator and hyaloidocapsular ligament which connects its posterior surface with the anterior hyaloid face of vitreous. Lens growth occurs at the equator where active mitosis is seen and the lens keeps on growing up to the age of 83 years and is covered by a capsule which is the basement membrane of the lens epithelium and is the thickest basement membrane of the body. When seen in the optical section on a slit lamp it is divisible into optical zones which indicate its growth pattern. The central zone is the embryonic nucleus which is made of posterior capsular epithelium which transforms to create it as an earliest development in the lens vesicle and that is the reason there is no epithelium over the posterior part of the capsule in later life. Covering the embryonic nucleus is the zone of foetal nucleus which indicates lens development up to the 9th month of intrauterine life. Overlapping the foetal nucleus is the infantile nucleus which represents lens development from the last month of intrauterine life to the age of puberty. Encasing the infantile nucleus is the adult nucleus, which represents lens development in later life, in addition to the area covering it, called the cortex. When a transparent lens becomes opaque (cataract) the structure becomes simple, i.e., a hard nucleus is surrounded by the soft cortex which is covered by the capsule. Being an avascular structure, it depends upon aqueous for its nourishment.

COMMON CLINICAL FEATURES OF LENS DISEASE

Decreased vision, misty or foggy vision, uniocular diplopia or polyopia, and white opacity in the pupil are among the common clinical features of lens disease.

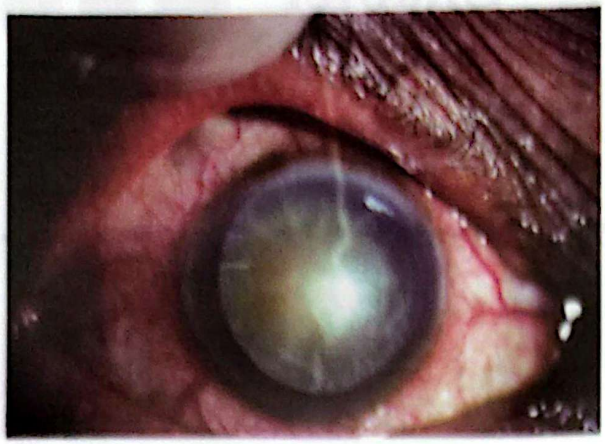


Fig. 11.2 Anterior dislocation of lens causing glaucoma

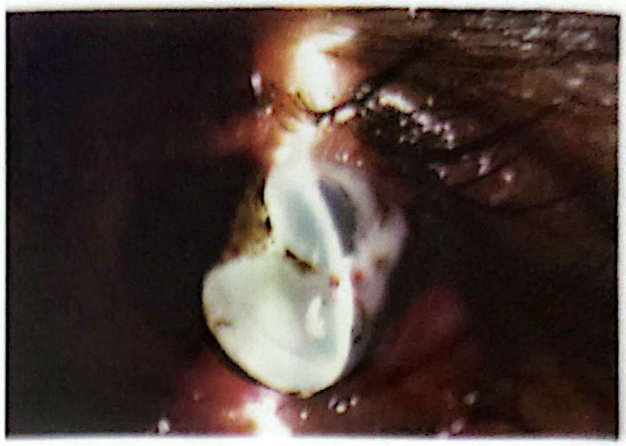


Fig. 11.3(B) Lens expulsion outside of the globe

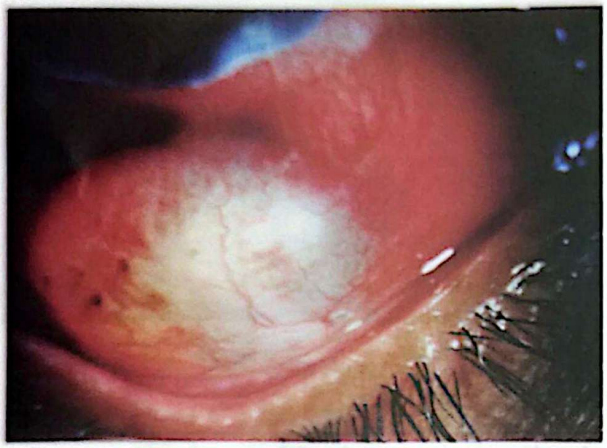


Fig. 11.3(A) Lenticele

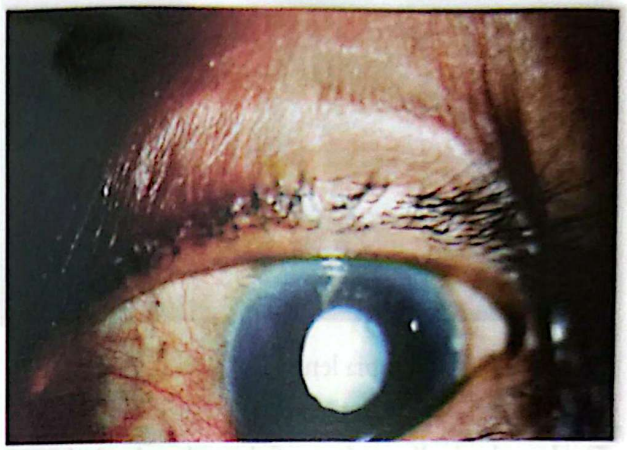


Fig. 11.4 Mature cataract

Ectopia Lentis

It is a partial or complete displacement/dislocation of the lens which occurs due to either maldevelopment (Fig. 11.1 A & B) or rupture of fibres of the suspensory ligament of the lens (Fig. 11.2). Partial dislocation called subluxation or complete dislocation called luxation is seen in a variety of hereditary diseases like Marfan syndrome, Weil-Marchesani syndrome, homocystinuria and Ehlers-Danlos syndrome. Lenticele is an extreme degree of traumatic lens dislocation where the lens is thrown out of the globe through an associated scleral tear and lies under Tenon's capsule or conjunctiva (Fig. 11.3 A). In cases of total corneal sloughing, the lens expulsion outside the globe can occur (Fig. 11.3 B). Treatment is to hold the lens in position when clear and not producing complications, like glaucoma, with miotic drops. When complications are anticipated, extracapsular extraction with IOL implantation may be performed by the help of fixing the lens capsule with a capsular tension ring.

Cataract

When the transparent lens develops opacification it is called a cataract (Fig. 11.4). Opacity in any part of the lens is called a cataract anatomically. Opacity in the central area of the lens which disturbs vision is called a cataract functionally. Biochemical definition of a cataract is coagulation of lens proteins in a part of the lens or in the whole lens. The importance of biochemical definition is for doctors treating the patients with cataract, as the lens proteins (crystallins and albuminoids) are unique in the body and they are sequestered proteins (the proteins which are not exposed to the immune system of the body during the period of embryogenesis) therefore, the immune system of the body recognises them as foreign proteins

Fig. 11.5 Congenital blue dot cataract

Fig. 11.7 Nuclear sclerosis in a senile cataract

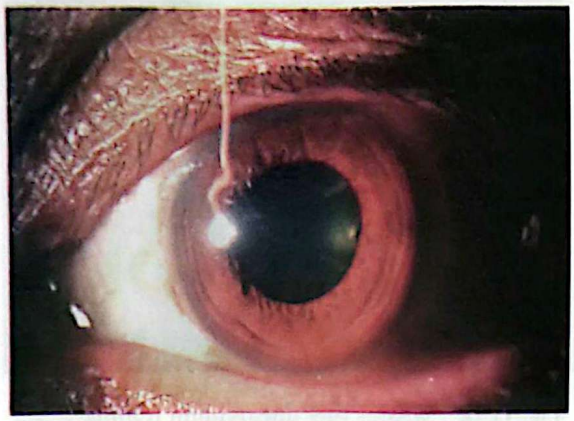


Fig. 11.6 Cortical Cataract

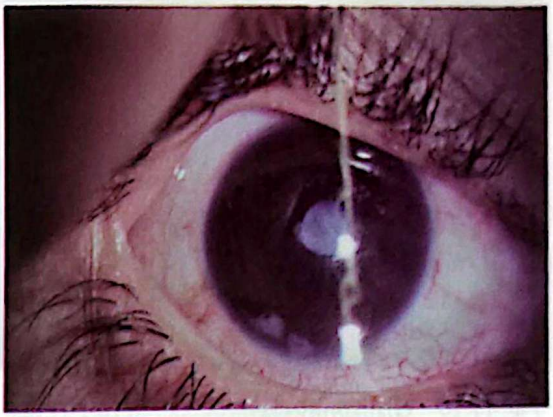


Fig. 11.8 Cataract formation after penetrating keratoplasty

hence there is intense uveal reaction when lens proteins leak out of the avascular lens, e.g., in phacolytic glaucoma and phacoantigenic uveitis.

Classification of a cataract

Classifying a cataract may be as simple as congenital (Fig. 11.5) and acquired or mature, immature and hypermature, or progressive and stationary or nuclear, cortical (Fig.11.6) and capsular or idiopathic and with cause. This oversimplification in classification may not include a wide variety of cataracts and we may not justify with classification of a disease which is the leading cause of reversible blindness throughout the world. Classifying cataracts according to cause may serve the purpose; therefore cataracts are classified under three main headings, i.e.,

A. Cataract associated with ocular and systemic disease.

B. Cataract associated with ocular but no systemic disease.

C. Cataract associated with no ocular and no systemic disease.

A. Cataract associated with ocular and systemic diseases

Embryopathies, e.g., drug induced disorders, radiation induced disorders and maternal infections like rubella (cataract, deafness, cardiac anomalies).

Metabolic, e.g., diabetes mellitus, galactosemia.

Skin disease, e.g., incontinentia pigmenti (cataract and uveitis), atopic dermatitis.

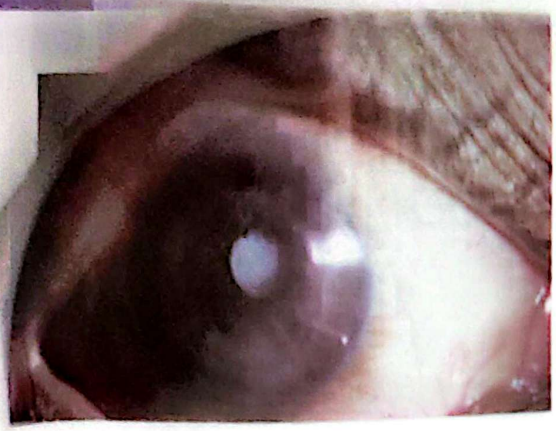


Fig. 11.9 Traumatic cataract

Fig. 11.11 Brown cataract

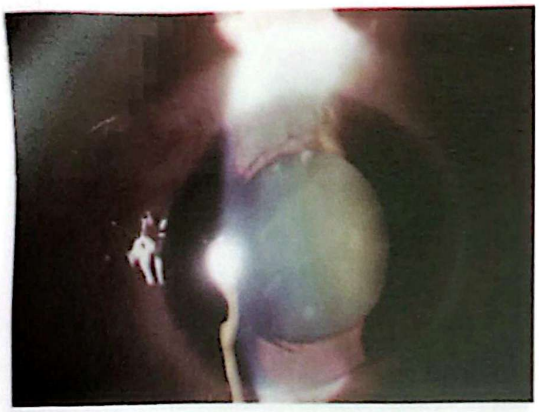


Fig. 11.10 Glaukomflecken

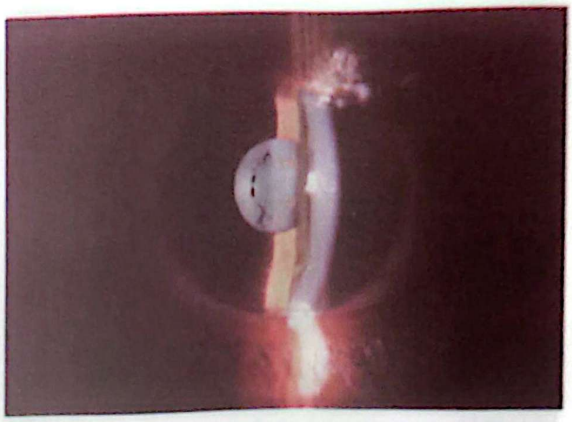


Fig. 11.12 Vossius ring due to blunt trauma

Muscle disease, e.g., myotonic dystrophy.

Neurologic disorder, e.g., Wilson's disease.

Bone disease, e.g., osteitis fibrosa.

Chromosomal disorder, e.g., Down syndrome.

B. Cataract associated with ocular but no systemic diseases

Congenital and acquired ocular conditions associated with cataract.

Congenital e.g. persistent hyperplastic primary vitreous, microphthalmia.

Acquired e.g. trauma (blunt, penetrating, radiation), uveitis, toxic (steroids, silicon oil), retinal detachment, retinitis pigmentosa, tumours.

C. Cataract associated with no ocular and no systemic disease

This group includes senile cataracts (Fig. 11.7) and all idiopathic congenital and acquired cataracts.

Mechanisms of cataract formation

A cataract can be formed by one of the following mechanisms:

1. Clear lens fibers may become opaque (Fig. 11.8), e.g., senile cataract, diabetic cataract, and traumatic cataract (Fig. 11.9).
2. Clear lens cells may become opaque, e.g., glaukomflecken (Fig. 11.10) (opaque anterior epithelial cells as a result of an acute attack of glaucoma).

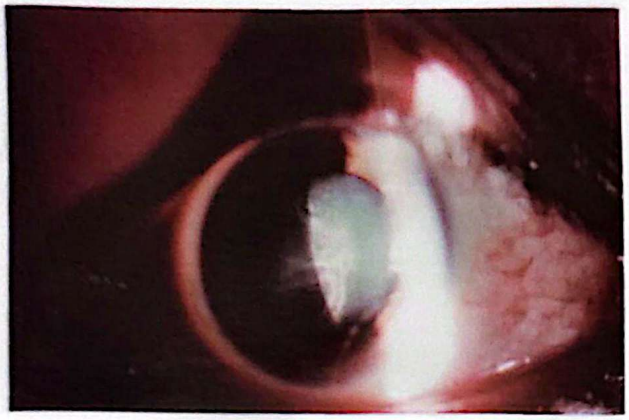


Fig. 11.13(A) Sutural cataract

Fig. 11.13(B) Dislocated lens

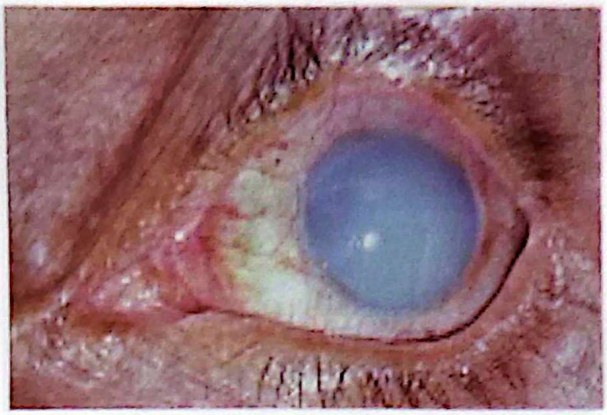


Fig 11.13(C) Phacolytic glaucoma

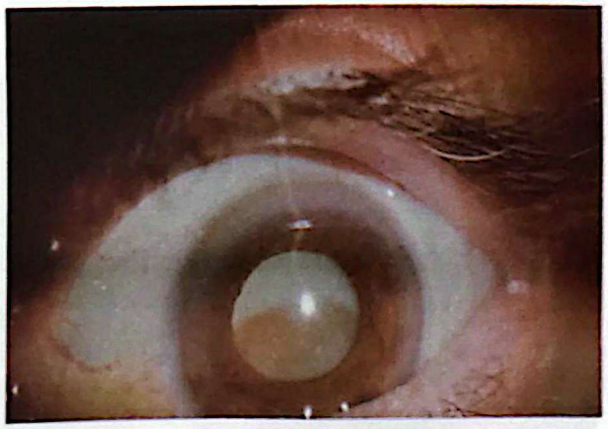


Fig 11.13(D) Morgagnian cataract

3. Deposition of endogenous material (Fig. 11.11), e.g., deposition of pigment in cataracta nigra (black cataract).
4. Deposition of exogenous material, e.g., chlorpromazine deposition leading to star shaped opacity, or deposition of iris pigment on the anterior surface of the lens after blunt ocular trauma as in Vossius ring (Fig. 11.12).
5. Formation of granular material instead of clear lens fibres, e.g., radiation cataract (lens epithelium has lost capability to produce lens fibres).
6. Formation of opaque lens fibres instead of clear lens fibres e.g. congenital and some developmental cataracts (Fig. 11.13 A).

Complications of hypermaturity

When a cataract is left unattended, it can progress to the stage of hypermaturity. When the capsule of the lens becomes impermeable, the lens becomes thin and may dislocate spontaneously (Fig. 11.13 B). When the capsule of the lens becomes permeable the lens may swell up and may cause phacomorphic glaucoma or may rupture causing phacolytic glaucoma (Fig. 11.13 C) or the nucleus may sink in the liquid cortex called Morgagnian cataract (Fig. 11.13 D).

Treatment of Cataract

Although surgical removal of a cataractous lens is the only option to treat cataract, in early stages, refractive error produced by a cataract is corrected by the prescription of glasses. Small central cataracts can jeopardise vision disproportionately, particularly in the day time and can be managed with dark glasses or mydriatic drops after

Fig. 11.14 Intracapsular Cataract Extraction

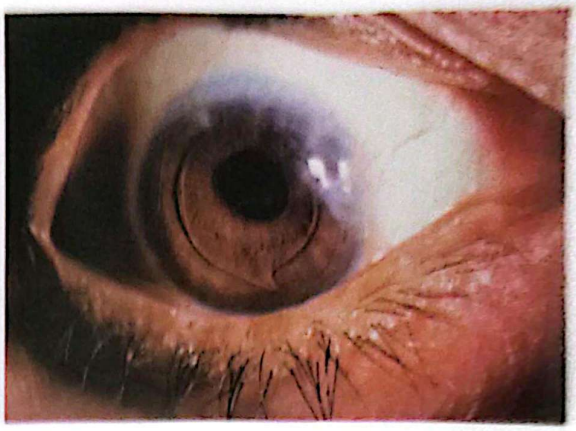


Fig. 11.16 Anterior chamber IOL implanted after encountering complication of posterior capsular rupture

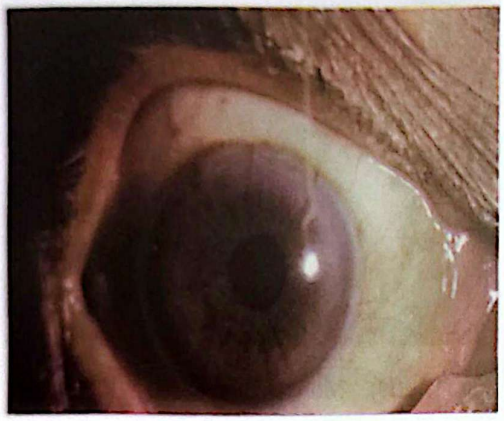


Fig. 11.15 Manual Extracapsular Cataract Extraction (ECCE) with posterior chamber IOL implanted and three 10 0 Nylon sutures placed are seen

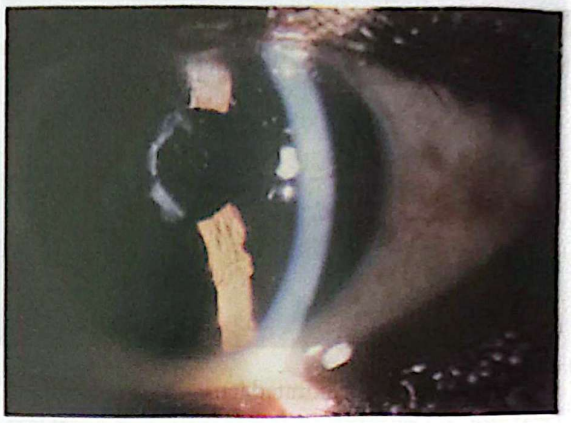


Fig. 11.17 PCO treated with YAG laser

performing gonioscopy to see if the angle is wide open. When the angle is narrow, surgery is the only option even in early stages. In the past, removal of the whole lens was performed surgically and the procedure was called intracapsular

cataract extraction (Fig. 11.14), which was associated with serious complications, therefore, this procedure has become obsolete. The surgical procedures currently being performed throughout the world are based upon the basic principle of extracapsular cataract extraction, where the posterior part of the capsule of the cataractous lens is retained in the eye and all other components are removed, including the anterior part of the lens capsule, lens nucleus and lens cortex.

1. Manual Extracapsular Cataract Extraction (ECCE)

(Fig.11.15) It is a time honored safe procedure which is performed through the corneoscleral limbus. An incision is given at the limbus and the anterior part of the capsule is cut open and removed by way of capsulotomy (removal of the anterior capsule through multiple small cuts) or by capsulorhexis (removal of the anterior capsule through a controlled circular tear). The hard nucleus is removed through a limbal incision and the wound is closed by nylon sutures (10 0). The remaining soft cortex is removed with the help of a two-way cannula. An intraocular lens (IOL) is implanted in the left-over posterior capsular bag to correct hypermetropia produced as a result

of cataract extraction. Complications include rupture of posterior capsule during surgery (when an anterior chamber IOL implantation may be an option (Fig. 11.16), postoperative endophthalmitis, glaucoma, astigmatism and others.

2. Phacoemulsification

It is performed with the help of an ultrasound machine which provides an ultrasonically vibrating tip utilised for breaking the hard nucleus into pieces and a suction mechanism to aspirate the emulsified nucleus and the cortex. The tip of the phacoemulsifier is introduced into the nucleus of the cataract for its emulsification after making a hole at the limbus with a keratome knife (for entry of the phacoemulsifier tip) and performing capsulorhexis in addition to mobilising the nucleus by hydrodissection and hydrodelineation. A foldable IOL is introduced through the hole to correct resultant aphakia. The beauty of this method is cataract removal through a hole at the limbus which does not require suturing. However, known complications of this method are damage to corneal endothelium due to ultrasonic vibration, rupture of the posterior capsule with dropping of the nuclear fragment into the vitreous which is very difficult to remove in addition to all complications which may occur with manual ECCE.

3. Limbal Lensectomy and Vitrectomy

It is performed to remove hard congenital cataracts. Removal of congenital cataracts should not be delayed as a dense amblyopia may occur. This method is a variant of pars plana vitrectomy which is not advisable in infants due to the fear of disinsertion of the retina from the ora serrata as the pars plana is not completely developed in them. The method requires a vitrectomy machine for removal of the cataract and anterior vitreous. Any of the complications can occur, as with other methods.

Aphakia

Absence of the lens from the pupillary axis resulting in high hypermetropia and loss of accommodation is called aphakia. Convex lenses in one of the forms like glasses, contact lenses or an intraocular lens is the treatment for aphakia.

Intraocular Lens (IOL) Types

Soft (foldable) IOLs are made up of acrylic, hydrogel or silicon and utilised in phacoemulsification. Hard IOLs are made up of PMMA (Polymethylmethacrylate) material.

Refractive types of IOL include spherical, toric (with astigmatic correction), multifocal and progressive.

IOLs can be fitted during cataract surgery in the posterior chamber (in-the-bag or in the iridociliary sulcus) or in the anterior chamber, if the posterior capsular support is not available or stitched to the sclera, called scleral fixation. IOLs can be implanted in aphakic eyes later after primary cataract surgery, called secondary IOL implantation.

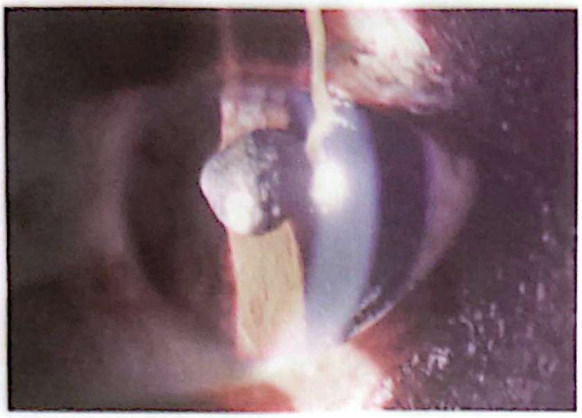
Accommodating IOLs provide some degree of accommodation and aspherical IOLs provide better contrast sensitivity.

IOLs can be placed in front of the transparent lens of the eye to correct high degree refractive errors, e.g., high myopia, called phakic IOLs.

Sometimes, an implanted IOL is of less or more power due to one or the other reason; therefore, another IOL can be placed over this IOL to increase or neutralise the power of this IOL. This placement of two IOLs in the same eye is called the"piggyback" approach.

Posterior Capsular Opacification (PCO)

Opacification of the left-over posterior capsule of the cataractous lens (Fig. 11.18 A) which holds the IOL occurs in a fairly large number of patients throughout the world. PCO causes decreased vision but responds well to Nd:YAG laser capsulotomy (Fig. 11.17) which may be performed at any time after six months of cataract surgery. Techniques utilised for Nd:YAG laser capsulotomy include First Crack Guided Conservative Posterior Capsulotomy



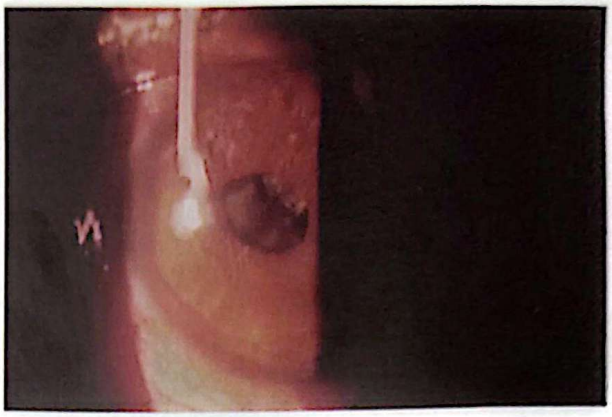


Fig. 11.18(A) Posterior Capsular Opacification (PCO)

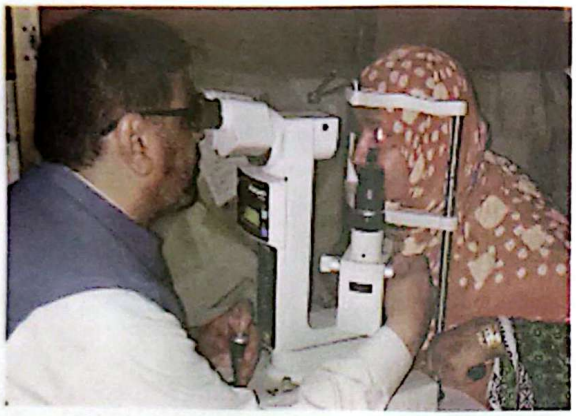


Fig. 11.18(B) Nd: YAG laser capsulotomy is being performed

Fig. 11.18(C&D) First Crack Guided Conservative Posterior Capsulotomy Using Nd: YAG laser capsulotomy is being performed

Using Neodymium: YAG Laser[26], Racquet Shaped Nd:Yag Posterior capsulotomy[28], Cross pattern method[29], Can-opener method[30], Inverted U method[31] and Circular with vitreous strand cutting method[32]. Complications of Nd: YAG laser capsulotomy include transient rise of IOP[40], transient uveitis, IOL pitting, macular oedema, and rarely retinal detachment.

Using Nd: YAG laser capsulotomy is being performed

Chapter 12

Glaucoma

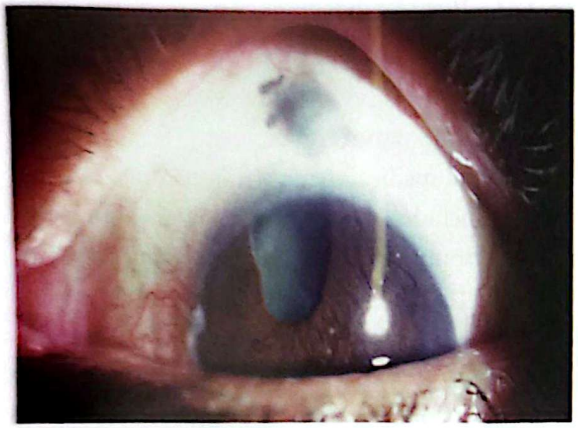


Fig. 12.1 Iridencleisis

Glaucoma is a group of disorders characterised by the presence of two or more signs among raised intra ocular pressure, optic disc cupping/optic disc sinking and characteristic visual field defects. Presence of all signs is not mandatory for the diagnosis of glaucoma, e.g., a glaucoma may be associated with normal intraocular pressure (Normal Tension Glaucoma), or the optic disc may be congested instead of being cupped (Acute Congestive Glaucoma). Historically, treatment of glaucoma was based on reduction of IOP by ligating the long posterior ciliary arteries to decreasing blood supply of the ciliary body, a procedure called cycloanaemisation or by making passages for aqueous to go out of the eye through entrapping the iris in the corneoscleral section called iridencleisis (Fig 12.1).

Aqueous humour is actively produced by ciliary processes in the posterior chamber at a rate of 2.1 ul/min. It passes through the pupil into the anterior chamber after bathing the lens. In the anterior chamber, it bathes the cornea and passes out of the eye through the angle of the anterior chamber where it encounters resistance. This resistance to the outflow of aqueous at the angle of the anterior chamber is the source of intraocular pressure (IOP). The IOP maintains the shape of globe and prevents intraocular tissues from swelling, e.g., at low IOP, choroid swells up and develops effusion. At the angle of the anterior chamber, 70% of aqueous flows out through trabecular meshwork to the canal of Schlemm and then into the blood via aqueous veins. The remaining 30% aqueous flows through interstitial spaces of the ciliary body towards the sclera, which is permeable to aqueous, and after crossing the sclera, finally reaches the orbital connective tissues from where it is taken up by blood circulation. This portion is termed as uveoscleral outflow. Normal IOP ranges between 9 mmHg to 21 mmHg. Rarely a healthy eye may have an IOP below 9 mmHg or above 21 mmHg. When the ciliary body stops producing aqueous, the eyeball shrinks to a condition called phthisis bulbi.

COMMON CLINICAL FEATURES OF GLAUCOMAS

Pain, decreased vision, red eye, cloudy cornea, symptoms related to visual field loss, halos around lights, premature presbyopia, neonate with unusually big eyes are among few clinical features of various types of glaucoma. A patient with glaucoma may be asymptomatic and diagnosed on routine eye testing.

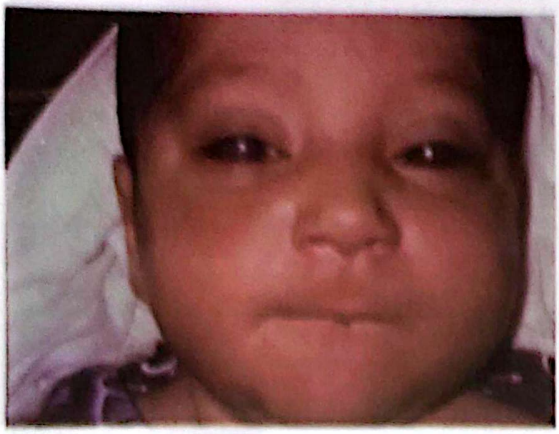


Fig. 12.2 Bilateral congenital glaucoma

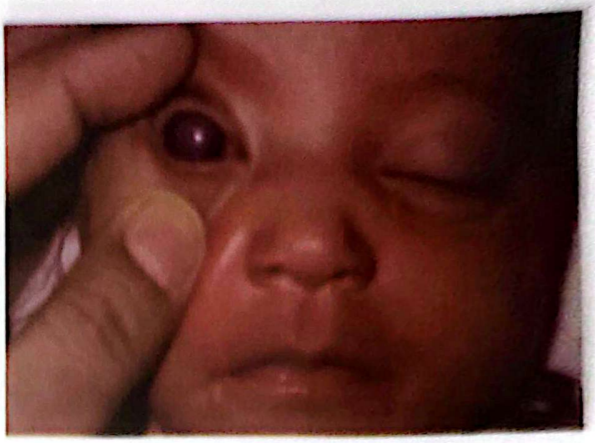


Fig. 12.3 Right buphthalmos

CLASSIFICATION OF GLAUCOMAS

Glaucomas may be classified into early onset and late onset glaucomas.

Early Onset Glaucomas

These include congenital glaucoma, infantile glaucoma and juvenile glaucoma.

Late Onset Glaucomas

These are classified into primary and secondary glaucomas.

Primary glaucomas are further classified into open angle and closed angle glaucomas

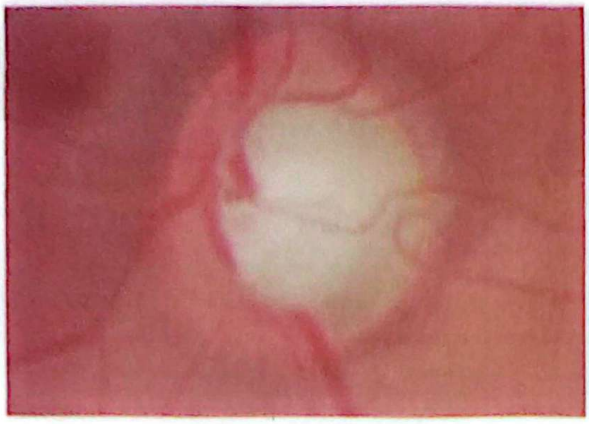
Secondary glaucomas are also further classified into open angle and closed angle glaucomas.

Congenital Glaucoma

It is the term allocated to glaucoma present at birth (Fig. 12.2). Usually both eyes are affected with unusually enlarged eyeballs thus also called "buphthalmos" or ox-eyed (Fig. 12.3). Corneal diameter may exceed 13 mm; normal average corneal diameter is 10 mm at birth and 12 mm in adults. IOP is raised and is the cause of enlargement of the globe as the infant's sclera and cornea are distensible. The cause of raised IOP is failure of development the of trabecular meshwork and the anterior chamber angle. The cause of corneal haze is due to epithelial oedema, resulting from raised IOP, and stromal oedema due to breaks in Descemet's membrane. Congenital glaucoma is sporadic but occasionally autosomal recessive inheritance is present. Treatment is initially with medical therapy followed by surgery which includes goniotomy, trabeculotomy, trabeculectomy with or without adjunctive[34] and artificial drainage devices, depending upon the severity of the condition and the surgeon's preference.

Infantile Glaucoma

It is not present at birth but becomes evident in the first few years of life. Most of the cases of infantile glaucoma are inherited as autosomal recessive and a few are sporadic. The cause of raised IOP is the same as congenital glaucoma but only differs in the stage of trabecular and anterior chamber angle development, i.e., although the aqueous outflow channels have not completely developed but are able to drain aqueous for the first few years to maintain normal IOP. The eyes are not enlarged and the cornea is not opaque, therefore it becomes difficult to even suspect glaucoma in these unfortunate children who may present with optic disc cupping and visual loss like primary open angle glaucoma. It is therefore recommended to check IOP and see the disc as a routine in children particularly those with a family history of glaucoma. Treatment is with IOP lowering drugs followed by surgery including goniotomy, trabeculotomy, trabeculectomy with or without adjunctive[34] and artificial drainage devices depending upon severity of the condition and the surgeon's preference.



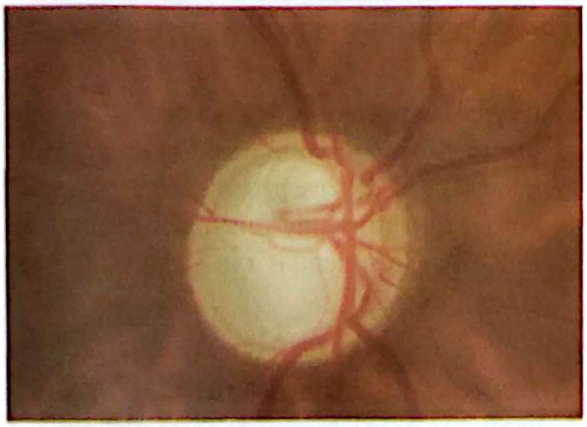


Fig. 12.4(A&B) Optic disc cupping in glaucoma

Juvenile Glaucoma

This occurs in older children and young adults up to the age of around 17 years. The cause of raised IOP is less maturity of the anterior chamber angle which functions for a limited period. Clinical presentation and treatment are the same as primary open angle glaucoma.

Primary Open Angle Glaucoma (POAG)

It is sometimes also called high pressure glaucoma and is characterised by raised IOP, slow loss of retinal ganglion cells with optic disc cupping and characteristic visual field defects. Clinically POAG may not produce any symptom till significant damage has occurred, however, rarely, onset may be early, i.e., before 40 years of age and then it may produce premature presbyopia or brow ache. Risk factors include age (there is gradual rise in IOP with advancing age due to aging of the trabecular meshwork), family history of glaucoma, race[37],

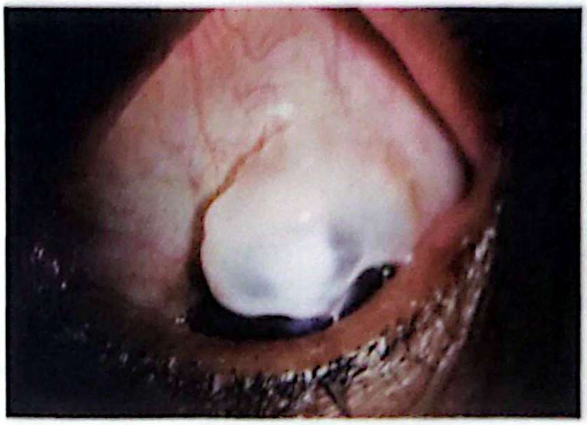


Fig. 12.5(A) Big unusual filtration bleb after trabeculectomy

blood group O-positive is frequently seen in patients with primary open angle glaucoma and A-positive frequently seen in primary angle closure glaucoma,[35] myopia, arteriosclerosis and raised IOP.

Diagnosis of POAG depends upon findings of optic disc cupping on fundoscopy (Fig. 12.4 A&B), raised IOP on applanation tonometry and characteristic visual field defects on perimetry depending upon the stage of the disease (enlargement of the blind spot, sickle-shaped scotoma, arcuate or Bjerrum's scotoma, ring scotoma, Roenne's nasal step and baring of the blind spot).

Treatment is with one or more topical IOP reducing agents like B-blockers (e.g., timolol), carbonic anhydrase inhibitors (e.g., dorzolamide), adrenergic derivatives (e.g., brimonidine), prostaglandin analog (e.g., latanoprost) and prostamide analogue (e.g., bimatoprost). When medical treatment is not effective due to a variety of reasons, surgical treatment is available which includes trabeculectomy (Fig. 12.5, 12.7) with or without adjunctive antimetabolite, e.g., mitomycin-c[36] and artificial drainage devices (Fig. 12.6). Trabeculectomy success may be facilitated by early suture lysis[37].

Primary Closed Angle Glaucoma (PCAG)

Also known as narrow-angle glaucoma, this occurs due to obstruction of aqueous outflow at the anterior chamber angle. PCAG passes through

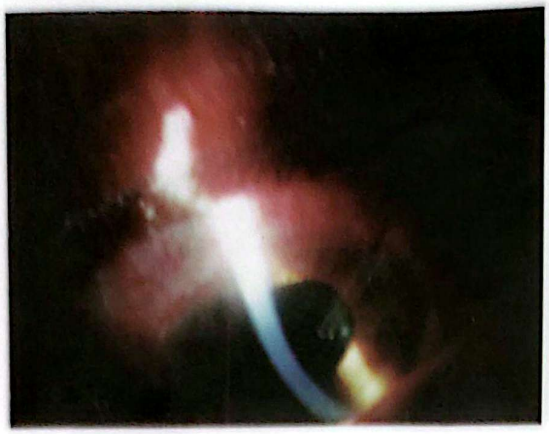


Fig. 12.6 Ahmad glaucoma valve in place

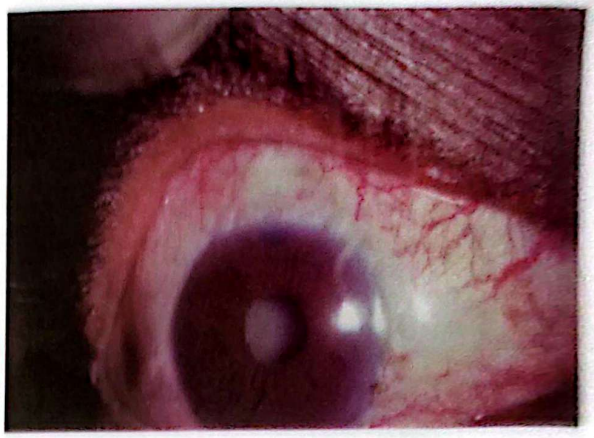


Fig. 12.7 Trabeculectomy performed in glaucoma with pseudoexfoliation of the lens

the following stages if left untreated; however, a stage may be skipped and a patient may present clinically at any stage.

1. Prodromal stage

It is the asymptomatic stage in which there is no abnormality but there is a danger of IOP rise at any moment consequent upon crowding of the iris in the anterior chamber angle due to enough pupillary dilation. Diagnosis of this stage requires gonioscopy (angle at grade 1) and provocative tests. The provocative tests like dark room test, prone provocative test, mydriatic test, Mapstone test, are all aimed at producing temporary iris crowding in the anterior chamber angle to see the effect on IOP. A rise of more than 8 mmHg on provocative testing is significant. Treatment includes either wait and watch or pilocarpine eye drops 1% to avoid iris crowding in the angle, prophylactic laser iridotomy or surgical peripheral iridectomy.

2. Stage of constant instability

It is the symptomatic stage in which IOP starts to rise intermittently and a history of halos, periods of decreased vision with pain are found. This stage results from frequent self-limited attacks of raised IOP consequent upon pupil dilation due to activities like watching a movie or dinning in a dimly lit background. For example, a person having an attack of raised IOP with associated headache may take a sleeping pill and attack is aborted due to sleep induced miosis. On gonioscopy these patients do have partially damaged angle but is still sufficiently functional to prevent IOP rise. Treatment is the same as in the prodromal stage.

3. Acute congestive stage

This occurs due to rapid rise in IOP to high levels. The patient presents with a red and painful eye with gross loss of vision, headache, nausea and sometimes vomiting. On examination, there is ciliary injection, the cornea is steamy, the anterior chamber is grossly shallow, the pupil is fixed mid dilated and vertically oval, and the IOP is very high. This stage results from either relative pupillary block mechanism, where due to already shallow anterior chamber and narrow angle a collection of aqueous under the iris cause an iris bombe's leading to iris crowding in the angle and blockage to aqueous outflow, or the plateau iris mechanism where an already anatomically anteriorly inserted iris occludes the angle on pupillary dilation. Treatment is medical, followed by surgery. Medical treatment is topical pilocarpine eye drops 2% to 4% four times a day and a B-blocker twice a day, intravenous mannitol 20% 250 ml or oral glycerol to abort the attack, acetazolamide 250mg four times a day orally and analgesics. Surgery is performed after IOP comes down to normal and is peripheral iridectomy, when the functioning angle is seen on gonioscopy

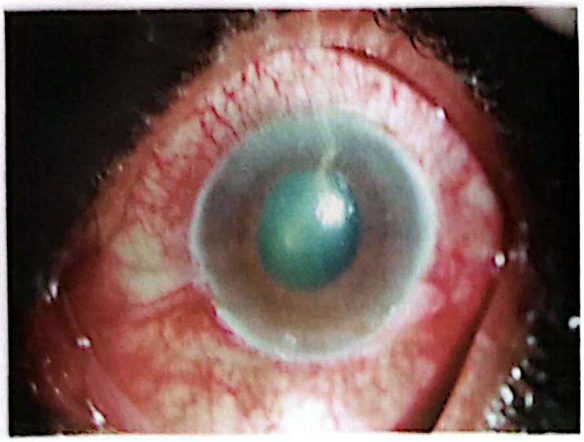


Fig. 12.8 Acute congestive glaucoma

Fig. 12.9 Flecks of lens material on the pupillary border in pseudoexfoliation syndrome

and trabeculectomy when the anterior chamber angle is nonfunctional due to peripheral anterior synechiae (Fig.12.8).

4. Chronic congestive stage

It may occur silently by skipping the acute congestive stage or may follow inadequately treated acute stage. Except closed angle on gonioscopy, other features are rhe same as POAG. Treatment is medical with pressure lowering agents followed by trabeculectomy.

5. Absolute stage

It is the stage of irreversible blindness and one should pray to Almighty Allah not to come across such patients as it creates a sense of guilt that a human is blind from a treatable disorder. Pain is treated by medical and surgical options. Medical treatment is with pressure lowering agents, mild steroids and atropine (which are contraindicated in earlier stages) in addition to analgesics. Surgical options include retrobulbar injection of absolute alcohol to denervate the eyeball or cyclocryopexy to destroy the aqueous producing factory, i.e., the ciliary body.

Secondary Open Angle Glaucomas

It is characterised by an open angle with raised IOP due to an apparent cause. This group includes the following conditions.

Phacolytic glaucoma

It is the result of a break in the capsule of a hypermature cataract leading to blockage of the anterior chamber angle with the lens cortex. The eye assumes a horrible look as white lens matter in the anterior chamber camouflages the black of the eye. Treatment is normalising IOP followed by immediate lens extraction.

Phacotoxic glaucoma

This results from micro breaks in the capsule of a cataractous lens and release of lens proteins which block the trabecular meshwork and excite a trabeculitis resulting in damage to the drainage apparatus of the eye and raised IOP. Treatment is the same as for phacolytic glaucoma.

Haemogenic glaucoma

Also called ghost cell glaucoma, occurs due to obstruction of the trabecular meshwork by the RBC envelopes (ghost RBCs). The red blood cells of fresh hyphaema (blood in the anterior chamber) passes through the trabecular meshwork easily due to their flexibility and elasticity but the red cells staying in the eye for longer periods, e.g., in vitreous haemorrhage, become inelastic and when they find a way to the anterior chamber angle they can-not pass out and get entangled causing a rise in IOP. Treatment is same as POAG.

Pigmentary glaucoma (PG)

It is mistakenly considered as a part of pigment dispersion syndrome (PDS). Pigment dispersion syndrome occurs as a result of an anatomical anomaly in the iris of some patients who possess a posteriorly bowing iris resulting in constant rubbing of the posterior layers of iris with the zonules of suspensory ligament of the lens. The pigment released passes into the anterior chamber and stains the anterior chamber angle and posterior surface of the cornea where it assumes the shape of a spindle named after the researcher Krukenberg. IOP remains normal in these patients as neither the iris pigment can block the spaces of Fontana, nor it excites inflammation. Few patients suffering from PDS may develop POAG where the only additional finding is pigmented angle and Krukenberg spindle therefore named as Pigmentary glaucoma. Treatment is same as POAG.

Pseudoexfoliation glaucoma

It is characterised by all signs of POAG and additionally greyish-white deposits over the lens surface, pupillary border (Fig. 12.9) and trabecular meshwork. Treatment is the same as POAG.

Steroid induced glaucoma

This occurs particularly in steroid responders and clinical features are same as POAG in addition to a history of steroid use and signs of the condition for which steroids were used, e.g., vernal keratoconjunctivitis. Treatment is withdrawal of steroids and, if IOP does not return to normal, same as POAG.

Glaucomatocyclitic crisis

Also known as Posner-Schlossman syndrome is a condition of transient rise of IOP due to an attack of trabeculitis in people in their bloom. On examination, IOP is usually above 50 mmHg, corneal epithelial oedema and a few kps (keratic precipitates) are found. Treatment is with IOP lowering drugs and topical steroids. These patients may be followed-up regularly.

Angle recession

Glaucoma results from blunt trauma to the globe. Angle recession is assumed on gonioscopy from the feature of widening of the ciliary body band in some part of the anterior chamber angle. Angle recession glaucoma is not an acute condition and it may take many years after trauma for IOP to rise. Once developed, angle recession glaucoma is refractory to treatment although treated like that of POAG.

Heterochromia iridis

It is a chronic uveitis with glaucoma as its complication. Glaucoma results from trabecular sclerosis and behaves like POAG in addition to difference in iris colour and symptomatology of chronic uveitis. Treatment is the same as of POAG.

Silicon oil induced glaucoma

This occurs due to blockage of the trabecular meshwork by emulsified silicon oil in the anterior chamber. Treatment is removal of silicon oil.

Secondary closed angle glaucomas

Include those closed angle glaucomas where the cause of angle closure is found. A few of them are:

Inflammatory glaucoma

Includes glaucomas caused by angle closure due to intraocular inflammation. Occlusion of the angle can occur rapidly (the iris blocks the anterior chamber angle in a circular fashion and blockage is usually reversible) or slowly (the iris blocks the anterior chamber angle at places and the blockage is usually irreversible). Treatment is with antiglaucoma medication followed by laser iridotomy or surgical iridectomy.

Neovascular glaucoma

Occurs as a complication of large ischaemic areas of the retina due to diabetic retinopathy and retinal vein occlusion. Angiogenic factors released by ischemic retina reach the iris and excite new vessel formation on the iris (rubeosis iridis) and

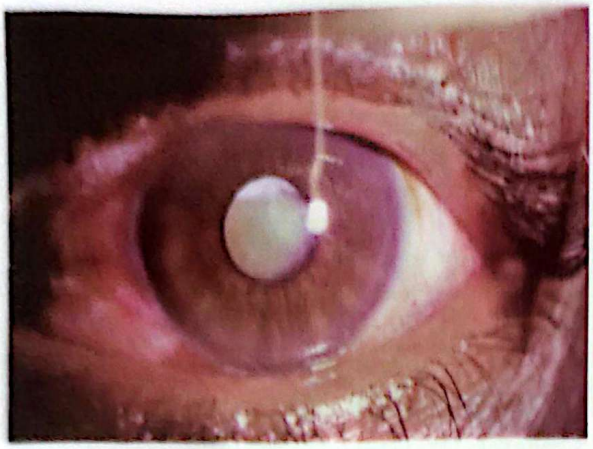


Fig. 12.10 Neovascular glaucoma with cataract

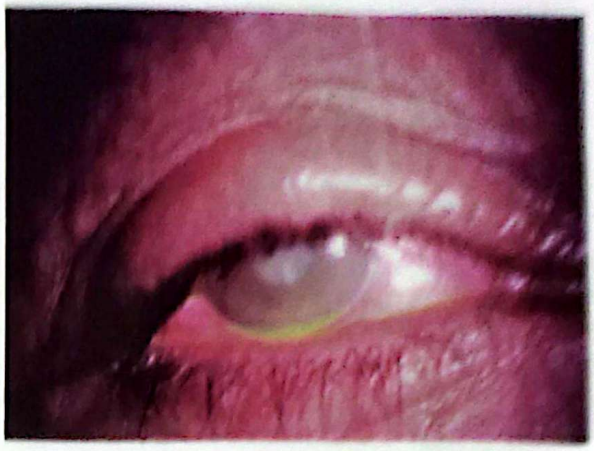


Fig. 12.11 Phacomorphic glaucoma

the anterior chamber angle where occlusion to aqueous outflow occurs resulting in neovascular glaucoma. Treatment is done by destroying the source of vasoproliferative factors with laser (retinal photocoagulation) in addition to medical therapy and surgical treatment (trabeculectomy with antimetabolite or artificial filtering shunts like the Ahmad glaucoma valve) when required (Fig. 12.10).

Phacomorphic glaucoma

Occurs due to lens swelling. Treatment is lens removal (Fig.12.11).

Malignant glaucoma

Occurs due to postoperative posterior misdirection of aqueous and pupillary block. Treatment is the control of IOP and peripheral iridectomy or intervention in the vitreous (Fig.12.12).

Glaucoma due to iris cyst

Iris cyst has been reported to cause pupillary block glaucoma rarely[39]. Nd:YAG laser have been used to treat this type of glaucoma by laser ablation of the cyst.

Normal tension glaucoma (NTG)

It is a unique variety of glaucoma in which IOP remains in normal range although visual field defects and optic disc cupping is present. It is this variety which stimulated the researchers to think of the glaucoma in terms of optic neuropathy.

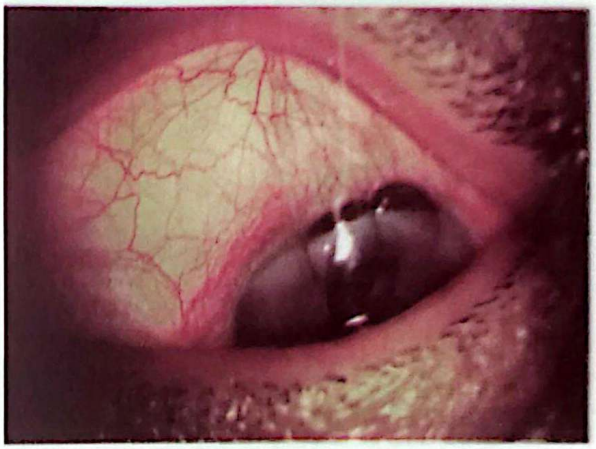


Fig. 12.12 Postoperative malignant glaucoma with iris prolapse

Treatment is aimed at keeping the IOP at the minimum, 30% reduction of IOP is desirable. B-1 selective blocker topical medication (betaxolol) is advisable to lower IOP in addition to increasing Optic nerve head blood flow. Trabeculectomy is desirable when field loss is progressive despite medication.

Ocular hypertension

It is the term given to persistently raised IOP with no disc and field changes. These patients are predisposed to glaucoma and must be kept under observation for development of glaucoma.

Chapter 13

Diseases of the Uveal Tract

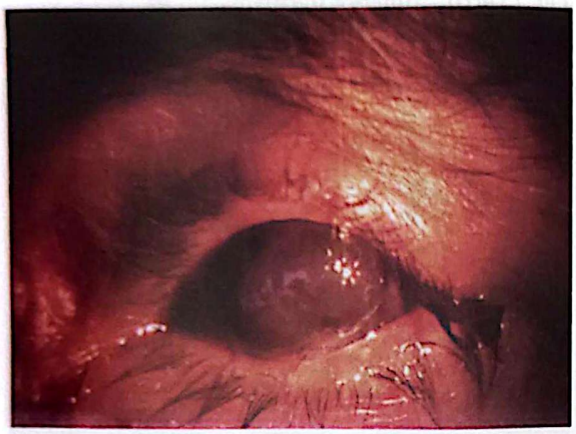


Fig. 13.1 Exposed uvea after total corneal destruction

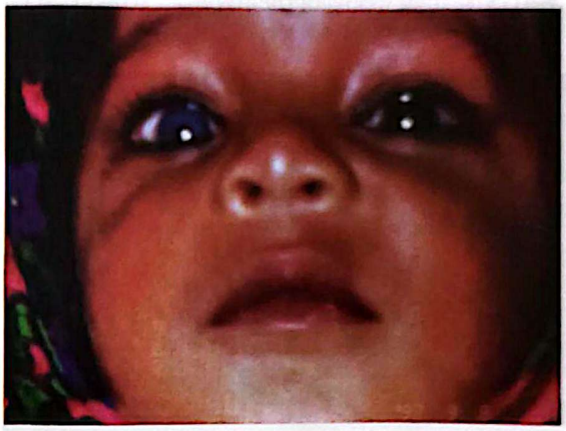


Fig. 13.2 Heterochromia iridis

The uveal tract consists of the iris, ciliary body and choroid and is protected by sclera and cornea. In the condition of corneal sloughing, uvea is exposed (Fig. 13.1). The iris and ciliary body are called anterior uvea and choroid is called posterior uvea. Blood supply of the iris, ciliary body and anterior half of the choroid comes from 7 anterior ciliary arteries and 2 long posterior ciliary arteries, whereas the posterior half of the choroid receives blood supply from 12 to 20 short posterior ciliary arteries. Pain receptors are found in the iris and ciliary body but not in the choroid therefore acute inflammations of anterior uvea are painful but that of choroid are painless. The main structural component of the uveal tract are blood vessels in addition to connective tissue, smooth muscles and epithelia; therefore this middle coat of the eyeball is also called the vascular coat. Embryologically the iris muscles are not derived from mesoderm but are derived from neuroectoderm, which gives rise to nervous tissue, therefore sphincter pupillae and dilator pupillae are unique in their development.

COMMON CLINICAL FEATURES OF UVEAL DISEASE

Pain, redness, blurred vision, floaters and change in color of the eye (Fig. 13.2) are among the few presenting features of uveal disease.

Coloboma of the Uveal Tract

This occurs as a result of nonunion of the embryological choroidal fissure and is seen at the site of the union, i.e., the inferior nasal quadrant. The coloboma (where tissues are missing) can extend from the optic nerve to the pupil or may be limited to a part e.g. iris coloboma (Fig.13.3).

Aniridea

This results from failure of anterior growth and development of the embryological optic cup due to a variety of causes, hereditary (autosomal dominant) and nonhereditary. An iris stump is usually visible on gonioscopy. Treatment is

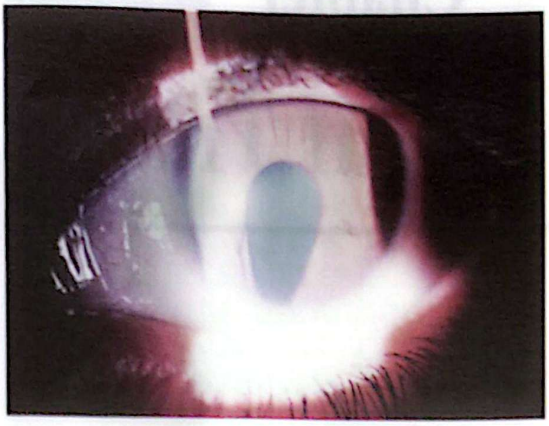


Fig. 13.3 Coloboma of the Iris

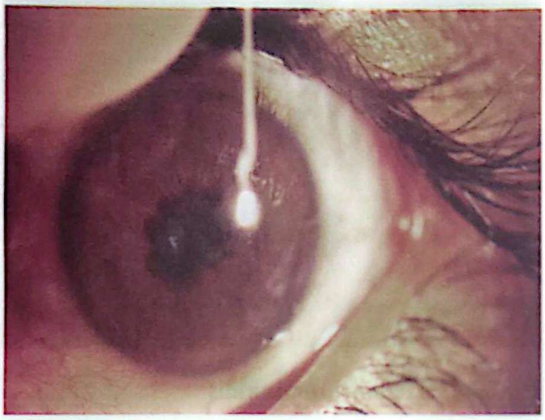


Fig. 13.4(A) Ectropion uveae

with colored contact lenses to control glare and observation for glaucoma which develops around adolescence in addition to genetic counseling.

Ectropion uveae

Presence of iris pigment epithelium on the anterior surface of the iris, pupillary border, is called uveal ectropion. It is an acquired condition but can be congenital. Acquired ectropion uveae is usually associated with neovascularization of the iris and neovascular glaucoma, but it may also be associated with the inflammatory or neoplastic process involving the iris.

Uveitis

Inflammation of the uveal tract is called uveitis and can involve the iris and ciliary body (anterior uveitis), parsplana part of the ciliary body (pars planitis or intermediate uveitis), choroid (posterior uveitis) or the whole uveal tract called pan uveitis.

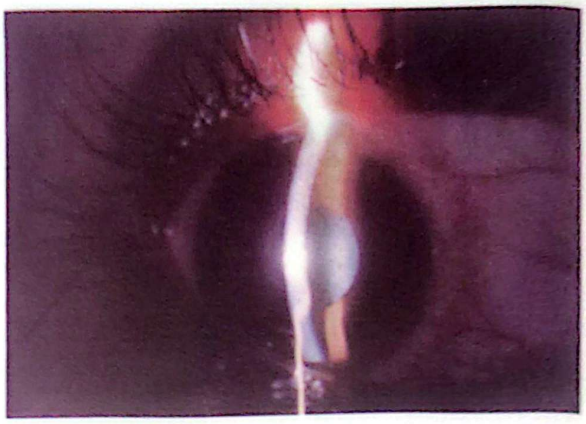


Fig. 13.4(B) Anterior uveitis

Fig. 13.5 Keratic precipitates in anterior uveitis

Signs of Anterior Uveitis

1. Flare is the earliest sign of uveal disease and is produced due to production of turbid aqueous humour instead of normal clear aqueous due to inflammation of the anterior uvea.
2. Presence of cells indicates uveitis (Fig. 13.4). In iritis, cells are present predominantly in the anterior chamber but not in the anterior vitreous. In cyclitis the density of cells is more in the anterior vitreous than in the anterior chamber. In iridocyclitis, density of cells is equal in the anterior vitreous and anterior chamber. Therefore, the presence of cells becomes a very useful sign to know which part of anterior uvea is predominantly affected.
3. Keratic precipitates (KPs) (Fig.13.5) are deposits of inflammatory debris over the endothelial side of the cornea mostly in the inferior half.

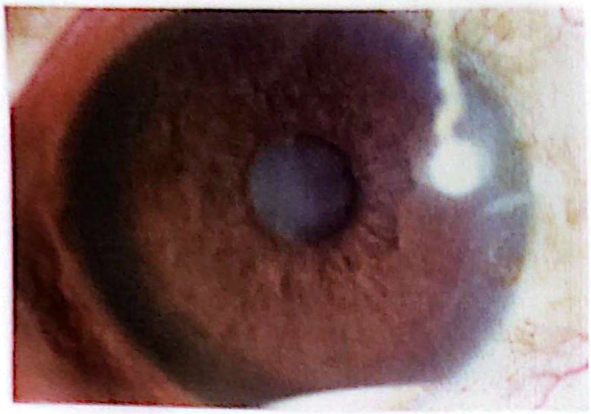


Fig. 13.6 Acute anterior uveitis

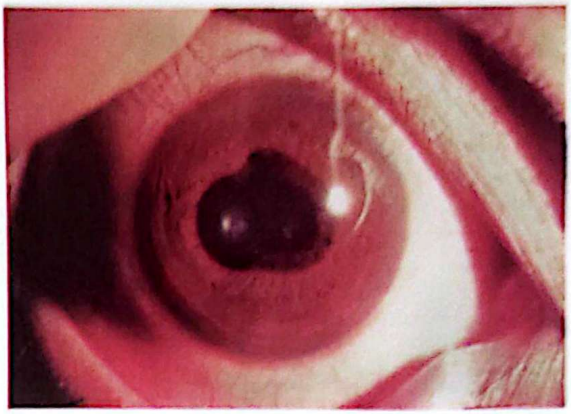


Fig. 13.8(A) Chronic Iridocyclitis with synechiae

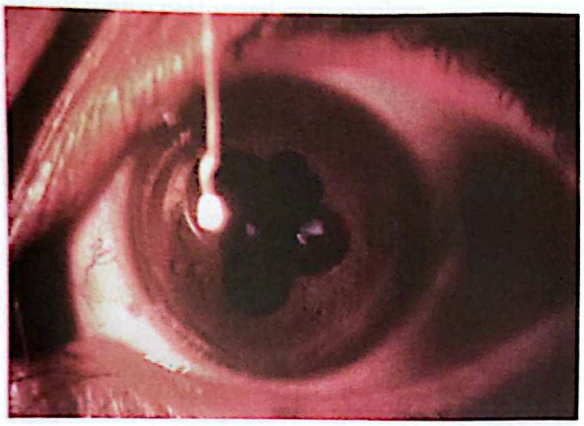


Fig. 13.7 Partially broken synechiae after use of mydriatic drops in anterior uveitis

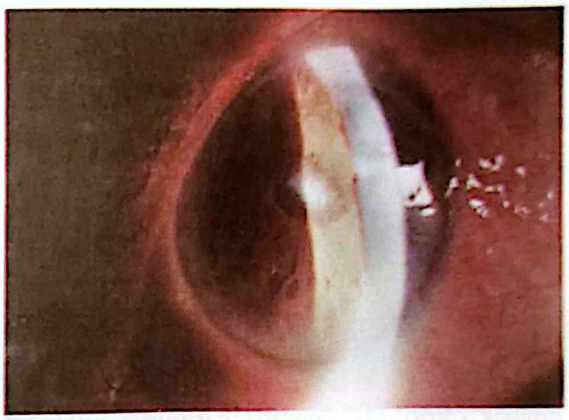


Fig. 13.8(B) Seclusio pupillae

4. Muddy iris (Fig.13.6) occurs due to deposition of inflammatory debris over the iris.
5. Inflammatory nodules which are called Koeppe's nodules are present along the pupillary border and Busacca nodules, and when present over the anterior surface of the iris, other than the pupillary margin.
6. Synechiae (Fig. 13.7,13.8 A) are adhesions between the pupillary border and the lens and occurs due to inflammatory debris acting as glue. Synechiae may also occur between **the pupillary border and the intraocular lens in pseudophakic eyes. They can be prevented with short acting mydriatic eye drops.**
7. **Posterior synechiae extending for 360° are called seclusio pupillae (Fig. 13.8 B) while occlusio pupillae** (Fig. 13.8 C) refers to a membrane obscuring the lens surface.

Signs of Intermediate Uveitis (Pars planitis)

Pars planitis is associated with aggregates of inflammatory cells in the vitreous called snowballs and the presence of white exudates (snowbanks) over the pars plana. Parsplanitis may indicate the presence of a systemic disease, therefore associated diseases like sarcoidosis, inflammatory bowel disease, multiple sclerosis, hepatitis C and Epstein–Barr virus (infectious mononucleosis) may be excluded in the affected patients.

Signs of Posterior Uveitis (Choroiditis)

- Flare and cells in the vitreous
- Vitreous deposits of inflammatory cells causing vitreous opacities
- Raised grayish patches on the fundus

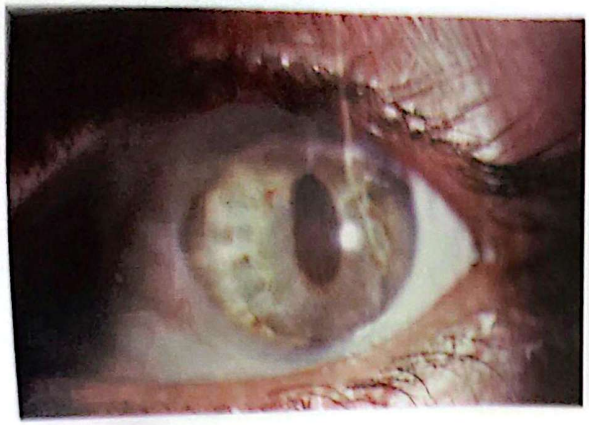


Fig. 13.8(C) Occlusio pupillae

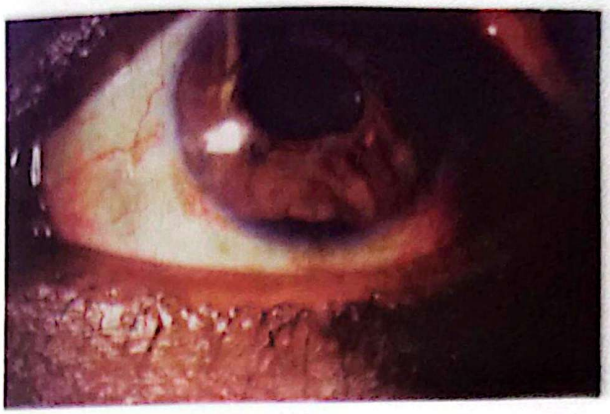


Fig. 13.10 Iridodialysis after severe trauma

Fig. 13.9 Collapsed iris cyst after YAG laser treatment looking like a melanotic lesion of iris

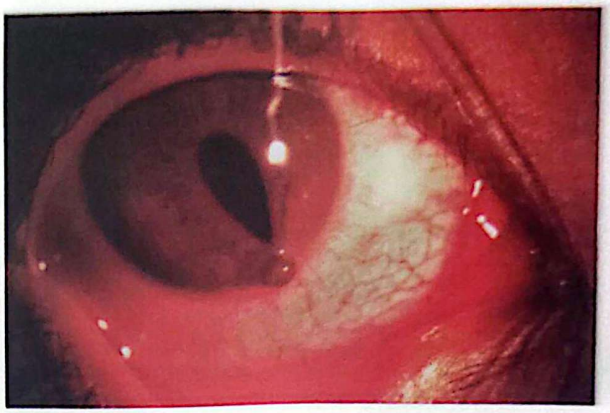


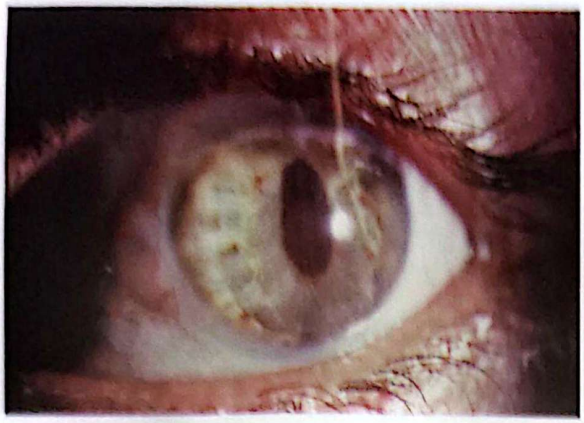
Fig. 13.11 Iris prolapse after penetrating trauma

associated with vitreous activity indicate active or hot lesions of posterior uvea

- Depressed lesions with pigmented borders on the fundus, without vitreous activity, indicate inactive or cold lesions of posterior uvea

CLASSIFICATION OF UVEITIS

- Uveitis associated with arthritis[41], e.g., ankylosing spondylitis, Still's disease, Reiter's syndrome
- Uveitis associated with viral infections, e.g., rubella, herpes simplex in children and herpes zoster in adults
- Uveitis associated with bacterial diseases, e.g., tuberculosis, leprosy, syphilis and Lyme disease
- Uveitis associated with fungus diseases, e.g., postoperative fungal endophthalmitis, panuveitis or endophthalmitis from Candida albicans in drug addicts, and histoplasmosis
- Uveitis associated with parasitic diseases e.g., toxoplasmosis and toxocariasis
- Uveitis associated with non-infectious systemic disease, e.g., sarcoidosis, ulcerative colitis, Crohn's disease, Whipple's disease, Vogt-Koyanagi-Harada syndrome and Behecet's disease
- Idiopathic uveitis, e.g., parsplanitis, Fuchs heterochromic uveitis, juvenile chronic anterior uveitis, multifocal choroiditis, serpiginous choroiditis and birdshot choroidopathy



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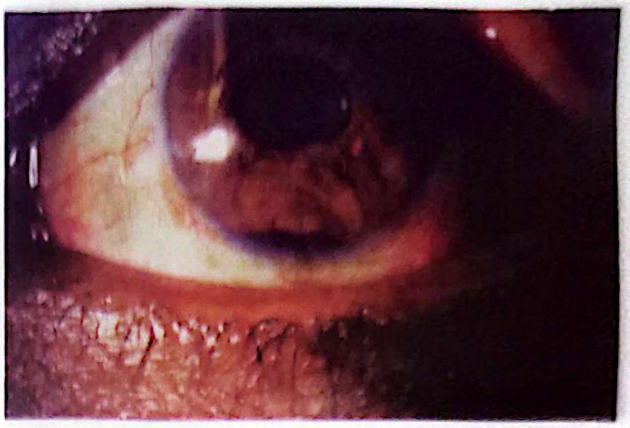

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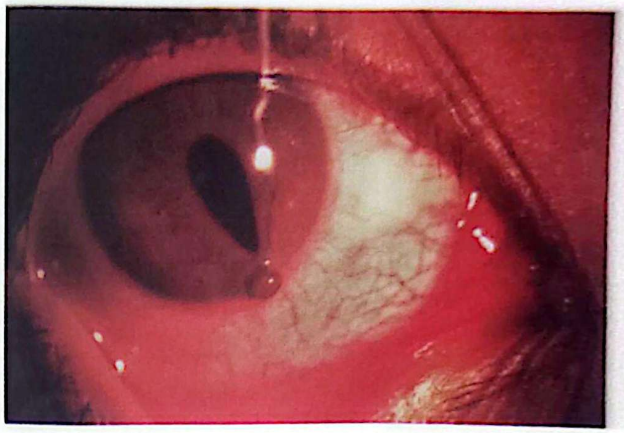

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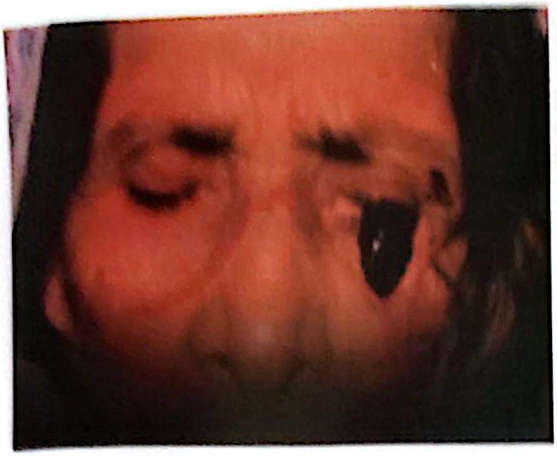


Fig. 13.12 Uveal prolapse after spontaneous expulsive hemorrhage due to hypertension in a patient with a perforated corneal ulcer

Treatment of Uveitis

It is aimed at controlling the inflammatory process with steroids, investigating and treating the underlying disorder and prevention of complications which may be a consequence of uveitis itself or as a result of drugs. Mydriatics are used to prevent synechae formation and to immobilise the inflamed parts to decrease intensity of inflammation.

Tumours of the Uveal Tract

These may be primary or secondary. Primary malignant tumour of the uveal tract is malignant melanoma which may arise from choroid, ciliary body or the iris, usually from preexisting nevi.

Any pigmented lesion (Fig. 13.9) in any part of the uveal tract should be watched closely for enlargement through regular visits and photography. Early detection and younger age are associated with better prognosis. Prognosis of malignant melanoma when detected late and in an older age group is not good. Diagnosis is suspected on history, slitlamp examination, fundoscopy, ultrasound A-scan and B-scan, indocyanine green angiography and evidence of metastasis, if present. Treatment options available are trans-scleral local surgical resection, transpupillary thermotherapy (with diode laser), laser photocoagulation, brachytherapy (application of radioactive plaque on sclera), radiotherapy, enucleation, exenteration and chemotherapy.

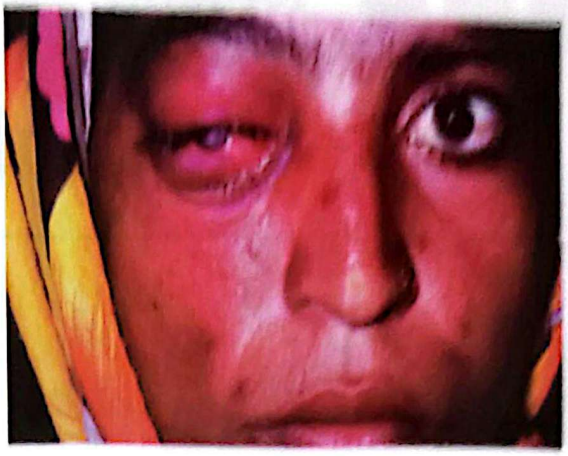


Fig. 13.13 Right panophthalmitis

Traumatic Uveal Damage

It may range from iridodialysis (Fig. 13.10) to partial (Fig.13.11) or complete uveal prolapse (Fig.13.12).

Panophthalmitis

It is a devastating condition of inflammation of the contents and coats of the globe with periocular involvement (Fig. 13.13). It is seen in severe bacterial infections. Treatment is evisceration of the globe (removal of contents and cornea, leaving behind the sclera as an inflammatory barrier).

Sympathetic Ophthalmitis

It is a rare, bilateral, autoimmune granulomatous uveitis following trauma to one eye. It is the most dreaded complication of unilateral penetrating eye injury involving uvea, as it can leave the patient completely blind. Sympathetic ophthalmitis can develop from days to several years after a penetrating eye injury. The injured eye is the exciting eye and the uninjured eye is the sympathising eye. Definitive prevention requires prompt (within the first 7 to 10 days following injury) enucleation of the injured eye. Immunosuppressive therapy is the mainstay of treatment after onset of sympathetic ophthalmitis.

Chapter 14

Diseases of Vitreous

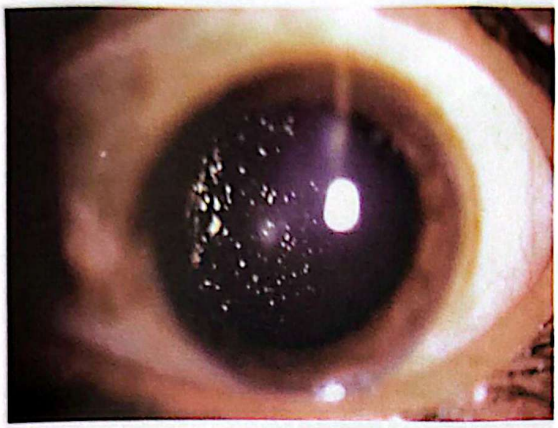


Fig. 14.1 Asteroid Hyalosis

Vitreous humour is a transparent gel like extracellular material amounting to approximately 4 ml in the human eye occupying almost two third of the volume and three fourth of the weight of the eyeball. It is composed of 99% water and 1% solids. It fills the vitreous chamber of the eyeball. Embryologically it is seen as early as the 1st month of intrauterine life when it is known as primary vitreous filling up the space between the future lens and retina and is made up of hyaloid vascular system (mesodermal) and fibrillar meshwork (somatic ectodermal). From the 4th month to the 7th month of intrauterine life, primary vitreous is replaced by secondary vitreous which is the adult vitreous. Around the equatorial area of the lens, the secondary vitreous transforms into the suspensory ligament of the lens and thus represents tertiary vitreous. Adult vitreous is composed of coiled up hyaluronic acid molecules, collagen fibrils, few cells (hyalocytes) and water containing electrolytes, amino acids, glucose and ascorbic acid. Structurally central vitreous is surrounded by vitreous cortex which is attached posteriorly with the margins the of optic disc and anteriorly with the ora serrata (vitreous base) and lens (hyaloidocapsular ligament).

COMMON CLINICAL FEATURES OF VITREOUS DISEASE

Loss of vision due to vitreous hemorrhage and floaters are among common clinical presentations of vitreous disease.

Posterior Vitreous Detachment (PVD)

It is the detachment of posterior vitreous cortex from its attachment on the optic disc. It occurs in the aged population but may occur at a younger age due to vitreous traction as a result of trauma or disease. Clinically, the patient may present with flashes and floaters. A circular opacity may be seen on the detached posterior vitreous called Fuch's ring which represents the attached area of vitreous with the optic disc in addition to optically empty space behind. Patients with PVD should undergo thorough fundus examination to exclude retinal breaks and areas of vitreoretinal traction.

Asteroid Hyalosis

It is a condition in which numerous creamy white snowball type of opacities are seen in the vitreous (Fig. 14.1) which result from aggregation of lipids containing calcium (saponified calcium soap). They are alarming for a young ophthalmologist more than the patient as they are usually symptom less and do not require treatment.

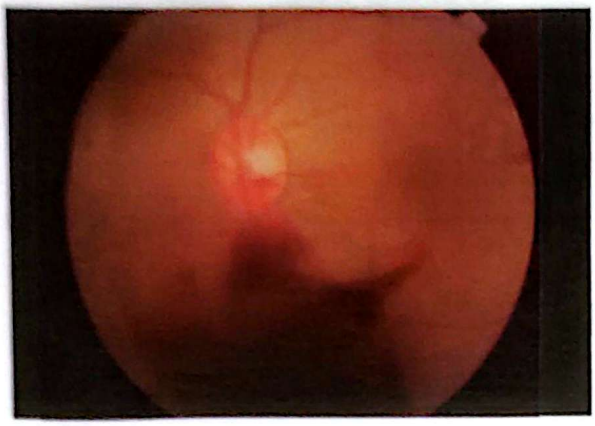


Fig. 14.2 Vitreous haemorrhage

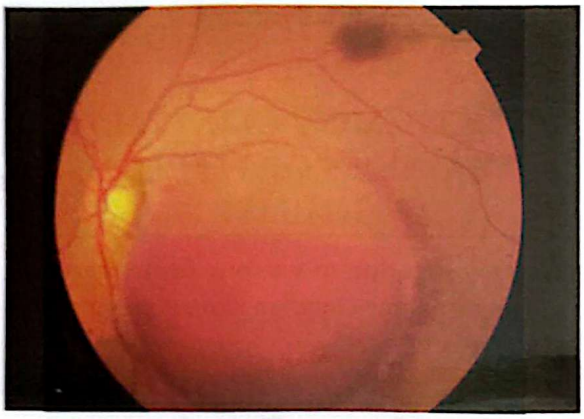


Fig. 14.3 Subhyaloid haemorrhage

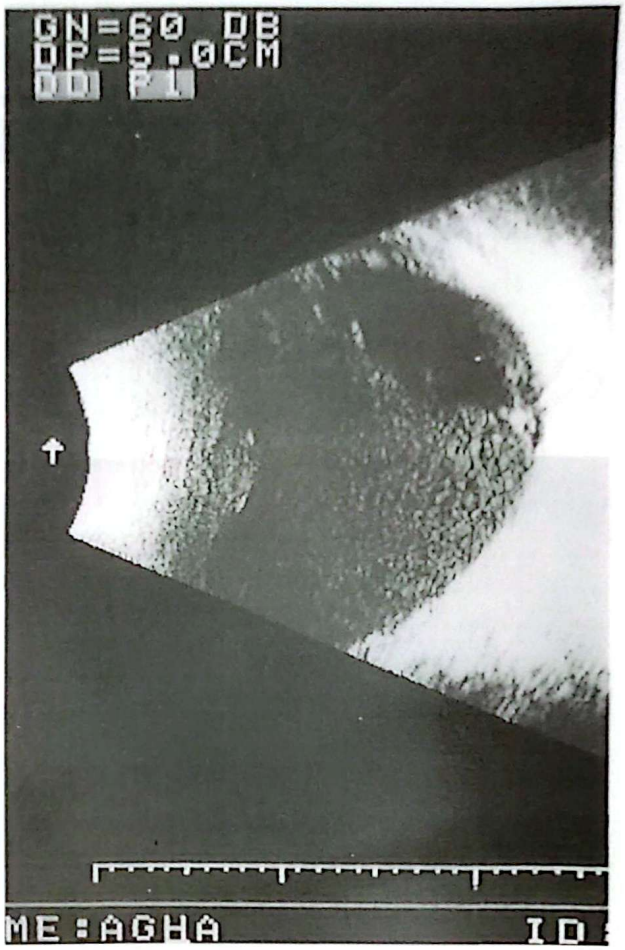


Fig. 14.4 B-scan showing Vitreous haemorrhage with intact retina

Synchysis Scintillans are coloured cholesterol crystals floating freely in the vitreous. They may rarely disturb vision.

Vitreous Haemorrhage

It is caused by rupture of abnormal or normal retinal blood vessels in a wide variety of conditions ranging from trauma to proliferative retinopathies. Vitreous haemorrhage can be preretinal, when blood is present between vitreous cortex (hyaloid membrane) and the internal limiting membrane of the retina (Fig. 14.3), or intragel when present inside the vitreous gel. All patients with vitreous haemorrhage should undergo ultrasound B-scanning (Fig. 14.4) to exclude retinal detachment in addition to detailed investigations to ascertain the cause for proper management of the disease process (Fig. 14.2).

Persistent Hyperplastic Primary Vitreous

This occurs due to failure of the embryological hyaloid vascular system to regress which is also associated with cataract and failure of the eyeball to grow, resulting in microphthalmia (small sized eyeball). The treatment is removal of the cataractous lens and aphakic correction with a contact lens. Prognosis is not good due to the associated underdevelopment of the retina.

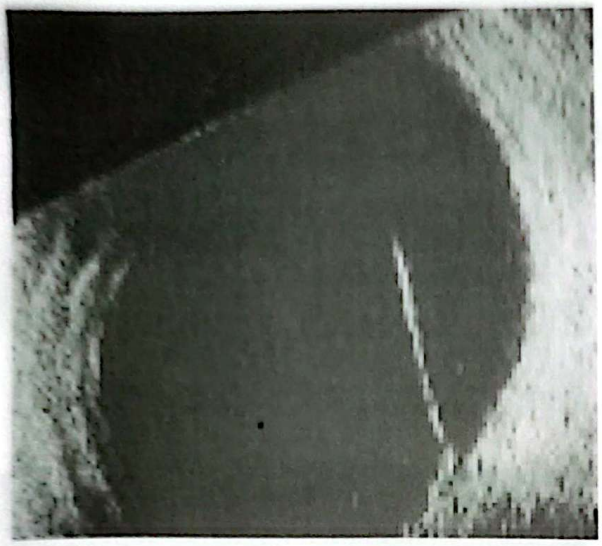


Fig. 14.5 Vitreous band causing traction on the retina as seen on B-scan Ultrasonography

Vitrectomy

It is the surgical removal of vitreous and vitreous bands (Fig. 14.5) with replacement. It is presently done with a sophisticated machine through the pars plana. Closed anterior vitrectomy is also done where required. Vitreous replacement can be done with a balanced salt solution, silicon oil, donor vitreous, hyaluronic acid and expanding gases like sulphur hexafluoride where internal tamponade is additionally required.

Chapter 15

Diseases of Retina

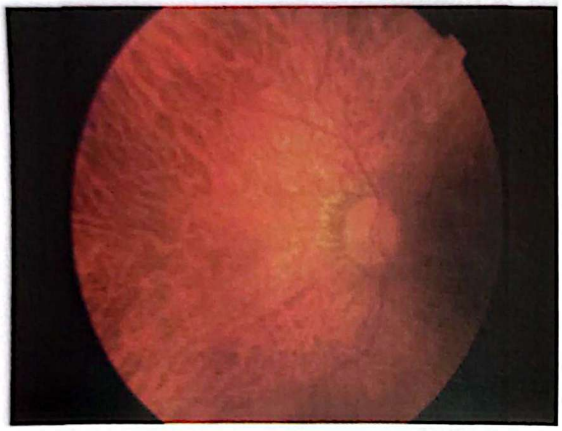


Fig. 15.1(A) Visible choroidal pattern on fundoscopy

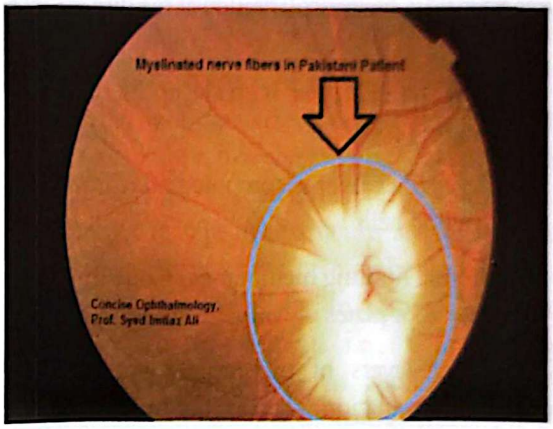


Fig. 15.1(B) Myelinated nerve fibres in the optic disc

The retina is the functional or neural coat of the eye ball and is protected from the outside by choroid and sclera, and from the inside by vitreous gel, and is thus packed perfectly. The retina develops from the embryological optic cup which is a folded neuroectoderm of the forebrain. The outer layer of the optic cup gives rise to retinal pigment epithelium; the other nine layers of retina are derived from the inner layer of the optic cup. In the adult human eye, the inner nine layers (neural retina) are transparent, therefore the retinal pigment epithelium is interposed between the neural retina and choroid. When retinal pigment epithelium is no longer capable of hiding the choroid, the choroidal pattern becomes visible on fundoscopy (Fig. 15.1 A) called tessellated fundus, erroneously.

The optic nerve receives myelination from oligodendrocytes up to its attachment with the sclera; however, sometimes myelination may extend forwards inside the optic nerve head (Fig. 15.1 B).

Under the light microscope, the retina is seen made up of 10 layers which are structurally described as under:

- Retinal pigment epithelium serves to absorb light and nourishes the adjacent outer layers
- Layer of rods and cones contains the outer segments of photoreceptor cells
- External limiting membrane is made up of outer foot plates of Muller fibres.
- Outer nuclear layer is made up of nuclei of photoreceptor cells
- Outer plexiform layer is made up of junctions between photoreceptor cells and bipolar cells
- Inner nuclear layer is made up of nuclei of bipolar cells
- Inner plexiform layer is made up of junctions between bipolar cells and ganglion cells

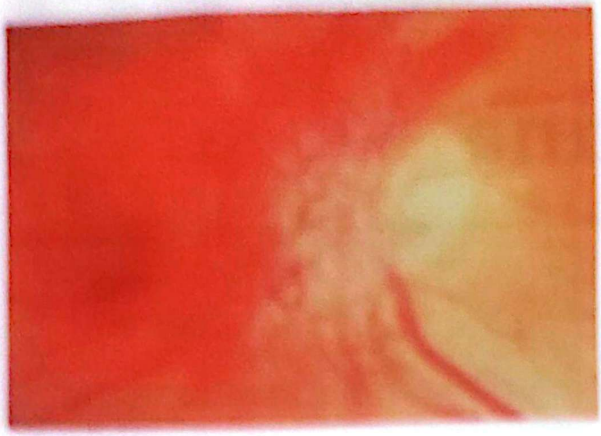


Fig. 15.1(C) Neovascularisation at the disc

- Ganglion cell layer is made up of nuclei of ganglion cells
- Nerve fiber layer is composed of axons of the ganglion cells
- Internal limiting membrane is made up of the inner foot plates of Muller cells

The outer layers of the retina depend upon choroidal circulation whereas, the inner layers of the retina are supplied by the central retinal artery. Cessation of any of the circulations is detrimental to the retina because there is no anastomosis between these two circulations.

The area between the upper and lower temporal vascular arcades is clinically regarded as the central retina (posterior pole) and the area outside this is called the peripheral retina. A central pit where the photoreceptor layer, containing only cones, is exposed to the light is called fovea (anatomically called foveola) surrounded by a disc diameter area called macula (anatomically called fovea centralis). The central retina is mainly concerned with visual acuity, colour vision and vision in bright light in addition to providing the central field of vision. The peripheral retina is mainly concerned with vision in dim light in addition to providing a peripheral field of vision.

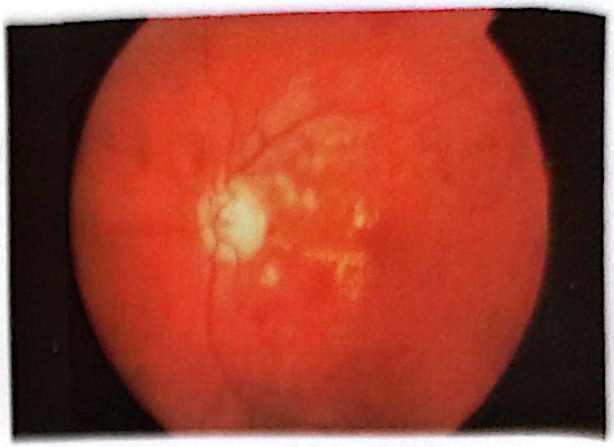


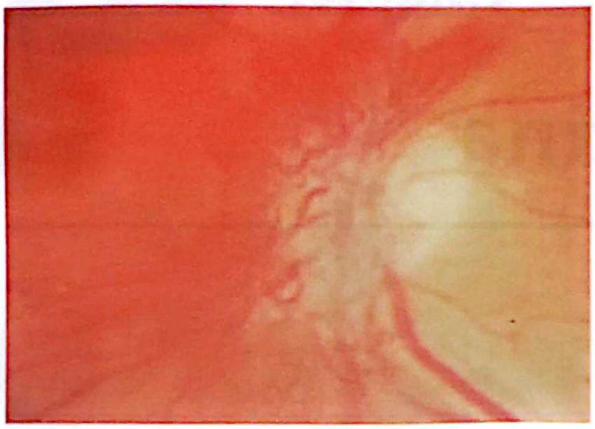
Fig. 15.2 Background diabetic retinopathy

COMMON CLINICAL FEATURES OF RETINAL DISEASE

These include flashes of light, metamorphopsia (irregular shapes of objects in view), micropsia (small sized image), macropsia (magnified image), and reduced visual acuity for far and near, impaired colour vision and night blindness.

Diabetic Retinopathy

This occurs as a result of diabetic microangiopathy affecting the retinal vasculature including precapillary arterioles, capillaries and venules. The result of diabetic microangiopathy is ischaemia of retinal neurons which in turn secrete vasoproliferative factors in a desperate attempt to produce more blood vessels for more blood circulation to increase the level of oxygen supply to suffering neurons. But this apparent good effort to oxygenate the ischaemic neurons turns into a nightmare when new vessels are produced as endothelial channels from the existing vessels called neovascularisation. Pericytes which normally strengthen the retinal capillaries are also decreased as a part of diabetic vascular changes leading to localised distensions called microaneurysms. The leakage of plasma proteins occurs from abnormal vessels leading to yellowish aggregations of proteins called hard exudates. The abnormal vessels may bleed inside the nuclear layer giving rise to circumscribed blood spots called dot haemorrhages or may bleed into the plexiform layer, giving rise to blood spots with fuzzy margins called blot haemorrhages or may



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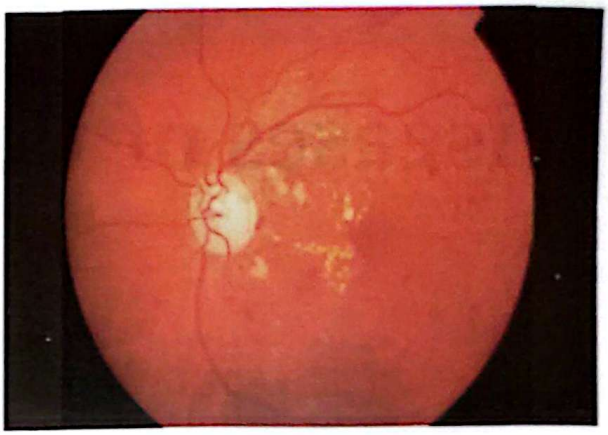

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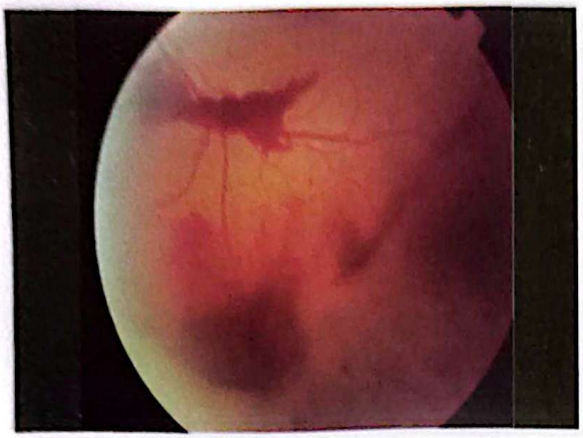


Fig. 15.3 Proliferative diabetic retinopathy

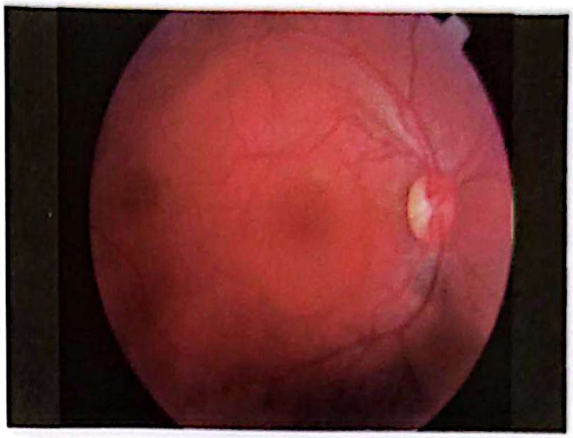


Fig. 15.4 Central serous retinopathy with RPE changes

bleed in a nerve fibre layer giving rise to flame shaped haemorrhages. When microangiopathy ends up in complete blockage of a precapillary arteriole, the area supplied by that arteriole undergoes nerve fibre layer infarction which is called a soft exudate or a cotton wool patch.

Neovascularisation may be seen on the retina called neovascularisation elsewhere (NVE) or on the optic disc called neovascularisation on the disc (NVD) (Fig. 15.1 C). Depending upon progression of diabetic retinopathy[42] it is classified into three stages. The first stage is background diabetic retinopathy[43] (Fig.15.2) characterised by the presence of microaneurysms, dot and blot haemorrhages and hard exudates. The second stage is preproliferative diabetic retinopathy which is characterised by aggravation of signs present in background diabetic retinopathy and presence of soft exudates, vascular anomalies and large blot haemorrhages which represent haemorrhagic infarcts.

The third stage is proliferative diabetic retinopathy PDR (Fig. 15.3) which is characterised by changes present in previous stages and NVD, NVE and vitreous haemorrhage. Treatment is close observation in the first two stages, except when macula is involved. When signs of PDR are seen, treatment is pan retinal photocoagulation[44] (PRP), anti-VEGF therapy and surgery.

Diabetic Maculopathy

It is the involvement of macula in the diabetic process which may develop in the form of nonperfusion of capillaries as seen on FFA, called ischaemic or dry maculopathy or leakage from capillaries called exudative or wet maculopathy. The leakage may be in a particular area of macula called focal maculopathy or may be generalised, called diffuse maculopathy. In some cases both elements may be present, i.e., nonperfusion in one area and leakage in another area of macula called mixed maculopathy. Treatment is application of laser after localisation with FFA to the leaking sites (microaneurysms) called direct laser or application of the laser around the fovea (excluding the papillomacular bundle) to stop entry of leaking fluid called macular grid laser, intravitreal injections of steroids[45] or anti-VEGF (anti-vascular endothelial growth factor) agents. The anti-VEGF agents commonly used presently include Pegaptanib (Macugen), Bevacizumab (Avastin), Ranibizumab Lucentis) and Aflibercept (Eylea). Laser treatment is only advised in diabetic maculopathy when hard exudates or oedema involves the fovea, a stage called clinically significant macular oedema (CSME).

Central Serous Retinopathy (CSR)

(Fig. 15.4) It is a relatively common macular disorder seen in young and middle aged males. CSR presents with sudden painless decrease in vision with metamorphopsia. On examination, mild RAPD may be present with a decrease in visual acuity to a few lines, often correctable with refraction, and a central scotoma detectable on the Amsler grid. Fundoscopy reveals loss of macular reflex with a raised dome shaped macular area

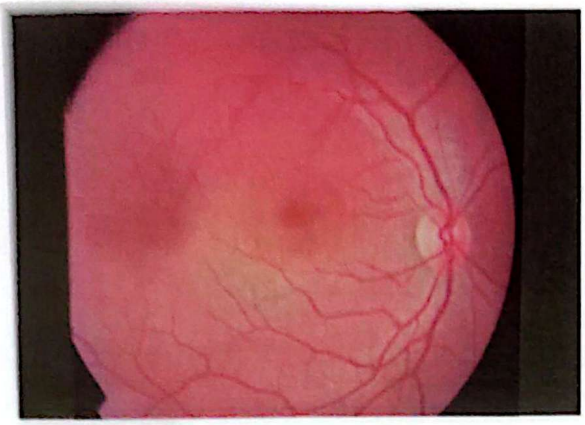


Fig. 15.5 Central serous retinopathy

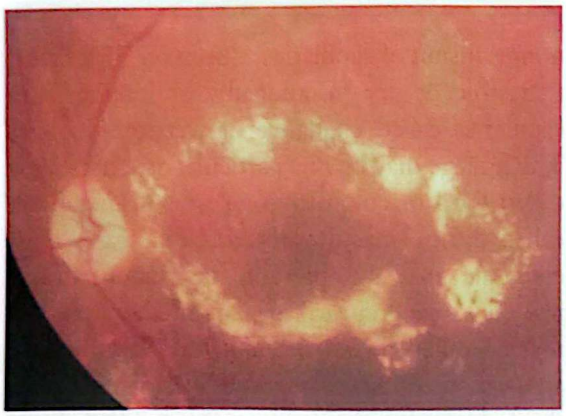


Fig. 15.6 Wet type of ARMD

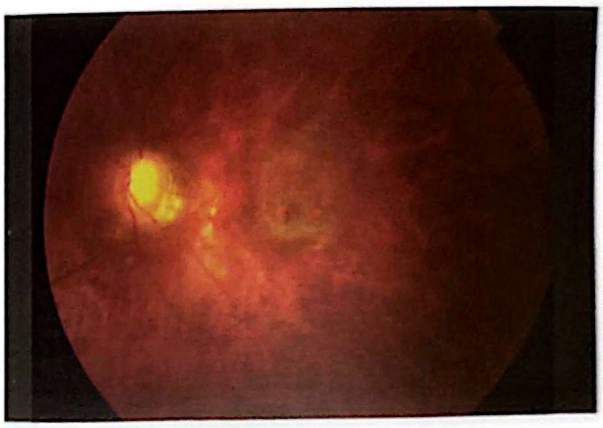


Fig. 15.7 Mixed type of ARMD

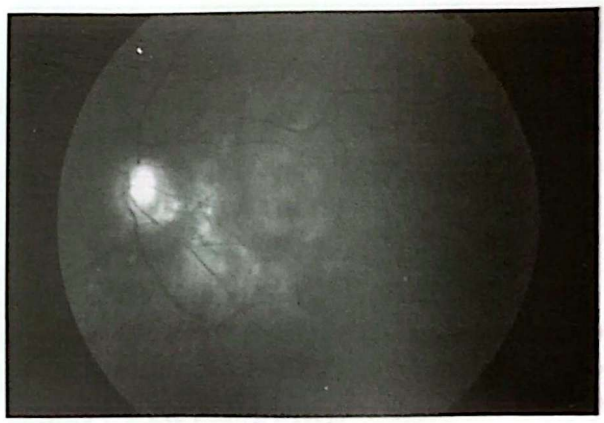


Fig. 15.8 Black & White fundus photograph of mixed type of ARMD

(Fig. 15.5). FFA, when done, shows a smoke-stack sign on the macular area due to vertical spread of dye from the site of the defect in RPE. Sometimes, instead of the common smoke-stack sign, the dye after passing through RPE defect spreads all around in a circular fashion called the ink-blot sign, but one Pakistani study has reported that the ink-blot sign is seen more frequently than the smoke-stack sign on FFA[46].

Treatment is wait-and-watch until resolution occurs and visual acuity returns to normal; which may take 6 to 12 months. When defect in RPE is away from the foveal avascular zone, laser photocoagulation may be carried out to seal the defect after 4 months, in an attempt to hasten resolution. Oral acetazolamide 125mg in the morning has been advocated to quicken the resolution.[47]

Age Related Macular Degeneration (ARMD)

Also called senile macular degeneration. This condition presents with metamorphopsia and progresses to loss of central vision, and occurs in an elderly population in three forms.

1. Dry ARMD or the atrophic type; accounts for majority of the cases.
2. Wet ARMD (Fig. 15.6, 15.9, 15.10) or subretinal neovascular membrane formation (SRNVM) or choroidal neovascularisation (CNV) is less common but associated with severe visual loss. 3. Mixed type_when both processes are combined (Fig. 15.7, 15.8).

Atrophic ARMD

It is associated with the deposition of drusen (deposition of abnormal material under RPE)

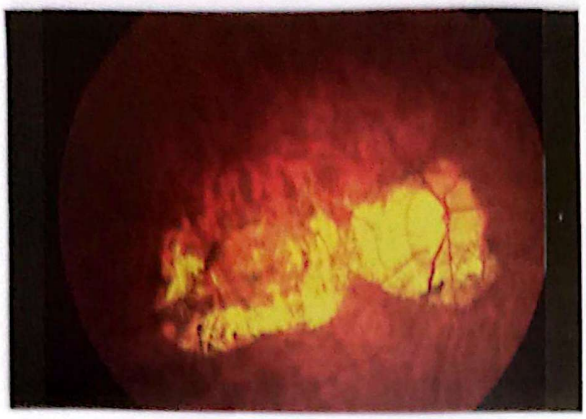


Fig. 15.9 Advanced stage of wet ARMD

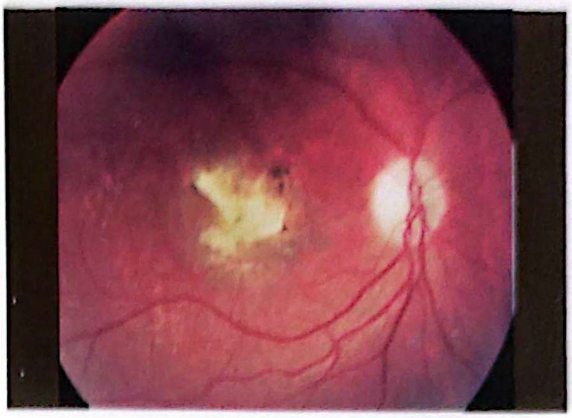


Fig. 15.11 Atrophic ARMD

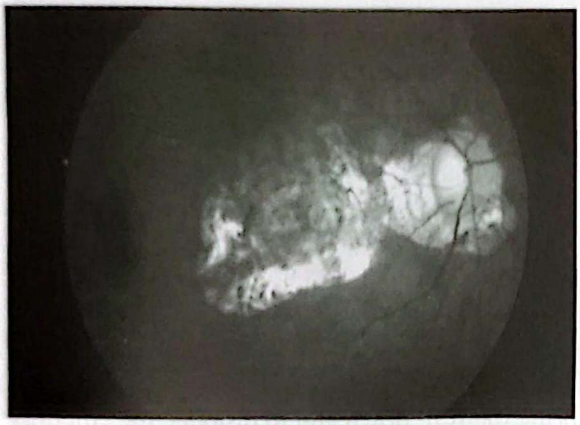


Fig. 15.10 Black & white fundus photograph of wet ARMD

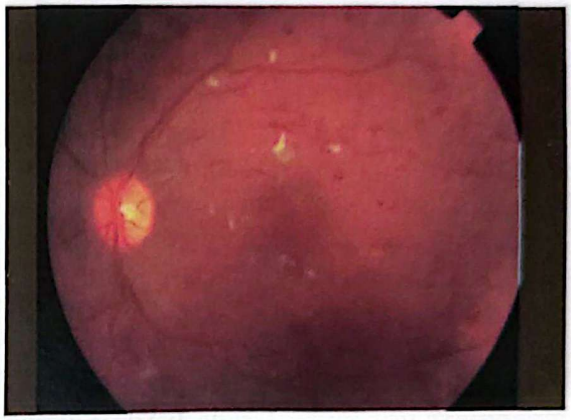


Fig. 15.12 Hypertensive retinopathy

visible as yellow deposits, atrophy of cones leading to visual loss and atrophy of RPE showing through the choroid underneath. Laser is not advisable in atrophic ARMD as it will cause more atrophy (Fig. 15.11).

Wet ARMD or SRNVM

Results from the growth of new vessels beneath the RPE (and later on over the RPE) from choriocapillaris through defects in Bruch's membrane. RPE may detach completely in the macular area due to exudation. Treatment is oral antioxidants, intravitreal injection of steroid or anti-VEGF medication, laser photocoagulation or photodynamic therapy (PDT) depending upon the stage of the disease.

Hypertensive Retinopathy

Occurs in long standing hypertension. Fundus changes occur due to rigidity and narrowing of arterioles which may press the veins on arteriovenous crossings called AV nipping. Sustained hypertension may damage the blood retinal barrier leading to increased vascular permeability resulting in the formation of hard exudates and flame-shaped haemorrhages in addition to disc swelling. Copper wiring followed by silver wiring of retinal arterioles indicates advanced stage. **Grading of Hypertensive retinopathy** is an important tool to understand the stage of disease and its management by an ophthalmologist, physician and cardiologist. The old method of grading has focused on retinal signs only, whereas, the new method of grading has included both retinal signs and associated symptoms of patients. The old one is **Keith-Wagener- Barker (KWB) Grading** which has

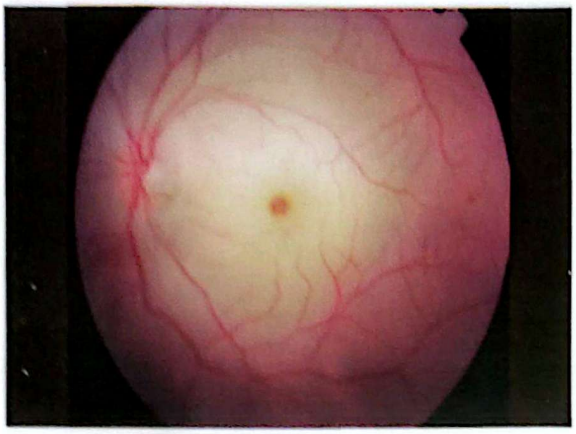


Fig. 15.13 Central retinal artery occlusion

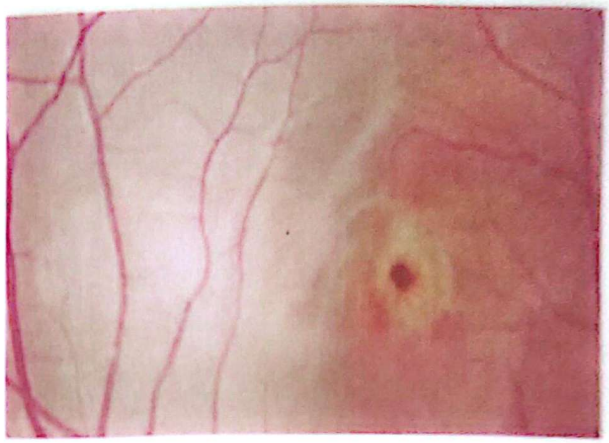


Fig. 15.14 Cherry red spot on macula in gangliosidosis

described four grades of hypertensive retinopathy. Grade 1: Generalised arteriolar constriction - seen as `silver wiring` and vascular tortuosities. Grade 2 Same as grade 1 + irregularly located, tight constrictions - Known as `(AV) nicking` or `AV nipping'. Grade 3: Same as grade 2 + with cotton-wool spots and flame-shaped haemorrhages. Grade 4 Same as the previous grades, but with swelling of the optic disc (papilloedema). A new one is **Imtiaz's grading of Hypertensive Retinopathy** which has described three grades of hypertensive retinopathy, **Grade 1:** Silver wiring of arterioles with AV nipping and headaches. **Grade 2:** Same as Grade 1 with flame-shaped haemorrhages, soft exudates and floaters. **Grade 3:** The same as Grade 2, with papilloedema and amaurosis fugax. A major aim of **treatment** is to prevent, limit, or reverse target organ damage by lowering the patient's high blood pressure and reduce the risk of cardiovascular disease and death. Topical medication, betaxolol, is given to increase optic nerve head blood flow (Fig.15.12).

Central retinal artery occlusion (CRAO) results in the sudden painless loss of vision. Fundoscopy reveals white retina with a **central cherry red spot** (Fig. 15.13) for almost a week followed by return of red fundus background with attenuated arteries and white optic disc.

Cherry red spot on macula is also seen in gangliosidosis[48] (Fig.15.14) in children. CRAO is caused by a variety of diseases involving vessel wall and blood circulation, including emboli from ruptured atheroma in the carotid artery in older individuals and emboli from cardiac valves in younger people, or thrombosis in disseminated intravascular clotting in a young lady with child birth related complications. Spasm of the central retinal artery may occur as a result of arteritis in older people and vasomotor instability in younger people. Treatment given within one hour is fruitful, after one hour partial recovery of peripheral vision occurs up to about 5 hours, but after that ischaemic necrosis of the inner layers of the retina leads to permanent loss of vision. Treatment includes immediate ocular massage to bring the IOP down and to dislodge the embolus in addition to allowing the patient to respire in a polythene bag (shopper bag available everywhere) in an attempt to increase PCO2 in the blood which results in vasodilatation. If the patient has the opportunity of immediate hospitalisation, then intravenous heparin and acetazolamide may be given.

Central retinal vein occlusion: It is seen typically in hypertensive people above the age of 60 years. Venous obstruction usually occurs at the level of the optic disc where the central retinal artery and vein share a common tunica adventitia (a thickened artery may compress the vein). Visual loss results from retinal hypoxia produced as a result of stagnation of blood circulation due to occlusion of venous bed extending to the level of capillaries. Bleeding starts from the capillaries due to ruptures in the walls of the capillaries as a result of raised blood pressure in capillaries and hypoxia of capillary walls. Clinically CRVO presents in two forms.

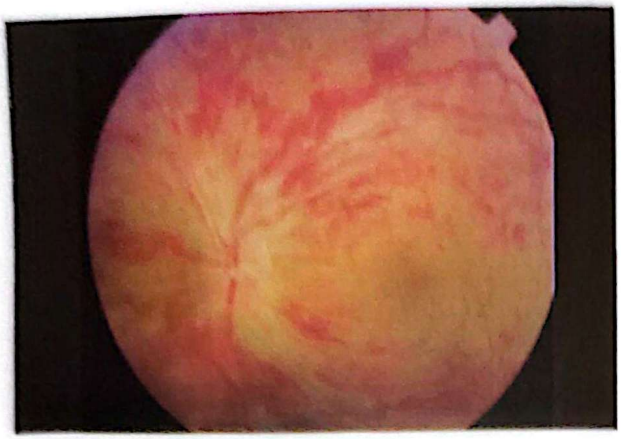


Fig. 15.15 CRVO (non-ischaemic)

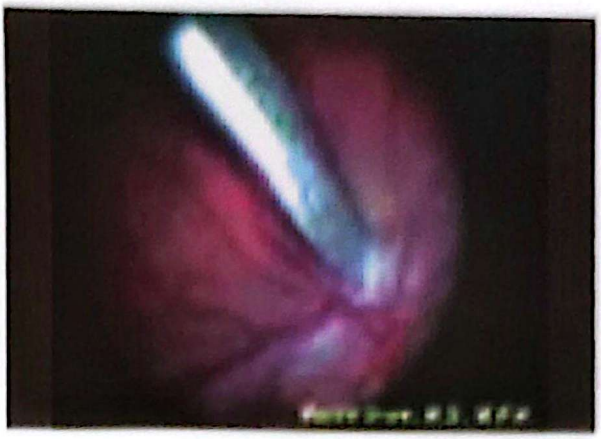


Fig. 15.17 Surgical radial optic neurotomy

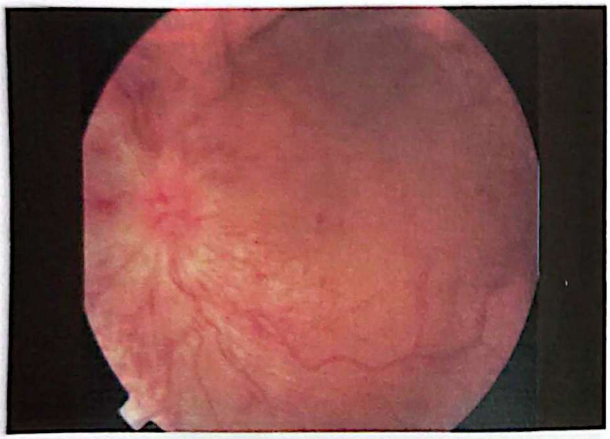


Fig. 15.16 CRVO (ischemic)

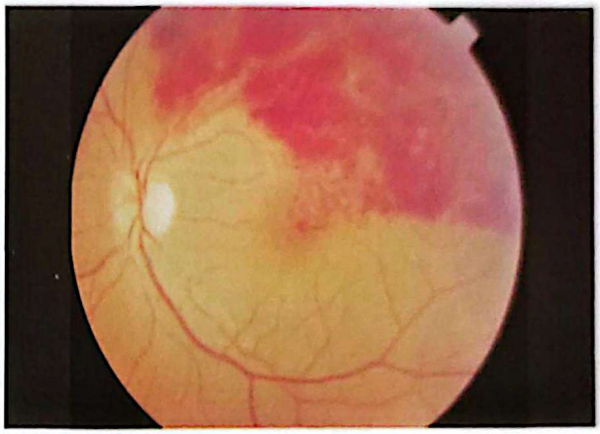


Fig. 15.18 Branch retinal vein occlusion (BRVO)

Non-ischaemic CRVO

This presents with moderate painless decreased vision. On examination, moderate relative afferent pupillary defect (RAPD) is present. Fundoscopy reveals a blot and flame-shaped haemorrhages in all four quadrants of the retina up to the periphery with the disc and macular oedema. Visual recovery is directly proportional to the level of visual acuity at the onset. FFA performed after resolution of haemorrhages shows good retinal capillary perfusion (Fig. 15.15).

Ischaemic CRVO

(Fig. 15.16) This presents with severe painless loss of vision. On examination, severe relative afferent pupillary defect (RAPD) is present. Fundoscopy reveals severe haemorrhage in all four quadrants of the retina up to the periphery with soft exudates and severe disc hyperaemia and oedema with macular involvement. Visual recovery is poor due to extensive retinal capillary nonperfusion resulting in severe retinal ischaemia as seen on FFA after resolution of haemorrhages. Treatment is surgical radial optic neurotomy (Fig. 15.17) in an attempt to restore the circulation. Patients should be examined regularly for the evidence of rubeosis (new vessels) on the pupillary border and anterior chamber angle, which may take three months to develop and cause neovascular glaucoma (90-day glaucoma). When neovascularisation is detected, the treatment is panretinal photocoagulation (PRP).

Branch Retinal Vein Occlusion (BRVO)

It is the occlusion of a branch of the central retinal vein and occurs usually in the elderly population. BRVO, when involving a main branch, presents with blurred vision and metamorphopsia due to involvement of macula. Fundoscopy reveals dilated

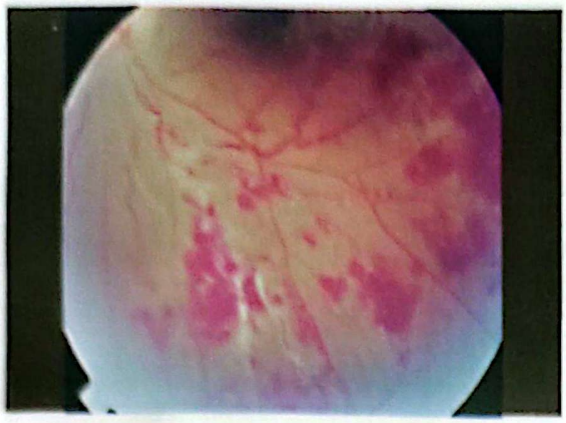


Fig. 15.19 Eales disease

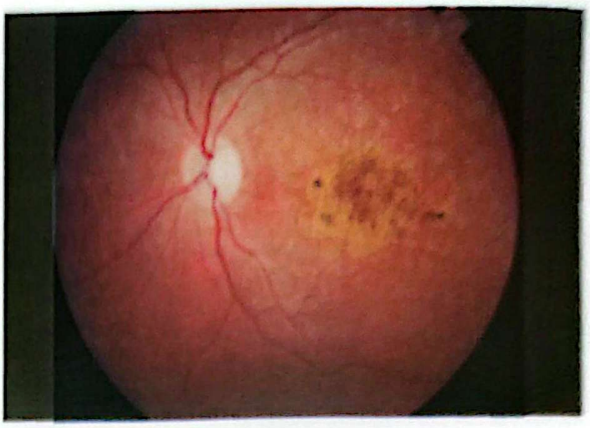


Fig. 15.21 Stargardt maculopathy

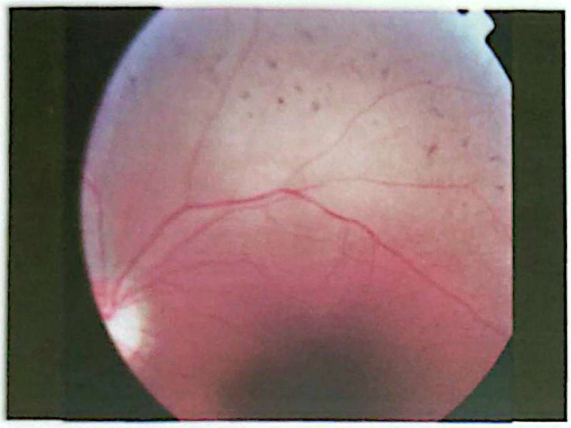


Fig. 15.20 Retinitis Pigmentosa

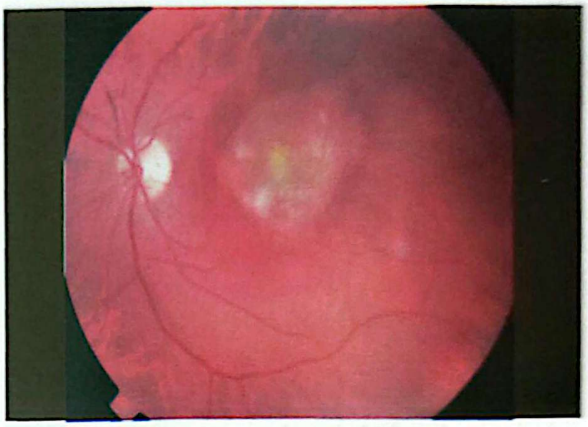


Fig. 15.22 Bull's eye maculopathy (Best disease)

and tortuous veins with a blot and flame shaped haemorrhages in the affected quadrant. Treatment depends upon the retinal capillary perfusion seen on FFA after resolution of haemorrhages. When retinal capillary perfusion is good, sectorial laser photocoagulation is recommended and visual recovery is good. When FFA shows capillary non-perfusion, laser is not recommended and prognosis is poor. One cause of decreased vision in BRVO is persistent macular oedema which should be treated with a macular grid laser (Fig. 15.18).

Eales Disease

It is the recurrent idiopathic inflammation of peripheral retinal veins[49] (retinal phlebitis) in males in their bloom leading to repeated episodes of vitreous haemorrhages followed by proliferative vitreoretinopathy, if untreated. Clinically there is a history of repeated attacks of unexplained visual loss in a young male from 15 to 25 years of age with spontaneous remissions. Fundoscopy reveals peripheral retinal venous sheathing, exudation and beading with or without vitreous haemorrhage (Fig. 15.19). Treatment is laser photocoagulation[50] of affected areas.

Retinopathy of Prematurity (ROP),

Previously called retrolental fibroplasia, is a proliferative retinopathy. It occurs in premature babies exposed to 100 percent oxygen which leads to severe vasoconstriction of retinal arterioles leading to retinal ischaemia followed by neovascularisation. Treatment is timely laser photocoagulation of the ischaemic retina.

Retinitis Pigmentosa (RP)

It is a hereditary retinal pigment epithelial dystrophy associated with photoreceptor degeneration with X-linked recessive, autosomal

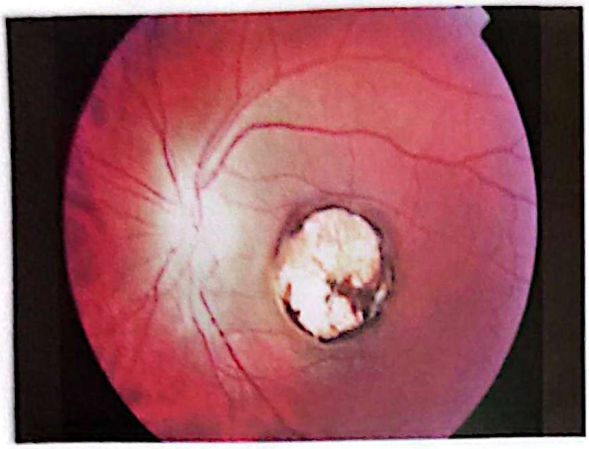


Fig. 15.23 Bull's eye maculopathy (congenital toxoplasmosis)

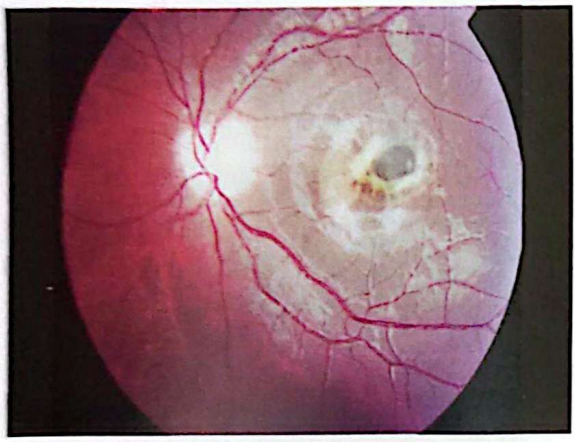


Fig. 15.24 Bull's eye maculopathy (chloroquine maculopathy)

dominant and autosomal recessive mode of inheritance. Dysfunction starts with rods followed by cones; therefore dim vision is affected first and patients complain of night blindness at puberty. Thereafter vision keeps on deteriorating till the patient becomes blind by 40 years of age in X-linked recessive forms, or is left with central vision in other forms. Fundoscopy reveals typical pigmentary changes with bone spicules appearance, waxy disc (due to atrophy with glial proliferation) and attenuated retinal vessels. Posterior subcapsular cataract is always associated. Management includes early detection with the help of history, examination and electrophysiological tests like ERG. Oral anti-oxidants, avoidance from strong lights, low vision aids, occupational and genetic counselling are offered to help these patients (Fig.15.20).

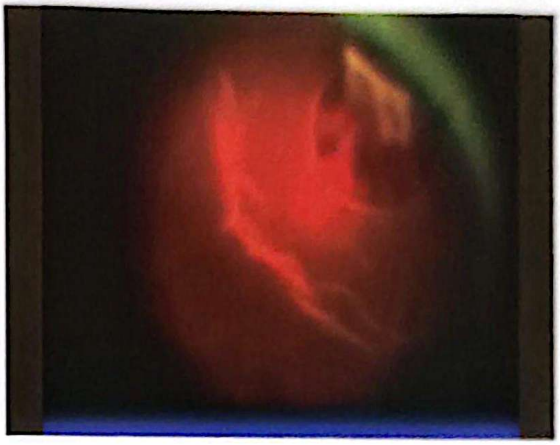


Fig. 15.25 Rhegmatogenous RD with horseshoe tear

Bull's Eye Maculopathy

This includes a wide variety of macular lesions which are characterised by a hypopigmented area around the fovea encircled by a hyperpigmented area in varying combinations, giving rise to a so-called bull's eye appearance over the macula. Drug toxicity including chloroquine (Fig. 15.24) and phenothiazines, Stargardt disease (Fig. 15.21), eclipse maculopathy due to gazing towards a solar eclipse, cone dystrophy, Batten disease, toxoplasmosis (fig. 15.23), Best disease (Fig. 15.22) and Bardet-Biedl syndrome are known causes of bull's eye maculopathy.

Acute Posterior Multifocal Placoid Pigment Epitheliopathy (APMPPE)

It is the vasculitis of choriocapillaris in young women characterised by pale white lesions at the posterior pole. Steroids in high doses may help in resolution, if given in the early stage.

Retinoschisis

It is a separation of the inner layers of neural retina from the outer layers, usually at the level of the outer plexiform layer. Retinoschisis usually starts from a pre-existing giant retinal cyst and is not associated with visual loss unless complicated by retinal detachment. It requires no treatment.

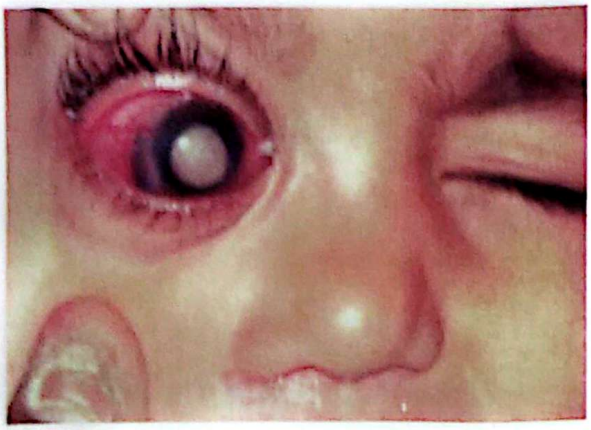


Fig. 15.26(A) Right Leukocoria due to retinoblastoma

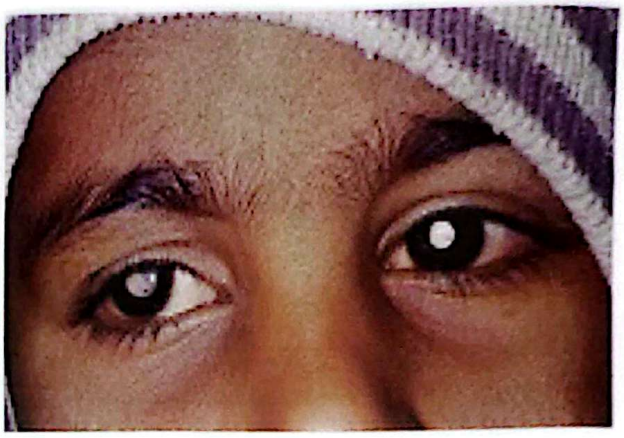


Fig. 15.26(B) Bilateral Leukocoria due to retinoblastoma

Retinal Detachment (RD)

It is a state of separation of the neural retina from RPE leading to visual loss. Four types of retinal detachment are seen clinically. 1. Rhegmatogenous RD (Fig. 15.25) is associated with a retinal hole or break through which fluid gets access into the potential space between neural retina and RPE resulting in RD. Treatment is reattachment of the retina by draining subretinal fluid (SRF) and sealing the hole by indenting the sclera over the hole by placing an explant or scleral buckle. 2. Exudative RD occurs due to massive exudation into the subretinal space as a result of inflammation. Treatment is steroid administration to resolve inflammation. 3. Tractional RD occurs due to centripetal pull or traction on the retina by fibrous tissue in the vitreous. Treatment is vitrectomy, internal tamponade (by silicon oil, expanding gases etc.) and sealing the retinal tears with endolaser. 4. Solid RD occurs as a result of ocular tumours like malignant melanoma and exophytic retinoblastoma. Treatment is directed towards the tumuor primarily.

Retinoblastoma

It is a malignant ocular tumour of infancy usually presenting between 1 to 2 years of age, taking origin from primitive photoreceptor cells. More than half of the cases are sporadic; however, about 40 percent of cases of retinoblastoma are transmitted through the autosomal gene located at region 14 of the long arm of chromosome 13. Clinical presentation may be in the form of strabismus, leukocoria (Fig. 15.26), orbital

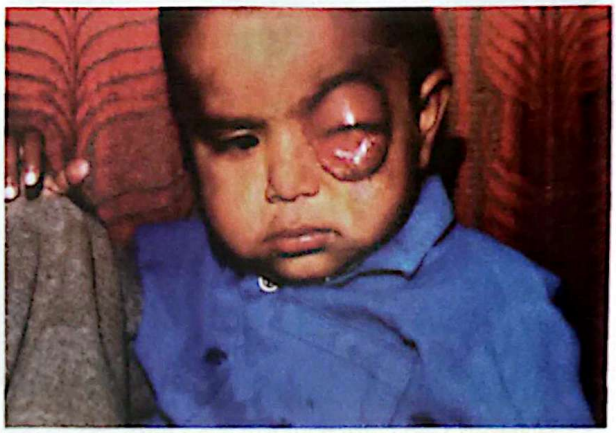


Fig. 15.27 Retinoblastoma-Left Eye

cellulitis (Fig. 15.27), proptosis (Fig. 15.29A), secondary glaucoma or uveitis, where signs of multifocal iris nodules and pseudohypopyon are seen. Clinically, when the tumour projects from the retina into the vitreous as a white mass it is called endophytic retinoblastoma and when the tumour grows in subretinal space causing retinal detachment, it is called exophytic retinoblastoma.

In addition to history and examination, investigations including B-scan ultrasonography and MRI or CT scan (Fig. 15.28) are helpful in diagnosis. In advanced cases, simply plain X-ray orbit for detecting calcification in the tumour may be helpful. Accurate diagnosis is established only after histopathology of tumour tissue. When the tumour is less than 4mm in diameter, laser photocoagulation or transpupillary thermotherapy is done for tumours posterior to the equator and cryotherapy is done for tumours anterior to the

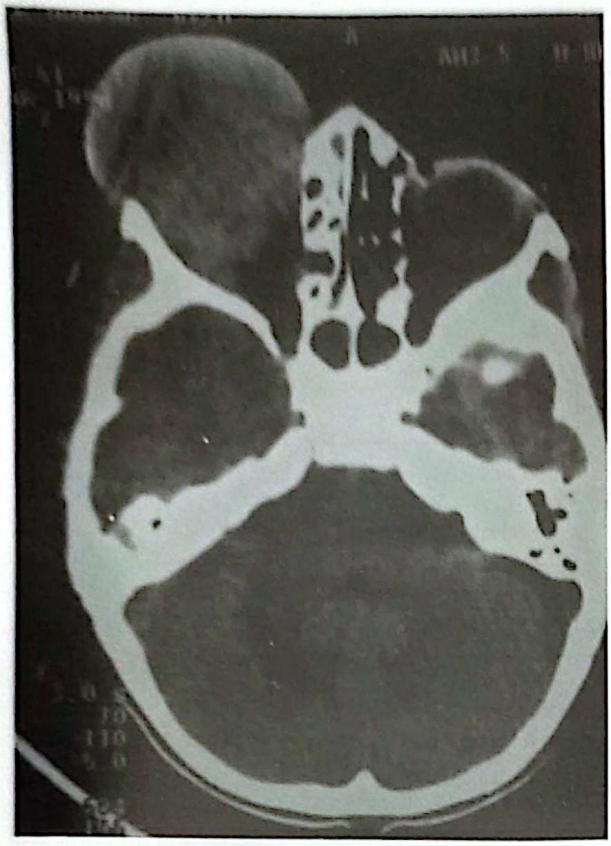


Fig. 15.28 T-scan of recurrent retinoblastoma

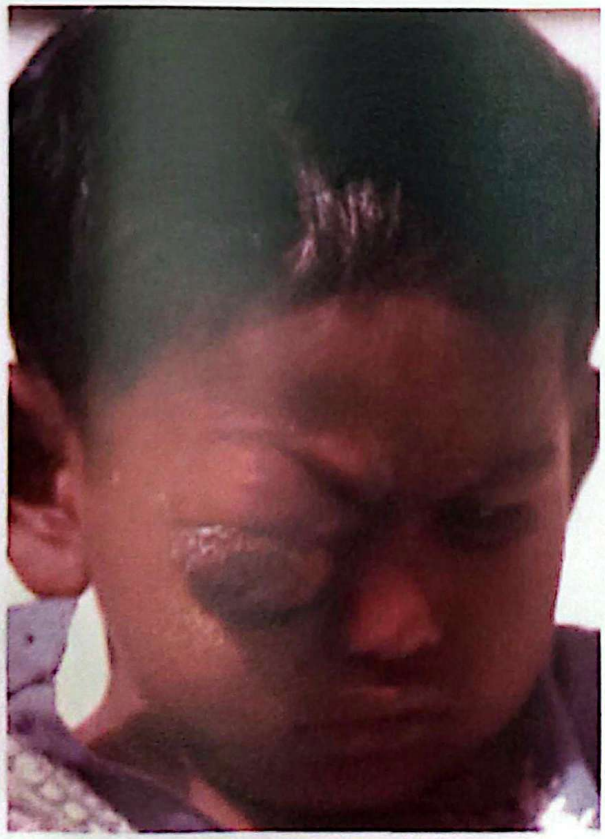


Fig. 15.29(A) Recurrent retinoblastoma

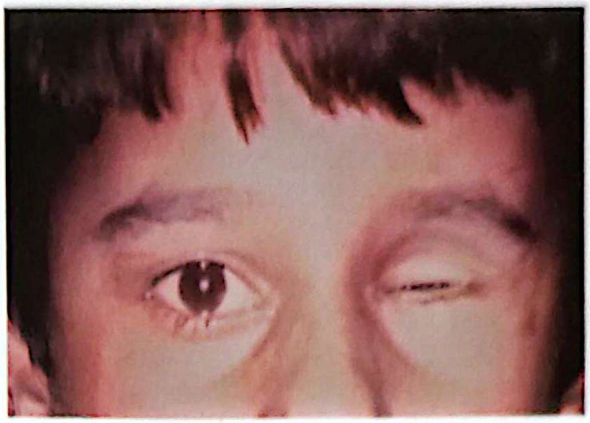


Fig. 15.29(B) Recovered case of Retinoblastoma after 12 years

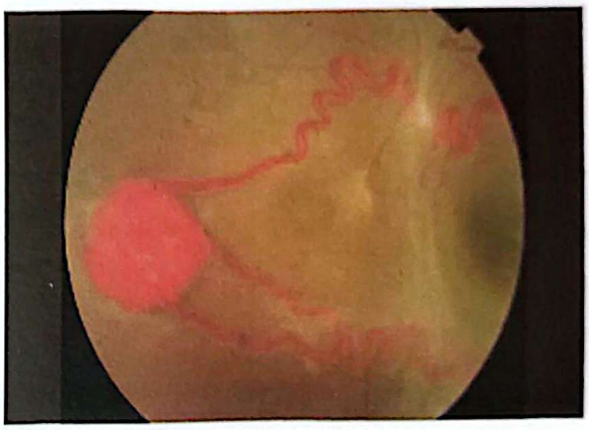


Fig. 15.29(C) Capillary haemangioma of the retina

equator. Treatment options for larger tumours[54] include brachytherapy, external beam radiotherapy, chemotherapy and enucleation of the affected eye with good results if the eyeball is intact and the optic nerve is not involved (Fig. 15.29 B).

Retinal vascular tumours

Vascular tumours of the retina are fortunately rare and occur as isolated tumours or associated with systemic disease. Retinal capillary haemangioma can occur as an isolated tumour (Fig. 15.29 C) or associated with von Hippel-Lindau (VHL) disease. Other retinal vascular tumours include cavernous haemangioma of the retina, retinal vasoproliferative tumour, and racemose haemangiomatosis of the retina or Wyburn–Mason syndrome.

Chapter 16

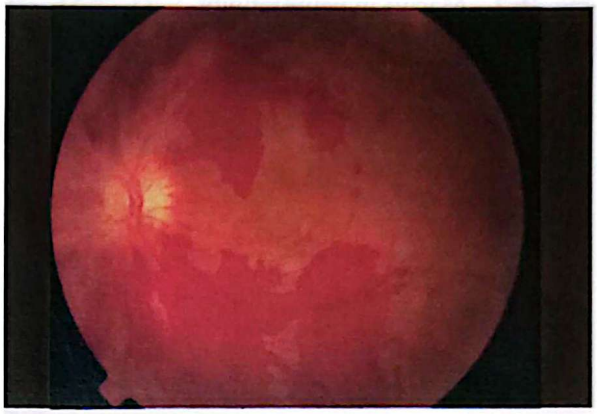
Diseases of Optic Nerve

Optic nerves are extensions of the CNS, thus, they are composed of second order neurons, myelinated with oligodendrogliocytes and covered by meningeal sheaths containing CSF in subarachnoid space but are erroneously labelled as the second pair of cranial nerves. In fact, the second pair of cranial nerves is represented by bipolar cells in the retina. The optic nerves passes from the eyeballs to the middle cranial fossa, by traveling through the orbits and optic canals, where they merge to form optic chiasma. The anatomical names given to various parts of the optic nerve are intraocular part (optic disc), intraorbital part, intracanalicular part and intracranial part.

COMMON CLINICAL FEATURES OF OPTIC NERVE DISEASE

Include loss of vision, colour vision impairment and pain on eye movements.

Inflammation of the optic nerve is seen clinically in three forms, retrobulbar neuritis (inflammation of the optic nerve posterior to the optic disc), papillitis (inflammation of optic disc (Fig. 16.1 A & B) and neuroretinitis (inflammation of the optic disc with involvement of the macula (Fig. 16.2). Severe visual loss is the main symptom of all varieties of optic nerve inflammations with additional symptom of pain on ocular movements in retrobulbar neuritis. Fundoscopy reveals disc hyperaemia in papillitis, disc hyperaemia with macular oedema and peripapillary haemorrhages in neuroretinitis and normal disc in retrobulbar neuritis. Viral infections are the common cause of papillitis and neuroretinitis. Demyelination as



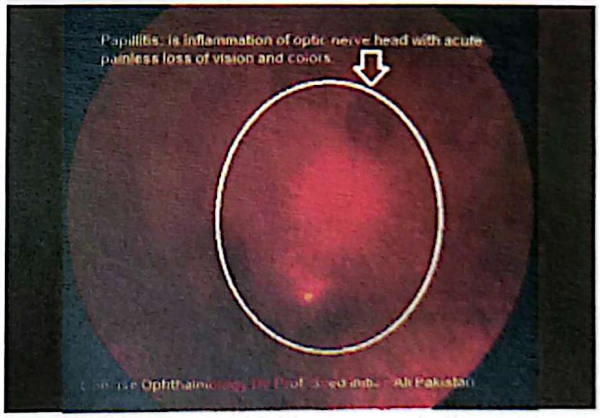


Fig. 16.1(A&B) Papillitis

in multiple sclerosis and drugs like ethambutol and isoniazid are among the common causes of retrobulbar neuritis. Treatment is not required in most cases as there is spontaneous recovery; however withdrawal of offending agents like ethambutol is required. Steroids may speed up recovery but their effect on final visual recovery is not proved.

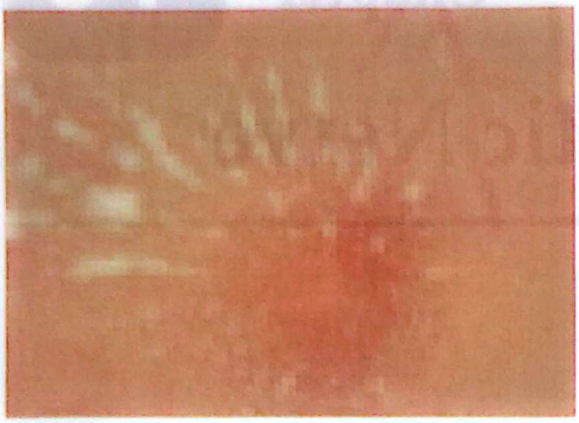


Fig. 16.2 Macula in neuroretinitis

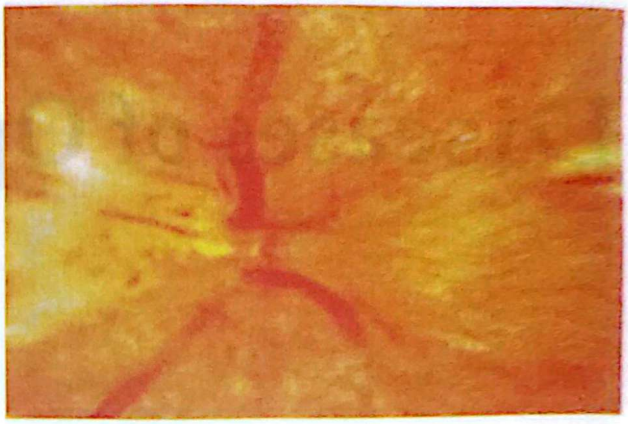


Fig. 16.4 Papilloedema (advanced stage)

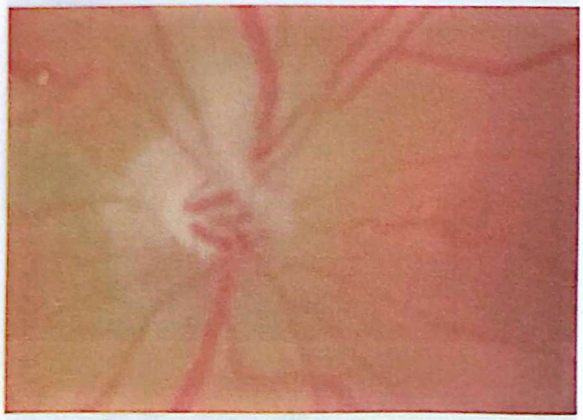


Fig. 16.3 Papilloedema (early stage)

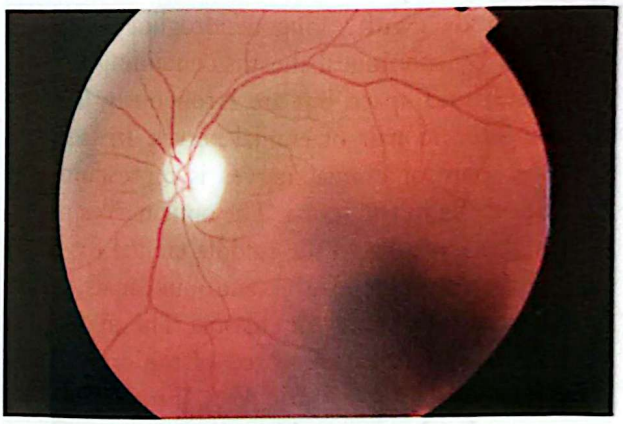


Fig. 16.5 Primary optic atrophy

Optic Neuritis

Anterior Ischaemic Optic Neuropathy (AION)

It is the necrosis of the optic disc due to occlusion of its blood supply from short posterior ciliary arteries which may result from arteritis (giant cell arteritis) called arteritic AION or arteriosclerosis (associated with hypertension) called non-arteritic AION. Clinical presentation is acute painless visual loss in one and rarely in both eyes in a middle aged or an old person. On examination, there is APD and associated features like jaw claudication, pain on combing hair, myalgia, headache in giant cell arteritis and hypertension or history of chain smoking in the non-arteritic form. Fundoscopy reveals a pale oedematous disc with few splinter haemorrhages on disc margins. Treatment includes administration of steroids in the arteritic form and control of hypertension with long term low dose aspirin to prevent future episodes of vascular occlusion in non-arteritic form. C-reactive protein level in the blood is raised in giant cell arteritis and should be done to differentiate between the two forms for initiating appropriate treatment.

Papilloedema

It is a bilateral passive optic disc oedema due to raised intracranial pressure resulting from space occupying lesions of the intracranial cavity. Papilloedema is not associated with visual loss unlike disc swelling due to inflammations which cause severe loss of vision. When there is obliteration of subarachnoid space around the optic nerves due to previous attack of meningitis, papilledema do not develop despite raised intracranial pressure. Other causes of papilloedema include malignant hypertension, blood dyscrasias, ocular hypotony and craniosynostosis. Pseudopapilloedema is seen

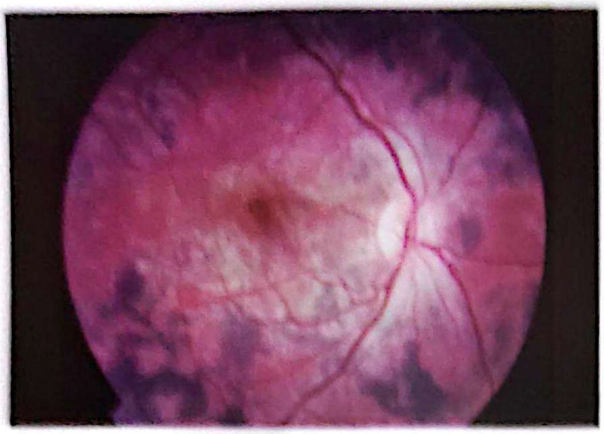


Fig. 16.6 Consecutive optic atrophy

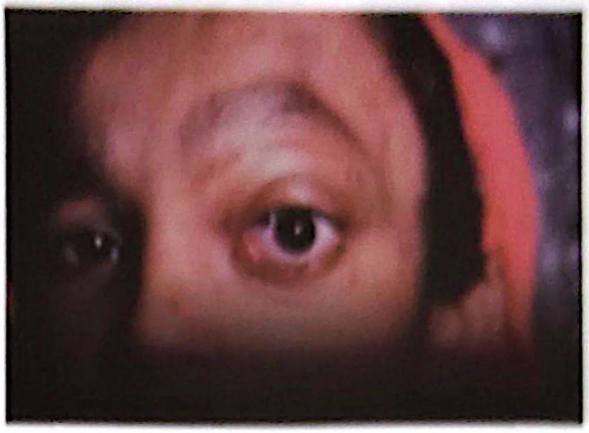


Fig. 16.7 Optic nerve glioma

in high hypermetropia, optic disc drusen and congenital disc anomaly. Treatment is directed towards the cause (Fig.16.3, 16.4).

Optic Atrophy

It is the end result of optic nerve insults characterised by a pale degenerated optic disc with loss of function. Four varieties are seen clinically.

Primary optic atrophy (Fig.16.5) is characterised by a white disc with distinct margins and normal appearing retina. It results from diseases which do not cause optic disc inflammation, e.g., drugs, alcohol, multiple sclerosis, trauma.

Secondary optic atrophy is characterised by a grey-white disc with blurred margins and retinal vascular sheathing. It results from papilloedema or papillitis.

Glaucomatous optic atrophy is characterised by evident optic disc cupping with nasal displacement of retinal vessels in the disc. It results from high intraocular pressure due to glaucoma.

Consecutive optic atrophy (Fig. 16.6) is characterised by a pale and waxy disc with distinct margins and associated retinal changes. It results from diseases of the retina, e.g., retinitis pigmentosa, central retinal artery occlusion, disseminated chorioretinal degenerations.

Optic Nerve Tumours

These include optic nerve glioma (Fig. 16.7) and meningioma. Treatment is surgical removal.

Chapter 17

The Pupil

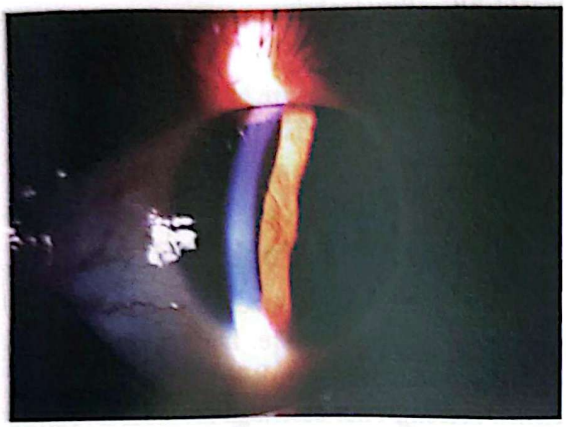


Fig. 17.1(A) Pseudoacoria with patches of iris atrophy

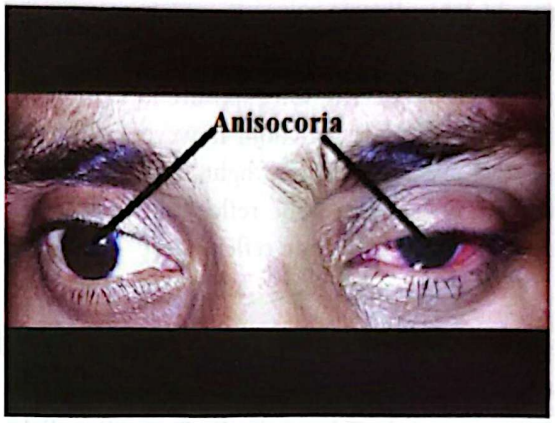


Fig. 17.1(B) Anisocoria

A reacting pupil is the sign of sight and life. Pupillary constriction is parasympathetic activity and pupillary dilation is sympathetic activity, therefore, when the parasympathetic system is alert, as in sleep, the pupils are constricted and when the sympathetic system is alert, as in a fight or flight, pupils are dilated.

COMMON CLINICAL FEATURES RELATED TO PUPILLARY ABNORMALITIES

These include amaurotic pupil, Marcus Gunn pupil, corectopia (ectopic pupil or pupil found other than central position), polycoria (more than two pupillary apertures seen in essential iris atrophy) or pseudo polycoria (Fig.17.2 A), pseudoacoria (absent pupil seen in pigmented pupillary membranes) (Fig.17.1 A), anisocoria (unequal pupils) (Fig.17.1 B) and irregular pupil (seen in anterior uveitis).

Pupillary Constriction

It is affected through four neurons: 1. Retina to the pretectal nucleus 2. Pretectal nucleus to the Edinger-Westphal nucleus 3. Edinger-Westphal nucleus to the ciliary ganglion 4. Ciliary ganglion to the sphincter pupillae.

Pupillary Dilation

It is effected through three neurons: 1. Posterior hypothalamus to the spinal cord (C8 to T2) 2. Spinal cord (C8 to T2) to superior cervical ganglion. 3. Superior cervical ganglion to dilator pupillae via carotid vessels, ophthalmic division of trigeminal nerve and long ciliary nerves.

Amaurotic Pupil or Afferent Pupillary Defect (APD)

It occurs in complete optic nerve dysfunction or atrophy. It is characterised by fairly dilated pupils if both eyes are affected but normal sized pupils

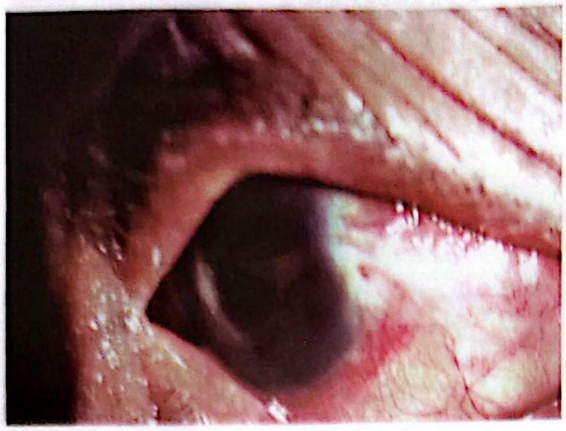


Fig. 17.2(A) Pseudo polycoria

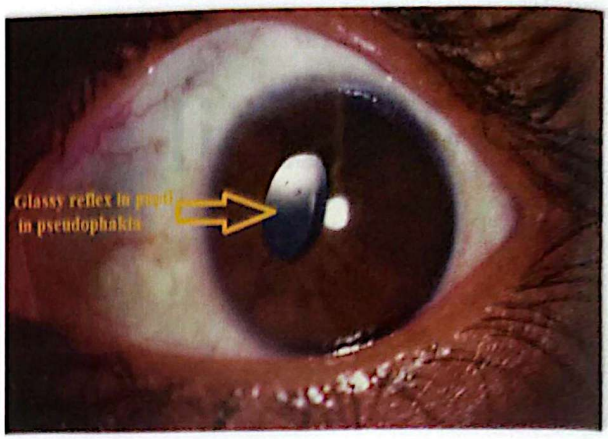


Fig. 17.2(B) Pseudophakia

if one eye is affected. On exposure to light, there is no pupillary constriction; however, when the normal eye is exposed to light, amaurotic pupil constricts (indirect light reflex) in addition to retained accommodation reflex.

Marcus Gunn Pupil or Relative Afferent Pupillary Defect (RAPD)

It is a partial disturbance in direct pupillary light reflex and occurs in partial dysfunction of the optic nerve or retina. It is a clinically significant test in diagnosis of the optic nerve or retinal disorders before permanent damage sets in. The swinging light reflex test is the best way of detecting RAPD.

Haemianopic Pupil

It is seen in hemianopia and is detected in straight gaze. Pupillary reaction is sluggish when exposed to light from the side of hemianopia.

Hippus

It is a rhythmic constriction and dilation of pupil when constantly exposed to light and is seen during prolonged slit lamp examination. It is a normal phenomenon.

Adie Pupil

It is a slow reacting pupil, i.e., there is obvious slow pupil constriction on accommodation and light exposure followed by slow dilation. It results from a wide variety of disorders associated with parasympathetic denervation of the pupil. When 0.25% pilocarpine drops are instilled, the affected pupil constricts more than the normal pupil due to denervation hypersensitivity.

Argyll Robertson Pupil (ARP)

It constricts on accommodation but fails to constrict on light exposure. During my student days I used to remember ARP as Accommodation Reflex Present. First described for neurosyphilis, ARP was later seen in a variety of conditions including alcohol addiction, insulin dependent diabetes mellitus, encephalitis etc.

Glassy pupil

Brilliant glassy reflex from the pupil is seen in cases of pseudophakia

Chapter 18

Ocular Motility Disorders

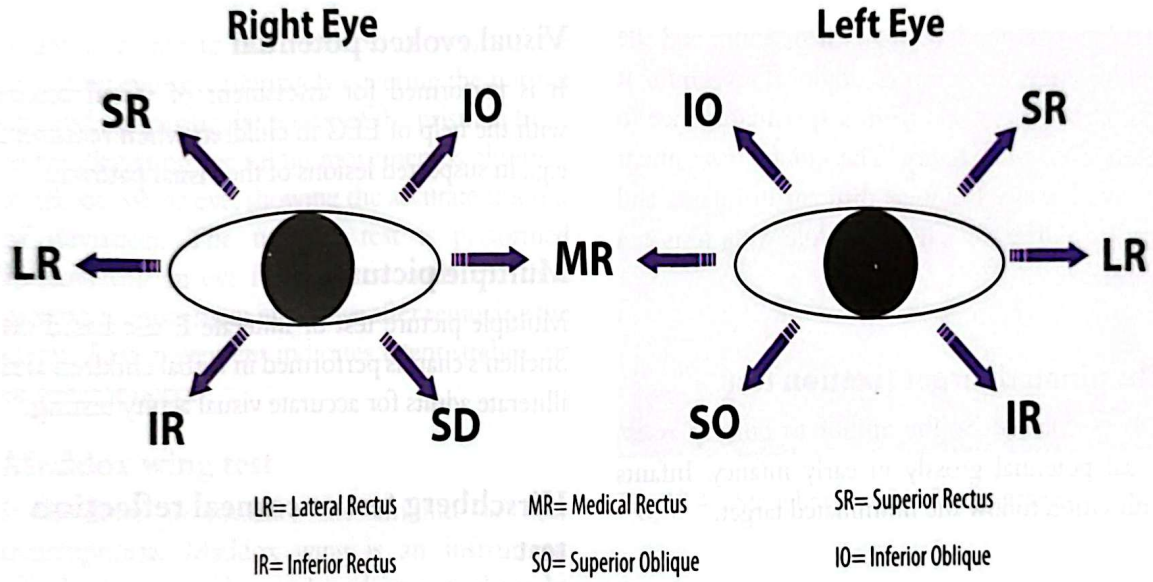


Fig. 18.1 Primary action of extra ocular muscles

Six extraocular muscles including four recti and two oblique muscles for each eye are responsible for ocular motility. Primary action of each extraocular muscle is shown alone.

COMMON CLINICAL FEATURES OF MOTILITY DISORDERS

These include binocular diplopia, confusion in viewing objects, difficulty in going downstairs and deviation of eye from a normal position.

Visual Axis

It is an imaginary line which connects an object in view with the fovea of both eyes in a normal state of vision. When visual axes of two eyes do not intersect at an object in view (fixation point), a condition of diplopia is produced and this misalignment leading to a state of unparallel eyes is called Strabismus.

Visual Acuity

It is the ability to locate and distinguish components of a target and identify it as a whole, e.g., in **Snellen's** visual acuity **chart** components

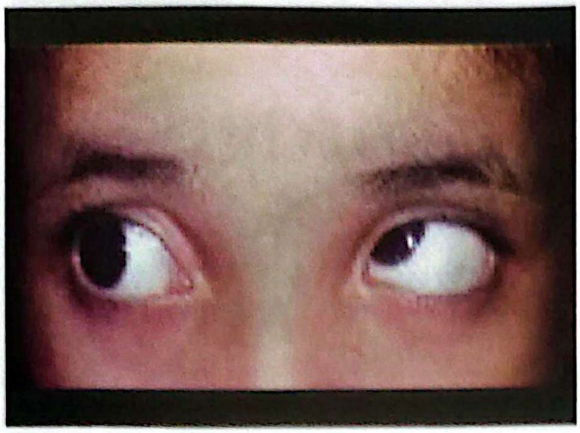


Fig. 18.1(A) Right superior rectus palsy

Fig. 18.1(B) Imtiaz zigzag pattern test is being performed

of a letter subtend an angle of one min and the whole letters subtend an angle of five min. it should be recognised from a specific distance to assess the visual acuity. The clinical assessment of visual acuity becomes difficult in infants and small children for whom the following tests can be performed.

Illuminated target fixation test

It is performed by the author to quickly assess visual potential grossly in early infancy. Infants with vision follow the illuminated target.

Preferential fixation

It is utilised for gross assessment of visual acuity. Imtiaz zigzag pattern test (Fig. 18.1 B) is performed with the help of zigzag black and white pattern developed by the author. Interest shown by the infant indicates state of visual acuity, e.g., if the infant moves his eyes only with the pattern, it indicates fair visual acuity. If the infant moves his head in addition to eyes after the pattern is moved to one side, it indicates fairly good visual acuity. When the infant tries to move his body in addition to his eyes and head, it indicates good visual acuity.

Hundreds and thousand's sweet test

It is performed on a child with the help of small coloured sweets. If he picks up the appropriate sweets on command, this indicates moderately good visual acuity.

Visual evoked potential

It is performed for assessment of visual acuity with the help of EEG in children when required, e.g., in suspected lesions of the visual pathway.

Multiple picture test

Multiple picture test or illiterate E test based on Snellen's chart is performed in verbal children and illiterate adults for accurate visual acuity testing.

Hirschberg test or corneal reflection test

It is performed to detect the deviation grossly and to exclude cases of pseudostrabismus. When a person is asked to fixate on a light, the corneal reflection of that light should be in the centre of each cornea normally. Any displacement of this corneal light reflection from the centre of the cornea indicates strabismus, e.g., when reflex is displaced medially on the cornea it indicates exotropia and when laterally, it indicates esotropia (Fig. 18.2).

Cover-Uncover test

This test is performed for near and far to look for heterophorias (latent or temporary deviations) and to measure heterotropias (permanent deviations). The cover test is performed for the deviating eye by covering the normal eye and then observing the movement of the uncovered deviating eye. Measurement of deviation is done with a prism bar (prism cover test) by placing the base of the

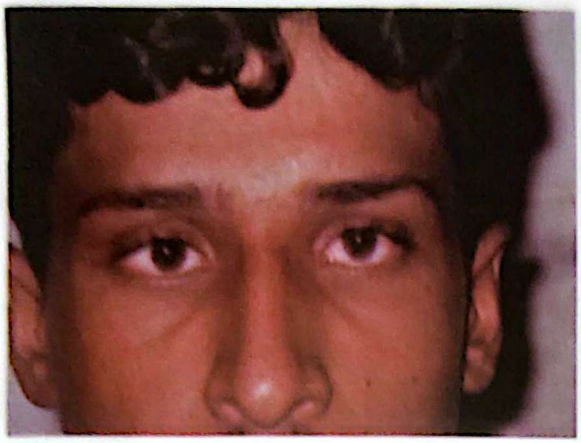


Fig. 18.2 Hirschberg test or corneal reflection test indicates right esotropia

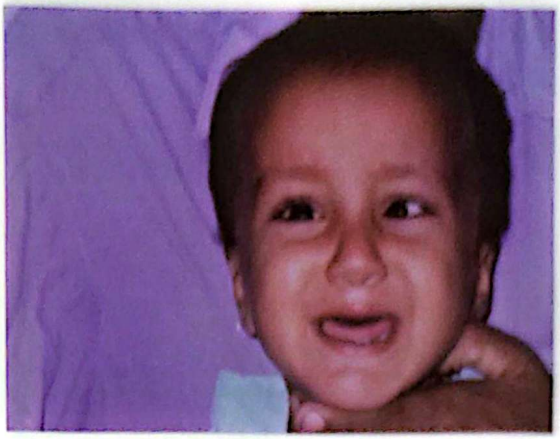


Fig. 18.3 Esophoria detected by chance during clinical examination

prism opposite to the deviation in front of the deviating eye and alternately covering the normal eye and increasing the power of the prism in front of the deviating eye till no movement is observed in the deviating eye, showing the accurate amount of deviation. The uncover test is performed by covering an eye for a few seconds and then observing movement in this eye after removing the cover. Any movement indicates latent strabismus or heterophoria.

Maddox wing test

It is done to measure the amount of near heterophoria. Maddox wing is an instrument which presents a white and a red arrow to the right eye and a cross of numbers to the left eye.

Maddox rod test

It is done to measure heterophoria for distance. A Maddox rod is available in all trial sets of glasses.

Amblyopia

It is a term given to unilateral or bilateral decrease in visual acuity that can not be explained on the basis of any organic lesion. It results from visual deprivation of any type, e.g., congenital cataract, prolonged unilateral cycloplaegia as with atropine, uncorrected refractive error, during period of visual immaturity, i.e., from birth to 5 years. Treatment is elimination of the cause at the earliest within the first 5 years of life and antiamblyopia therapy before the first nine years of life which include intermittent eye patching and fusion exercises with an amblyoscope.

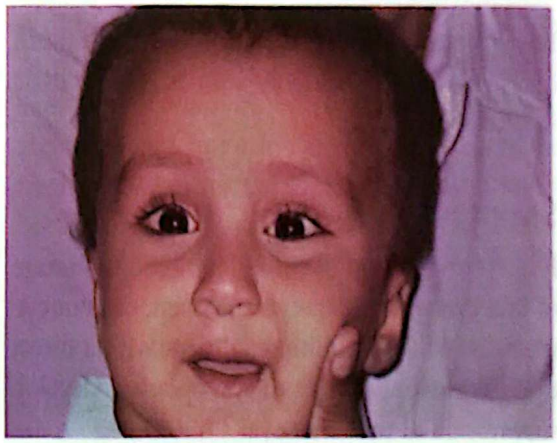


Fig. 18.4 Straight eyes after few moments

Fusion

It is the ability of occipital cortex to integrate two similar images from both eyes into one image (sensory fusion) and ability of the two eyes to remain aligned (motor fusion).

Orthophoria

It is a normal state of the eyes where eyes remain parallel for far vision without requiring fusion and retain normal convergence for near.

Heterophoria

It is the deviation of the eye which is prevented by motor fusion. When motor fusion is disrupted, e.g., covering one eye, heterophoria becomes apparent (Fig. 18.3, 18.4).

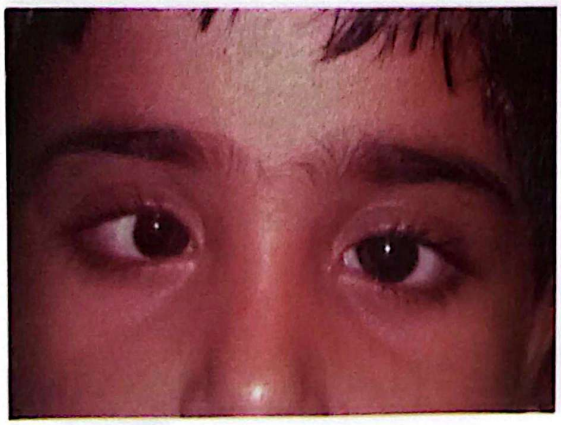


Fig. 18.5 Esotropia before surgery

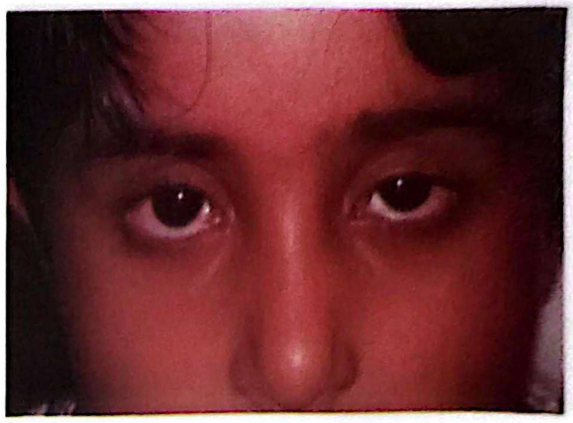


Fig. 18.6 After strabismus surgery

Concomitance

It is a condition of equal deviation in all positions of gaze and is commonly seen in nonparalytic squints.

Suppression

It is a phenomenon of cortical inhibition of image of one eye when both eyes are open, e.g., an eye specialist while examining a patient with a direct ophthalmoscope with both eyes open will develop suppression of the other eye.

Paralytic or Nonconcomitant Strabismus

It results from paresis (weakness) or paralysis of one or more extraocular muscles in which the movement of the globe is restricted in the direction of paralysed muscle. It results from tumours, aneurysms, head trauma, multiple sclerosis, neuropathies, thyroid myopathy, myasthenia gravis etc. Treatment is directed towards the cause followed by muscle strengthening operations when required.

Nonparalytic or Concomitant Strabismus

It is characterised by the presence of deviation with normally functioning extraocular muscles and the same angle of deviation in all positions of gaze. It is evident in three forms **1. Nonparalytic esotropia 2. Nonparalytic exotropia. and, 3. A and V deviations**

Nonparalytic esotropia

It is accommodative, non-accommodative, mixed and with sensory deprivation.

Accommodative esotropia is a familial condition which becomes apparent around the first two years of life with hypermetropia of +4 to +6 dioptres. The child's eye becomes straight with corrective glasses. Treatment is full refractive correction, near addition in form of bifocal glasses when esotropia for near is detected and miotic drops in patients who do not wear glasses.

Nonaccommodative esotropia

It is a familial condition which becomes apparent at birth. Refractive correction has no effect on the amount of deviation. Treatment is occlusion therapy and surgical correction of deviation after excluding organic causes of sensory deprivation.

Mixed accommodative and non-accommodative esotropia

It is a familial condition with both elements present. The accommodative element is corrected with glasses and the nonaccommodative element is corrected by strabismus surgery (Fig.18.5, 18.6).

Esotropia associated with sensory deprivation

Esotropia associated with sensory deprivation due to cataract, corneal opacity, persistent hyperplastic primary vitreous, retinal or optic nerve disease or anisometropia is obvious when the cause is

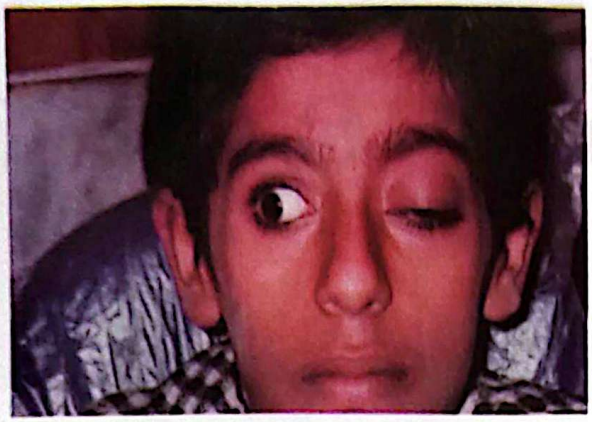


Fig. 18.7 Left oculomotor nerve palsy

established. It is treated by treating the cause, e.g., cataract extraction with IOL implantation.

Nonparalytic exotropia

It is familial, accommodative and acquired.

Familial exotropia

Presents as intermittent exotropia in school children followed by constant exotropia. Treatment is surgical correction of exotropia.

Accommodative exotropia

Occurs in myopes who do not use glasses. Treatment is refractive correction.

Acquired exotropia

Occurs due to defective vision in one eye. Blind eyes or eyes with defective vision tend to develop exotropia after 15 years of age as a rule. Treatment is surgery.

A and V deviations

A and V deviations are seen when deviation is more in upgaze or downgaze due to extraocular muscle underaction or overaction. Treatment is surgical correction.

Microtropia

It is a small degree strabismus less than 8 prism dioptres not associated with diplopia due to central suppression scotoma.

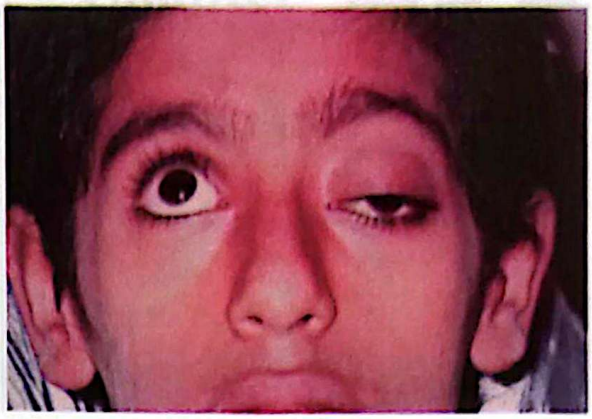


Fig. 18.8(A) Left oculomotor nerve palsy

Oculomotor Nerve Palsy

It is seen frequently in a clinical setting. In complete third nerve palsy, ophthalmoplegia is extensive, leaving behind only two actions, i.e., abduction and intorsion on downgaze effected by the lateral rectus and superior oblique muscles because all extraocular muscles except the lateral rectus and superior oblique are supplied by the oculomotor nerve. Aneurysm of the posterior communicating artery, diabetes mellitus, trauma, hypertension, tumours and vasculitis are among the causes of third nerve palsy. The third nerve derives its blood supply from vasa nervosa which come from adjacent internal carotid and ophthalmic arteries except pupillomotor parasympathetic fibres which run in the trunk of the oculomotor nerve but derive their blood supply from blood vessels of pia mater. Therefore, posterior communicating artery aneurysm, head trauma with intracranial haematoma and intracranial tumours cause third nerve palsy with fixed pupil due to involvement of pial vessels, whereas microangiopathy of vasa nervosa due to diabetes and hypertension lead to infarction of the trunk of the oculomotor nerve sparing pupillomotor fibres leading to oculomotor palsy with sparing of pupillary reflex, a very valuable sign in clinical setting for differentiating surgical from medical lesions (Fig. 18.7, 18.8 A).

Abducent Palsy

It leads to restriction of abduction of the globe because the sixth nerve supplies the lateral rectus muscle. Sixth nerve palsy is frequently

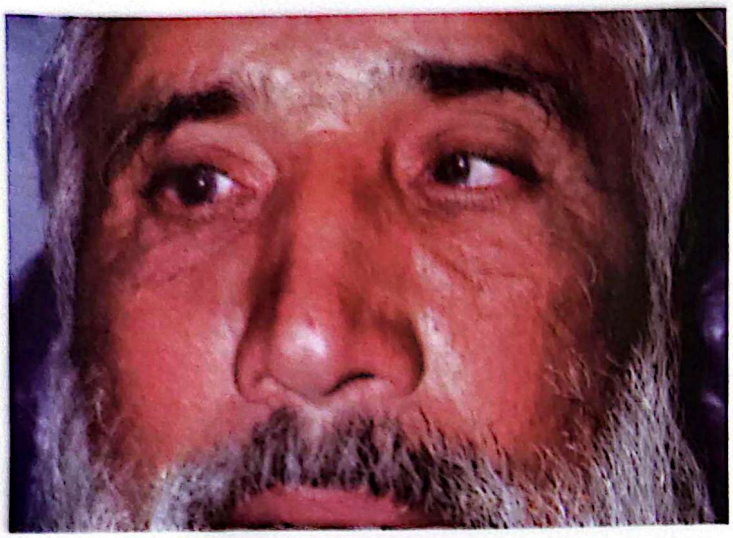


Fig. 18.8(B) Right abducent palsy

seen in ophthalmic practice due to the diplopia it produces on lateral gaze and due to its long intracranial course; thereby its implication in a wide variety of intracranial lesions ranging from pseudotumour to acoustic neuroma, nasopharyngeal tumours and cavernous sinus lesions. Diabetes mellitus and hypertension are among common medical causes of sixth nerve palsy. In recent sixth nerve palsy the affected eye is esotropic because of overaction of medial rectus whereas in old sixth nerve palsy, the eye is straight due to contracture of the medial rectus. To exclude acoustic neuroma as a cause of sixth nerve palsy clinically, corneal sensitivity and hearing must be checked (Fig. 18.8B)

Chapter 19

Neuro-Ophthalmology

MYASTHENIA GRAVIS

Myasthenia Gravis (Fig. 19.1) is an autoimmune disease in which there are autoantibodies against the postsynaptic acetylcholine receptors at the neuromuscular junction. It commonly affects females between the ages of 20 to 40 years and is characterised by excessive fatigability of the skeletal muscles.

Ocular features

This disease starts in the eye lid and extraocular muscles as they are the fine muscles and then it may spread to become generalised. The ocular features are:

1. Ptosis

It is bilateral, may be asymmetrical and worse towards the end of the day when the patient is tired.

2. Diplopia

It is variable; may be present in any direction but may start in upward movement first.

Investigations

Tensilon Test

This is used to confirm the diagnosis of myasthenia gravis. There is improvement in weakness of the muscles following the intravenous injection of edrophonium (Tensilon) (Fig. 19.2) which is a fast acting anticholinesterase.

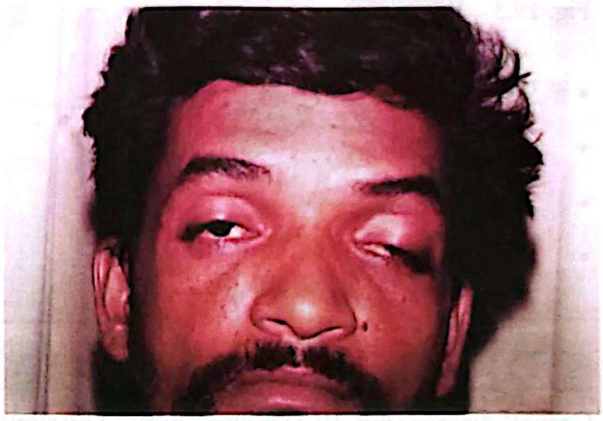


Fig. 19.1 Before the test

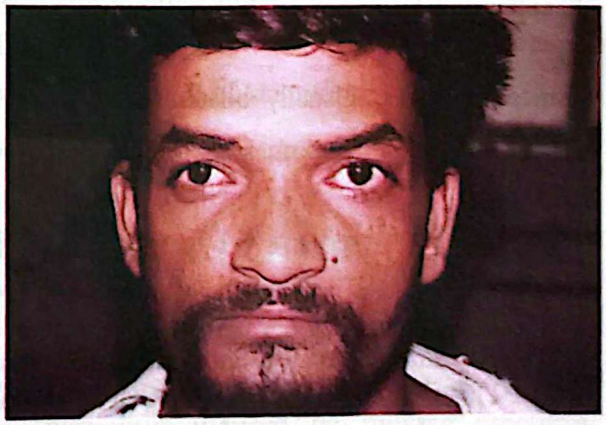


Fig. 19.2 After the test

Electromyography

Antibodies

Anti-acetylcholine receptor antibodies are present in about 90% of patients with myasthenia gravis.

Treatment

- Pyridostigmine(Mestinon) - this is a long acting anticholinesterase drug

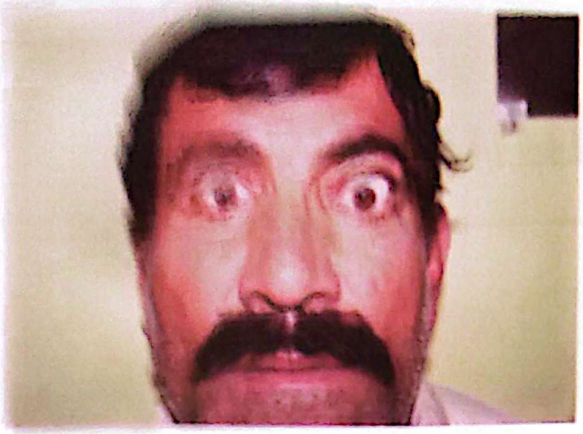


Fig. 19.3 Bilateral disease

Fig. 19.4 Unilateral disease

- Systemic corticosteroids
- Cytotoxic drugs
- Thymectomy
- Plasmapheresis

THYROID OPHTHALMOPATHY

Graves' disease (Fig. 19.3) is characterised by signs of hyperthyroidism and eye signs. Ophthalmic Graves' disease or euthyroid eye disease is characterized by eye signs but without signs of hyperthyroidism.

Clinical Features: Usually bilateral but can be unilateral (Fig. 19.4).

Eye Lid Signs

Lid retraction

The upper lid is elevated and sclera becomes visible. This is due to Muller's muscle over-action due to sympathetic over-stimulation.

Lid lag

There is a slow descent of the upper lid while moving the eyeball from up-gaze to down-gaze.

Presentation includes staring and frightened appearance of the eyes, fine tremors of closed lids and infrequent blinking.

Proptosis

Proptosis is due to proliferation of orbital fat and connective tissue.

Myopathy

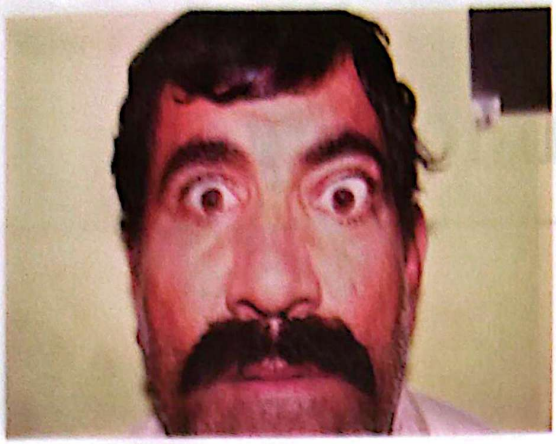
Enlargement of extraocular muscles in the early stages and fibrosis in later stages leads to the symptom of diplopia.

Complications

- Optic nerve compression may cause blindness.
- Exposure keratitis
- Dry eyes

Treatment

- Mild cases only require reassurance and regular follow up visits.
- Artificial tears to lubricate the eye
- Systemic corticosteroids in case of optic nerve compression and acute chemosis and proptosis
- Radiotherapy, if not responding to systemic corticosteroids
- Surgery for orbital decompression, extraocular muscle imbalance and blephroplasty



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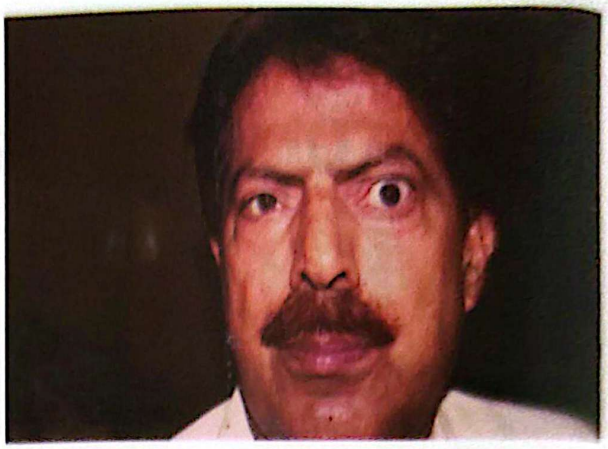

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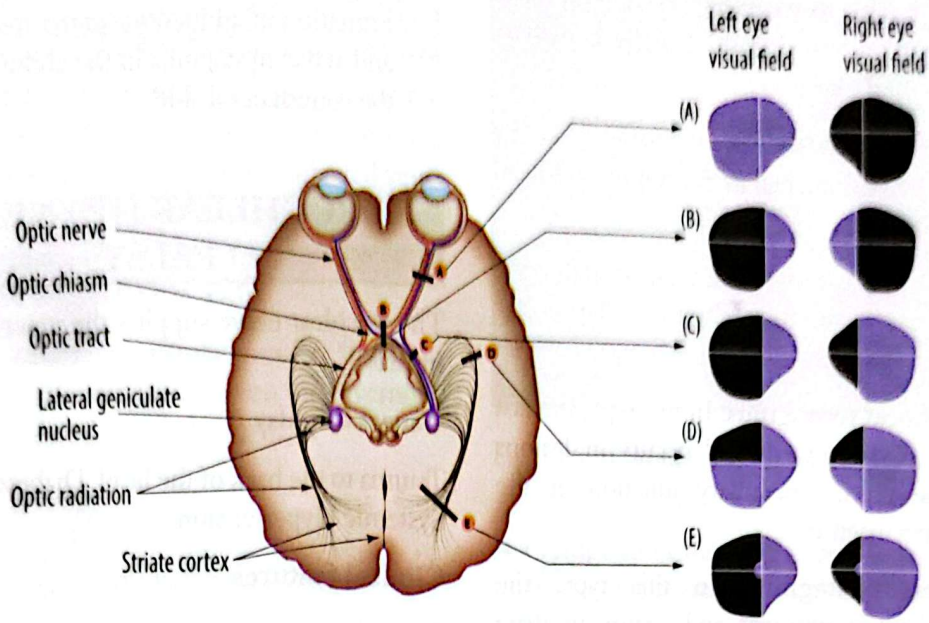

Fig. 19.5 Visual Pathway lesions

Visual Pathway (Fig. 19.5) Consists Of the:

- Optic Nerve
- Optic Chiasma.
- Optic Tract
- Optic Radiations
- Visual Cortex

Visual Pathway lesions

- Optic Nerve lesion - Uniocular loss of vision

  Causes - Optic neuritis, Ischaemic optic neuropathy
- Optic Chiasma lesion - Bitemporal hemianopia

  Cause - Pituitary Tumour
- Optic Tract lesion - Contralateral Homonymous hemianopia

  Cause – Stroke (CVA)
- Optic Radiation lesion - Contralateral Homonymous quadrantanopia

  Cause - Stroke (CVA).
- Visual Cortex lesion-Contralateral Homonymous hemianopia with macular sparing

  Cause - Stroke (CVA)

NYSTAGMUS

Nystagmus is the rhythmic involuntary to-and-fro oscillation of the eyes. It is an important sign indicating central nervous system disease. It can be jerky, pendular or mixed nystagmus.

Clinical types

- Physiological Nystagmus
- Pathological Nystagmus

Physiological Nystagmus

- **End point nystagmus:** Fine jerky nystagmus in the extreme gaze positions
- **Optokinetic nystagmus:** It is a jerk nystagmus induced by moving repetitive stimuli across the visual field
- **Vestibular nystagmus:** It is jerky nystagmus caused by altered input from vestibular nuclei to horizontal gaze centers. Cold or hot water in the ear will induce vestibular nystagmus.

Pathological Nystagmus

- **Ocular diseases:** Congenital cataracts, macular hypoplasia, albinism and Leber's congenital amaurosis

- **Congenital nystagmus:** Present at birth without any eye disease. Usually hereditary and the jerky type
- **Spasmus nutans:** This is characterised by nystagmus, abnormal head position and head nodding
- **Ataxic nystagmus:** It occurs in an abducting eye in association with internuclear ophthalmoplegia
- **Downbeat nystagmus:** In this type, the fast phase is downwards and occurs in lesions of the cervico-medullary junction at the foramen magnum
- **Upbeat nystagmus:** In this type, the fast phase is upwards and occurs in drug intoxication and posterior fossa lesions
- **See-saw nystagmus:** One eye rising and intorting while the other eye falls and extorts. It occurs in bitemporal hemianopia in chiasmal lesions

GAZE PALSIES

- **Gaze** is the binocular movement of eyes. It can be horizontal gaze or vertical gaze
- **Saccades:** This is binocular movement to move the eyes from one object to another
- **Pursuit:** This is binocular movement to maintain fixation on the target. **Example:** A bird is flying; the watching person first locates the bird with saccades and then follows it with pursuit
- **Frontal gaze palsy:** Frontal supranuclear gaze palsy is usually seen after acute frontal insult such as frontal haematoma or infarct or trauma. The eyes are deviated towards the side of the lesion
- **Bilateral Pontine gaze palsy:** This occurs in infarcts, haemorrhages and demyelination. There is ipsilateral loss of conjugate gaze to saccades, pursuit, oculo-kinetic, vestibule-ocular and caloric testing
- **Internuclear ophthalmoplegia (INO):** The lesion is ischemia or demyelination in the medial longitudinal fasciculus. There is limitation of adduction on the ipsilateral side and ataxic nystagmus in the abducting eye on the contralateral side

TROCHLEAR (4TH) NERVE PALSY

The trochlear nerve supplies the superior oblique muscle.

Causes of palsy

Trauma to the back of the head, Diabetes Mellitus, Systemic Hypertension.

Clinical features

Diplopia is worse during looking down, particularly when walking downstairs and reading.

Hypertropia (upward squint)

Treatment

Control diabetes and hypertension.

Palsy usually recovers within three months.

Migraine

Paroxysmal attacks of unilateral headache with visual disturbances and vomiting. Commonly occurs in young to middle aged females with a family history. Usually begins at puberty and continues intermittently to middle age. The precipitating factors could be some foods like chocolate and cheese, emotions, oral contraceptives and menstruation.

The characteristic attack starts with a sense of ill health and is followed by visual aura (shimmering lights, scotomas) usually in the field opposite to the side of the succeeding headache. Then the throbbing unilateral headache starts and is associated with anorexia, nausea, vomiting, photophobia and then withdrawal. There may be transient hemiparesis or sensory symptoms.

Treatment

- Precipitating factors may be removed
- Simple analgesics and antiemetics
- Prophylactically beta blockers may be given

CARATICO-CAVERNOUS FISTULA

An arteriovenous fistula is an abnormal communication between previously normal veins and arteries. The blood in the affected veins becomes arterialized; the intravenous pressure rises and venous blood flow may be altered in both rate and direction (Fig. 19.6).

A carotid cavernous fistula results from an abnormal communication between the cavernous sinus and the carotid arterial system.

Causes

Trauma - fracture of the base of the skull

Spontaneous - rupture of either an intracavernous aneurysm or of an atherosclerotic internal carotid artery in a hypertensive individual.

Clinical features

- Chemosis, redness and dilatation of episcleral blood vessels
- Proptosis-typically pulsatile, associated with thrill and bruit
- Ophthalmoplegia commonly with sixth nerve palsy
- Raised intraocular pressure

Treatment

- Some fistulas close spontaneously
- Balloon catheter embolisation is done in persistent fistulas

OCULAR MANIFESTATIONS OF VASCULAR DISEASE

Amaurosis Fugax

This includes transient painless loss of vision lasting a few minutes and recovering rapidly without residual deficit. The loss of vision is often described as a curtain coming downwards or upwards over one eye. Uniocular amaurosis fugax

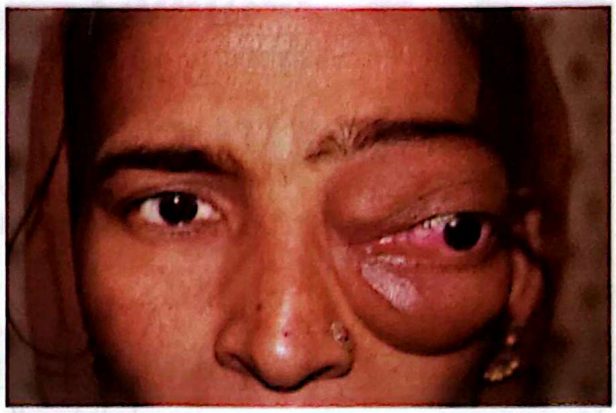

Fig. 19.6 Carotico-Cavernous Fistula

is a symptom of transient ocular ischemia which may affect the retina, the choroid or both.

Bilateral amaurosis fugax happens in cerebral ischaemia, involving the visual pathway.

- Causes
- Emboli from carotid atheroma
- Systemic Hypertension
- Diabetes Mellitus
- Polycythaemia

Treatment

- Low dosage aspirin as antithrombotic agent 300 mg/day
- Treat the cause

OCULAR MANIFESTATIONS OF BRAIN TUMOURS

Ocular manifestations of brain tumours depend upon the site of the tumours in the brain.

Pituitary Adenoma

It typically presents with bitemporal hemianopia. The patient finds difficulty in seeing from the sides.

Acoustic Neuroma

This is also called cerebello-pontine angle tumour. It is a neurofibroma of the acoustic (8th) cranial

nerve. It involves the 5th, 6th, and 7th cranial nerves and causes paralysis of these nerves.

Subfrontal Tumor

This tumour presents with Foster-Kennedy syndrome. There is optic atrophy on one side and papilloedema on the other side.

MENINGIOMA

Meningiomas arise from meningothelial cells of arachnoid at multiple intracranial sites, in the optic canal and optic nerve sheath. Meningiomas typically occur in females in the forties.

Optic nerve sheath meningioma presents with proptosis and loss of vision. Meningioma of the middle cranial fossa may cause papilloedema with raised intracranial pressure.

DIPLOPIA

Seeing two images of a single object.

Binocular Diplopia

Diplopia present when both eyes are open.

Causes

- Paralysis of cranial nerves 3rd, 4th and 6th.

This type of double vision occurs when the image of an object does not fall on the corresponding points of the retina in both eyes, e.g., the image of an object falls on the macula in the right eye, and outside macula in the left eye.

Uniocular Diplopia

Diplopia present when only one eye is open.

Causes

Major causes includes incipient cataract, lens subluxation, mild corneal opacity.

This occurs when more than one image of the same object fall on the different parts of the retina in one eye. There may be Diplopia (two images), Triplopia (three images) or Polyopia (many images).

Chapter 20

Diseases of the Lacrimal System

The lacrimal system is responsible for keeping the eyeballs wet and thus perform a crucial function. A fish can not survive outside water similarly survival of the globe is not possible without a wetting system. The lacrimal wetting system is made up of the secretory portion and drainage portion. The **secretory portion** is made up of **reflex and basic secretors,** which secrete around 2.2 ul of lacrimal fluid per minute out of which 0.85 ul/min evaporates in standard conditions of temperature and humidity and the rest is drained by the lacrimal drainage system into the nose. The drainage portion has two components, an active component and a passive component. Total capacity of the lacrimal drainage system is around 100 ul/min, therefore during lacrimation the level of tear production must exceed 100 ul/min before actual tearing over the cheeks starts. The **Reflex secretor** is a lacrimal gland with its bigger orbital portion and smaller palpebral portion, and secretes aqueous fluid on demand, providing both aqueous and mucus components. The conjunctival basic secretors are responsible for constant wetting of the eye, therefore they seem to have more importance in terms of basic secretion. Dacryoadenectomy (surgical removal of the lacrimal gland) will not produce a dry eye, whereas damage to the basic secretors end up in a dry eye. Basic secretors form the continuous three layered tear film. The deeper mucin layer, which keeps the tear film attached with the corneal epithelium, is maintained by mucin secretors which include goblet cells, crypts of Henle and glands of Manz. The middle aqueous layer, which forms the bulk of the tear film, is maintained by the glands of Wolfring and Krause. The superficial oily layer, which prevents rapid evaporation of tears, is maintained by the tarsal glands, glands of Zeis and glands of Moll. **Lacrimal drainage portion's** active component comprises of muscular action of the lids which close the lid margin from the lateral to the medial side, thereby pumping the lacrimal fluid into the puncta with every blink, an action called **zipper action**. The passive draining component is made up of the upper and lower canaliculi, common canaliculus, lacrimal sac and nasolacrimal duct which passes through the bony nasolacrimal canal and opens into the nose through the inferior meatus. The wetting of the corneal surface is achieved by way of constant maintenance of the tear film through the blinking reflex which is triggered by formation of dry spot on the corneal surface. Therefore, the blinking rate depends upon the time of dry spot formation. More viscous tear film leads to delayed dry spot formation and therefore lesser rate of blinking as in Lions the blink rate is one blink per minute. Tear film of monkeys is less viscous so their blink rate is 44 blinks per minute. Humans possess moderately viscous tear film so the rate is 12 to 15 blinks per minute.

COMMON CLINICAL FEATURES OF LACRIMAL DISEASE

These include lacrimation (overproduction of tears), epiphora (drainage obstruction), foreign body sensation (dry eye), upper lid swelling (lacrimal gland inflammation or tumor) and discharge over the medial canthus (dacryocystitis).

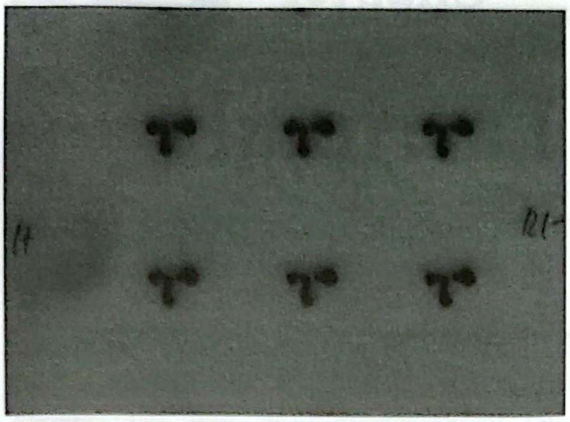

Fig. 20.1 Dacryoscintography showing Right blocked nasolacrimal duct and Left patent nasolacrimal duct

Fig. 20.2 Dacryoscintigraphy is in progress

Investigations for Patency of the Lacrimal Drainage System

Routine investigations performed for evaluation of lacrimal passages are as under.

Chloramphenicol test

It is a simple test of patency performed by the author. A drop of chloramphenicol eye solution is instilled in the conjunctival sac of the affected side and normally the patient experiences bitter taste in the throat in a few minutes indicating passage of the solution into the throat and patent lacrimal drainage system.

Fluorescein test

It is performed with fluorescein eye drops 2% or with a fluorescein strip. Normally, fluorescein instilled into the conjunctival sac disappears from the conjunctival sac in a minute and stains the swab placed in the corresponding nostril indicating patent lacrimal passage. If the dye does not appear in the nose, syringing is done, if the dye then appears in the nose, it indicates abnormality in an active component, e.g., punctal ectropion or atresia. If the solution does not pass in the nose on syringing, it indicates passive draining component blockage, e.g., canalicular or nasolacrimal duct block.

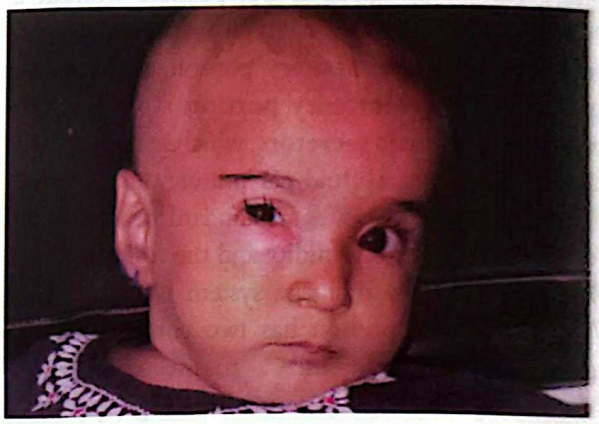

Fig. 20.3 Right nasolacrimal duct block (NLD block)

Dacryoscintigraphy

It is performed by instilling a dilute solution of radioactive marker into the conjunctival sac and taking a dacryoscintogram which indicates a patent or blocked passage. Dacryoscintigraphy has almost replaced Dacryocystography (Fig. 20.1, 20.2).

Nasolacrimal Duct Block (NLD Block)

(Fig. 20.3) It is a common cause of epiphora in children and adults. NLD block in children is usually due to non-canalisation of the nasolacrimal duct, which is the last to canalise. When the infant presents with tearing on the first few days of life, NLD block should not be included in the diagnosis as tear production is not sufficient to give rise to epiphora; therefore, in these cases

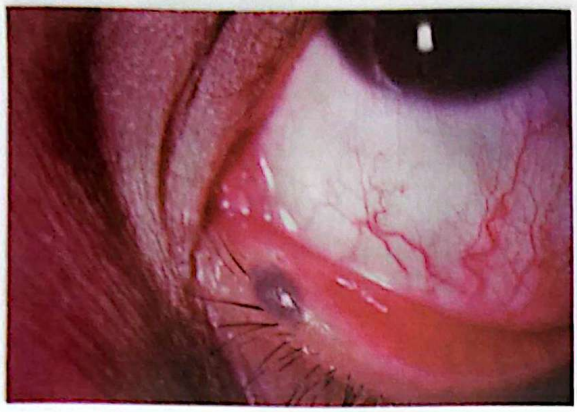

Fig. 20.4 Nevus near the lacrimal punctum

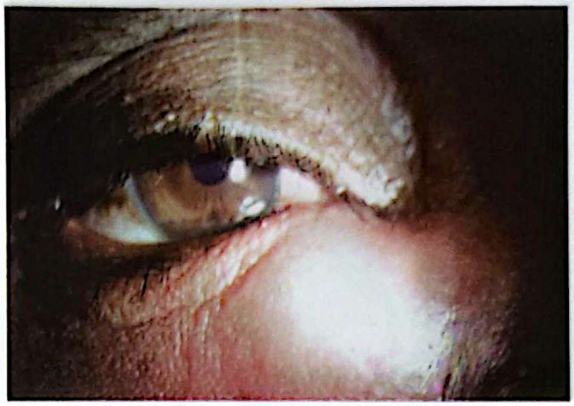

Fig. 20.6(A) Acute dacryocystitis

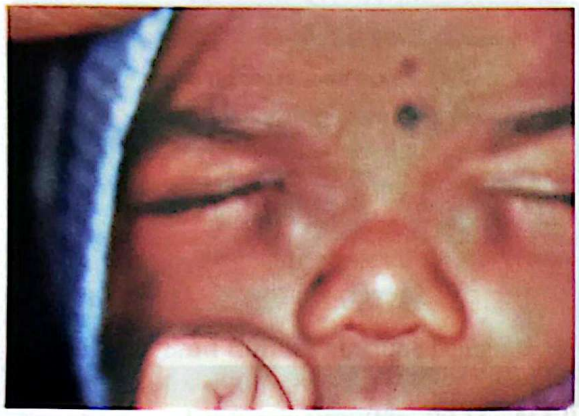

Fig. 20.5 Bilateral congenital dacryocystitis

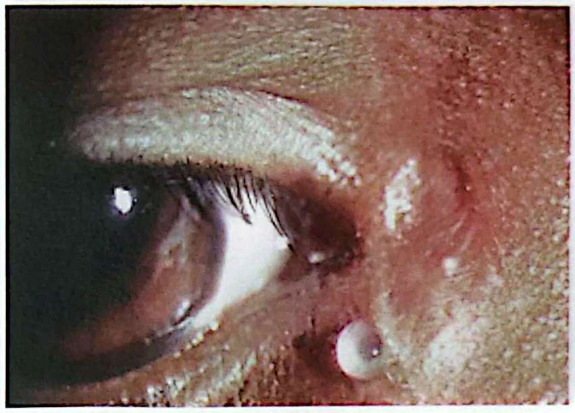

Fig. 20.6(B). Lacrimal fistula

congenital glaucoma and infection should be ruled out first. Spontaneous recovery due to continued process of canalisation occurs in the majority of cases in the first three months of life, but after this period spontaneous recovery usually does not occur. NLD block in adults is usually due to idiopathic stenosis of the nasolacrimal duct or due to irradiation and trauma. Rarely an isolated nevus is found associated with epiphora (Fig. 20.4). Treatment is avoidance of infection, massage at the medial canthus and probing of the lacrimal passages. If the condition still does not resolve, then dacryocystorhinostomy (DCR) is performed with or without intubation.

Dacryocystitis

It is the inflammation of the lacrimal sac usually as a result of stasis and obstruction of the nasolacrimal duct. It may be congenital (Fig. 20.5) or acquired, acute or chronic.

Acute Dacryocystitis (Fig. 20.6 A) is acute suppurative inflammation of the lacrimal sac associated with painful swelling over the medial canthus with systemic symptoms including malaise and fever. Treatment is started with antibiotics and anti-inflammatory drugs after taking a swab for culture and sensitivity. Lacrimal obstruction should be treated after resolution of an acute attack. If acute dacryocystitis is left untreated, it can end up in complications like orbital cellulites, lacrimal fistula (Fig. 20.6 B)

Chronic dacryocystitis

It is the recurrent and persistent inflammation of the lacrimal sac presenting with epiphora and conjunctivitis or with mucocele (Fig. 20.9 A). Treatment is surgery, i.e., DCR with or without intubation[51,52] (Fig. 20.7, 20.8).

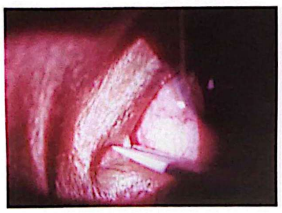

Fig. 20.7 DCR tube is being cut

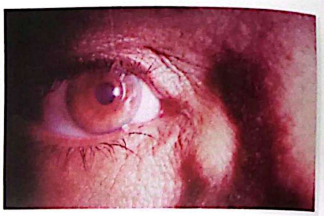

Fig. 20.9(A) Mucocele

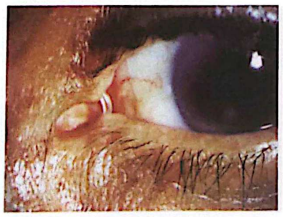

Fig. 20.8 DCR tube in place

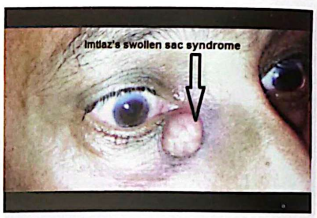

Fig. 20.9(B). Imtiaz's swollen sac syndrome

Imtiaz's swollen sac syndrome

Imtiaz's swollen sac syndrome (Fig. 20.9 B) resembles chronic dacryocystitis but is the result of temporary canalicular obstruction due to either foreign body in the canaliculus, e.g., an eye lash or swollen punctum due to inflammation. History is short, i.e., a day or two without previous history of epiphora and may or may not be associated with pain. It resolves completely with conservative treatment, i.e., systemic NSAIDs and antihistamines and topical broad spectrum antibiotics. I have not seen remission in any of my cases.

Dacryoadenitis

It is an acute inflammation of the lacrimal gland[53] usually associated with systemic disease as a part of glandular inflammation, as in mumps. Suppurative inflammation of the lacrimal gland may end up in a lacrimal abscess. Clinically, lacrimal gland swelling is tender and downward displacement of the lateral part of the lid margin gives it an S-shaped curve. Dacryoadenitis associated with systemic disease resolves spontaneously, however surgical drainage is required for lacrimal abscess.

Dry Eye (Keratoconjunctivitis Sicca)

Results from deficiency of one or more components of tear film due to atrophy of glands or due to obstruction of ducts of glands as a result of cicatrisation. In a clinical setting, dry eye is more dangerous than tearing and is more difficult to treat. Patients present with foreign body sensation and burning to the extent that they may not be able to open the eyes. On examination, punctate erosions of the cornea are seen over the lower half of the cornea on fluorescein staining, minute comma shaped filaments are seen attached with the corneal surface. Tear film break up time is decreased to around 4 seconds. Schirmer's test

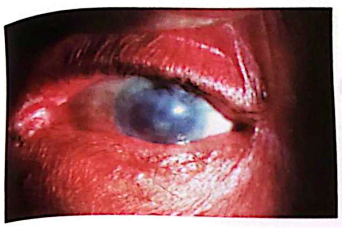

Fig. 20.10 Dry eye

Fig. 20.11 Lacrimal gland adenocarcinoma

is done with Whatman No. 41 filter paper strip placed in the anesthetised conjunctival sac by inserting it over the lateral part of the lower lid and watching the wetting after 5 minutes. Less than 8mm of wetting of the strip, excluding the folded area, indicates dry eye. Treatment is frequent instillation of artificial tears in the day time and gel at bed time, occlusion of puncta to conserve tears and moist chamber goggles (Fig. 20.10).

Lacrimal gland tumours include benign mixed cell tumour and malignant tumours including lymphoma, adenocarcinoma (Fig. 20.11, 20.12), adenoid cystic carcinoma and squamous cell carcinoma. Treatment is removal through the orbital approach followed by radiotherapy.

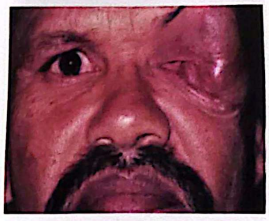

Fig. 20.12 Recurrent adenocarcinoma of the lacrimal gland

Chapter 21

Diseases of the Orbit

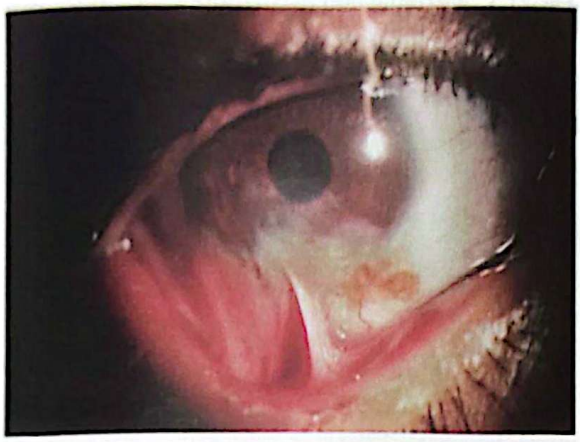

Fig.21.1 Symblepharon

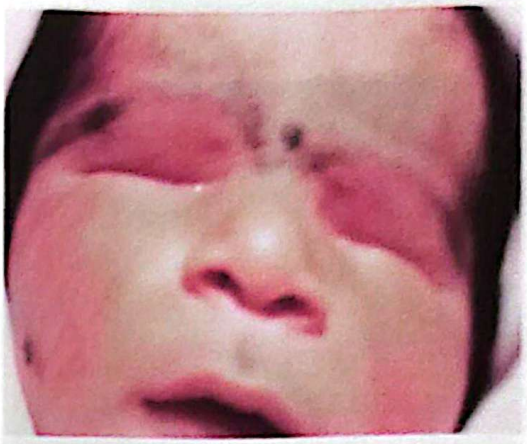

Fig. 21.2(A) Bilateral congenital anophthalmos

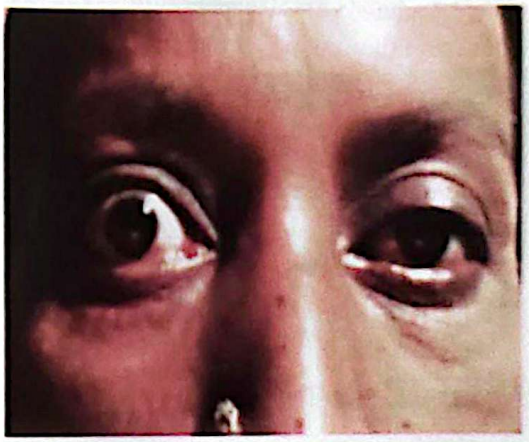

Fig. 21.2(B) Right proptosis

The orbit is a pyramidal cavity with its base in front and opens to the space and apex behind, providing passage to nerves and vessels. The roof of the orbit is the floor of the anterior cranial fossa containing frontal lobes of the cerebral hemispheres. The floor of the orbit is the roof of the maxillary sinus. The medial wall of the orbit is the lateral wall of the ethmoidal sinuses. The lateral wall of the orbit is the medial wall of the temporal fossa anteriorly, and related to the temporal lobe of the cerebral hemisphere behind. Looking into the intimate relation of the orbit to the surrounding, it occupies a central position where it can affect or get affected, from any structure, with a wide variety of disorders.[55]

COMMON CLINICAL FEATURES OF ORBITAL DISEASE

Conjunctival chemosis and hyperaemia, proptosis (Fig. 21.2B), globe displacement in various directions, lid oedema, symblepharon (Fig. 21.1) and widening of the palpebral fissure.

Congenital Anophthalmos

Congenital anophthalmos (Fig. 21.2 A) or empty orbits with no eyeballs present at birth may be seen in a clinical setting but histological evidence of vestiges of ocular tissues is present.

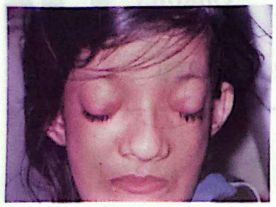

Fig. 21.3 Ocular hypertelorism

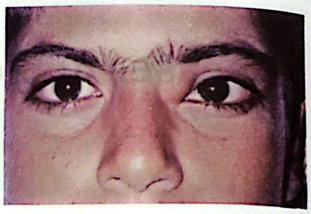

Fig. 21.5 Dermoid cyst

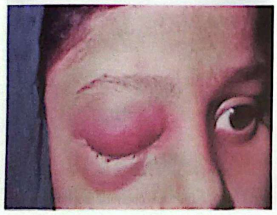

Fig. 21.4 Right Orbital cellulitis

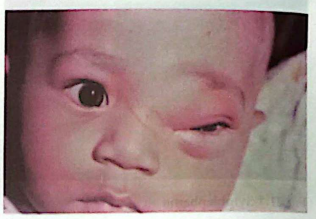

Fig. 21.6 Lymphangioma

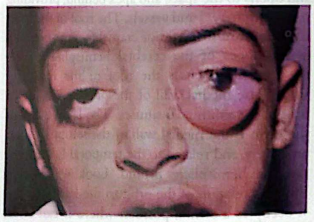

Fig. 21.7 Proptosis in acute lymphocytic leukaemia

Ocular Hypertelorism

It is a condition in which the distance between the two eyes is abnormally more than the general population. Ocular hypertelorism is commonly associated with chromosomal anomalies and mental deficiency (Fig.21.3).

Orbital Cellulitis

(Fig.21.4). It is an acute inflammation of the orbital soft tissue[56] (postseptal orbital cellulitis) or periorbital tissue (preseptal orbital cellulitis). It usually occurs due to the spread of infection from an already present focus of infection, e.g., ethmoiditis, maxillary sinusitis or a boil in the lid and trauma. The onset is rapid with pain and mechanical ptosis due to swelling in preseptal cellulitis. In postseptal orbital cellulitis, which is more dangerous, pain, conjunctival chemosis and hyperaemia is associated with proptosis. Treatment should be prompt otherwise complications may occur particularly in the postseptal variety which include cavernous sinus thrombosis, optic nerve involvement or spread of infection to the cranial cavity causing meningitis or brain abscess. After taking a swab from conjunctival secretions for culture and sensitivity and blood samples for complete blood picture and blood culture, intravenous appropriate antibiotics are given with nonsteroidal anti-inflammatory drugs. Further tests like CT-scan or MRI may be required to see extensions in to the surrounding areas.

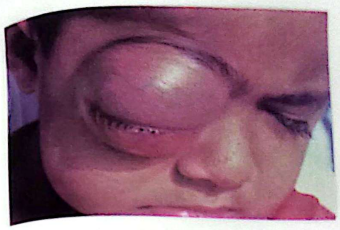

Fig. 21.8 Rhabdomyosarcoma in a child

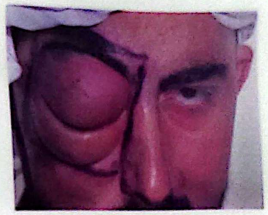

Fig. 21.10 Advanced stage of a pseudotumor

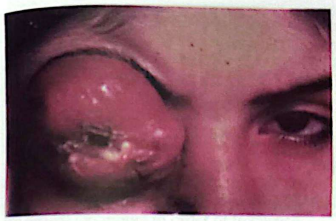

Fig. 21.9 Rhabdomyosarcoma in an adult female (a rare presentation)

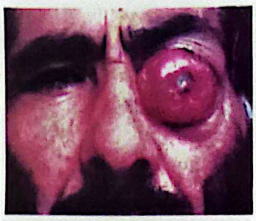

Fig. 21.11 Lymphoma left orbit

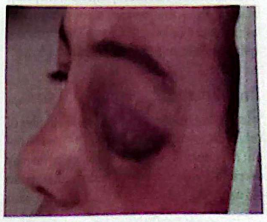

Fig. 21.12 Fungal sinusitis involving orbit

Proptosis (Exophthalmos)

It is the abnormal forward displacement of the globe usually due to a space occupying lesion of the orbit. It may occur at any age and may be unilateral or bilateral.

In children, proptosis may occur due to capillary haemangioma, orbital varices, dermoid cyst (Fig. 21.5), hydatid cyst[57], lymphangioma (Fig. 21.6), leukaemia (Fig. 21.7), tumours like retinoblastoma, rhabdomyosarcoma (Fig. 21.8, 21.9), neurofibroma, neuroblastoma, optic nerve glioma and shallow orbits as in craniosynostosis.

In adults, proptosis may occur due to thyrotoxicosis, cavernous haemangioma, optic nerve meningioma, optic nerve glioma, nasopharyngeal carcinoma, lymphoma (Fig. 21.11), lacrimal gland tumours, intraorbital spread of sinus tumours or fungal infections (Fig. 21.12), pseudotumour[58] (Fig. 21.10) and carotid-cavernous fistula (pulsating proptosis).

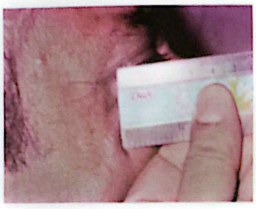

Fig. 21.13 Measurement of proptosis

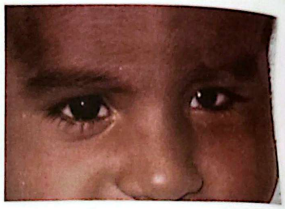

Fig. 21.14 Left Microphthalmos

Measurement of proptosis (Fig. 21.13) helps in assessment of progression or remission in proptosis and is done simply with a glass scale (present in the geometry box of students) placed on the lateral orbital margin and noting the distance up to the apex of the cornea which is normally 1.6 cm.

Besides routine blood and urine tests, thyroid function tests (T3, T4, TSH), orbital ultrasound B-scan, orbital X-ray, orbital venography, CT scan and MRI are helpful investigations in reaching at a diagnosis. Treatment includes medical therapy for infections and pseudotumour, surgical decompression of orbits in dysthyroid orbitopathy, surgical removal of tumours when present and intravascular ballooning through interventional radiology in carotid-cavernous fistula.

Enophthalmos

It is an abnormally sunken eye due to atrophy of orbital fat, microphthalmos (Fig. 21.14) and blow-out fracture of orbit. Absorption of orbital fat may occur in anorexia associated with diseases, or in old age.

Cryptophthalmos

It is a rare congenital anomaly in which the skin of the brow passes over the malformed eye. It is associated with abnormalities of the orbit (Fig.21.15, 21.16).

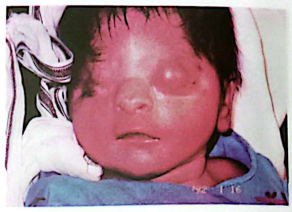

Fig. 21.15 Bilateral cryptophthalmos

Blow-Out Fracture

It occurs due to sudden rise in intraorbital pressure, as in a playing ball trauma, causing fracture of the medial wall or floor of the orbit. Patients present with periorbital ecchymosis and oedema with nasal bleed. On examination, anaesthesia in the distribution of infraorbital nerve and diplopia may be present. Enophthalmos becomes evident after resolution of oedema. X-ray orbit (Water's view) may show a fracture. A CT scan is more informative. Most of the patients improve with conservative treatment; a few may require surgical intervention.

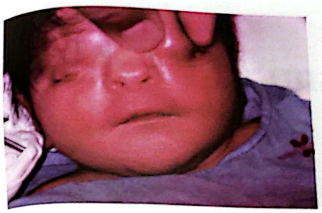

Fig. 21.16 Bilateral cryptophthalmos

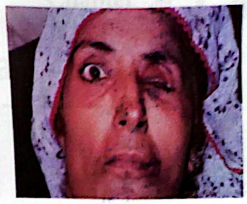

Fig. 21.17 Left empty orbit with phantom ocular pain

Phantom Pain

(Fig.21.17) Phantom pain of the eye[59] is a rare condition in which the patient starts complaining of pain in the eye in an empty orbit. Treatment is reassurance and provision of ocular prosthesis (Fig. 21.18).

Metastatic orbital tumours involving the orbit and adjacent areas include breast carcinoma, neuroblastoma, lung cancer, prostrate carcinoma, gastrointestinal carcinoma, renal cell carcinoma and thyroid carcinoma in order of frequency.

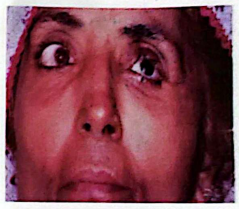

Fig. 21.18 Left ocular prosthesis in place

Chapter 22

Ocular Trauma

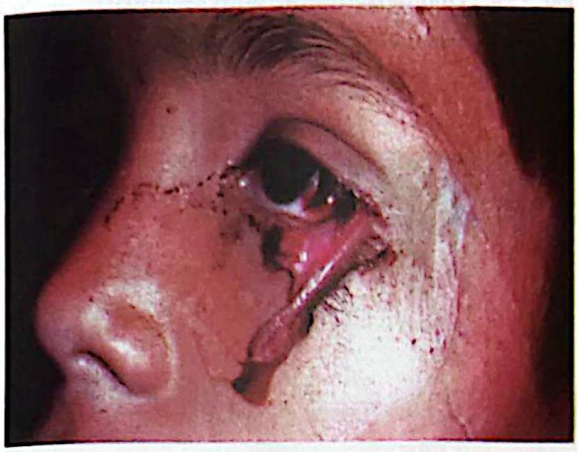

Fig. 22.1 Lower lid cut

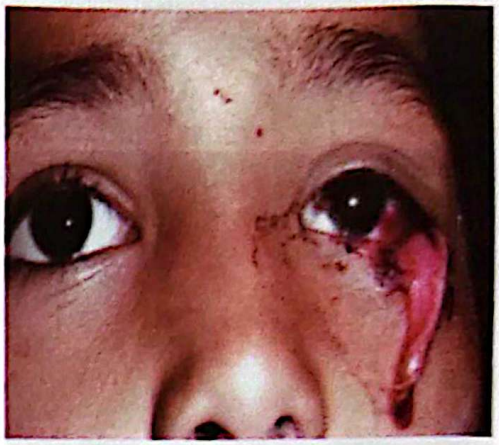

Fig. 22.2 Lower lid cut

Trauma, whether physical or chemical can be a serious threat to vision if not treated appropriately and in a timely fashion. The most obvious presentation of ocular injuries is redness and pain.

Eyelid Trauma

Lid trauma may vary from a trivial scratch to a serious lid cut (Fig. 22.1 & 22.2). Lid injuries should be repaired as soon as possible due to danger of corneal exposure. A grey line on the lid margin should be approximated first to regain lid anatomy.

Hyphaema

A hyphaema is the presence of blood in the anterior chamber, a common finding after blunt eye trauma. If the bleed is large, the blood will settle out in a layer at the bottom of the anterior chamber (AC) (Fig. 30.2). If the entire AC is filled with blood it is known as an "8-ball hyphaema". Most of the time, however, the bleeding is microscopic and can only be seen on the slit lamp as "red cells" floating in the aqueous fluid.

Blood typically clears well, though you can get staining of the cornea if the blood is persistent or coexists with high intraocular pressure. Patients are encouraged to sleep with their head elevated (to help the blood settle) and to avoid straining.

Medical treatment involves steroids (to decrease the inflammatory response) and a cycloplegic dilating drop to help with photophobia. As with iritis, this dilation also keeps the iris from sticking to the underlying lens and forming synechiae. Trabeculectomy is a good surgical option for total hyphaema.

Angle Recession

Blunt trauma may lead to damage to the angle of the anterior chamber which is known as angle recession. Angle recession may lead to secondary glaucoma.

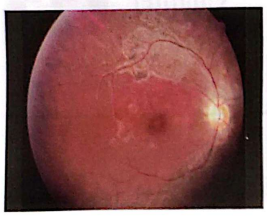

Fig. 22.3(A) Areas of retinal pigment atrophy following commotio retinae.

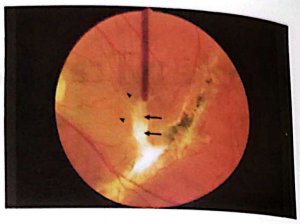

Fig. 22.4 Choroidal rupture with secondary neovascular membrane and subsequent scarring of the macula

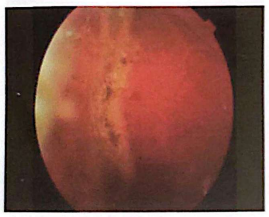

Fig. 22.3(B) Choroidal rupture

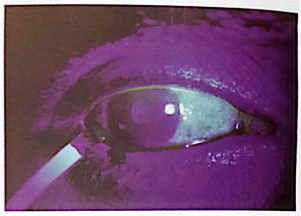

Fig. 22.5 Fluorescein staining showing corneal abrasion seen in cobalt blue light

Commotio Retinae

Commotio retinae (Fig. 22.3 A) is a countercoup injury to the retina. It can occur centrally or peripherally, and when it involves the macula, it is called Berlin's oedema. The affected area becomes white and opaque usually hours after the trauma. On careful examination, most of the opaqueness and whitening is in the outer retina and the blood vessels are clearly seen. These areas may undergo atrophy of the retinal pigment epithelium. There is no known treatment. Prognosis is usually excellent. In rare cases the retinal contusion causes cystoid macular oedema that may, in turn, progress to a macular hole.

Traumatic choroidal rupture

Choroidal ruptures (Fig. 22.3 B, 22.4) are breaks in the choroid, the Bruch's membrane, and the retinal pigment epithelium that result from blunt ocular trauma.

After blunt trauma, the ocular globe undergoes mechanical compression and then sudden hyperextension. Because of its tensile strength, the sclera can resist this insult; the retina is also protected because of its elasticity. The Bruch's membrane does not have enough elasticity or tensile strength; therefore, it breaks.

Choroidal neovascular membranes may occur in these areas months or years later.

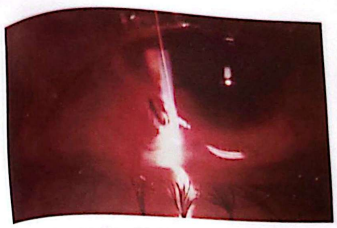

Fig. 22.6 Scleral perforation near limbus

Fig .22.7 Corneal laceration with prolapse of iris into the wound

Retinal Dialysis

Retinal dialysis and retinal tears may occur after trauma to the eyeball. This may lead to retinal detachment.

Corneal Abrasion

Corneal abrasion (Fig. 22.5) is probably the most common eye injury. Abrasion is a defect in the surface of the cornea that is limited to the epithelial layers and does not penetrate the Bowman membrane. In some cases, the bulbar conjunctiva is also involved. Corneal abrasion results from physical or chemical trauma.

Corneal abrasions occur in any situation that causes epithelial compromise. Examples include corneal or epithelial disease (e.g., dry eye), superficial corneal injury or ocular injuries, (e.g., those due to foreign bodies), and contact lens wear. Spontaneous corneal abrasions may be associated with corneal dystrophy or recurrent corneal erosion syndrome.

Spontaneous healing is the rule in abrasions. During corneal healing of a lesion, corneal epithelial cells become flattened, they spread, and they move across the defect until they cover it completely. Cell proliferation, which is independent of cell migration, begins approximately 24 hours after injury. Stem cells from the limbus also respond by proliferating to give rise to cells which migrate to heal the corneal defect and proliferate to replenish the wounded area.

Penetrating Trauma

Penetrating trauma is far more common in males as compared to females. Frequent causes are assaults, accidents and sports.

Extent of injury is determined according to the size of the object, its speed and its composition. Extent of injury may be determined by diagnostic tools like ultrasound B-scan[®] and CT scan and may range from a minor laceration to the globe, with or without a cataract to a major injury resulting in total disruption and loss of the eyeball.

Treatment depends upon the extent of damage and associated complications specially endophthalmitis or panuveitis.

Lacerations

Most corneal scratches only involve the surface of the epithelial layer. If the injury goes deeper into the stroma, then it is known as a laceration. With any laceration, you want to ensure that the cornea has not perforated. Corneal integrity can be checked with the "Seidel test". A strip of fluorescein paper is placed over the wound and checked if dye flows down the corneal surface, indicating leaking aqueous fluid.

If a patient is "Seidel positive" than it is an open-globe injury (Fig. 22.7) and surgical intervention is required.

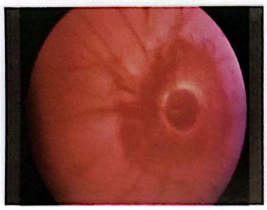

Fig. 22.8 Optic nerve avulsion

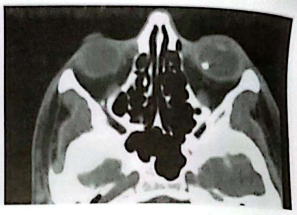

Fig. 22.10 CT scan showing left intraocular foreign body and another foreign body at the right orbital apex

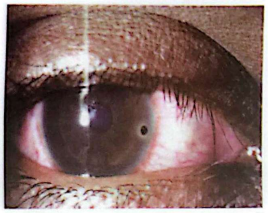

Fig. 22.9 Superficial corneal iron foreign body with surrounding rust ring

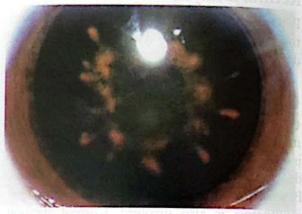

Fig. 22.11 Siderosis leading to cataract due to iron deposition in the lens epithelial cells.

Optic Nerve Avulsion

It typically occurs when an object intrudes between the globe and orbit resulting in displacement of the eye. On examination, a cavity is present in place of the optic nerve head (Fig.22.8).

Corneal Foreign Body

Small pieces of metal can fly into the eye and stick onto the cornea causing small abrasions and discomfort. Metal rusts quickly and will form a rust ring within a day. Foreign bodies can be easily removed at the slit-lamp using a 26- gauge needle (Fig. 22.9).

Residual rust ring can also be removed with the needle or with the help of a 'burr' if available. Topical antibiotics are given to prevent infection until the area is healed.

Intraocular Foreign Body

Small metal fragments can enter the eye at high speed and leave little or no signs of injury. This may take place in working places where metal is hammered. These workers should wear goggles to protect their eyes. Foreign bodies can cause mechanical effects, introduce infection or exert toxic effects on intraocular structures.

Mechanical effects include cataract, retinal haemorrhages and tears. Metal is very toxic to the retina and can destroy the retinal cells if not detected. If there is any suspicion for penetrating injury, a thin-slice CT scan of the orbit (Fig. 22.10) or ultrasound scan to look for metal pieces not obvious on clinical examination should be ordered. Avoid MRI in this setting to avoid creating a moving projectile inside the eye.

Any intraocular foreign body should be removed with the help of magnets or forceps depending on its location.

Siderosis

Siderosis refers to deposition of iron in tissues where it undergoes dissociation and exerts a toxic effect on cellular enzymes resulting in cell death. Iron deposits in intraocular epithelial structures like the lens, iris, ciliary body and retina. This happens when iron foreign bodies are not removed from the eye (Fig. 22.11).

On examination there will be decreased visual acuity, injection, heterochromia, secondary glaucoma, anterior capsular cataract, degenerative pigment dispersion in the retina.

Traumatic Optic Neuropathy

Optic neuropathy refers to sudden visual loss resulting from a blunt or penetrating trauma to the orbit, resulting in optic nerve injury. The fundus is normal and there is no sign other than a relative afferent pupillary defect.

Chapter 23

Ocular Pharmacology

STEROIDS

Steroids play an important role in ophthalmology. These substances include glucocorticoids mineralocorticoids and sex hormones. Only the glucocorticoids are of importance in ophthalmology. They are 21-C compounds secreted by the adrenal cortex.

Mechanism of Action

They reduce inflammation by reduction of leukocytic and plasma exudation, maintenance of cellular membrane integrity with inhibition of tissue swelling, inhibition of release of lysosome and suppression of circulating lymphocytes.

Pharmacological Preparation

They are administered in the form of eye drops, ointments, injections (subconjunctival, sub-tenon, retroequatorial, retrobulbar, intracameral, intravitreal, intralesional) and systemically in the form of tablets and injections.

Indications

In topical preparation, they are used in uveitis, scleritis, vernal catarrh, phlycten, allergic keratoconjunctivitis, cystoid macular oedema and following refractive, corneal and intraocular surgery. In systemic preparations, they are indicated in corneal graft rejections, scleritis, posterior uveitis, sympathetic ophthalmia, papillitis, reterobulbar neuritis, anterior ischemic optic neuropathy, VKH syndrome, orbital pseudotumor, malignant exophthalmos, Herpes Zoster Ophthalmicus and orbital lymphangioma. Nowadays, the intravitreal injections of steroids are given to treat Exudative Age Related Macular Degeneration, diabetic retinopathy and cystoid macular edema.

Currently the available formulation of steroids for ocular use are **dexamethasone** (Decadron), **prednisolone** (Pred Forte), **fluorometholone** (FML, Floroptic), **loteprednole** (ALREX, LOTEMAX), **Medrysone** (HMS) and **Rimexolone** (BEXOL).

Side Effects

The ocular side effects of systemic steroids include cataract, glaucoma, activation of infection, herpetic keratitis, delayed wound healing, CRVO and papilloedema, while the systemic complications are mental changes, Cushing Syndrome, diabetes mellitus, hypertension, reactivation of tuberculosis and other infections, peptic ulcer, renal failure and osteoporosis. The side effects of the topical steroids include cataract, glaucoma, dry eye, ptosis and reactivation of eye infections.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

These are a heterogeneous group of anti-inflammatory, analgesics and anti-pyretics which irreversibly block cyclo-oxygenase enzyme and hence they inhibit the release of prostaglandins and other inflammatory chemical mediators like kinins, lymphokine and lysosomal enzymes etc.

Topical Preparations

For ophthalmic use, NSAIDS are available in the form of drops and include; Diclofenac (DICNY, DIFSOM), Bromfenac (XIBROM), Nepafenac (Nevanac, venac), flurbiprofen (Ocufen), ketorolac (keterosan, ketro).

Indications

Episceleritis

Oxyphenbutazone 100 mg TDS and indomethacin **(INDOCID)** 25 mg BD are effective.

Scleritis

NSAIDS are effective in diffuse and nodular scleritis.

Cystoid macular oedema

Topical and systemic NSAIDS like keterosan, nepafenac sodium and oral preparations of diclofenac, flurbiprofen are effective.

Uveitis

NSAIDS are useful in long term therapy of recurrent anterior uveitis initially controlled by steroid therapy. Phenylbutazone is most effective in uveitis due to ankylosing spondylitis.

Spring catarrh and allergic keratoconjunctivits

Ketorolac is very effective.

To **maintain Dilatation of the pupil during cataract extraction:** Flurbiprofen is used preoperatively every five minutes.

Postoperative inflammations: NSAIDS reduce postoperative inflammation. Nepafenac sodium is used to relieve pain after corneal and cataract surgery.

Immunosuppressive and antimitotic agents: The commonly used immunosuppressive and antimitotic agents in ophthalmology are:

1. 5-Fluorouracil
2. Mitomycin-C
3. Daunorubicin
4. Interferon α-2a
5. Interferon α-2b
6. Cyclosporin

5-Fluorouracil

(5-Fu Inj Urotral) It blocks the mitosis of fibroblasts. It is used as adjuvant in glaucoma filtration surgery per operatively, it is used as a single application of 3 x 2 mm piece of cellulose sponge soaked with 50 mg/ml 5-FU and placed at the trabeculectomy bed site under the conjunctival flap for 60 seconds. The sponge is then removed and the area is irrigated with BSS or Ringers solution. Also, sub-conjunctival injection of 5-FU can be given during filtration surgery where 0.05 cc of 10 mg/cc or 0.1 cc of 5mg/cc solution of 5-FU is injected subconjunctivally with 27G needle 180 degree from the filtration site. Postoperatively, S/C injection of 0.1 cc of 5mg/cc solution of 5-FU is given 90 to 180 degrees away from the bleb over a one week period. It is a very safe, effective and cheap drug but should not be used if corneal grafting is also done. Also, it is not practicable to use in children due to repeated general anaesthesia.

Mitomycin-C

(Inj MMC 2mg) It is an anti-fibro proliferative agent. A 3x2 mm cellulose sponge soaked in 0.2 - 0.4 mg/cc MMC is applied for 3-5 minutes to the trabeculectomy bed and then irrigated profusely to wash it.

MMC is also used topically after pterygium excision, excimer laser surface ablation and certain procedures of removing corneal opacities. It is also used to treat certain conjunctival and corneal tumours.

Daunorubicin

(Daunoblastina) It is an anti-fibroblastic and cytotoxic drug .It is more effective and less toxic than 5-FU and MMC. The application procedure is the same as for MMC. The recommended dose is 0.2mg/cc. No localised thinning or avascular bleb has been reported with DAUNORUBICIN.

Interferon alpha 2a

(Inj. Inferon). It is used as intralesional injections in the treatment of capillary haemangioma.

Interferon alpha 2b

(Inj. Uniferon 3 million and 5 million)

It is used in cases of epithelial cell tumour of corneal and conjunctival origin like basal cell carcinoma, squamous cell carcinoma, and transitional cell carcinoma etc. It is used as topical eye drops 1 million/cc and as intralesional/perilesional injection of 1 million I.U.

Cyclosporin

(C-spore Eye Drops) This is a T-cell immunosuppressor and hence suppresses the delayed-type (Type 4) Hypersensitivity. It is used topically in refractory cases of Uveitis, Sympathetic Ophthalmia and keratoconjunctivitis sicca.

IMMUNOSUPPRESSIVE AGENTS

These are used in ocular inflammatory diseases. They include:

Antimetabolites, i.e., **methotrexate and azathioprine.**

Alkylating agents, i.e., **cyclophosphamide (Inj ZYCRAM** 500mg and 1gm and **chlorambucil.**

Antibiotic, i.e., cyclosporine- A

Antimetabolites: Methotrexate (Cytotrexate Tab 10mg & 25mg) and azathioprine

They are used in Rheumatoid arthritis, Pemphigoid, Sympathetic ophthalmia, Behcet's disease, Pars planitis, VKH Syndrome.

Azathioprine

It interferes with purine metabolism and hence blocks synthesis of nucleic acid. The dose is 100–200 mg/day in single or divided doses starting from 1–2 mg/kg/day then to gradually increase to 2.5mg/kg/day. CBC, white cell counts are noted regularly.

Methotrexate

It interferes with folate activity and blocks nucleic acid synthesis. The oral dose is 2.5mg - 15mg until a therapeutic response and then at maintenance dose. Regular renal, hepatic and hematologic screening in required.

Alkylating Agents

These are T-cell suppressing agents.

Cyclophosphamide

Start with an empty stomach dose of (200mg) 1–2 mg/kg/day. TLC is taken every day until seven days. At this point dose is reduced by 25-50 mg to stabilize the TLC at about 3000 cell/μl then TLC and DLC are taken weekly.

Chlorambucil

Start with a dose of 0.1–0.2 mg /kg and increase every 4th day to a total dose of 10-12 mg/day while screening TLC, DLC, and CBC as for cyclophosphamide.

Cyclosporine-A

It is an immunomodulating agent. Its topical use is associated with decreased inflammatory markers to the lacrimal gland, increased tear production and improved comfort and vision. It is, therefore, approved topically for chronic dry eye. Orally, its dose is 2.5-5mg/kg per day given in an olive oil-ethanol solution with fruit juice. The maximum dose is 10 mg/kg/day.

Monthly or weekly CBC and urine analysis is done to monitor the side effects. The dose is adjusted to keep the TLC at 3000-4000/μl and continued for one year to induce remission before being stopped.

DRUGS USED IN OPHTHALMIC SURGERY

Viscoelastic substances, i.e., Hyaluronate, chondroitin sulphate and HPMC have the properties of viscosity, elasticity, cohesiveness and adhesiveness. Therefore, they are used in anterior segment surgery to maintain spaces, protecting surfaces and moving tissues.

Ophthalmic glue Cyanoacrylate tissue adhesive (Dermabond, Isodent) is applied in corneal ulcers, perforation and breaks in the form of a liquid which polymerizes into a solid plug. Fibrinogen Gel (tissue) is used to firmly paste the conjunctiva, amniotic membrane and lamellar corneal grafts.

Band Keratopathy

Disodium, EDTA or edetate, disodium (endrate) is applied over the corneal stroma, after removing the epithelium, to chelate the calcium deposits from the stroma.

Anterior Segment Gases

SF6, and perfluoropropane gases are injected into the anterior chamber to maintain the AC reformation after trabeculectomy for internal tamponade to prevent Descemet membrane detachment.

Vitreous Substitutes

These include gases, **perfluorocarbon liquids and silicon oil.** With the exception of air, the gases like SF_6, CF_4, $C2F_6$, $C4F_8$ and C_3F_8 are expansible due to their interaction with systemic O_2, CO_2 and Nitrogen. So, they are used to internally tamponade the retina in cases of TRD, membrane peeling procedures, PVR and RDs.

The perfluorocarbons are used in the management of giant retinal tears, PVRs, PDR, removal of dislocated crystalline lenses or intraocular lenses, drainage of sub-retinal macular haemorrhages and choroidal or expulsive haemorrhages. They are retinotoxic if they remain in contact with the retina for a long time.

Silicon oil is extensively used for long term tamponade of retina. It is kerato-phako-retinotoxic and also results in glaucoma.

Surgical Haemostatic Agents

Thrombin, a coagulating factor, when given intravitreally, can assist in controlling intraocular haemorrhage during vitrectomy.

It can also be applied via soaked sponges to exposed conjunctiva and sclera, where bleeding becomes a problem.

Topical aminocaproic acid gel (caprogel)

It is used to prevent re-bleed in cases of hyphaema.

Tissue Plasminogen Activator (t-PA)

It is used during intraocular surgeries to lyse the blood and to assist in evacuation of hyphaema, sub-retinal clots or non-clearing vitreous haemorrhage.

Botulinum Toxin A

It is used in the treatment of squint, blephrospasm, spasmodic torticollis, hemifacial spasm, Meige's syndrome, facial wrinkles and migraine headache. It prevents the release of acetylcholine at the neuromuscular junction and so, temporarily paralyses the injected muscle. Ptosis and diplopia are common complications.

Alcohol (Ethanol) and Chlorpromazine (Largactil Inj)

Reterobulbar injection of absolute or 95% alcohol (ethanol) or chlorpromazine causes damage to the ciliary nerves and so relieves the pain in patients of painful blind eye. It may damage other orbital nerves resulting in total ophthalmoplegia or neuroparalytic keratitis. Since the injection itself is painful, it should be preceded by a local anaesthetic injection.

ANTIBACTERIAL AGENTS

Drugs utilised for treating bacterial infections are either produced by microorganisms (antibiotics, e.g., penicillins) or prepared synthetically (antimicrobials, e.g., sulfonamides).

Penicillin. Penicillin G is used to treat syphilis and infections caused by streptococci, pneumococci and gonococci. Penicillinase resistant penicillin like cloxacillin, floxacillin, and methicillin are used systemically in treatment of infections caused by the H.influenzae, Proteus mirabilis and Neisseria species.

Cephalosporins are produced by Cephalosporium acremonium. They are structurally and pharmacologically related to penicillins. First generation cephalosporins like Cefazolin are bactericidal against Gram positive bacteria. Second generation cephalosporins like Cefuroxime, third generation cephalosporins like Cefotaxime and fourth generation cephalosporins like Cefquinome are increasingly effective against Gram negative bacteria as well.

Fluoroquinolones. They are a family of synthetic antibacterial agents utilised in treating infections caused by Gram positive bacteria, Gram negative bacteria and anaerobic organisms in the body including eye infections. First generation fluoroquinolones, e.g., nalidixic acid, second generation fluoroquinolones, e.g., ciprofloxacin, third generation fluoroquinolones e.g. moxifloxacin and fourth generation fluoroquinolones, e.g., trovafloxacin have a place in treating infections in and around the eye.

Aminoglycosides including gentamicin, tobramycin and neomycin are effective against Gram negative bacteria, especially Pseudomonas species, and are also effective against Gram-positive bacteria.

Tetracyclines including oxytetracycline (Terramycin), doxycycline (Vibramycin), methacycline (Rondomycin) and minocycline (Minocin) are effective against Gram negative and gram positive bacteria in addition to Chlamydia, Mycoplasma, Rickettsia and amoebae.

Erythromycin is effective against Chlamydia trachomatis.

Vancomycin is a useful antibiotic against Gram positive bacteria including resistant strains of Staphylococcus aureus.

Fusidic acid is the most effective drug available against staphylococcus aureus infections.

Sulfonamides like sulfacetamide, sulfisoxazole and combination like cotrimoxazole are utilised in the treatment of Trachoma and prevention of ophthalmia neonatorum.

ANTIVIRAL AGENTS

Currently no antiviral agent is available for the treatment of viral conjunctivitis caused by **Adenovirus**. However, the following drugs are available against Herpes Zoster and Herpes Simplex virus induced keratitis and keratouveitis:

Acyclovir (Zovirax, Viracyl) 3% ointment, 800mg tab five times a day. Oral acyclovir is of clear benefit in reducing the risk of recurrent herpetic keratitis and also indicated in cytomegalovirus induced retinitis.

Valacyclovir (Valtrex) 500mg tab BID for seven days.

Gancyclovir is indicated intravitreally in viral retinitis caused by CMV, HZO and AIDS.

Glucocorticoids are contra-indicated in herpetic epithelial keratitis while they are indicated in disciform keratitis.

ANTIFUNGAL AGENTS

Yeast fungi (Candida) and Filamentous fungi (Fusarium, Cephalosporium and Aspergillus) cause keratomycosis, scleritis, canaliculitis, mucormycosis and endophthalmitis for which various antifungal agents are used which are as follows:

Natamycin (Natasan, Ophth-natamycin) 5% drops are most effective against filamentous fungi which are the commonest cause of fungal keratitis around the world.

Amphotericin B (FUNGIZONE INFUSION) is used as a 0.15% topical solution and 1mg subconjunctival injection for 1-2 doses at 24-48 hour intervals for fungal keratitis, 5µg intravitreal injection for fungal endophthalmitis.

Miconazole: It is a broad spectrum antifungal drug used as 1% ophthalmic solution instilled as one drop/hour, and tapering over several

weeks. Also it can be given as subconjuctival injection 5-10mg every 48 hours for 2-3 doses. It can also be given intravitreally 10 μg injection. It is available in oral gel form as **MICORAL GEL** 20 gm and can be used as ophthalmic gel.

Ketoconazole (Nizoral) is effective against Yeast, Fusarium and Aspergillus. Oral tablets of 200mg as a single dose of 800mg/24hrs and then, once the symptoms disappear, to be continued for a week. For topical preparation, concentration ranges from 1-5 %.

A combination therapy of Nizoral orally and Miconazole in topical or subconjunctival injection is very effective against fungal corneal ulcers.

Fluconazole (Diflucan) is very effective against yeast. The oral dose (Diflucan capsule) is 200-600mg/day for 3 weeks in candidiasis while 10-12 weeks for cryptococcus. 3% ophthalmic solution is used as topical drops 1 drop 4 hourly to be continued for 3 weeks. This solution is highly effective in fungal corneal ulcer and abscesses which outclasses it over all other antifungal drugs in eradicating all species of fungi. Fluconazole can also be given intravitreally in a dose of 100μg which is non-toxic to the retina. It can also be given intravenously as infusions.

Itraconazole (SPORANOX CAP 100mg) 200mg can be given orally twice daily for yeast and fungal keratitis and endophthalmitis.

ANTIGLAUCOMA DRUGS

The following drugs are used in the treatment of Glaucoma.

Pilocarpine: It is the most commonly and widely used oculohypotensive drug. It is a direct acting Parasympathomimetic drug. It causes contraction of ciliary body muscles which pulls the scleral spur and trabecular meshwork so that the aqueous outflow is enhanced.

Indications and contraindications

It is used in chronic open angle glaucoma, acute angle closure glaucoma, chronic synechial angle closure glaucoma and following cyclodialysis surgery. It is primarily contraindicated in inflammatory glaucoma, malignant glaucoma or known allergic cases.

Dosage

Available in 0.25 to 10% solution (Pilocar 2%, Isopto Carpine 2%, Piloptic).

Pilocarpine is also available in combination with **epinephrine** (E-Pilo 1 & E-Pilo 6), with beta blockers **(Pilogan & Piloptic)** and with **physostigmine**.

PILOCARPINE OCULAR THERAPEUTIC SYSTEM

(Ocusert). P-20 Ocusert (equal to 0.5-1% pilocarpine drops), P-40 Ocusert (equal to 2-4% pilocarpine drops.

Side effects

These include corneal oedema, miosis, decreased vision, drug induced myopia, brow ache, retinal detachment, iris atrophy, iris cyst, contraction in peripheral visual fields, conversion of OAG with narrow angles into partial angle closure glaucoma, due to anterior shift of the iris lens diaphragm, cataract and punctal stenosis of the nasolacrimal duct.

Physostigmine (Eserine)

It is a reversible cholinesterase inhibitor and so enhances the effects of endogenous acetylcholine. It is used in glaucoma, accommodative esotropia and louse and mite infestations of eyelashes. It is available as 0.25% ointment. It is contraindicated in narrow angle glaucoma without iridotomy, inflammatory glaucoma and hypersensitivity reactions. The ocular side effects are the same as that of pilocarpine.

Echothiophate

It is the most frequently used agent because of its long duration of effect. It irreversibly inactivates cholinesterase. It is indicated in subacute or chronic angle closure glaucoma after iridectomy or where surgery is refused or contraindicated and in certain non-uveitic secondary glaucoma.

ALPHA ADRENERGIC RECEPTOR AGONISTS

1. Apraclonidine

(Iopidine, 0.5, 1% solution) It is a selective alpha adrenergic agonist. It reduces aqueous production by beta-2 receptor stimulation. It is used to control the acute rise of IOP in conditions like anterior segment laser surgery, laser iridotomies and glaucoma, as its onset of action is within an hour. The side effects include burning, itching, upper lid elevation and conjunctival blanching.

2. Brimonidine Tartrate

(Alphagan 0.15,0.2% solution) Being a highly selective alpha-2 receptor agonist, it decreases aqueous humour production like beta-blockers and increases uveoscleral outflow like prostaglandins. The side effects include burning, stinging, ocular hyperaemia, headache and oral dryness.

3. Epinephrine

(Glaucon, Epifrin 0.1,0.5,and 2% solution). It is a sympathomimetic drug (Alpha & Beta stimulant). It decreases aqueous production and increases aqueous outflow. It is primarily used in POAG. The side effects include topical allergy, cystoid macular oedema, canalicular destruction and clave pigmented conjunctival deposits.

4. Dipivefrin

(Propine 0.1% solution) It is a pro-drug of epinephrine so that it is converted to epinephrine inside the eye. Its penetration is 17 times more than that of epinephrine. It is used in POAG and ocular hypertension. It is contraindicated in narrow angle glaucoma. The side effects are less than that of epinephrine.

ADRENERGIC BLOCKING AGENTS (BETA BLOCKERS)

These include **Timolol** (Timoptol, Timoptic) (0.25 and 0.5% solution), **Levobunolol** (0.5% solution Betagan), **Carteolol** (1% solution, Ocupress), **Metipranolol** (0.3% solution, Optipranolol). They all are beta-1 (cardiac) and beta- 2 (smooth muscle-pulmonary) receptor blocking agents while **Betaxolol** (0.25% solution, Betoptic-S) is cardio selective. They reduce the production of aqueous and lower IOP. They are used in open and narrow angle glaucoma and ocular hypertension. The side effects include conjunctival hyperaemia, ocular irritation and punctate epithelial corneal erosions. They are contra-indicated in asthma, chronic obstructive lung disease, congestive cardiac failure, patients of AV block and bronchitis.

CARBONIC ANHYDRASE INHIBITORS

They are non-bacteriostatic **sulphonamides** that non-competitively inhibit the enzyme carbonic anhydrase which results in decreased production of aqueous humour, and so lower the IOP. They are used in various acute secondary glaucomas, primary and secondary open and closed angle glaucomas. These agents include:

1. Acetazolamide (Diamox, AZM, Acemox 250mg tablets)

It is also used as an injection, 500mg per vial, and 5% topical ophthalmic drops.

The common side effects include malaise, metallic taste, anorexia, gastric upsets, numbness, paraesthesia of extremities, renal colic, pruritus, urticaria, rashes and blood dyscrasias. It is contra-indicated in hypersensitivity cases, patients with depressed sodium and potassium levels and patients with significant respiratory disease, as it results in metabolic and respiratory acidosis. Other systemic CAIs in use are **Dichlorphenamide, Methazolamide** and **Ethoxzolamide**.

2. Dorzolamide

Its ophthalmic drops (2% solution) are available as monotherapy (Azopt) or combined with beta blockers **(Co-Dorzal, Synigan, Co-sopt, Timorex-D and Dortim).**

There are no side effects except for occasional cases of burning and ocular irritation.

3. Brinzolamide

(1% ophthalmic solution) It is more comfortable than topical Dorzolamide and also safe and well tolerated.

Prostaglandins

Latanoprost: (Xalatan, Velatan 0.005% solution)

It is a prostaglandin F2α analogue. It increases uveoscleral outflow around the obstructed Trabecular Meshwork. It is used once daily with IOP reduction up to 35%. There is no contraindication except for hyper-sensitivity reactions. It may result in conjunctival hyperaemia and darkening of the iris colour. This eye colour change occurs after years and is permanent. It can also result in eye discomfort, foreign body sensation, ocular pain and pruritus. It is used as monotherapy or in combination with beta-blockers (XalaCom, Timoprost).

Bimatoprost

(Lumigan, 0.03%) is ophthalmic solution which belongs to prostamide, the fatty acid amide family, and is a potent hypotensive agent. It enhances uveoscleral outflow and hence lower IOP. The side effects are as with latanoprost.

Unoprostone (Rescula 0.15% eye drops) and **Travoprost (Travaton** 0.004% eye drops).They are Prostaglandin Analogues. The dosage and side effects are the same as that of Latanoprost.

HYPEROSMOTIC AGENTS

They are used in immediate control of IOP in acute glaucoma cases. Sometimes, they are used intra-operatively to dehydrate the vitreous prior to anterior segment surgical procedures; when given, they increase plasma tonicity which draws water out of the eye. The side effects of these agents include severe systemic hypertension aggravation, nausea, vomiting and diuresis, fluid electrolytic imbalance, acidosis, pulmonary oedema and hyperglycaemic crisis. They are contraindicated in acute pulmonary oedema, cardiac failure, dehydration, renal failure, oliguria and anuria. These agents are:

Glycerine (oral glycerine 120ml pack) mixed with fruit juice.

- The standard dose is 1-1.5g/kg body weight.
- Topical glycerine is used to clear corneal oedema for better view of intraocular structures

Mannitol (25% solution)

- The standard adult dose is 0.5-2g/kg body weight.

Isosorbide: (45% mint flavored solution in 220ml pack)

- The dose is 1-2g/kg body weight given 2-4 times a day

Hypertonic saline: Like topical glycerine, it is a topical hyperosmotic agent **(OPHTH HYPERTONIC SALINE eye drops)** which effectively dehydrates the cornea in cases of corneal oedema due to acute surge of IOP or in cases of endothelial decompensation. This is used to visualise the filtration angle for gonioscopy or goniotomy, to clearly view the iris to perform laser iridotomy and to see the anterior chamber reaction.

CALCIUM CHANNEL BLOCKERS

Nifedipine: (Adalat) and verapamil (calan) inhibit calcium influx in vascular smooth muscles and increase the blood flow. They are useful in low tension glaucoma by increasing blood flow to the optic nerve head.

OCULAR HYPOTENSIVE LIPIDS

Two new anti-glaucoma agents belonging to this class are on clinical trial. They are well tolerated and have favourable ocular and systemic safety with an efficacy of 26% after administering a single drop once a day.

NEUROPROTECTIVE AGENTS

Retinal ganglion cell apoptosis start with blocking of transport of neurotrophins from the brain to retinal ganglion cells. There are agents which prevent this cascade of events. These are neuroprotective agents which include:

1. Brimonidine

It binds the cell receptor and signals the cell to carry on with neuroprotective function.

2. NMDA Antagonists

These are direct acting non-IOP lowering neuroprotective agents. They block the increase in glutamate which is a cause of cell death in glaucoma. One of these compounds is **Memantine (ZEXA)** which is an effective agent for the prevention of glaucomatous optic neuropathological progression, especially in patients of normotensive and low tension glaucoma. Other neuroprotective agents to prevent retinal ganglion cell apoptosis are **Eliprodil, L-deprenyl** and **Riluzole**.

ANAESTHETICS IN OPHTHALMIC PROCEDURES

These include **Cocaine, Proparacaine (Alcaine 0.5%)** and **Tetracaine** eye drops and Lidocaine gel. (Xylocaine gel).

Proparacaine and **Tetracaine** eye drops are used to perform tonometry, to remove foreign bodies from conjunctiva and cornea, superficial corneal surgeries like refractive surgery, placement of intra-corneal rings, conjunctival surgeries, cataract surgery and to manipulate the nasolacrimal canalicular system. **Cocaine** is used intranasally in combination with topical anaesthesia for nasolacrimal system cannulation.

Lidocaine (Xylocaine) and **bupivacaine (Albocain, Bupivacaine)** are used for peribulbar and retrobulbar injections.

Intracameral anterior chamber injection of 1% preservative free Lidocaine can be used for a cataract operation.

General anaesthetics and **sedatives** are used for strabismus, DCR, detachments, enucleation, posterior segment surgeries, orbitotomies and for examination in children. Most of such agents result in reduction of intraocular pressure while **ketamine** is associated with the rise of IOP. Therefore, ketamine **(Ketalar)** and other depolarising anaesthetic agents like **succinylcholine (Muselax)** are contraindicated in cases of repair of a ruptured globe. Intravenous analgesia like **Nelbuphine (kinz)** is also effective for eye surgeries.

DRUGS AND DYES USED FOR OCULAR DIAGNOSIS

The commonly used drugs for ocular examination and diagnosis are:

1. **Tropicamide:** (Mydriacyl 1% drops) used topically to dilate the pupil for posterior segment examination.
2. **Epinephrine:** (10 % Isonephrine, Ethifrin) It is used topically to dilate the pupil for posterior segment examination. Also used as a pharmacological provocative test to diagnose glaucoma, to diagnose ptosis due to Horner's syndrome, to identify the lost muscle during squint surgery and to differentiate scleritis from episcleritis by blanching of superficial vessels.
3. **Pilocarpine:** (Pilocar 2% eye drops) It is used to constrict the pupil to see the Kayser-Fleischer ring by gonioscopy while in 0.125% concentration drops, it is used to diagnose Holmes-Adie syndrome (Adie's tonic) pupil).
4. **Edrophonium:** It is used intravenously to diagnose myasthenia gravis in the Tensilon test. After an IV injection of 0.3 mg atropine and then an IV test dose of 0.2 ml (2 mg) of edrophonium, 0.8 ml (8 mg) of edrophonium is injected intravenously. The ptosis recovers within 30-60 seconds and lasts for 5 minutes.
5. **Physostigmine** it is used to diagnose myasthenia gravis when edrophonium is not available (Stigma Test). The side effects are the same as that of Acetylcholine. Therefore, its injection

is preceded by IV Atropine injection to minimise the side effects.

6. **Cocaine:** When 4% drops of cocaine are instilled, a normal pupil dilates while Horner's pupil does not dilate.
7. **Hydroxyamphetamine:** (1% eye drops, Paredrine) On instillation, Horner's pupil with preganglionic lesion will dilate while postganglionic Horner's pupil will not dilate.
8. **Cycloplegics:** Atropine, Homatropine, Cyclopentolate
9. **Glycerine:** It is used to see the details of the anterior chamber, iris and angles in patients with corneal oedema.
10. **Fluorescein:** (2% Fluorescein drops and Ophth Fluorescein strips): It stains epithelial defects by pooling, does not stain dead or dying cells and mucous, can promote growth of pseudomonas and stains soft contact lenses. It detects epithelial defects of the cornea and conjunctiva, aqueous humour leak (Seidele test), presence of corneal and conjunctival foreign body and the patency of the nasolacrimal system. It is also used for applanation tonometry and for the fit of rigid and semi-rigid contact lenses. Its use is contraindicated when the soft contact lenses are worn.
11. **Fluorexon** (0.35% topical drops **Fluoresoft**). It is a high-molecular weight fluorescent solution. It is used when fluorescein drops or strips are contraindicated, i.e., when soft contact lenses are worn. It can also be used to do applanation tonometry without removing the contact lenses.
12. **Fluorescein** injection (10%, 25%) It is used as an intravenous injection for fundus fluorescein angiography to diagnose retinal vascular abnormalities, CME, CSCR, CNV, PEDS and ischaemic retina. The side effects include nausea, vomiting, itching, skin and urine discolouration, and very severe pain at the extravasation site. Powder fluorescein mixed with water or fluorescein injection in a citrus drink can be taken orally to study retinal abnormalities (after 45-60 minutes).
13. **Rose Bengal.** It stains dead and dying cells and mucin, a brilliant red. It is applied as drops or a strip to evaluate keratoconjunctivitis sicca, corneal abrasions, foreign body detection and herpes simplex keratitis. There is no need of actual break in the epithelium for positive staining with this dye.
14. **Lissamine green** Like Rose Bengal, this stains mucus fibrils, dead cells and degenerated cells. Vacuoles in the mucus thread remain unstained.
15. **Indocyanine green** This unique nontoxic ophthalmic dye is used as a single bolus IV injection (40mg in 2cc of aqueous solvent followed by a bolus of 5cc of normal saline) for ophthalmic angiography, digital indocyanine green video angiography (ICG-V) and ICG-angiography guided laser photocoagulation. In ICG-V the imaging of choroid is enhanced markedly so that occult choroidal NV is well demarcated. Also, ICG-V can detect drusen with risk of exudation. ICG-angiography allows identification and treatment of feeding vessel in subfoveal NV.
16. **Trypan Blue:** This is a lens capsule strainer without affecting the corneal endothelium and without staining the lens matter. It is therefore used for capsulorrhexis in mature and hyper mature cataract and to eliminate the chances of hitting the rhexis with a Phacotip. It is injected into the anterior chamber after the air is injected and the bevel should be towards the capsule and below the air bubble. It is then left for one minute and then washed with viscoelastic. The blue capsule stands clearly demarcated from the underlying white cataract.
17. **Verteporfin (Visudyne)** Verteporfin is a photosensitive dye which is administered intravenously for photodynamic therapy (PDT), the drug/light combination for the treatment of wet ARMD. The drug reaches the choroidal circulation and is activated by non-thermal diode laser at wave length of 689nm, which results in generation of free radicals which cause vessel damage and then platelet activation, thrombosis and occlusion of CNV. The dye is selectively taken and retained by CNV so that the surrounding normal healthy tissue is not damaged. The drug is available in a single use 15 mg vial in powder form. 7.0ml of sterile water is injected into the vial for reconstitution. Saline solution should not be used because the dye precipitates in saline. 5 % dextrose is added to dilute the reconstituted solution to a total infusion volume of 30ml. The dose of the dye is 6mg per m² (BSA). The drug is given at the infusion rate of 3ml per minute in a total duration of 10 minutes. Then the laser is delivered at an interval of 5 min after the start of verteporfin infusion for 83 seconds through a slit lamp to the affected retina through a standard ophthalmological contact lens. The half-life of the drug is 5 to 6 hours with predominant excretion in faeces. The side effects include injection site reaction, headache, visual disturbances and photosensitisation. The patient should avoid exposure of the skin or eyes to direct sun light and even indoor bright light for 5 days.

ANTI-VASCULAR ENDOTHELIAL GROWTH FACTOR (ANTI-VEGF) MEDICATIONS

Anti-VEGF agents are used for treatment of ocular diseases caused by neovascularisation, e.g., age related macular degeneration, diabetic retinopathy, CRVO, retinopathy of prematurity, neovascular glaucoma and corneal neovascularisation. Vascular endothelial growth factor has been demonstrated to cause neovascularisation[63], therefore anti-VEGF medications were utilised to control and treat the undesirable effects of neovascularisation.

Following anti-VEGF medications are commonly used presently.

Pegaptanib sodium (Macugen) is a 28-nucleotide RNA aptamer that binds specifically to the VEGF-A165 isomer, the major pathological VEGF protein in the eye.

Bevacizumab (Avastin) is a full-length recombinant humanised anti-VEGF monoclonal antibody. It is a large-sized molecule (molecular weight: 148 kDa) and has twice the half-life than ranibizumab. Bevacizumab bind and neutralise all the biologically active forms of VEGF.

Ranibizumab (Lucentis) is an engineered, humanised, recombinant antibody fragment (Fab) active against all VEGF-A isoforms. It lacks the Fc domain and has a shorter half-life than other anti-VEGF agents.

Aflibercept (Eylea) also known as VEGF Trap-Eye, is a recombinant fusion protein comprising the key VEGF-binding domains of human VEGF receptors 1 and 2. Aflibercept was found to bind VEGF with a greater affinity than that of bevacizumab or ranibizumab[64].

SYSTEMIC DRUGS CAUSING OCULAR SIDE EFFECTS

Many systemic drugs produce adverse effects on ocular tissues. These effects may be mild to vision threatening. These include the following;

Conjunctiva

- **Tetracyclines** cause yellow discolouration of the light exposed portion of the conjunctiva.
- **Systemic minocyclines** cause blue-grey scleral pigmentation in the interpalpebral area.
- **Gold** when used for arthritis, produces golden to violet discolouration of conjunctiva due to its deposits as a part of the condition called chrysiasis.
- **Phenothiazine** results in brown pigmentary deposits.

- **Cornea**
- **Gold** treatment results in chrysiasis and induces gold to violet discolouration due to its deposits in anterior cornea (a reversible condition on discontinuation) and with higher doses of gold, the deposits occur in the posterior cornea which is a permanent condition. However it does not affect the vision.
- **Phenothiazine** like chlorpromazine and thioridazine get deposited in Descemet's membrane and the posterior cornea and results in browning of the cornea. The deposits typically do not affect vision and persist even after discontinuation.
- **Amiodarone:** It is a cardiac medicine which deposits in inferior corneal epithelium as brown coloured pigments arranged in a whorl-like pattern called cornea-verticillata. The deposits do not affect the vision.
- **Chloroquine, (Neivaquine Basoquin), hydroxychloroquine, Plaquenil atovaquone** and **indomethacin**. They also result in cornea-verticillata.
- **Vitamin–D:** Its excessive use results in band-shaped keratopathy.
- **Isotretinoin** (Accutane) causes dry eye.
- **Sulfonamides** (Septran) is notorious for Stevens-Johnson syndrome.
- **Steroids:** Their use is associated with Herpes-simplex keratitis.

Anterior Chamber

- Rifabutin is used in combination with clarithromycin or fluconazole in patients of AIDS for the treatment of opportunistic infection due to mycobacterium avium. This results in iridocyclitis with hypopyon which resolves on stopping the medicines.
- Chloral hydrate, morphine, neostigmine cause miosis; while amphetamine, tricyclic agents cause mydriasis.
- Cydophir: It is used for cytomegalovirus retinitis in patients with AIDS. It causes acute anterior uveitis with marked fibrinous exudates but few cells.

Lens

- **Steroids** are notorious for causing posterior sub-capsular cataract.
- **Allopurinol** (used in gout). **Gold** (used in rheumatoid arthritis), **Busulphan (malarone** used in chronic myeloid leukaemia) and **Chlorpromazine (Largeactil)** (used in schizophrenia and psychosis) are cataractogenic.

Retina

- **Hydroxychloroquine, Plaquenil and chloroquine (Nivaquine),** anti-arthritis and anti-malarial drugs, can cause irreversible vision threatening macular toxicity (Bull's eye maculopathy) by concentrating in RPE and choroid.
- **Phenothiazine: Thioridazine** (Melleril) used to treat schizophrenia and psychosis cause salt and pepper pigmentary changes, plaque like pigmentation and focal or generalised loss of RPE and choriocapillaris.
- **Sildenafil (Viagra, Penegra 50, 100-mg tab)** this drug, used to achieve and maintain penile erection, causes light sensitivity and blue vision and anterior ischaemic optic neuropathy. The drug inhibits PDE6, which controls the level of cyclic GMP in the retina. Similar visual disturbances are produced by vardenafil and **tadalafil**.
- **Tamoxifen (Tamoxifeno tab, Nolvadex tab).** It is used in patients of breast cancer. It causes retinopathy characterised by bilateral fine, superficial, yellow crystalline deposits in the inner retinal layers, while punctate grey lesions in the outer retinal layers.
- **Canthaxanthin:** A carotenoid, used for sun tanning, deposits at the posterior poll of the retina as tiny glistening yellow deposits arranged symmetrically in a doughnut shape.
- **Nitrofurantoin:** It is an antibiotic, used for urinary tract infection, which causes toxic crystalline retinopathy.

Chapter 24

Ocular Anaesthesia

Adequate anaesthesia is of prime importance in success of surgery on the eye. General anaesthesia is an ideal anaesthesia[61] for an eye surgeon but it has its own limitations, e.g., many patients requiring eye surgery may never reach at the fitness level for general anaesthesia, additional time is required by an anaesthetist for inducing anaesthesia and then reawakening the patient, which may end up in a benefit to a lesser number of deserving patients, less availability of anaesthetists and trained paramedics. Postanaesthetic irritated behaviour of the patient is also dangerous for a freshly operated eye. Local anesthaesia, however, is safest for the patient and readily available.

AIM OF LOCAL ANAESTHESIA

It adequately provides analgesia (painless surgery), adequate akinesia (restriction of movements) and a quiet and relaxed patient. The long known analgesic technique of **retrobulbar** injection of a local anaesthetic agent (lignocaine 2%) is now almost abandoned throughout the world due to its complications including retrobulbar haemorrhage, globe perforation, optic nerve injury and serious complications like brain stem anaesthesia. The long known **facial block** to produce akinesia by paralysing orbicularis oculi with infiltration of a local anaesthetic around the facial nerve or its branches is also becoming less popular due to severe pain, which the patient never forgets throughout his later life. Patients complain of temporal pain on mastication afterwards.

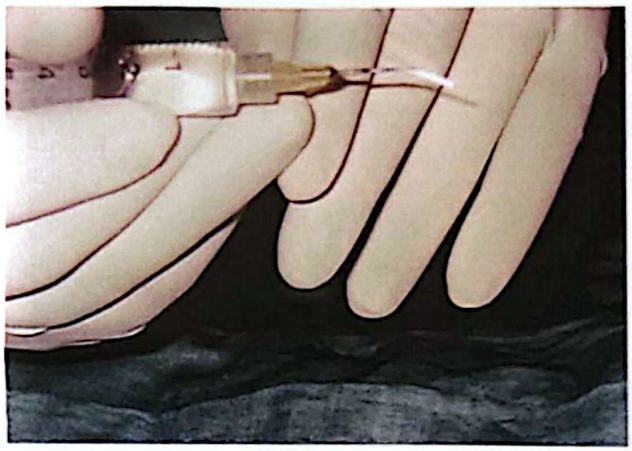

Fig. 24.1 Blunt cannula for spraying an anaesthetic into the sub-Tenon space

TOPICAL ANAESTHESIA

It is the surface anaesthesia produced by instillation of local anaesthetic drops in the conjunctival sac, e.g., proparacaine hydrochloride eye drops of 0.5% strength are used to remove foreign bodies from cornea and conjunctiva. Topical anaesthesia has been used recently in intraocular surgery like phacoemulsification.

Sub-Tenon anaesthesia is being used frequently for anaesthetising the globe to perform all types of intraocular surgery including cataract surgery, glaucoma surgery and posterior segment surgery. It produces excellent analgesia and is safe[62]. Two millilitres of lignocaine 2% is introduced in the sub-Tenon space through a small opening created in the inferonasal or inferotemporal quadrant 5 minutes prior to surgery, with the help of a blunt cannula (Fig. 24.1).

PERIBULBAR ANAESTHESIA

It is another method of achieving good ocular analgesia. It requires 5 millilitres or more of anaesthetic drug into the orbit around the globe.

Chapter 25

Lasers in Ophthalmology

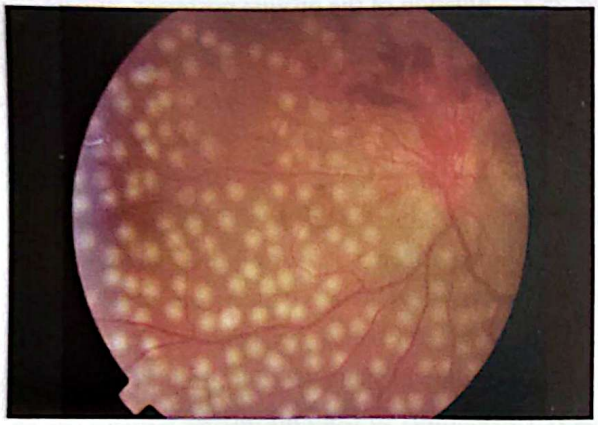

Fig. 25.1 A view of the fundus just after panretinal photocoagulation

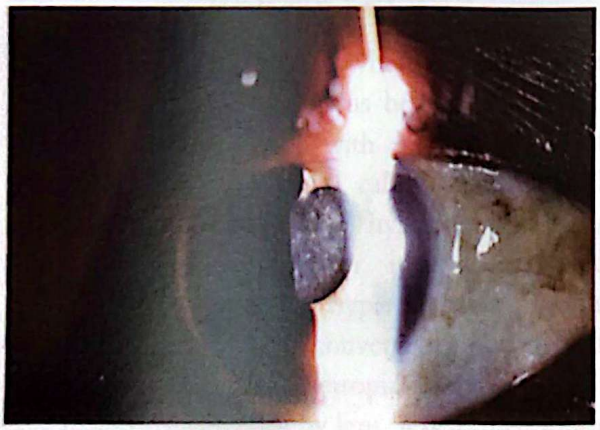

Fig. 25.2 Posterior capsular opacification

Light Amplification by Stimulated Emission of Radiation abbreviated as Laser, has now occupied an important position in the diagnosis and treatment of eye disease. The following types of lasers are in use in clinical practice.

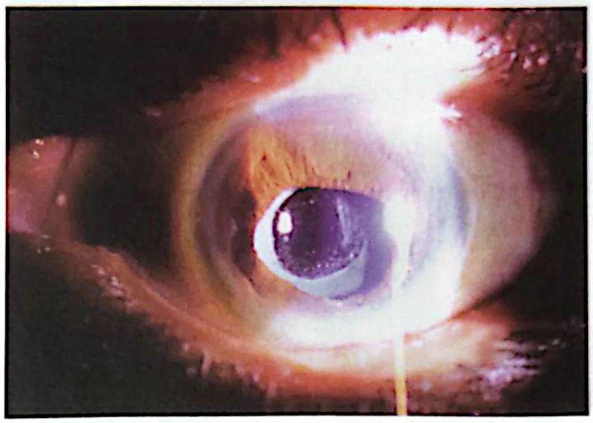

Fig. 25.3 YAG laser capsulotomy performed in a very thick posterior capsule

PHOTOCOAGULATION

It is produced by the laser which works through its heating effect and is widely used. A green laser with 532 nm emission is used particularly in macular lesions as green light is least absorbed by xanthophyll, but is well absorbed by melanin and haemoglobin. An Argon laser with 488-515 nm emission (blue-green) is effective in peripheral retinal lesions. A Krypton laser with 577 nm emission works well in red lesions. A Diode laser with infrared emission is utilised in thermotherapy (Fig.25.1).

PHOTODISRUPTION

(Fig. 25.3, 25.4) It is produced by the laser which works through its disrupting effect and is used to perform posterior capsulotomy in the opaque posterior capsule (Fig. 25.2) after cataract surgery. A Nd:YAG laser with 1064 nm emission is applied on the posterior capsule resulting in plasma

formation at that point. This plasma formation creates an acoustic wave that disrupts nearby tissue.

PHOTODECOMPOSITION

It is produced by an excimer laser which works through its decomposing effect on covalent bonds in the tissue polymers thereby producing clean cuts. This laser is used in photorefractive keratectomy (PRK) or laser in-situ keratomileusis (LASIK) for correction of myopia.

PHOTOEVAPORATION

It is produced by a dye laser which is used in evaporating surface lesions on eyelids mostly for cosmetic purpose.

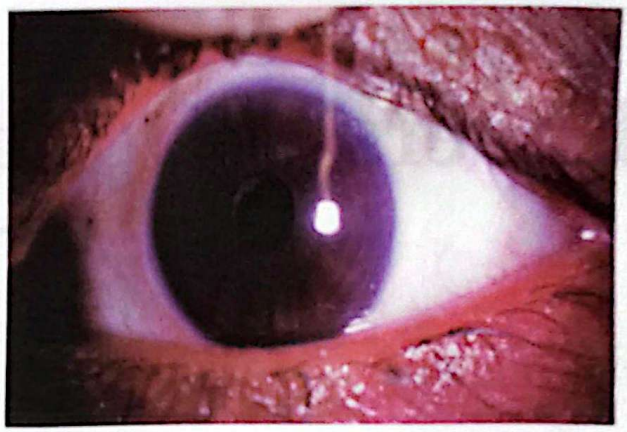

Fig. 25.4 Ablation of the opaque posterior capsule with a YAG laser

Chapter 26

Errors of Refraction

Normally, the total converging power of the eye (60 dioptres) and axial length of the eye (24 mm) are so aligned that parallel rays of light come to a point focus on the retina to see the clear image of the object in view, called emmetropia. Any misalignment between the converging power and axial length of the eye ends up in parallel light rays falling either in front or behind the retina leading to blurred vision, called refractive error or ametropia. Clinical assessment of refractive error is performed by the method of retinoscopy (Fig. 26.1).

Fig. 26.1 Retinoscopy is being performed for assessment of refractive error

HYPERMETROPIA

It is an error of refraction where parallel rays of light come to a point focus behind the retina. A human child is born with +2.5 to +3 D of hypermetropia normally called physiological hypermetropia. When hypermetropia is due to short axial length, it is called axial hypermetropia. When hypermetropia results from decreased corneal convergence power, it is called curvature hypermetropia and when from decreased convergence by lens, it is termed index hypermetropia. Latent hypermetropia is normally neutralised by the tone of ciliary body but becomes manifest in cycloplegia. Hypermetropia is kept corrected to see clearly in young people and is called facultative hypermetropia. The residual hypermetropia which cannot be overcome by effort of accommodation is called absolute hypermetropia. Hypermetropia is corrected by convex (notified as +) glasses, contact lenses or PRK.

MYOPIA

It is an error of refraction where parallel rays of light come to a point focus in front of the retina. Myopia may be due to increased corneal curvature, called curvature myopia, or due to longer axial length, called axial myopia. Myopia resulting from increased converging power of the lens, e.g., in hyperglycaemia and senile nuclear sclerosis, is labeled as index myopia. When myopia is best corrected with glasses, concave glasses (notified as -) it is called simple myopia. When myopia cannot be corrected with glasses due to pathological changes in the retina (myopic degeneration), it is called pathological myopia. In clinical settings, a patient with simple myopia is seen to have refractive error up to 6 dioptres and higher refractive error is seen in pathological myopia but scientifically a low myope may have pathological myopia and a high myope may have simple myopia. Contact lenses, PRK and LASIK are the methods of myopic correction in addition to concave glasses in spectacles. Radial keratotomy

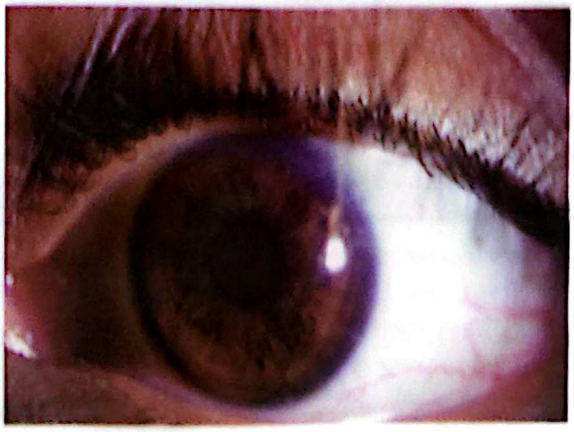

Fig. 26.2 Cart wheel appearance on the cornea after radial keratotomy previously done for myopic correction assessment of refractive error

(Fig. 26.2) was performed with a diamond knife but is now practically replaced by excimer laser keratotomy (PRK and LASIK).

ASTIGMATISM

It is an error of refraction where a point focus can not be formed on the retina but there are two focal lines separated from each other. The distance between these two focal lines is called focal interval. When one focal line falls on the retina and the other behind the retina, the condition is called simple hypermetropic astigmatism. When one focal line falls on the retina and the other in front of the retina, the condition is called simple myopic astigmatism. When both focal lines fall behind the retina, the condition is labelled as compound hypermetropic astigmatism. When both focal lines fall in front of the retina, the condition is termed compound myopic astigmatism. When one focal line falls in front of the retina and the other one behind the retina, the condition is called mixed astigmatism. Astigmatism is corrected with convex cylindrical glasses in hypermetropic astigmatism and concave cylindrical glasses in myopic astigmatism. Correction with contact lenses and refractive surgery are other treatment options.

ANISOMETROPIA

It is a condition of different refractive states in two eyes. Normally up to 3 dioptres of difference can be tolerated but the difference of more than 3 dioptres is not acceptable to the cerebral cortex and thus the fusion of two images each from an eye do not occur due to aniseikonia (different sized images).

APHAKIA

It is a condition of absence of the lens in the pupillary axis. In aphakia, hypermetropia is associated with loss of accommodation. Most common causes of absence of lens are surgical removal of cataractous lens and trauma. A deep anterior chamber and Iridodonesis (shaky iris on movement) are physical signs of aphakia. In modern ophthalmic practice, aphakia is seldom seen because cataract extraction is always followed by intraocular lens implant during the same sitting (primary IOL implantation). The eye with IOL implantation is called pseudophakic.

PRESBYOPIA

It is the inability to read or see near objects distinctly due to decreased amplitude of accommodation as a result of aging process. Presbyopia is seen clinically in emmetropes around 40 years of age when addition of convex lenses for near of +0.75 are prescribed. With growing age +0.50 is added after passage of a 5-year tenure so that at the age of 60 years +2.50 lenses are required.

Chapter 27

Differential Diagnosis

RED EYE

Acute conjunctivitis

Acute keratitis

Acute iridocyclitis

Acute congestive glaucoma

Sub conjunctival hemorrhage

Corneal foreign body

Episcleritis

Scleritis

PSEUDOPROPTOSIS

High axial myopia

Buphthalmos

Asymmetry of orbits

Facial asymmetry

UNILATERAL PROPTOSIS

Thyroid ophthalmopathy

Orbital pseudotumour

Orbital cellulitis

Optic nerve glioma

Hemangioma

Dermoid cyst

Retrobulbar Hemorrhage

BILATERAL PROPTOSIS

Thyroid ophthalmopathy

Orbital pseudotumour

Cavernous sinus thrombosis.

Leukaemia

MADAROSIS

Blepharitis

Trachoma

Burns

Following radiation

Chronic skin diseases

Idiopathic

TRICHIASIS

Trachoma

Spastic entropion

Blepharitis

Burns

Trauma

LACRIMATION

Reflex trigeminal irritation

Keratitis

Iritis

Cyclitis

Glaucoma

Reflex visual irritation

Reflex psychogenic stimulus (crying)

Acute congestive glaucoma

Trichiasis

Entropion

Conjunctivitis

EPIPHORA

Punctal stenosis

Punctal eversion

Punctal occlusion

Canaliculitis

Lacrimal Sac stenosis

Lacrimal sac atrophy

Nasolacrimal duct obstruction

Inferior turbinate hypertrophy

Lower lid ectropion

Facial nerve palsy

DRY EYE SYNDROME (KERATOCONJUNCTIVITIS SICCA)

Sjögren's syndrome

Sarcoidosis

Lymphoma

Leukaemia

Amyloidosis

Vitamin A deficiency

Trachoma

Stevens-Johnson syndrome

Ocular pemphigoid

Chemical burns

Chronic conjunctivitis

Radiotherapy

Exposure keratitis

FOLLICULAR CONJUNCTIVITIS

Adeno viral keratoconjunctivitis

Herpes simplex conjunctivitis

Molluscum contagiosum

Trachoma

Inclusion conjunctivitis

Toxic reaction to topical instillation of drugs

Pilocarpine

Idoxuridine

Physostigmine

NODULE AT THE LIMBUS (FIG. 27.1)

Pinguecula

Pterygium

Phlyctenula

Vernal keratoconjunctivitis

Episcleritis

Scleritis

Papilloma

Granuloma

Dermoid

Epithelioma

Squamous cell carcinoma

CILIARY CONGESTION

Acute keratitis

Acute iritis

Acute iridocyclitis

Cyclitis

Glaucoma

Reflex visual irritation

Reflex psychogenic stimulus (crying)

Acute congestive glaucoma

Trichiasis

Entropion

Conjunctivitis

EPIPHORA

Punctal stenosis

Punctal eversion

Punctal occlusion

Canaliculitis

Lacrimal Sac stenosis

Lacrimal sac atrophy

Nasolacrimal duct obstruction

Inferior turbinate hypertrophy

Lower lid ectropion

Facial nerve palsy

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NODULE AT THE LIMBUS (FIG. 27.1)

Pinguecula

Pterygium

Phlyctenula

Vernal keratoconjunctivitis

Episcleritis

Scleritis

Papilloma

Granuloma

Dermoid

Epithelioma

Squamous cell carcinoma

CILIARY CONGESTION

Acute keratitis

Acute iritis

Acute iridocyclitis

Acute congestive glaucoma

Trauma

DIMINISHED CORNEAL SENSATION

Herpes simplex keratitis

Herpes zoster ophthalmicus

Neuroparalytic keratitis

Trigeminal neuralgia

Exposure keratitis

Contact lens wearer

Corneal dystrophies

PANNUS FORMATION

Trachoma

Phlycten

Vernal conjunctivitis

Use of contact lens

Blind degenerative eyes

Fuchs' dystrophy

DEEP VASCULARISATION

Syphilitic interstitial keratitis

Tubercular interstitial keratitis

Diabetes mellitus

Chemical injury

PERIPHERAL CORNEAL ULCERS

Marginal keratitis (catarrhal ulcer)

Rosaceae keratitis

Phlyctenular keratitits

Mooren's ulcer

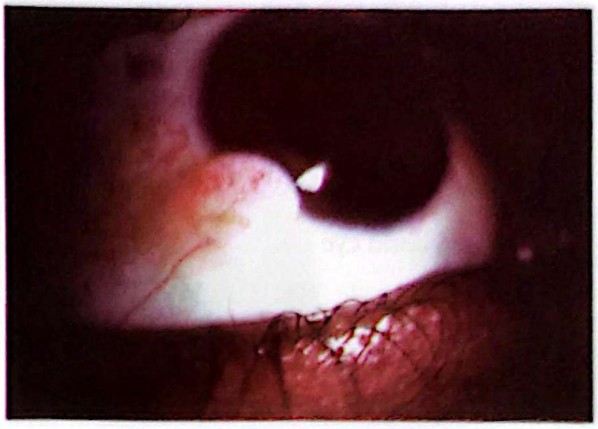

Fig. 27.1 Nodule at the Limbus

DEEP KERATITIS

Herpetic stromal keratitis

Disciform keratitis

Keratitis profunda

CAUSES OF HYPOPYON ULCER

Pneumococcus

Pseudomonas pyocyanea

Moraxella

E–coli

Streptococci

MICROCORNEA

Congenital

Ehlers- Danlos syndrome

Weill-Marchesani's syndrome

Nanophthalmos

Rubella syndrome

MEGALOCORNEA

Congenital

Buphthalmos

Keratoglobus

Marfan's Syndrome

Lowe's Syndrome

RUBEOSIS IRIDIS

Central retinal vein occlusion

Diabetes mellitus

Eales disease

Degenerated blind eye

Uveitis

Absolute glaucoma

Longstanding RD

Intraocular tumours

DEEP ANTERIOR CHAMBER

Aphakia

High Myopia

Buphthalmos

Posterior dislocation of the lens

SHALLOW ANTERIOR CHAMBER

Angle closure glaucoma

Intumescent cataract

Adherent leukoma

High hypermetropia

MYDRIASIS

Physiological

Women

Myopia

People with blue iris

During emotional state

Pathological

Glaucoma

Trauma

3rd nerve palsy (pupil non-sparing)

Optic neuritis

Optic atrophy

Amblyopia

Mydriatics

MIOSIS

Physiological

Men

Hypermetropes

In brown eyes

In the very young and the old

Pathological

Iritis

Miotics

Orbital lesions

Horner's syndrome

Pontine lesions

LEUKOCORIA (FIG. 27.2)

Cataract

Retinoblastoma

Endophthalmitis

Retinal detachment

Cyclitic membrane

Coats' disease

Persistent hyperplastic primary vitreous

Retinopathy of prematurity

Chorioretinal coloboma

Vitreous bands

SUDDEN PAINLESS LOSS OF VISION

Central retinal artery occlusion

Central retinal vein occlusion

Vitreous haemorrhage

Retinal detachment

Optic neuritis

SUDDEN PAINFUL LOSS OF VISION

Acute congestive glaucoma

Acute iridocyclitis

Acute keratitis

Chemical injury

Mechanical injury

GRADUAL PAINLESS LOSS OF VISION

Cataract

Primary open angle glaucoma

Age related macular degeneration

Diabetic retinopathy

Retinitis pigmentosa

Corneal dystrophy

Optic atrophy

Keratitis

Uveitis

Scleritis

UNIOCULAR DIPLOPIA

Dislocated lens

Immature cataract

Polycoria

BINOCULAR DIPLOPIA

Myasthenia gravis

Thyroid ophthalmopathy

Blow out fracture

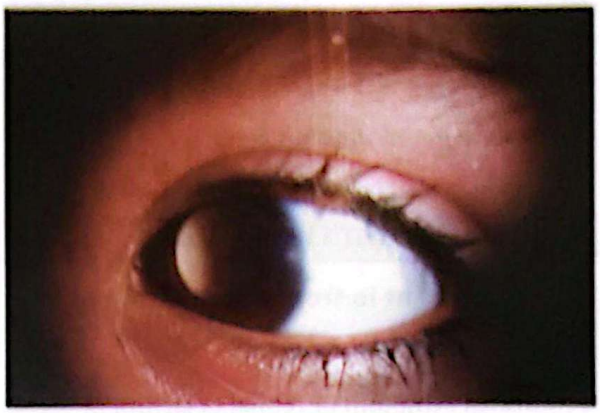

Fig. 27.2 Leukocoria

3rd nerve palsy

4th nerve palsy

6th nerve palsy

HALOES

Acute congestive glaucoma

Intumescent cataract

Mucopurulent conjunctivitis

NIGHT BLINDNESS

Vitamin-A deficiency

Retinitis pigmentosa

Primary open angle glaucoma

Peripheral cortical cataract

Panretinal photocoagulation

DAY BLINDNESS

Central nuclear cataract

Central corneal opacity

Central vitreous opacity

Congenital deficiency of cones (rare)

FLOATERS

(Black spots moving in front of the eyes)

Vitreous haemorrhage

Vitreous degeneration

Choroiditis (exudates in vitreous)

Posterior vitreous detachment

PHOTOPSIA

(flashes of light in front of the eyes)

Posterior vitreous detachment

Prodromal symptom of retinal detachment

Vitreous traction bands

PAINFUL RED EYE

Keratitis

Iridocyclitis

Acute congestive glaucoma

Scleritis

Corneal foreign body

CONJUNCTIVAL FOLLICLES

Viral conjunctivitis

Chlamydial conjunctivitis

Hypersensitivity to topical medication

CONJUNCTIVAL PAPILLAE

Trachoma

Allergic conjunctivitis

Contact lens wear

HYPOPYON

Bacterial corneal ulcer

Fungal corneal ulcer

Iridocyclitis

Endophthalmitis

Panophthalmitis

Pseudohypopyon

CHERRY RED SPOT

Central retinal artery occlusion

Tay–Sachs disease

Niemann–Pick disease

Commotio retinae

RETINAL HAEMORRHAGE

Diabetic retinopathy

Hypertensive retinopathy

Central retinal vein occlusion

Branch retinal vein occlusion

Eales disease

Sickle cell disease

Retinopathy of AIDS

Age related macular degeneration

COTTON WOOL SPOTS (SOFT EXUDATES) IN THE RETINA

Hypertensive retinopathy

Diabetic retinopathy

Central retinal vein occlusion

Retinopathy of AIDS

HARD EXUDATES ON THE RETINA

Diabetic retinopathy

Hypertensive retinopathy

Retinal vein occlusion

Papilledema

Coats' disease

NEOVASCULARISATION OF THE RETINA

Diabetic retinopathy

Central retinal vein occlusion

Branch retinal vein occlusion

Central retinal artery occlusion

Sickle cell retinopathy

Eales disease

TUBULAR VISION

Glaucoma in advanced stage

Retinitis pigmentosa

Panretinal photocoagulation

CENTRAL SCOTOMA

Optic neuritis

Compressive optic neuropathy

Tobacco amblyopia

Macular hole

BITEMPORAL HEMIANOPIA

Pituitary tumour

Suprasellar aneurysms

Craniopharyngioma

Glioma of the third ventricle

HOMONYMOUS HEMIANOPIA

Optic tract lesion

Lateral geniculate body lesion

Lesions involving total fibres of optic radiation

Visual cortex lesion (usually sparing of the macula)

DISC OEDEMA

Papillitis

Papilloedema

Ocular hypotony

Central retinal vein occlusion

Tumours of the optic nerve

Thyroid ophthalmopathy

Metastasis – Leukaemia

PAPILLOEDEMA

Intracranial space occupying lesions – tumour, abscess and cyst

Impaired CSF circulation – aqueductal stenosis

Meningitis

Benign intracranial hypertension

Malignant systemic hypertension

Vitamin-A toxicity

Chapter 28

Instruments

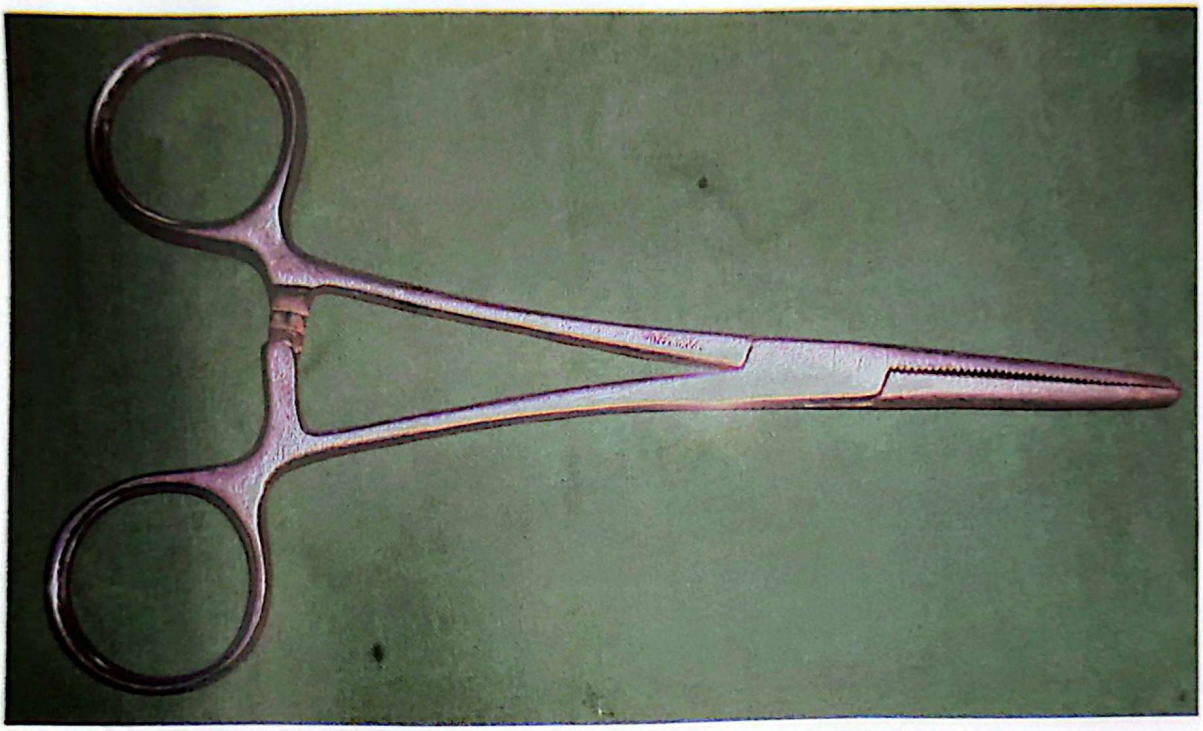

Fig. 28.1 Artery Forcep straight: Used for grabbing a bleeding vessel during eye surgery, and to hold the skin and muscle.

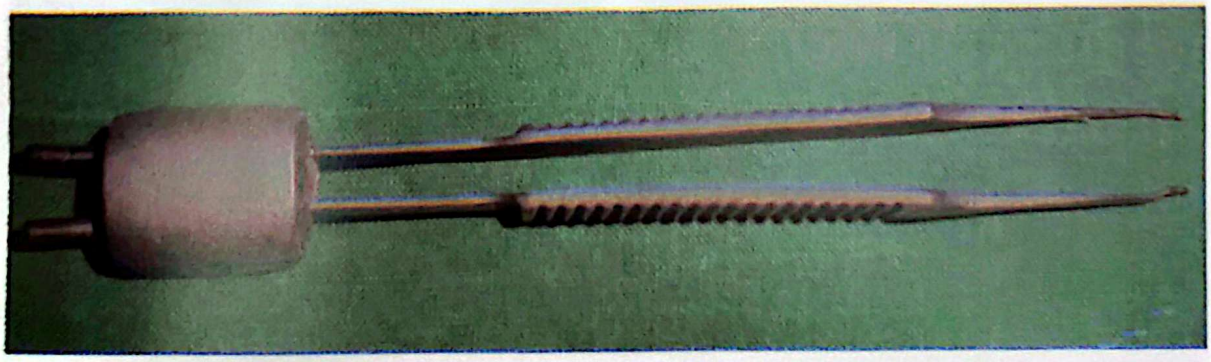

Fig. 28.2 Bipolar Cautery Forcep: Used for cauterisation of bleeding vessels to stop bleeding during surgery

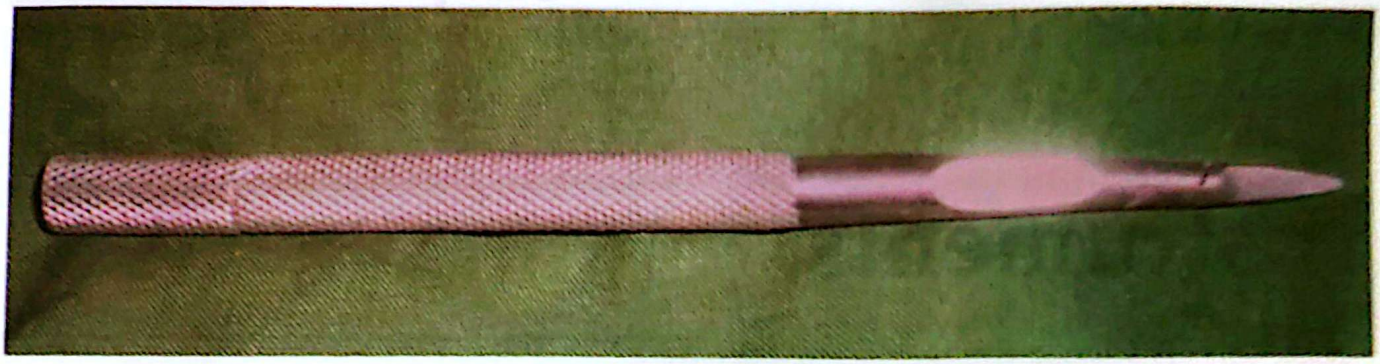

Fig. 28.3 Blade Breaker Holding Knife used in Superficial Corneal and Scleral Incisions

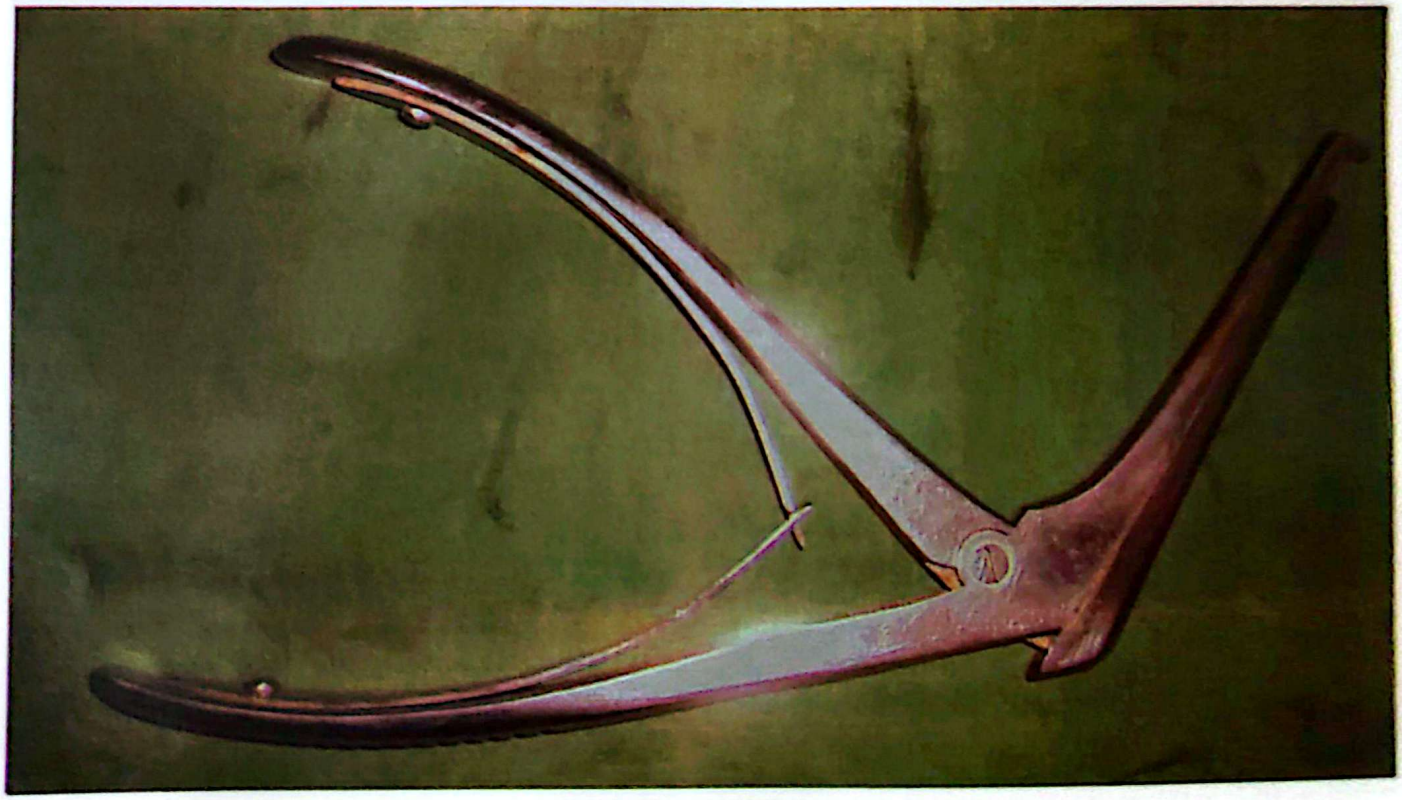

Fig. 28.4 Bone punch used for formation of a hole in the nasal wall during DCR surgery

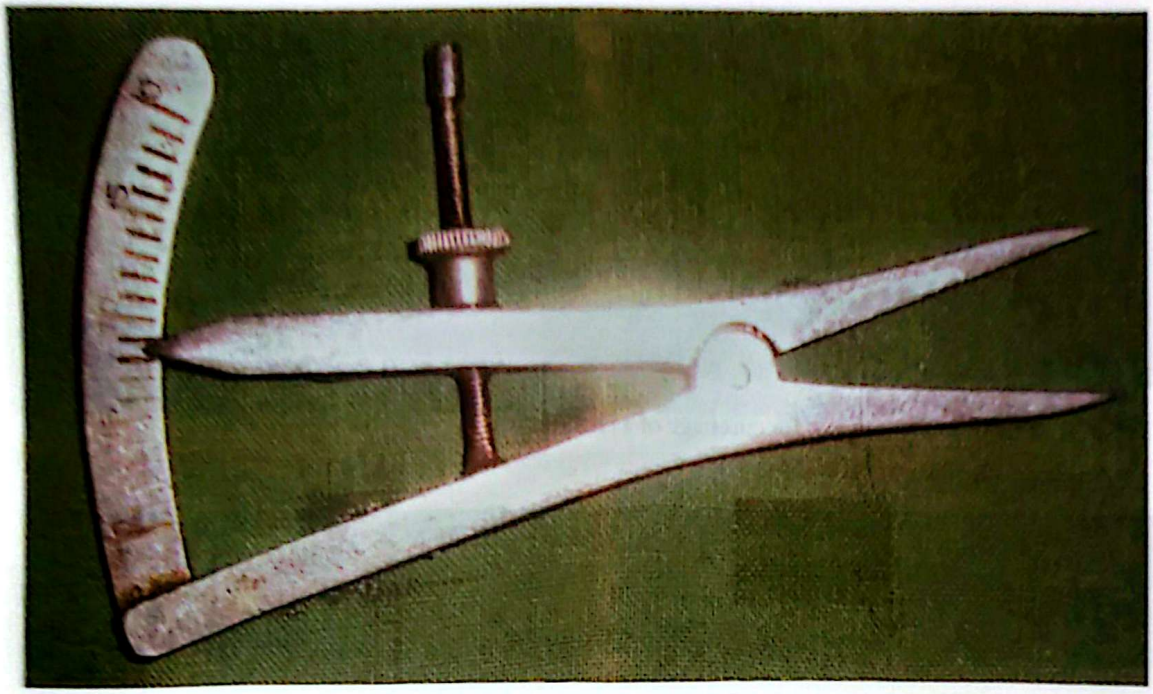

Fig. 28.5 Castroviejo Caliper utilised for measurements during surgery of a squint, ptosis, retinal detachment and pars plana vitrectomy

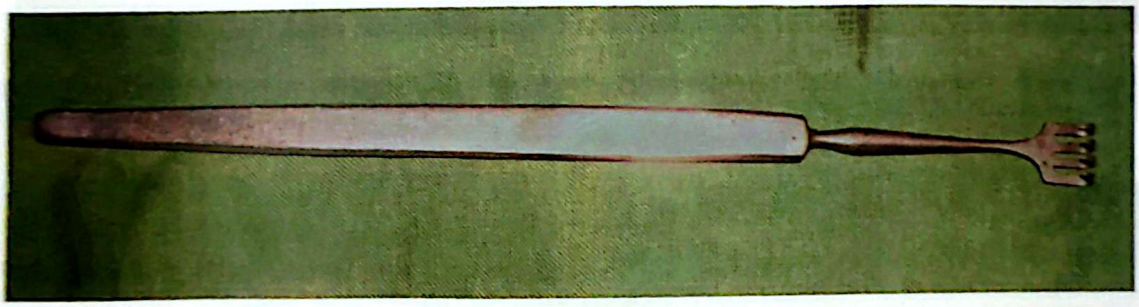

Fig. 28.6 Cat's paw lacrimal wound retractor: Used for retracting the soft tissue during DCR and Lid Surgery

Fig. 28.7 Chalazion Clamp: Used for holding and everting a chalazion

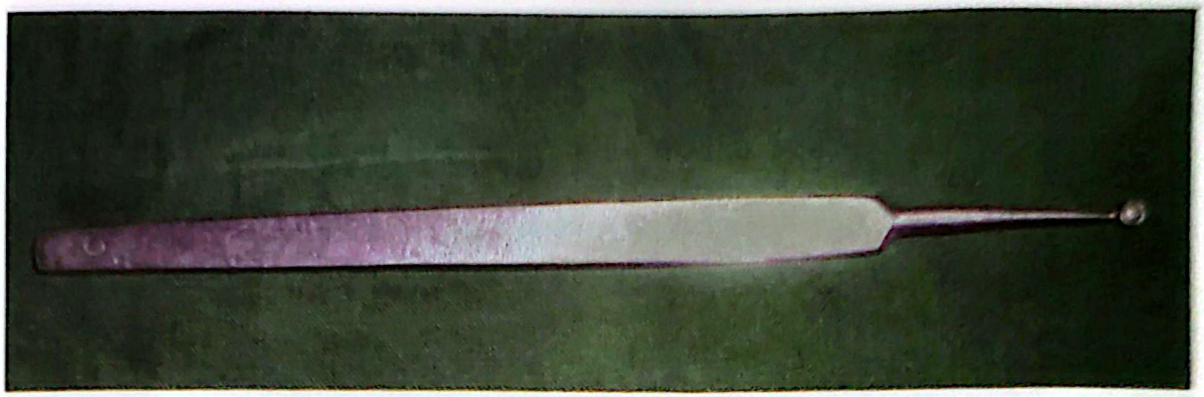

Fig. 28.8 Chalazion Scoop. Used for curettage of a chalazion

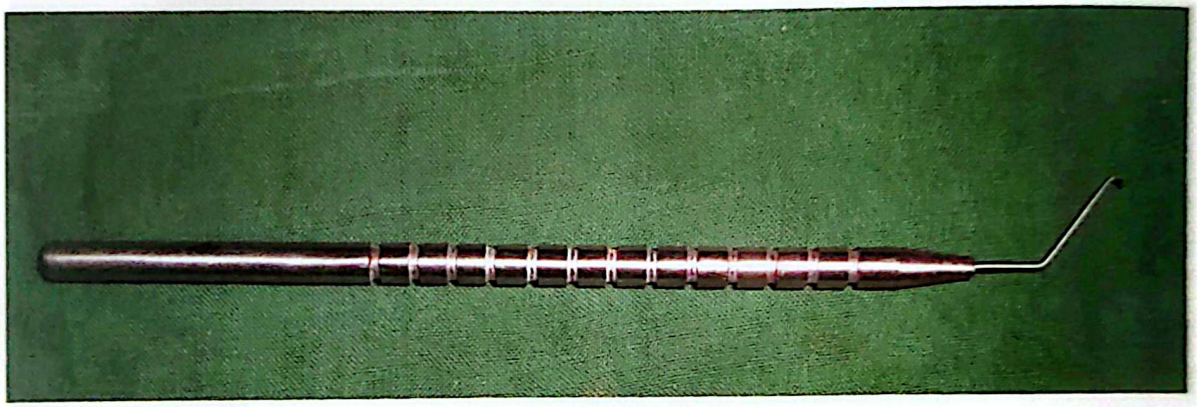

Fig. 28.9 Chopper used for holding and chopping the lens nucleus during phacoemulsification surgery

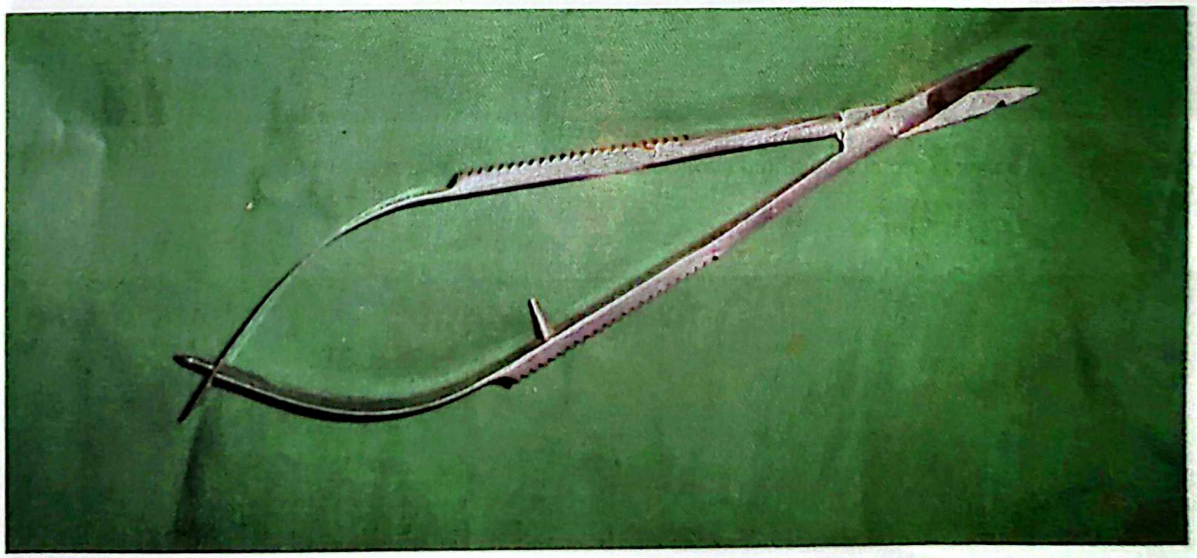

Fig. 28.10 Conjunctival scissor. Used for cutting the conjunctiva

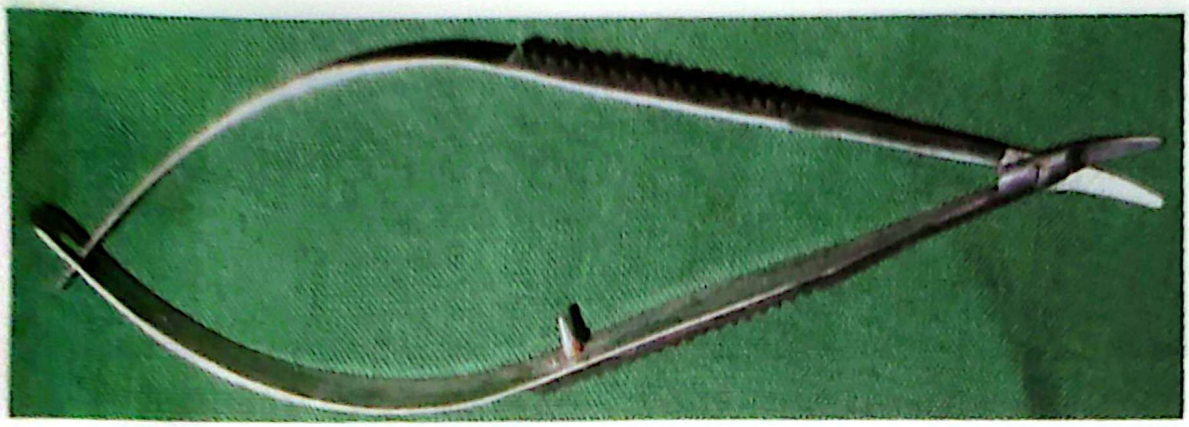

Fig. 28.11 Corneal Scissors: used for full thickness corneal incision during cataract surgery

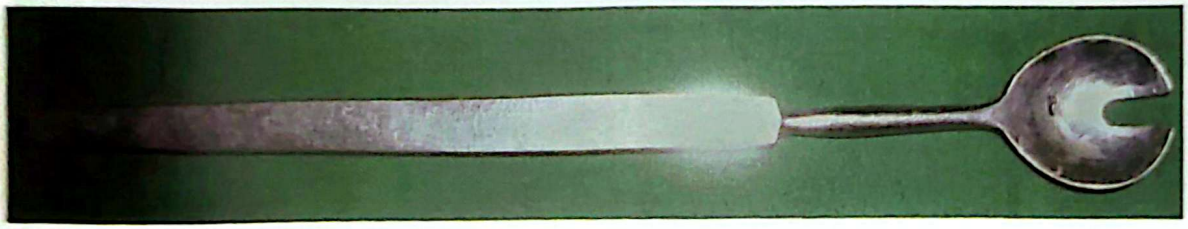

Fig. 28.12 Enucleation Scoop used for holding the optic nerve during enucleation

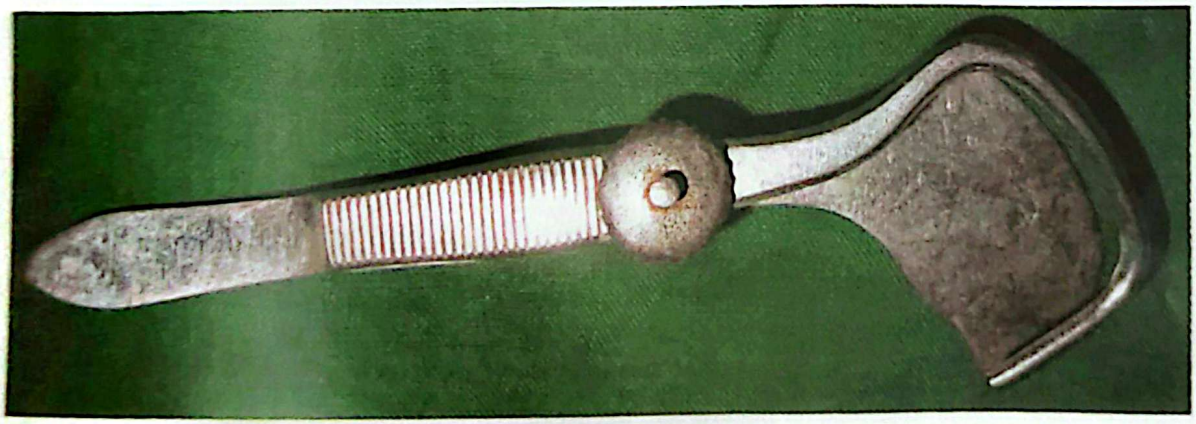

Fig. 28.13 Entropion Clamp used during entropion surgery

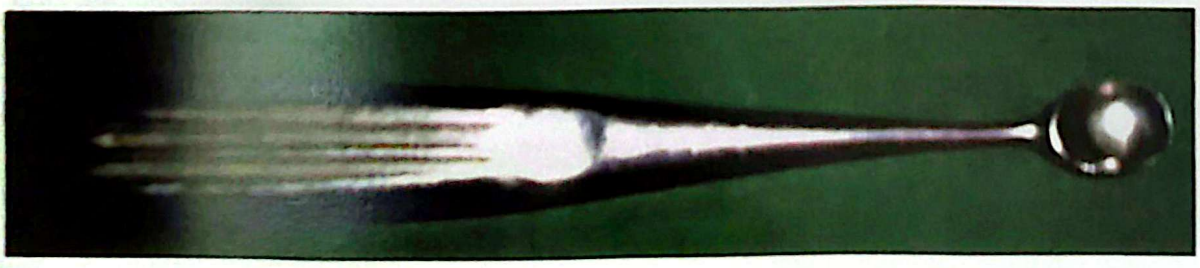

Fig. 28.14 Evisceration scoop: Used for removal of intraocular contents during evisceratio

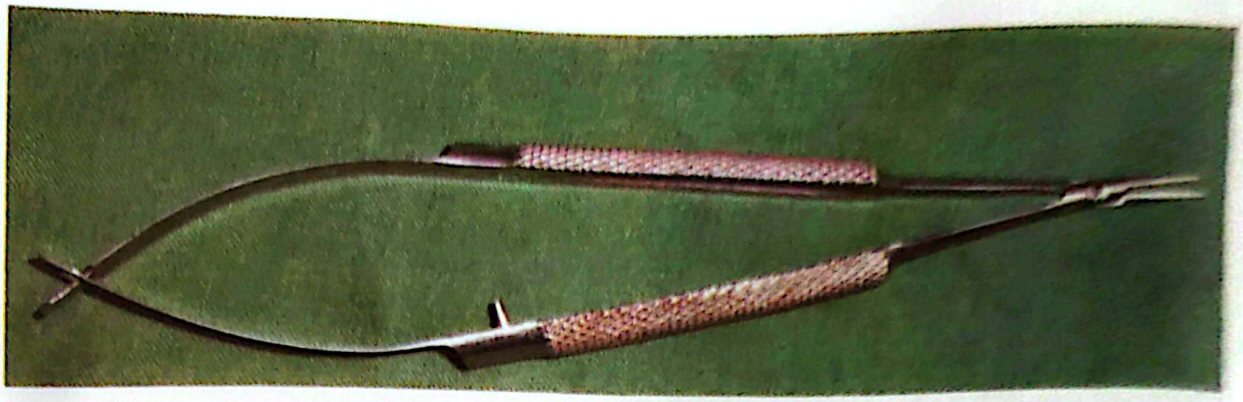

Fig. 28.15 Fine needle holder. Used for holding fine needles and sutures

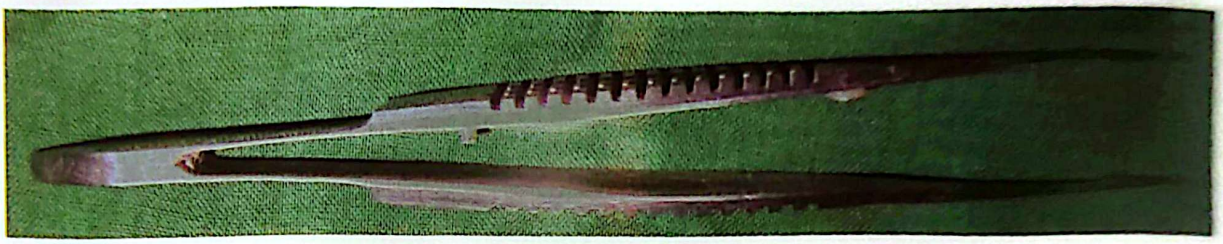

Fig. 28.16 Fine plain forcep used for tying the fine sutures and holding conjunctiva

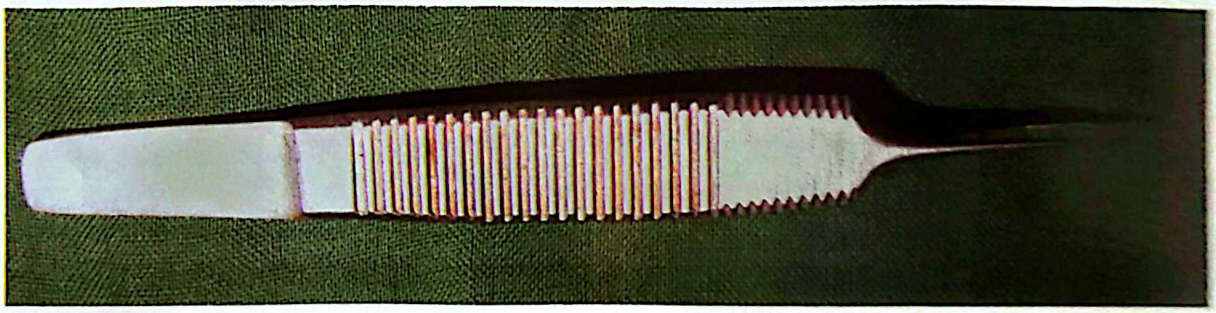

Fig. 28.17 Fine tooth forceps used for holding the cut edge of a cornea

Fig. 28.18 Iris Repositor: Used for repositioning a prolapsed Iris

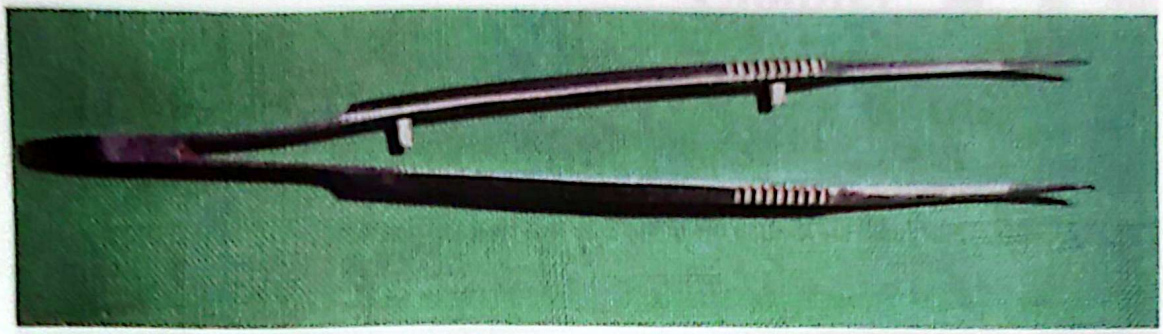

Fig. 28.19 Kelman-McPherson Forceps used for tying the fine sutures and placing IOL

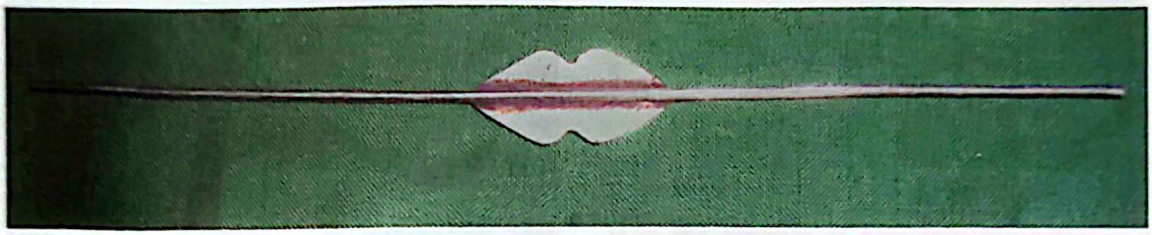

Fig. 28.20 Lacrimal Probe used for probing a blocked nasolacrimal duct

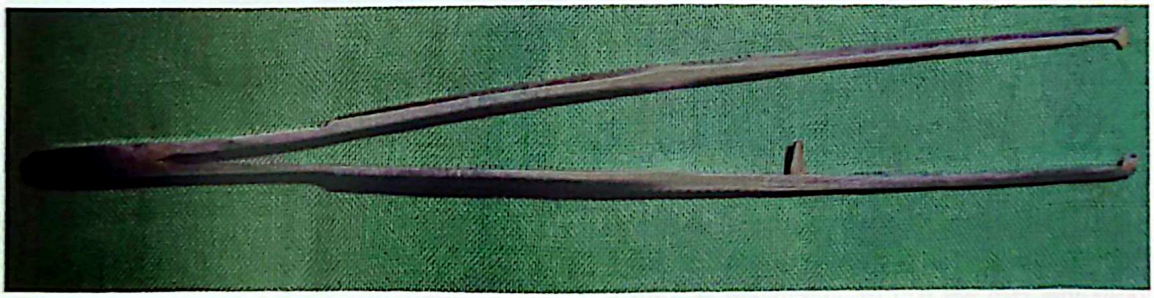

Fig. 28.21 Large Tooth Forcep used for holding the lid and rectus muscle tendon

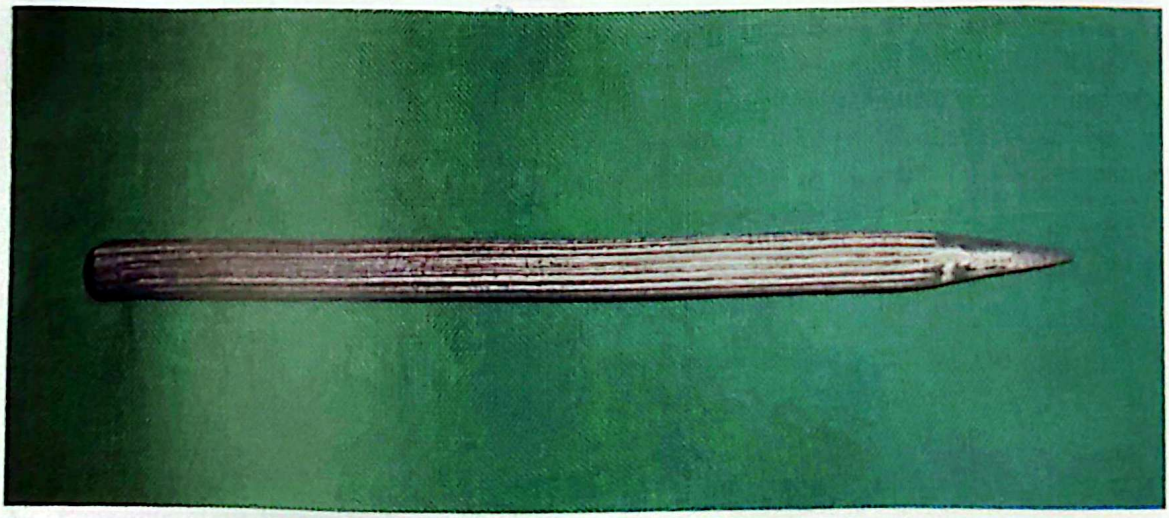

Fig. 28.22 Lacrimal Punctum Dilator used to dilate the lacrimal punctum

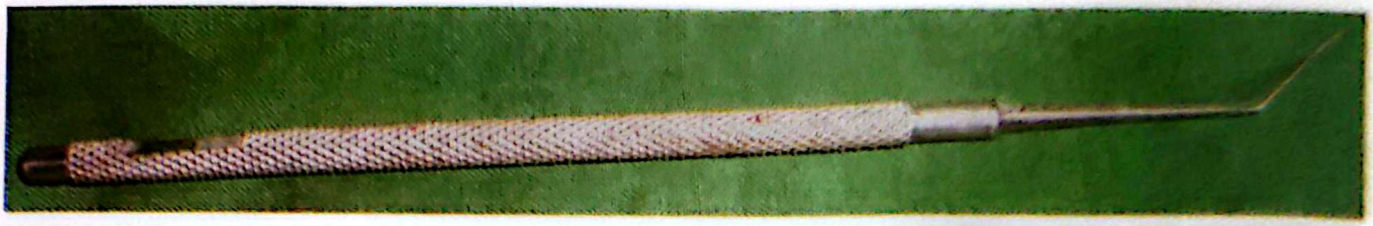

Fig. 28.23 Lens Dialer used for rotating the intraocular lens (IOL) in position

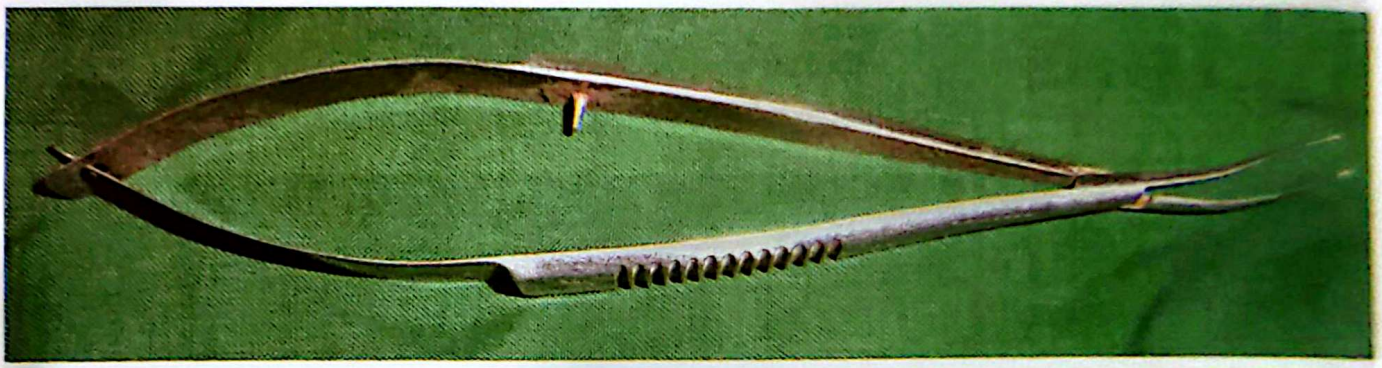

Fig. 28.24 Lens Holding Forceps used for intraocular lens (IOL) implantation

Chapter 29

Community Ophthalmology

Fig. 29.1 A Glimpse of a Hajj Congregation

Community ophthalmology can be defined in simple terms as the ophthalmic health care offered to a population instead of an individual patient, e.g., applying measures to prevent the spread of contagious eye diseases during Hajj congregation (Fig. 29.1). Community ophthalmology service requires team work which includes ophthalmologists, optometrists, orthoptists, ophthalmic nurses, opticians, general practitioners (GPs), technicians, health care assistants and pharmacists.

The basic purpose of practicing community ophthalmology is to assess the severity of the problem of eye diseases. This is done by carrying out cross-sectional studies or surveys to collect the basic data. Once we have assessed the prevalence and incidence of various eye diseases in the population, then we have three goals:

1. Promote normal vision
2. Preserve healthy eyes
3. Prevention of eye diseases leading to blindness

Prevalence

It is the number of cases of a disease at a particular time and this is divided by the population at risk.

Incidence

It is the number of new cases occurring in a given time period and this is divided by the total population at risk at the beginning of the period. The time period is conveniently taken as one year and the population at risk is usually taken at the beginning of the period of the measurement, or sometimes at the end of the follow up period.

Cross- sectional studies

In a cross-sectional study a sample is drawn from a defined population and investigated in as short a time as practicable, so that the data will show the status of the sample members at a particular point in time. The findings are then used to make estimates for the population.

The usual primary objectives are to estimate the prevalence of disease for the population and to assess the need for the health services. Generally the main aim is to provide the necessary data for planning future health services and implementing programs accordingly.

There are two methods for survey or cross-sectional studies:

1. Simple Random Sampling
2. Cluster Sampling

Simple random sampling

The easiest way to understand this method is to consider a population which is listed; for example a voters list of a town or a village, and we select and examine every 10th or every 20th person from that list. Similarly, the list of persons attending an eye camp; and we select and examine every 5th person and compile a data of diseases of the eyes prevailing in that population. This is a random selection and everyone on the list had a chance of being selected initially. The list of the target population is known as the sampling frame and each member in the list is called a sampling unit.

This sampling can be used to carry out studies to assess the general pattern of eye diseases in the population or to see the prevalence of one particular disease such as glaucoma or diabetic retinopathy.

Cluster sampling

In cluster sampling population members are divided into clusters of individuals and a sample of clusters is chosen to yield a sample of the population members.

There are three main varieties of cluster sampling.

1. **One Stage Cluster Sampling:** The population is divided into a number of primary clusters and no further division is carried out. All the population members within the cluster are included in the final sample.
2. **Two Stage Cluster Sampling:** In this procedure, a number of clusters are selected from the primary clusters but the individuals within a chosen cluster are selected according to the method used in simple random sampling.
3. **Multi-Stage Cluster Sampling:** This method is used when the study has to be carried out in a big region or country. First this is divided into large primary sampling units like provinces and then smaller convenient clusters are chosen. Out of these clusters members of the population are selected by simple random selection. Regarding community ophthalmology a few parameters should be known for day to day usage while carrying out various studies:

BLINDNESS AND LOW VISION

Blindness

It is defined as a best corrected visual acuity of 3/60 in the better eye or a visual field in each eye to less than 10 degree from the point of fixation.

Low vision

It is defined as best corrected visual acuity (BCVA) of less than 6/18 but equal to or better than BCVA of 3/60 in the better eye. It has been divided into various categories.

Category 1 BCVA 6/18 to 6/60

Category 2 BCVA 6/60 to 3/60

Category 3 BCVA 3/60 to 1/60 or finger counting at 1 meter

Category 4 BCVA 1/60 to perception of light

Category 5 No perception of light

Chapter 30

Ocular Emergencies

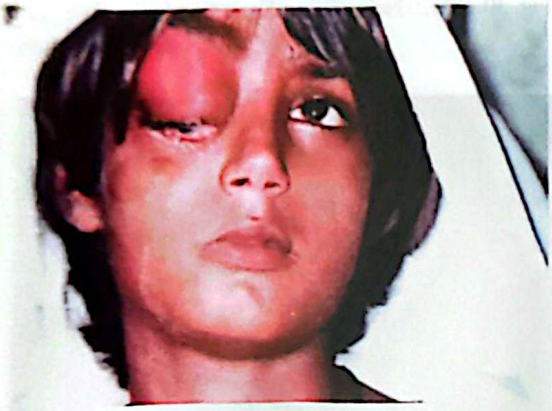

Fig. 30.1 Orbital cellulitis

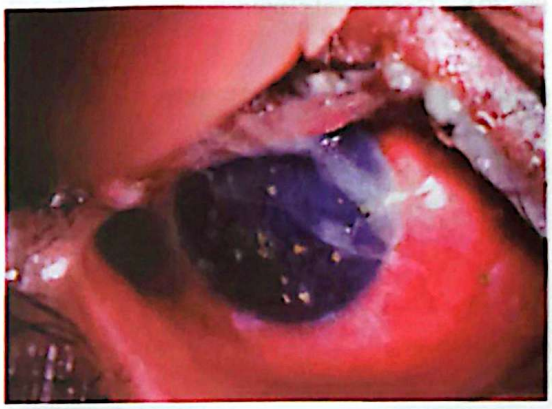

Fig. 30.3 Cracker blast trauma of the globe

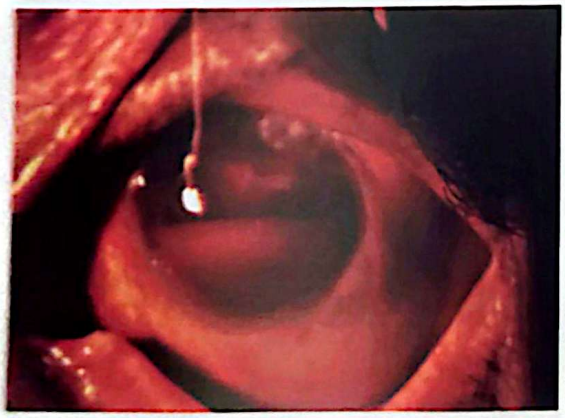

Fig. 30.2 Hyphaema

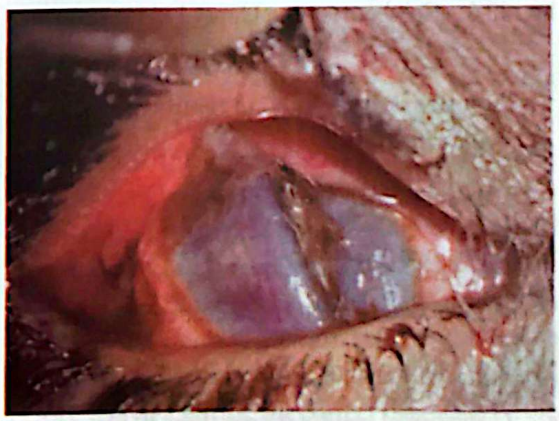

Fig. 30.4 Severely damaged eyeball due to penetrating trauma

Although medical students may erroneously think that emergencies do not exist in clinical ophthalmic practice; indeed, they do exist. Following are the ocular emergencies.

- Central retinal artery occlusion
- Chemical burns of the eyes (Fig. 30.12)
- Acute congestive glaucoma
- Endophthalmitis (Fig. 30.13)
- Orbital cellulitis (Fig. 30.1)
- Perforation of the eyeball (Fig. 30.3, 30.4)
- Cavernous sinus thrombosis

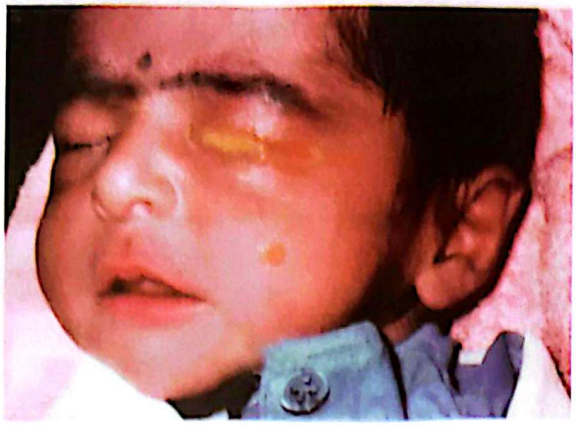

Fig. 30.5 Gonococcal conjunctivitis

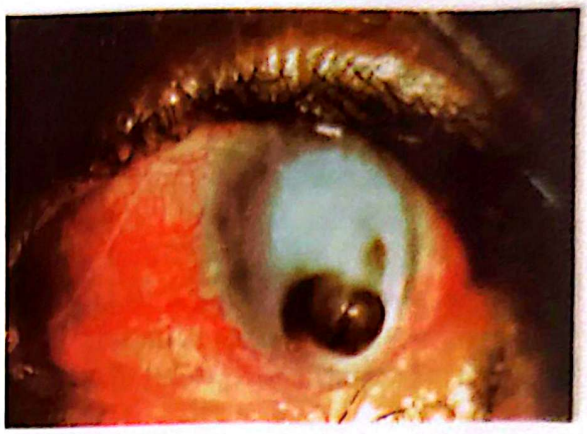

Fig. 30.7(A) Necrotising Keratitis

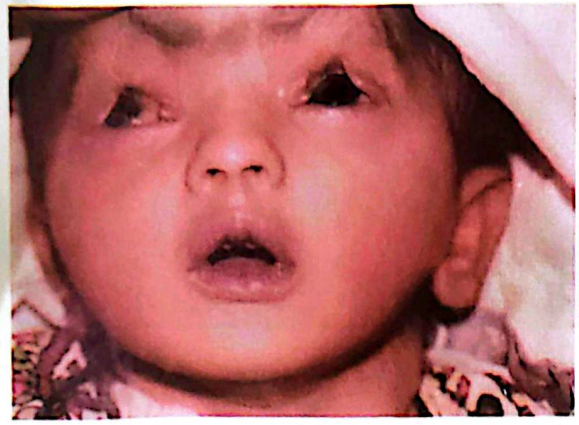

Fig. 30.6 Coloboma of the Upper Lid

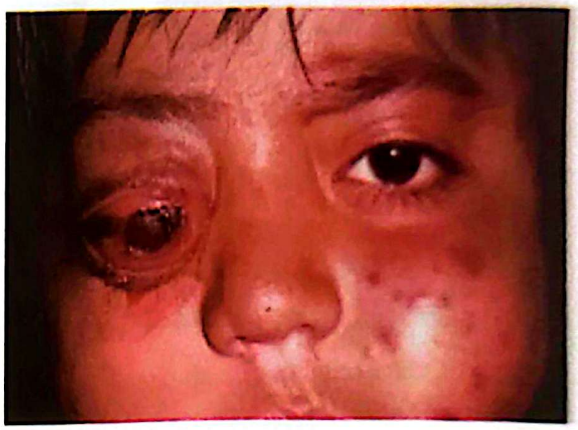

Fig. 30.7(B) Acute Myeloid Leukemia

- Anterior ischaemic optic neuropathy due to giant cell arteritis
- Central retinal vein occlusion
- Optic neuritis
- Retinal detachment threatening the macula
- Retinal dialysis
- Gonococcal conjunctivitis (Fig. 30.5)
- Necrotising keratitis (Fig. 30.7 A)
- Lid cuts exposing the cornea
- Coloboma of the lid involving more than half of the upper lid (Fig. 30.6)
- Proptosis with corneal exposure
- Retraction of the lid with corneal exposure (Fig. 30.8, 30.9)
- Bilateral advanced open angle glaucoma
- Intraepithelial carcinoma of conjunctiva (Fig. 30.10)
- Intraocular tumours
- Acute lymphocytic *leukaemia* (ALL)
- Acute myeloid *leukaemia* (AML) (Fig 30.7 B)
- Anisometropia in children under 5 years of age
- Unilateral ptosis covering the pupil, in children under 5 years of age (Fig. 30.11)

CHEMICAL BURNS OF THE EYE

Involving conjunctiva and cornea pose a real emergency in clinical ophthalmic practice. Alkali burns cause more damage than acid burns because of their rapid intraocular penetration through

Fig. 30.8 Right corneal opacification due to chronic exposure

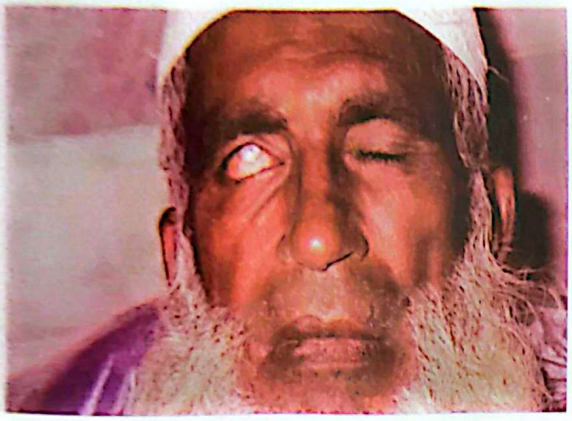

Fig. 30.9 Lid retraction as a cause of chronic exposure keratopathy

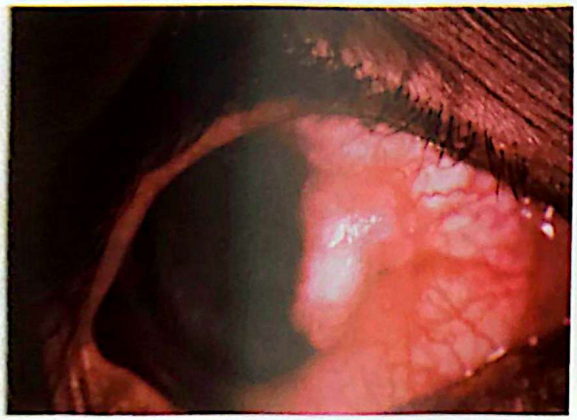

Fig. 30.10 Intraepithelial carcinoma of conjunctiva

cornea. Acid burns cause rapid initial damage by coagulation of proteins, thereby producing a sheet of necrotic tissue which does not allow further penetration of acid. Alkalis soften the tissue by disrupting cell membranes and keep on

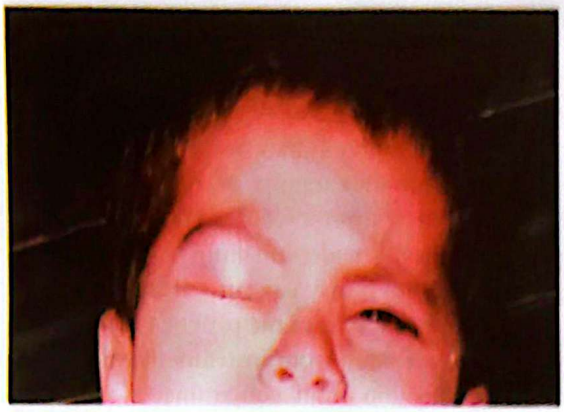

Fig. 30.11 Right unilateral ptosis

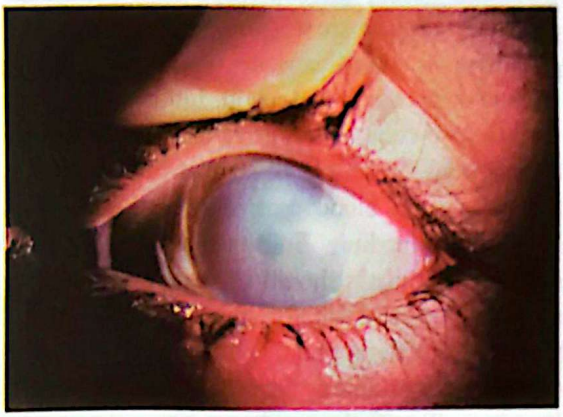

Fig. 30.12 Chemical burns of the eye

penetrating into the deeper tissues till removed. Treatment includes immediate copious irrigation with an available source of water because the extent of permanent damage is directly related to the time lapse preceding irrigation. Irrigation in the hospital should be done by everting the lids to remove any hidden particle in addition to conjunctival drip. Litmus paper should be utilised, before use of a buffer, for correct diagnosis of the type of chemical, i.e., acid or alkali, as the history may be misleading. The treatment of complications should be instituted, including infection, uveitis, glaucoma and cataract. A bandage contact lens may be placed over the cornea whenever required. Chemical burns may lead to scarring, ending up in corneal opacification, dry eye, symblepharon and entropion requiring multiple surgeries (Fig. 30.12).

ENDOPHTHALMITIS

It is vision threatening inflammation of intraocular tissues and should be treated vigorously and timely to save useful vision. It may occur postoperatively, due to penetrating trauma or through a blood borne infection from a focus away from the eye, called metastatic endophthalmitis. Postoperative endophthalmitis may occur from airborne infection, infection from the patient's conjunctiva or eyelids, infection from instruments, drapes or sutures, infection from contaminated irrigating fluid or gel and infection from an assistant or a surgeon's gloves. Patient with postoperative bacterial endophthalmitis presents with pain, decreased vision, lid oedema, and conjunctival chemosis on the first postoperative day. On examination, there is loss of fundus reflex, corneal oedema and hypopyon. Lid margin swab, conjunctival swab, anterior chamber paracentesis and vitreous tap should be sent to the laboratory for culture on blood agar, chocolate agar and Sabouraud's medium. Smears from conjunctival or corneal exudates should be seen with a Gram stain in the ward lab for immediate treatment with appropriate antibiotic. Intravitreal injection of a diluted antibiotic like vancomycin should be given. Fortified eye drops like tobramycin and ofloxacin should be instilled with one-hour interval into the conjunctival sac in addition to atropine to relax the ciliary body and pupil. Early surgical intervention with vitrectomy is a useful tool to save the eye (Fig.30.13).

Fig. 30.13 Endophthalmitis

Chapter 31

Optics

Optics is a branch of physical science which deals with the study of light and its interactions with matter. A Muslim scientist named Abu Ali al-Hassan Ibn al-Haytham (965-1040 C.E.) is considered the father of modern optics because of his outstanding contributions to optics and scientific methods. In this chapter, we shall be approaching the science of optics in a systematic way and for that purpose we have divided this chapter into three sections, i.e., general optics, physiological optics and clinical optics.

GENERAL OPTICS

Electromagnetic Spectrum

It is the entire frequency range of electromagnetic waves from the shortest ionising radiation (10^{-16} m) to the longest radio waves (10^{6} m). In this chapter our focus will be on the visible part of this spectrum, called visible light.

Visible Light

It is a small fraction of the electromagnetic spectrum which gives visual sensation to the human eye, having a wavelength of approximately between 400 and 700 nm. The main source of visible light is the Sun.

Nature of Light

In optics, we are supposed to study about the properties and behaviour of light but ironically until now scientists are unable to understand what actually light itself is. Should we consider light a wave or a particle is a question that cannot be answered clearly because in some instances light behaves like a wave having frequency, speed and wavelength and in other instances it behaves like a particle, called a photon, which is a discrete packet of energy. It seems that light possesses dual nature, i.e., in composition it has a particle-like nature but in propagation it has a wave-like nature. But for the purpose of simplification, light is diagrammatically represented as a straight arrowed line called a ray.

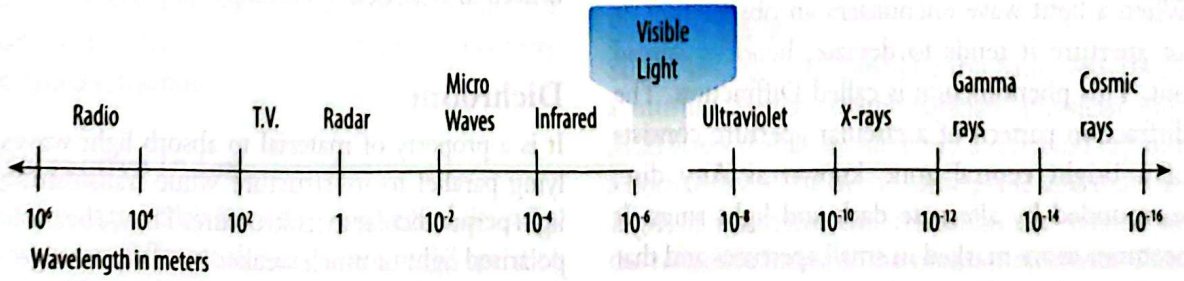

Fig. 31.1 Electromagnetic Spectrum

Properties of Light

Unlike the lack of understanding in the nature of light, the properties and behavior of light is well understood and extensively studied. Light waves are known to have their specific wavelength, frequency, direction of propagation, orientation of electrical field and a certain amount of energy within individual quanta. Shorter wavelengths have a higher frequency and greater energy.

Coherent Light

When all waves of light are exactly in phase, i.e., their crests fall on each other and so do their troughs, it is said to be coherent light. Laser light is an example of coherent light. Light waves that are out of phase are called incoherent and the fraction of a wavelength by which one wave leads the other is called the phase difference.

Interference

If two light waves travelling along the same path are in phase, their resultant wave is a summation of the two waves (i.e., brighter light), this type of interference is called constructive interference. If two light waves of equal amplitude are out of phase by half of a wavelength, they cancel each other out (i.e., no light), it is called destructive interference. If the phase difference between two waves is less than half of a wavelength, then the resultant wave will be of intermediate amplitude and phase. Anti-reflective coating is an example of destructive interference; and Optical Coherence Tomography (OCT) uses interference to detect changes in the coherence of light.

Diffraction

When a light wave encounters an obstruction or an aperture it tends to deviate, bend or spread out. This phenomenon is called Diffraction. The diffraction pattern of a circular aperture consists of a bright central zone known as Airy disc, surrounded by alternate dark and light rings. It becomes more marked in small apertures and that is why, with smaller pupils, an imperfect image is formed and visual acuity is reduced.

Scattering

When light interacts with particles of similar wavelength it is scattered. The shorter the wavelength, the higher is the scattering. The sky looks blue because the gas molecules around 18 miles above the earth's surface scatter blue light. With larger and irregularly spaced particles all wavelengths are scattered, that is why the clouds look white. In corneal oedema, corneal scarring, cataracts and posterior capsular opacification visual function is reduced due to the scattering of light, called glare.

Polarisation of Light

Each light wave has an electrical field with a particular orientation. In non-polarised light, the electrical field of each wave has random orientation, while in a polarised light all electrical fields have the same orientation. Ordinary light can be used to create polarised light by passing it through a polarising agent. A polarising agent only transmits light of one particular orientation and blocks all the rest, therefore two polarising agents, if kept at right angles to each other are capable of blocking all light. Polarised light is used in the Titmus stereotest to assess binocular vision and it is also used to produce Haidinger's brush entropically.

Birefringence

It is a property of substance to transmit light waves lying parallel to its molecular structure and to redirect light waves lying perpendicular to its structure. Birefringent materials have two refractive indices. They change the state of polarisation by splitting any unpolarised light into two polarised beams at right angles to each other. A crystal of quartz has this property. Birefringence is used in Polarised microscopy.

Dichroism

It is a property of material to absorb light waves lying parallel to its structure while transmitting light perpendicular to its structure. Thus, a beam of polarised light of much weaker intensity emerges. Polaroid, a dichroic substance is commonly used in preparation of sunglasses.

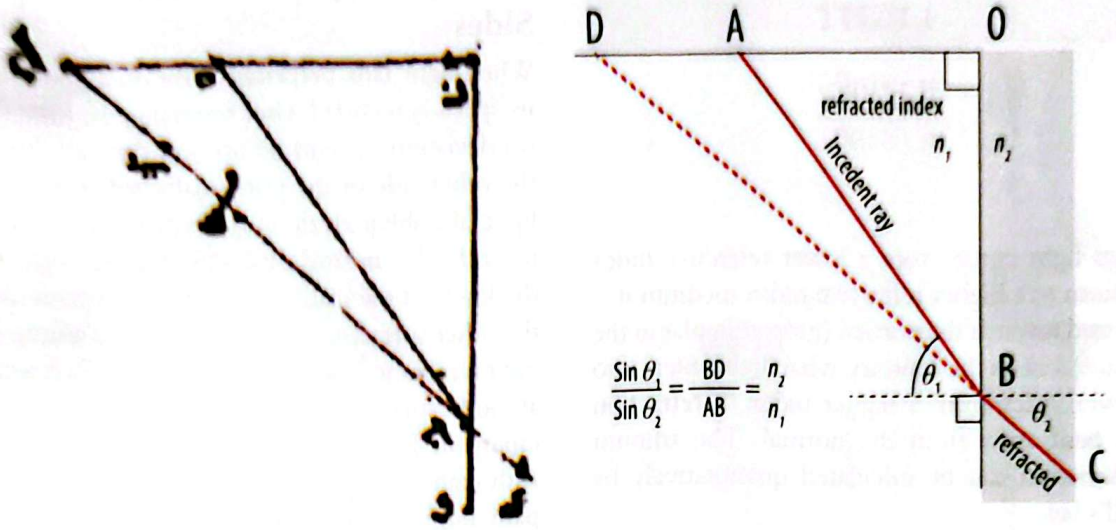

Fig. 31.2 Ibn Sahl's law with Arabic annotations translated.

Reflection of Light

When light meets a body in its passage, it exhibits any one of three behaviours. Light is absorbed in the bodies which are opaque and transmitted through the bodies which are transparent. But when light is bounced back from a body, this is called reflection. This behaviour of light is best exhibited by mirror surfaces; however, it occurs to some extent at all surfaces. The light reflected from the anterior surface of the cornea is used by instruments like Placido's disc, to examine the regularity of corneal curvature and by a Keratometer, to measure the radius of curvature.

Refraction of Light

When light passes from one transparent medium to another of different optical density, its speed is changed. The more dense the medium the slower the light passes. If light strikes a body at an angle, then not only is it slowed down but its direction is also changed. This phenomenon of the bending of light as it enters in a medium of different density is called refraction.

Refractive Index

Different materials have different optical densities and hence different abilities to retard the speed of light. If we take the medium of air as a standard and compare the velocity of light in any substance to that in air, then we can measure the refractive power of that substance, called its refractive index. Refractive index is the ratio of the speed of light in the air to speed of light in that material. Refractive indices of different materials are given in the table below:

Material	Refractive index (n)
Air	1.00
Water	1.33
Aqueous	1.33
Cornea	1.37
Crystalline lens	1.38-1.41
Crown glass	1.52
Diamond	2.5

Snell's Law

Snell's law (first described by Ibn Sahl (940-1000) a muslim Persian mathematician, physicist Fig. 31.2) states that an incident ray, a refracted ray and a normal ray, all lie in the same plane and that the angle of incidence and refraction are related to the refractive index of the media concerned. This relationship can be expressed by the following equation:

DIRECTION OF DEVIATION OF LIGHT

$$\frac{n_1 = \sin\theta_2}{n_2 = \sin\theta_1}$$

When light enters from a lower refractive index medium to a higher refractive index medium it is deviated towards the normal (perpendicular to the surface) and on the contrary, when light enters into a lower index from a higher index of refraction it is bent away from the normal. The amount of deviation can be calculated quantitatively by Snell's law.

Critical Angle

The angle of incidence at which the refracted light is bent exactly 90° away from the normal is called the critical angle. The critical angle for tear film-air interface is 48.5° and for crown glass-air interface is 41°.

Total Internal Reflection

When the angle of incidence exceeds the critical angle, the light is not refracted into the lower density medium, instead it is reflected back into the higher refractive index medium. This inability of light to emerge out is called total internal reflection. The visualisation of the anterior chamber angle with the naked eye is not possible because of the total internal reflection, and that is where gonioscopy comes to our rescue.

Refraction by a Plate with Parallel Sides

When light falls perpendicularly on a glass plate its speed is retarded while traversing the plate but no deviation of light occurs and the light leaves the other side of the plate unaffected. But when light falls obliquely upon a glass plate it is refracted towards the normal and proceeds through the thickness of the plate in a different direction until the other surface of the plate is reached where the light is refracted away from the normal. Refraction at both surfaces is equal and opposite so, the emergent light is parallel to the incident light. Although the light is displaced from its original path, but it travels in the same direction as before.

Refraction by Prisms

A prism is defined as a medium bounded by two plane surfaces which are inclined at a certain angle. When light enters a prism it deviates towards the normal and hence redirects itself to the base of the prism. When light leaves a prism it deviates further towards the base since it is refracted away from the normal on reentering a rarer medium. Hence the total deviation of light transmitted through a prism is towards the base of the prism and the image is displaced towards the apex of the prism.

Refraction by a Spherical Surface

When parallel light encounters a spherical surface its individual rays will be bent to a different degree and all rays will meet at a point. The distance of

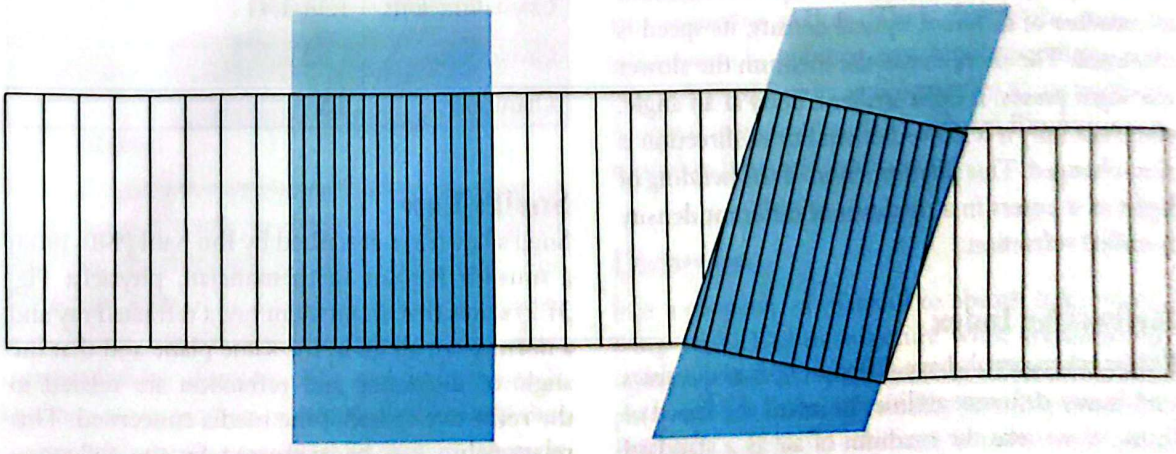

Fig. 31.3 Refraction of light by a plate

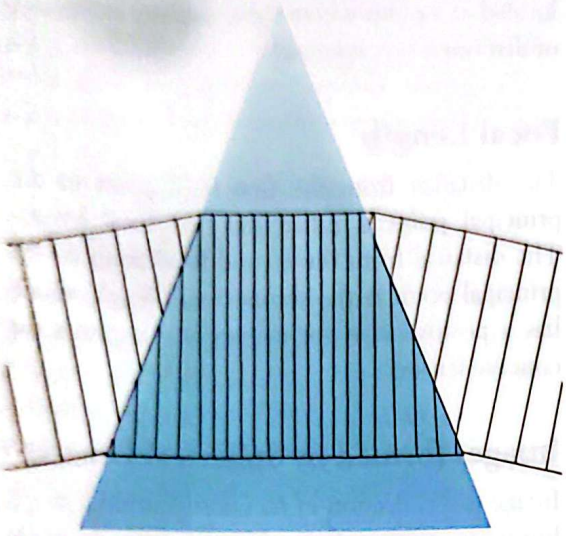

Fig. 31.4 Refraction of light by a prism

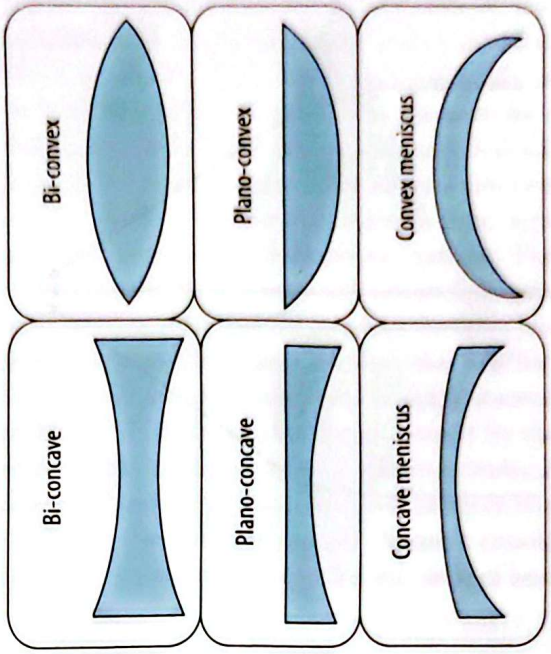

Fig. 31.5 Different forms of spherical lenses

this point from the refracting surface depends on the difference between the refractive indices of the two media and the radius of curvature of the refracting surface. The refracting power of a spherical surface may be calculated with the help of this equation:

$$Ds = \frac{n^1 - n}{r}$$

where $n^1 - n$ = difference in refractive index, and r = radius of curvature of the sphere in metres.

Refraction by Spherical Lenses

A lens is defined as a medium bordered by two curved surfaces. When each surface of a lens forms part of a sphere such a lens is called a spherical lens. Spherical lenses sometimes do have one plane surface because a plane surface can be considered part of a sphere with infinite radius. Basically there are two forms of spherical lenses, i.e., convex or plus lenses and concave or minus lenses.

Convex lenses converge light and concave lenses diverge light. In order to understand, let us suppose that a convex lens is a combination of two prisms placed base to base. If two parallel rays of light pass through them they can be brought to a focus. Similarly two prisms if placed apex to apex can refract light in a diverging manner like a concave lens.

Cardinal points of thin spherical lenses:

Principal Axis

A small central part of the lens may be considered to have parallel sides so the ray of light passing through it continues its straight path without deviation. The undeviated line of the ray passing through the centre of the lens is called the principal axis of the lens.

Principal Plane

The total power of a spherical lens is the sum of two surface powers, if the thickness factor is neglected. Therefore, refraction can be thought of as occurring at the principal plane of the lens which is a plane that lies at the centre of the lens perpendicular to the principal axis. In ray diagrams, the type of lens is shown by an appropriate symbol at each end of the principal plane.

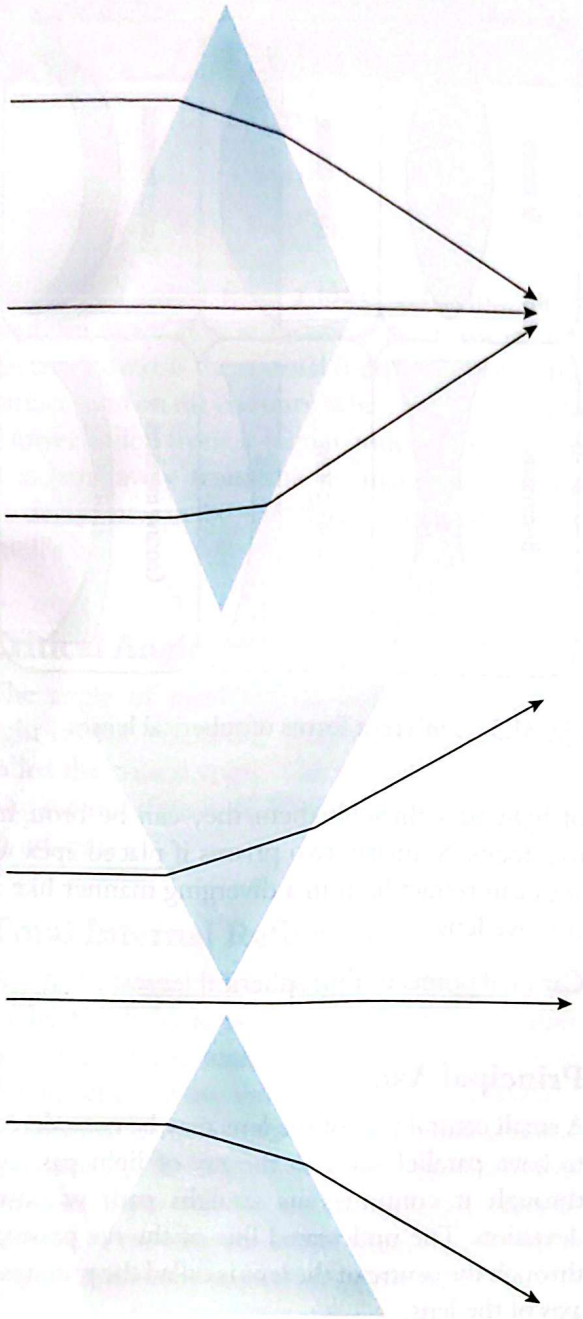

Fig. 31.6 Refraction of light by two prisms

Principal Point

The point at which the principal axis and principal plane intersect is the called principal point, or nodal point.

Principal Focus or Focal Point

A point of origin of rays which become parallel after refraction by the lens is called the first or primary or anterior principal focus. A point at which parallel rays meet after getting refracted by the lens is called the second or posterior principal focus. For a diverging lens, the principal focus is located at a point where the imaginary extensions of diverging rays intersect.

Focal Length

The distance from the first focal point to the principal point is called the first focal length. The distance from the second focal point to the principal point is the second focal length which has a positive sign for convex and negative for concave lenses.

Images formed by Spherical Lenses

Image is a collection of foci corresponding to all luminous points of an object. Parallel incident rays are converged by convex lenses at secondary focus where the image is formed. Divergent rays are focused at a point beyond the secondary focus. Rays emanating from the primary focus will emerge as parallel, and the image is formed at infinity. If the source of light rays is brought even nearer, then the rays will be divergent and the image is formed beyond infinity, i.e., no real image is formed but a virtual image is seen by the observer on the opposite side of the lens. Concave lenses form images through same principles but they never form a real image because these lenses always diverge rays of light. Their images are always virtual, erect and diminished in size.

Thin Lens Formula

This equation is used to calculate the image distance for any given lens:

$$\frac{1}{v} \quad \frac{1}{u} \quad = \frac{1}{f}$$

where v is the distance of the image from the principal point, u is the distance of an object from the principal point, and f is the second focal length.

Central Ray

A ray drawn from the top of the object passing through the principal point undeviated is called a central ray. It is used to obtain additional information about image size (i.e., magnified or

minified) and image orientation (i.e., upright or inverted). Initially, extra-axial extension of an object is drawn to indicate its height, a central ray is traced from the tip of the object, image distance is located through lens formula and image is drawn from optical axis up or down to the central ray.

Lens Power

The strength or ability of a lens to converge or diverge light is called lens power. The power of a lens can be measured in a unit called a dioptre, which is the inverse of the focal length of a lens in metres. Lenses with shorter focal length are more powerful than lenses with longer focal length. If the focal length of a converging lens is 1 metre, it has a refractive power of +1 dioptre (D) and if the focal length of a diverging lens is 25 cm, it has a power of -4 D.

Vergence of Light Rays

It is the measure of the degree of convergence or divergence of rays in terms of dioptres. The reciprocal of an object and image distance gives us the vergence of the object and image rays respectively. Converging rays have plus vergence, diverging rays have minus vergence, and parallel rays have no vergence. Vergence of light is changed by introducing a lens in their path. We can rewrite thin lens formula in terms of vergence of light rays:

$$U + D = V$$

where U is the vergence of light entering the lens, D is the vergence of light added by the lens or lens power, and is the vergence of light leaving the lens.

Refraction by Cylindrical Lenses

Cylindrical lenses have two surfaces, one plane and the other cylindrical. Unlike spherical lenses which have same refracting power in all meridians, cylindrical lenses have different refracting powers in two meridians 90° apart. It produces a focal line rather than a focal point. There are two forms of cylindrical lenses, i.e., pure cylindrical lenses and sphero-cylindrical or toric lenses.

A pure cylindrical lens has power only in one meridian and no power in an other meridian which is called its axis. Light is refracted only in the meridian perpendicular to the axis to make a focal line parallel to the axis. A sphero-cylindrical lens has power in two meridians but to a different extent. Light, when it passes through these type of lenses produces a characteristic pattern. The meridian with more power will refract light rays more and bring them to focus before the other less powerful meridian. There are thus two line foci and the distance between them is called interval of Sturm. The geometrical figure formed by the rays of light emerging from a sphero-cylindrical lens is called Sturm conoid. Halfway between two focal lines the cross section of Sturm's conoid becomes circular, which is called the circle of least confusion.

Refraction by Thick Lenses

A thick lens has greater separation between two refracting surfaces by lens substance and therefore its mathematical analysis is more complicated than the ideal thin lens. It has been simplified by introduction of cardinal points.

Cardinal points of thick lenses:

Principal Planes

These are two hypothetical planes, such that the incident ray at the first principal plane leaves the second at the same vertical distance from the principal axis.

Principal Points

The points where principal planes intersect the principal axis at right angles, are called principal points.

Nodal Points

Any ray passing through the first nodal point leaves the lens from the second nodal point undeviated. When the medium on both sides of the thick lens is the same, nodal points coincide with principal points.

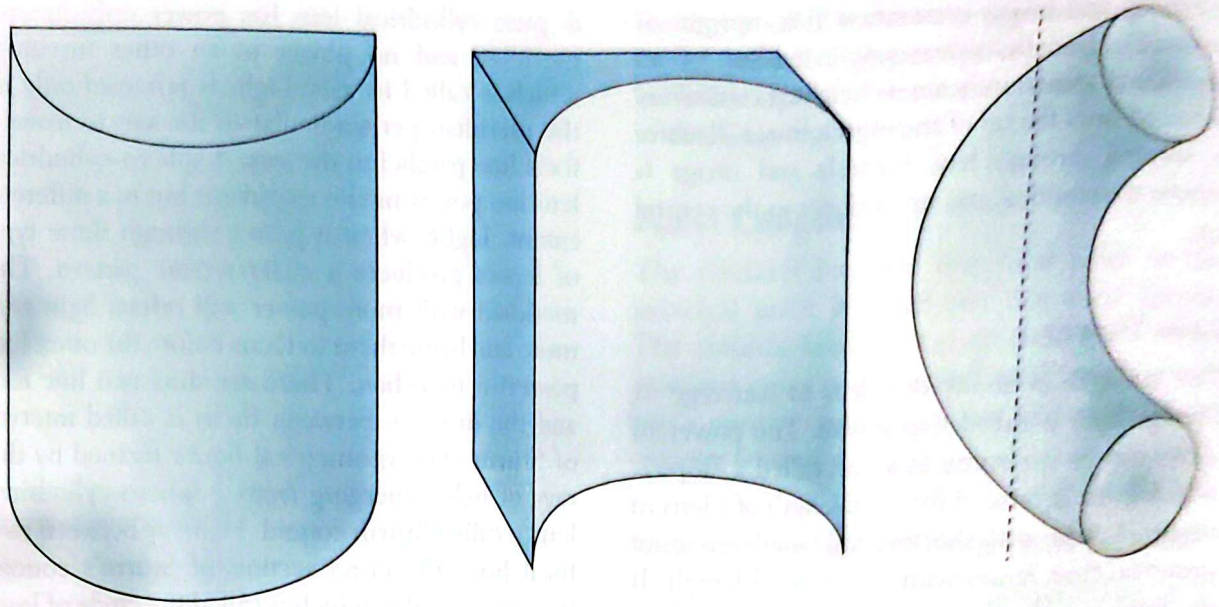

Fig. 31.7 Cylindrical Lenses

Principal Foci and Focal Lengths

Principal foci in thick lenses have the same definition as for thin lenses, but the focal lengths are calculated from corresponding principal points. In some thick lenses principal points may lie outside the lens substance and it becomes necessary then to distinguish between true focal lengths and vertex focal lengths. Vertex focal lengths, measured by instruments such as a focimeter, are the distances measured from the principal foci to the vertex or central surface of the lens, and furthermore, the anterior and posterior vertex focal lengths are not equal to each other.

PHYSIOLOGICAL OPTICS

Optical Components of the Eye

The understanding of the optics of a normal human eye needs some description of its individual elements constituting the optical system of the eye.

Cornea

The first structure that meets light on its way to the retina is the cornea. The anterior surface of the cornea is approximately spherical in shape having about 8 mm radius of curvature and it contributes to two-third of the total refractive power of the eye (i.e., 40-45 D) and the credit for this goes to the air-cornea interface. The importance of the air-cornea interface comes to light when a swimmer opens his eyes underwater to find his vision blurred and for that reason uses goggles to keep air in front of the cornea. Corneal stroma of a normal human eye scatters 10% of incident light, which affects the quality of retinal image as compared to the much transparent stroma of an eagle's eye which gives it Snellen acuity of 6/1.5.

Iris and Pupil

The iris, an adjustable diaphragm of the eye, controls the amount of light admitted to the eye by changing pupillary diameter from 8 mm in very dim light to 1.5 mm in very bright light. The extreme size of the pupil may affect the retinal image quality negatively by causing optical aberrations in a large pupil and diffraction in a small pupil. Generally a pupil with a diameter of 2.4 mm gives the best retinal image where the effects of aberrations and diffractions are balanced.

Crystalline Lens

It is responsible for about one third of the eye's refractive power that is 16 to 20 D which is less than the cornea because the refractive index

difference between aqueous, vitreous and lens substance is not as marked as the difference at the air-cornea interface. The anterior and posterior surfaces have radii of curvatures 10 mm and 6 mm respectively. The refractive index is not distributed uniformly inside the lens substance and this variation in refractive indices (i.e., higher in the nucleus, lower in the cortex) is responsible for neutralising the spherical aberration caused by the cornea.

Retina

The retina is a photosensitive tissue where the optical elements of the eye forms real, inverted images which are reinverted in the cerebral cortex. The finest details in the retinal image can only be resolved in the foveal macular region. Scattering of light inside the eye is prevented by black pigment epithelium, Müller cells, orientation of photoreceptors, yellow macular pigment and configuration of foveal pit.

Reduced Schematic Eye

The complex optical system of the eye can be reduced without introducing any appreciable error to make the analysis easier. The reduced eye is a single refracting surface with one principal plane, one nodal point, 5.5 mm away

$$\frac{\textit{Object height}}{\textit{Retinal Image height}} = \frac{\textit{Object distance from nodal point}}{17mm}$$

from the principal point, index of refraction 1.33, refractive power of +60 D and axial length of 22.5 mm. We can use the schematic eye to determine retinal image height for any object by the formula:

Visual Angle

The nodal point of the reduced eye acts as the optical centre, and rays can pass through it without getting refracted. Two rays from the extremities of an object passing through the nodal point enclose an angle which is known as the visual angle. It is equal to the angle subtended by the retinal image at the nodal point. As an object approaches the eye it appears larger because it subtends greater visual angle.

Emmetropia

When parallel rays of light strike a physiologically normal eye and are focused on the retina without any accommodation, the condition is called emmetropia. This state is achieved if the axial length of an eye is appropriate for its dioptric power.

Accommodation

The ability of the eye to change its focus and increase its converging power is called accommodation. In an emmetropic eye, with fixed power, parallel rays coming from distant objects are focused on the retina while diverging rays from near objects are focused behind the retina to form a blurred image. To overcome this problem the crystalline lens, which is held stretched under tension, assumes a more globular shape through ciliary muscle contraction increasing its curvature, thickness and converging power. The increase in curvature affects mainly the anterior surface of the lens, which moves slightly forward towards the cornea.

There are certain terms related to the process of accommodation:

Far Point of Accommodation

The farthest distance away at which an object can be seen clearly without accommodation is called the far point or punctum remotum. The far point of an emmetropic eye is located at infinity.

Near Point of Accommodation

The nearest point at which an object can be seen clearly with maximum accommodation is called the near point or punctum proximum.

Range of Accommodation

The distance between the far point and the near point is called the range of accommodation.

Amplitude of Accommodation

The difference between the dioptric power of the eye at rest and when fully accommodated is called the amplitude of accommodation.

Synkinesis

The associated action of convergence and pupillary contraction in concert with accommodation is called synkinesis. The close physiological association between these synkinetic movements can be understood by the fact that the same nerve, the third cranial nerve, supplies all the muscles carrying out these actions, (i.e., medial recti, sphincter papillae and ciliary muscle).

ACCOMMODATIVE CONVERGENCE/ ACCOMMODATION RATIO (AC/A RATIO)

For each dioptre of accommodation there is a fixed number of prism dioptres of convergence and this AC/A ratio is relatively constant for each individual. The normal range for AC/A ratio is 3:1 to 5:1.

Physiological Optical Defects

Every lens system has its own inherent defects and the eye is no exception to this rule. Diffraction causes limitation in retinal image quality and in an eye with 2 mm of pupillary diameter, the diameter of airy disc is 0.01 mm. Dispersion of light by the lens into its individual colours is called chromatic aberration which reduces the definition of a retinal image, but to a smaller degree. A spherical aberration occurs when the peripheral rays are brought to focus earlier than central rays. This phenomenon becomes evident when the pupil is widely dilated, otherwise its effects are negligible. Images formed in the peripheral portions of the retina are less clearly defined than those in the central portion of the retina. Some important peripheral aberrations are coma, oblique astigmatism and distortion of the image; but their effects are neutralised to a large extent by the peculiar shape of the eye.

CLINICAL OPTICS

Refractive errors have been discussed earlier in chapter 26. In this section we shall be discussing visual acuity, spectacle lenses, contact lenses and low visual aids.

Visual Acuity Testing

Pin-Hole Test

Visual acuity is reduced not only due to refractive error but also due to ocular pathology and neurological disease. The pin-hole test is a useful method to determine the cause. When a patient looks through a small hole in an opaque disc his visual acuity is either improved (i.e., in refractive errors), not improved (i.e., in ocular pathology) or may even become worse (i.e., in macular disease).

Minimum Angle of Resolution (MAR)

This is the minimum angular separation between two object points subtended at the nodal point that the eye can distinguish as separate. These angles are measured in arc minutes. The minimum angle of resolution is inversely proportional to the visual acuity.

Snellen Visual Acuity Test

Although there are several methods of measuring visual acuity but the Snellen acuity test is most commonly used. It is based on the theory that the smallest object resolved by the eye subtends an angle of one minute of the arc at the nodal point. The test employs a chart with rows of letters of diminishing size. Each letter at a certain specified distance subtends an angle of 5 minutes, but each arm of the letter subtends an angle of 1 minute.

LogMAR Visual Acuity

The most standardised system of measuring visual acuity is based on the logarithm of the minimum angle of resolution. These tests (e.g., the Bailey-Lovie test) are more precise because there is regularity in the size progression and spacing of letters.

Vernier Acuity

It is the ability of the eye to detect the smallest break in a line. Normally an offset of 3 to 5 seconds of arc is discernable which is less than the separation of photoreceptors, therefore, it is also called hyperacuity.

Spectacle Lenses

Lenses have many uses in ophthalmology, for patients in the form of spectacles, contact lenses and low vision aids; and for ophthalmologists in the form of essential components of instruments.

Prescription of Spectacles

The optical characteristics of a pair of ophthalmic lenses are specified by writing a prescription. A prescription for the right (OD) and left (OS) eyes includes the sphere power (sph) and cylinder power (cyl) with an appropriate sign, axis of cylinder, add power if additional power is required for near vision, and in case of indication of prism, its magnitude and orientation is specified.

Transposition of Lenses

The alteration of prescription from one form of the lens to another optically equivalent form is called transposition of the lens.

Simple Transposition

In the simple transposition of spherical lenses the lens power is given by the algebraic sum of the surface powers. For example, a +3.00 D spherical lens can be transposed as +1.5+1.5, 0+3.00, -1.50+4.50-6.00+9.00, etc. In simple transposition of cylinders the astigmatic lens is described in such a way as to express the cylinder in an opposite power. This change in description is accomplished by following these steps:

1. Sphere and cylinders are added to give new power of the sphere
2. Cylinder sign is changed retaining its numerical power
3. Cylinder axis is rotated through 90°

Toric Transposition

Toric transposition is carried out in the following manner:

1. Prescription is transposed to change the sign of the cylinder to the same sign of the base curve
2. Spherical surface power is calculated by subtracting the base curve from the sphere
3. Axis of the base curve is specified at right angles to the cylinder
4. Cylinder is added to the base curve keeping its axis at right angles to the base curve

Identification of Lenses

Method of Neutralisation

Clinically, spectacle type can be detected and even its power can be measured by neutralising with a lens of equal power and opposite sign. You can determine the type of lens by drawing two lines crossed at right angles to each other and observing the images while looking through the spectacles. When spherical lenses are moved from side to side or up and down, the arm perpendicular to the direction of movement also appears to move. In convex lenses 'against movement' is observed, while 'with movement' is observed in concave lenses. Astigmatic lenses cause distortion of the cross unless their axes coincide with the cross lines. When rotated, they cause a 'scissoring movement' which is absent in spherical lenses. Prisms displace a line of the cross but the direction of displacement is constant. Base up prism displaces the image downward, and base down prism displaces it upward.

Geneva Lens Measure

This instrument measures the radius of curvature of a surface. It has two fixed legs and a central movable leg which is deflected, by an amount depending on the curvature, when placed on a surface. The radius of curvature is then converted into dioptres for a known refractive index. It is usually calibrated for ordinary crown glass and needs correction if any other glass is employed.

Effective Power of Lenses

In a hypermetropic eye, if the correcting convex lens is moved away from the eye, the image is brought forward, thus lens effectivity is said to be increased. In a myopic eye, if the correcting minus lens is moved further away from the eye,

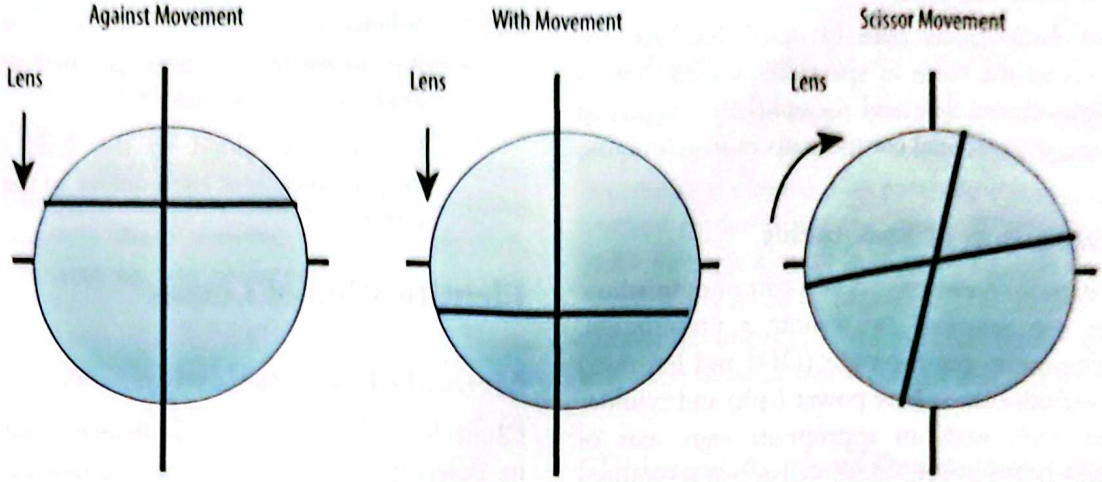

Fig. 31.8 Method of identification by Hand

the image is brought forward and effectivity of the lens is said to be reduced.

Back Vertex Distance

The distance between the back of the lens and the cornea is called the back-vertex distance. It is calculated in situations where the lenses are to be worn at a different distance, e.g., due to a high bridge nose, deep-set eyes or when power adjustment is required for contact lenses because the position of lenses in front of the eye affects the optical correction, the closer you get to the retina the more power you are going to need to correct the same ametropia. BVD must be given with all prescriptions over 5 dioptres.

Contact lenses

There are mainly two types of contact lenses, soft contact lenses and hard contact lenses. Nowadays, hard contact lenses have been replaced by rigid gas permeable (RGP) contact lenses. Soft contact lenses are used as bandage contact lenses in surface corneal diseases, small perforations and ulcers, as well as in treating refractive errors and for cosmetic purposes. RGP contact lenses are used most commonly for treating Keratoconus. Compared to spectacles the contact lenses give a wider field of view, lower optical aberrations and better cosmetic appearance but are difficult in handling, needing meticulous cleaning and disinfection. Moreover, they increase the chances of complications like, corneal trauma and infections.

Low Vision Aids

Low vision aids are devices used for improving visual performance in low vision patients and enabling them to carry out their daily academic and social activities. Low vision aids include high power spectacles, hand held magnifiers, stand magnifiers, telescopes, video magnifier systems, closed-circuit televisions, computers and tablets.

Telescopes

A telescope is an optical instrument made by combining two or more lenses to improve the resolution of an object by increasing the size of the image on the retina. A simple telescope consists of an eyepiece and an objective lens. There are two types of telescope, astronomical or Keplerian telescope, and the Galilean telescope. An astronomical telescope has two convex lenses, the objective lens having lower power than the eyepiece. Both the lenses are separated by the sum of their focal lengths. The image formed by an astronomical telescope is real and inverted. The Galilean telescope has a concave eyepiece and a convex objective lens and the distance between them is determined by the difference in their focal lengths. The image produced by the Galilean telescope is real and erect. The Galilean telescope

is cheaper, smaller and lighter as compared to the astronomical telescope, but the astronomical telescope gives a better optical quality and greater visual field. Sometimes a reverse Galilean telescope is also used in which the eyepiece is convex and the objective lens is concave. It is used in patients with peripheral field loss, e.g., glaucoma, retinitis pigmentosa.

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