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6th Edition

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Essentials of
Ophthalmology

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Essentials of Ophthalmology

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Dedicated to

Dr Bani Biswas

Who still loves me even after extensive hours with this book.

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Preface to the Sixth Edition

In the world of medicine, there is only one constant—change. Therefore, it has been essential to bring out the sixth edition of this book. Another constant—the appreciation and feedback from the readers, who are not just undergraduates, but also postgraduates and practising ophthalmologists from all around the Indian subcontinent, has encouraged me to improve and update the book. As a teacher, it has been a pleasure to see the readership of *Essentials of Ophthalmology* grow and now after eighteen years, and with over 75,000 copies sold, the book is stepping into adulthood!

Ophthalmology has changed considerably over the years, especially with the advancement of technology, which reflect the improvement in surgical techniques, innovation in treatment regimens, and even in the way we diagnose and manage diseases. The highlights of this edition are—plenty of new colored diagrams and photographs, latest medical and surgical management of some diseases, and updates on newer investigations. The new look and the use of American spelling is to make the book reach out to a wider range of international readers. However, a few things have not changed in this edition, like its predecessors, is in four color. The bulleted format of the book, detailed illustrations, a selection of slit-lamp examination, and such features which facilitate revision have been retained.

Finally, this edition marks the handover of the book from Current Books International to Jaypee Brothers Medical Publishers. I take this opportunity to thank my old publisher for taking care of this book for so many years, and extend my thanks to my new publisher as well, who has published four of my ophthalmology books previously. Thanks are also due to all the doctors and staff members of Disha Eye Hospitals for their continued support and involvement. A special thanks to Dr Aditya Pradhan, my cornea fellow who has helped me with some notes. Dr Soham Basak, new budding ophthalmologist, has also helped me with his insights and suggestions and as always, thanks to my wife Dr Bani and my daughter Sohini.

I hope this edition enhances your understanding of the subject. Readers are welcome to send in more constructive criticism.

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Preface to the First Edition

Most medical colleges do not have a required undergraduate teaching course in ophthalmology. Consequently, the students are often sadly deficient in this subject, unless they are specially interested. In adition, an acute shortage of time, prevents the students from reading large text books on this subject. Many students complain that most of the available books are either too brief or too extensive. It is with the hope of bridging this gap that I have written this book.

This book focusses on the information in a concise, step-wise, outline format which is essential for getting a true picture of the subject. The arrangement of the subject matter is to-the-point, and up-to-date with plenty of line diagrams which are easily reproducible during examination. Though, this book is essentially meant for the undergraduate students, I hope, it will be very useful to the postgraduate students as well as practising ophthalmologists, for a quick revision of the subject.

In conclusion, I shall be happy if this book proves itself to be a valuable guide both for the undergraduate and postgraduate students, especially during their examination. Any critical comment and suggestion from the students and teachers, regarding this effort, will be appreciated.

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February, 1997

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I am extremely grateful to my teacher, Prof VK Dada, Ex-Chief, RP Center for Ophthalmic Sciences, AIIMS, New Delhi, who had spent his valuable time reading the manuscript and writing the foreword for this book.

The material of this book is drawn from many sources—Parsons' Diseases of the Eye, Kanski's Clinical Ophthalmology, Trevor-Roper's The eye and its Disorders, Newell's Ophthalmology: Principles and Concepts, Wolff's Anatomy of the Eye and Orbit, Bruce-Shield's A Study Guide of Glaucoma, Abram's Practice of Refraction. I express my sincere gratitude to all authors and respective publishers. For any instance, in which I have omitted to mention its source, it is hoped that the author will accept this blanket of acknowledgments.

I am greatly indebted to Dr Amitava Kundu, who has helped me to compile the manuscript. I gratefully thank my wife, Dr Bani Biswas and my children Soham and Sohini, for their cheerful acceptance of many evenings and holidays sacrified, so that I could complete this book. Finally, I would like to convey my gratitude to my publisher Mr Jitendar P Vij (Group Chairman), Mr Ankit Vij (Group President), Ms Chetna Malhotra Vohra (Associate Director-Content Strategy) and all technical staff.

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1 CHAPTER Embryology and Anatomy

EMBRYOLOGY OF THE EYEBALL

The human eye originates from neuroectoderm, surface ectoderm and the extracellular mesenchyme which consists of both neural crest and mesoderm. Ocular development in the human embryo begins around 3rd week into embryonic life and continues through the 20th week of life (Table 1.1).

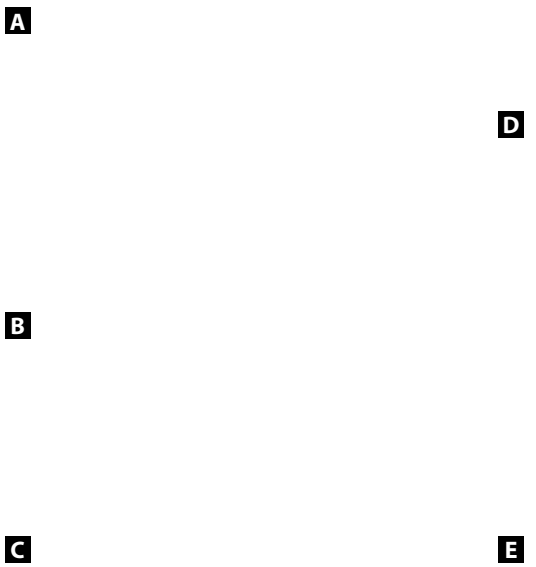
- Both the sensory and pigmentary layers of retina are developed from the neuroectoderm. These layers continue anteriorly to give rise to ciliary epithelium and the pigmented layer of the iris. The neuroglial and neural portion of the optic nerve are also originate from neuroectoderm.
- The surface ectoderm is the primordia of the crystalline lens (Table 1.1), the conjunctiva, corneal epithelium and the eyelids with the epithelium of their glands.

TABLE 1.1: Primordia of ocular structures

Surface ectoderm	Neuroectoderm	Mesoderm	Neural ectoderm
• Conjunctival epithelium • Corneal epithelium • Eyelids—epithelium, glands, eyelash, skin • Caruncle • Lacrimal system • Lacrimal gland (also from neural crest) • Crystalline lens • Vitreous (also mesoderm)	• Neurosensory retina • Optic nerve, axons, glia • Retinal pigment epithelium	• Extraocular muscle • Fat (also neural crest) • Iris sphincter and dilated muscles • Iris stroma • Sclera (also neural crest) • Vascular endothelium • Vitreous (also surface ectoderm)	• Bones—midline/ inferior orbital bones; parts of orbital roof and lateral rim • Orbital connective tissue • Choroidal stroma • Ciliary ganglion • Cornea-stroma and endothelium • Extraocular muscles sheath and tendon • Fat (also mesoderm) • Iris pigment epithelium • Melanocytes • Optic nerve sheath • Sclera (also mesoderm) • Trabecular meshwork

- ***Eyelids:*** Both from surface ectoderm and mesoderm
- ***Zonules*** (tertiary vitreous): Surface ectoderm and mesoderm
- ***Bruch's membrane:*** Neural ectoderm and mesoderm

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Figs 1.1A to E: The development of the eye
Solid red—neural ectoderm; ***dotted area***—mesoderm; ***hatched area***—surface ectoderm

- Neural crest cells are themselves derived from the ectoderm and lie close to the neural tube. They are responsible for development of sclera, cornea—Descemet's membrane and endothelium and connective tissue, and bony structure of the orbit.
- The mesoderm is the primordia of the extraocular muscles, endothelial lining of blood vessels of the eye, choroidal blood vessels, sclera and choroid, vitreous, zonular fibers, iris sphincter/dilated muscles, and iris stroma.
- Eye development is initiated by the master control gene Pax-6. The Pax-6 gene locus is a transcription factor for the various genes and growth factors involved in eye formation.

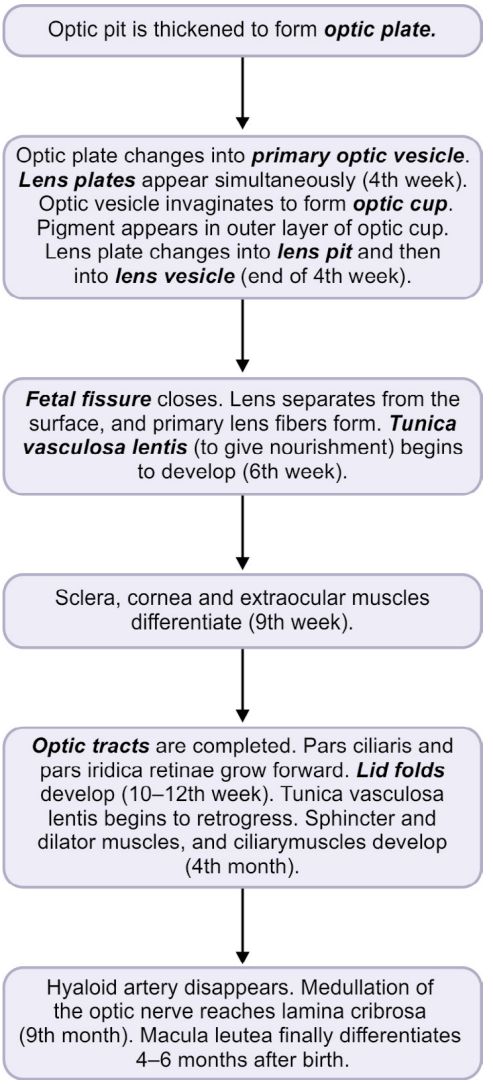
THE EYE AT BIRTH

- Orbit is more divergent (50°) as compared to adult orbit.
- Eyeball is 70% of adult length, being almost fully developed at the age of 8 years.
- Cornea is 80% of its adult size, being fully developed at the age of 3 years.
- The newborn is hypermetropic by +2.5D.
- Pupil is small and does not dilate fully.
- Anterior chamber is shallow and the angle is narrow.

DEVELOPMENT (FIGS 1.1A TO E)

On either side of the cephalic end of forebrain, a lateral depression appears, known as ***optic pit*** (3rd week).

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GROSS ANATOMY OF THE EYEBALL (FIG. 1.2)

- It is not a true sphere, but consists of the segments of two modified spheres, one in front of the other.
- The cornea is more convex than the rest of the globe (7.8 mm radius as opposed to 12 mm).
- The anteroposterior diameter ***(axial length)*** is about 24 mm, while the vertical is 23 mm, and the horizontal is 23.5 mm.
- The eyeball is shorter in hypermetropes but longer in myopes.

THE GLOBE

- ***Three concentric layers or tunics:***
  1. ***Outer supporting layer:*** It consists of transparent cornea, opaque sclera and their junction, the ***limbus***.
  2. ***Middle vascular layer:*** It is called uvea, and consists of choroid, ciliary body and the iris.
  3. ***Inner neural layer:*** It is called retina, composed of two parts (1), a sensory portion and (2) a layer of pigment epithelium.
- ***A crystalline lens:*** The transparent structure is located immediately behind the iris, and is supported by fine fibers, called ***zonules***.
- ***Three chambers:***
  1. ***Anterior chamber:*** It is located between the iris and the posterior surface of the the cornea. It communicates with the posterior chamber through the pupil.
  2. ***Posterior chamber:*** It is minute in size, bounded by the lens and zonules behind, and the iris infront.
  3. ***Vitreous cavity:*** It is the largest and located behind the lens and zonules, and adjacent to the retina throughout.

THE CORNEA

Cornea (Fig. 1.3) is elliptical from front, being 12 mm horizontally, and 11 mm vertically. Posteriorly, it is circular with a diameter of 11.5 mm.

- ***Thickness:*** 0.5 mm at the center and 1 mm at the periphery.
- ***Radius of curvature:*** Anterior surface 7.8 mm and of posterior surface 6.5 mm.
- ***Refractive index:*** 1.37.
- Cornea constitutes the anterior one-sixth of the eye.

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Fig. 1.2: The human eyeball

- It is the main refracting surface of the eye. ***Dioptric power*** is about + 43.0 to + 45.0 D.

Structures

It consists of five layers:

1. ***Stratified squamous epithelium***
   - 5–6 cells deep, continuous with the conjunctival epithelium.
   - The basal cells are tall with oval nuclei.
   - The intermediate layers (2–3 cells deep) are polyhedral wing cells.
   - The surface layers (about 2) are very flat, but not keratinized.
   - The corneal epithelium rests on a basement membrane, secreted by the basal cells.
2. ***Bowman's layer (anterior elastic lamina)***
   - Anterior condensation of substantia propria.
   - Once eroded, it does not regenerate and leaves behind a superficial corneal scar.

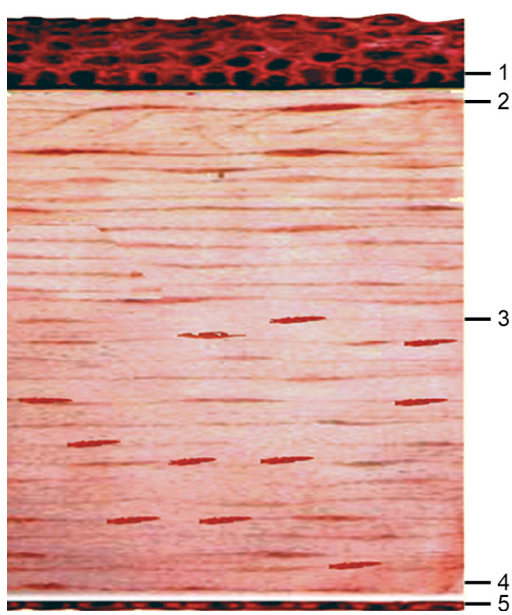


Fig. 1.3: Transverse section of the cornea
1—Epithelium; **2**—Bowman's membrane; **3**—Stroma; **4**—Descemet's membrane; **5**—Endothelium

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3. ***Substantia propria (stroma)***
   - Comprising 90% of corneal thickness.
   - Composed of collagen fibrils arranged in sets of lamellae, lying parallel to the surface.
   - They form a crystalline lattice, in a ground substance of glycoprotein and mucopolysaccharides.
   - Scattered between the lamellae are the ***keratocytes***, and a few wandering leukocytes and macrophages.

Keratocytes (corneal fibroblasts) are specialized fibroblasts residing mainly in the corneal stroma. They play the major role in keeping the cornea transparent, healing its wounds, and synthesizing its components. In a silent healthy cornea, keratocytes stay dormant, coming into action after any kind of injury or inflammation.

Some keratocytes underlying the site of injury, even a light one, undergo apoptosis immediately after the injury. Other neighboring keratocytes, when acted upon by the same molecules, become active, proliferate and start synthesizing matrix metalloproteinases (MMPs) which is required for tissue remodeling. These activated cells are designated as "active keratocytes".

Keratocytes are developmentally derived from the cranial population of ***neural crest cells***, from where they migrate to settle in the mesenchyme. ***Keratan sulfate*** produced by keratocytes is thought to help maintain optimal corneal hydration. The average keratocyte density in the human stroma is about 20500 cells per mm^3. The highest density is observed in the anterior 10% of the stroma. The number of keratocytes declines with age, at a rate approximately 0.45% per year.

Keratocytes may play a role in ectatic corneal disorders. In keratoconus, excessive keratocyte apoptosis happens as a major pathological event.

Pre Descemet layer (Dua's layer): A new layer was suggested by Harminder Singh Dua, et al. in 2013. It is hypothetically 15μ thick, the fourth corneal layer from top, and located between the corneal stroma and Descemet's membrane. This layer is very strong and impervious to air, despite its thinness. This is still a controversial issue, as the existence of pre Descemet stromal tissue remaining after pneumodissection is well known.

4. ***Descemet's membrane (posterior elastic lamina)***
   - Strong, homogenous and very resistant membrane.
   - It readily regenerates after an injury.
   - It is secreted by endothelial cells and essentially their basement membrane.
   - In old age, it may bear some warty elevations called ***Hassall-Henle's bodies*** at its periphery.
5. ***Endothelium***
   - A single layer of flattened polygonal endothelial cells.
   - Continuous with the endothelium over the anterior surface of the iris.
   - It does not regenerate in human being.

Nerve Supply

The corneal nerves, are derived from the long and short ciliary nerves, branches of the ophthalmic division of trigeminal nerve.

They form the ***pericorneal plexus*** just outside the limbus, and then pass onto the cornea as 60–70 trunks. They loose their myelin sheaths after a millimeter or two, and reach the cornea. Cornea does not have proprioceptive sensation.

Blood Supply

Cornea is avascular, but the corneoscleral limbus is supplied by the anterior conjunctival branches of anterior ciliary arteries and form a perilimbal plexus of blood vessels.

THE SCLERA

- It is a dense tough fibrous envelope that covers ***posterior five-sixths*** of the eye.
- Sclera has two large openings, the anterior (the corneal window) and the posterior (for optic nerve).
- Structures piercing the sclera are:
  - Four vortex veins (4 mm behind the equator).

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- Long and short ciliary nerves and vessels.
- Anterior ciliary nerves and vessels.

- Sclera is ***thickest*** posteriorly surrounding the optic nerve (1 mm), and is ***thinnest*** just posterior to the insertion of recti muscles (0.3 mm).

Structures

Sclera has three parts:

1. ***Episclera:*** It is the loose fibrous tissue, containing numerous fine capillaries.
2. ***Sclera proper:*** It is a dense network of collagen fibers. The sclera is white, because of variable diameter and irregular arrangement of collagen fibers of the stroma.
3. ***Lamina fusca:*** It is the inner layer, located adjacent to the choroid.

Blood Supply

It is from the episcleral and choroidal vessels. Anterior to the insertion of recti muscles, the anterior ciliary arteries form a dense episcleral plexus. These vessels become congested in ciliary congestion.

Nerve Supply

Short ciliary nerves posteriorly, and long ciliary nerves anteriorly, provide sensory innervation. Because of generous innervation, inflammation of the sclera is extremely painful.

THE LIMBUS

- Transitional zone between the cornea and the sclera. It is 1–2 mm wide.
- Its internal boundaries are scleral spur posteriorly, and the Schwalbe's line.
- Its external boundaries are by sclerolimbal junction posteriorly, and corneolimbal junction anteriorly.
- Sclerolimbal junction (Fig. 1.4) is the only consistent land mark of the limbus, used during surgery.

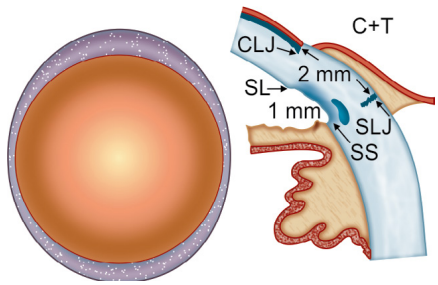


Fig. 1.4: Surgical anatomy of the limbus
C+T—Conjunctiva and Tenon's capsule; **SL**—Schwalbe's line; **SS**—Scleral spur; **CLJ**—Corneolimbal junction (at the termination of the Bowman's membrane); **SLJ**—Sclerolimbal junction (junction of the white sclera and the translucent bluish limbus)

- Limbus contains trabecular meshwork internally, through which the aqueous humor leaves the anterior chamber.

Angle of the Anterior Chamber

- Anterior chamber is bounded infront by the cornea, behind by the anterior surface of iris and part of the anterior surface of the lens which is exposed at the pupil (Figs 1.5A and B).
- The peripheral recess of the anterior chamber is known as ***angle of the anterior*** chamber, which is also known as the ***cockpit of glaucoma***.
- It is bounded ***anteriorly*** by the corneosclera, and ***posteriorly*** by the root of the iris and the ciliary body.
- At this part in the inner layers of sclera, there is a circular venous sinus (sometimes broken up into more than one sinus) called ***canal of Schlemm***. It is of great importance in the drainage of aqueous. The Schlemm's canal is lined by endothelial cells.
- At the periphery of the angle, between Schlemm's canal and the recess of anterior chamber, there lies a loose meshwork of tissues, known as ***trabecular meshwork***.
- Trabecular meshwork is almost triangular in shape. Its apex arises from the termi-

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A B

Figs 1.5A and B: A. The angle of the anterior chamber; **B.** The anterior segment of the eye
E—Corneal and conjunctival epithelium; **D+E**—Descemet's membrane + Endothelium; **TM**—Trabecular meshwork; **SC**—Schlemm's canal; **SVP**—Scleral venous plexus; **CM**—Ciliary muscle; **CP**—Ciliary processes; **L**—Lens; **I**—iris; **S**—Sclera; **CS**—Corneal stroma; **AC**—Anterior chamber; **PC**—Posterior chamber; **Z**—Zonules.

nation of Descemet's membrane and the adjacent part of corneal stroma, and its base merges into the tissues of the ciliary body and the root of the iris.

- The trabecular meshwork is made up of circumferentially disposed flattened bands and each is perforated by numerous oval openings. Through these tortuous pas sages, communication exists between the Schlemm's canal and anterior chamber.
- The junction between these cells are not 'tight', and the cells themselves have pores for aqueous drainage.
- Angle of the anterior chamber is best visualized by a gonioscope. Normally, the angle is wide open, and is about 20–45°. If the angle is less than 10°, there will be every chance of developing angle-closure glaucoma.

THE UVEA

The middle coat, or uvea means grape consists of three parts:

1. ***Anterior:*** Iris, a free circular diaphragm with a central opening called pupil.
2. ***Intermediate:*** Ciliary body
3. ***Posterior:*** Choroid.

Functions

- The iris with its central opening, pupil, controls the amount of light entering the eye.
- The ciliary body secretes aqueous humor and contains smooth muscles responsible for changing the shape of the lens during accommodation.
- The choroid, a vascular layer, provides the blood supply to the retinal pigment epithelium and the outer half of the sensory retina.

THE IRIS AND THE PUPIL

- The iris lies in front of the lens and the ciliary body. It separates the anterior chamber from the posterior chamber. Pupil is situated just inferior and slightly nasal side of the center.
- Its periphery or 'root' is attached to the anterior end of the ciliary body.
- Anterior surface of the iris is divided into two zones (Fig. 1.6):
  1. Central pupillary zone and
  2. Peripheral ciliary zone.
- Their junction is a circular ridge called ***collarette*** (Fig. 1.7) which marks the embryonic site of the minor vascular circle of iris, from which the embryonic pupillary membrane originated.
- The ciliary zone is marked by many ridges and crypts, but the pupillary zone is relatively flat.

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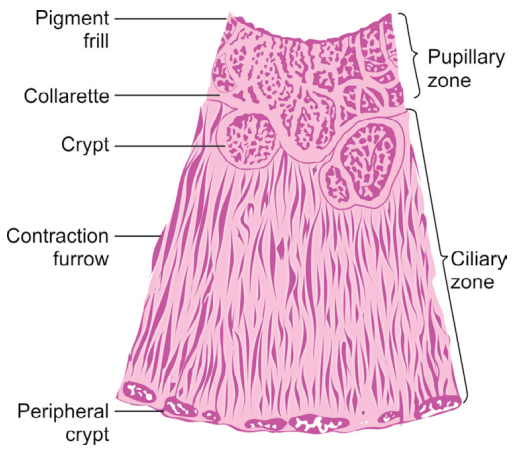


Fig. 1.6: Surface pattern of the iris

Structure

- ***Anterior endothelium:*** It is continuous with the corneal endothelium.
- ***Stroma:*** It consists of spongy connective tissue with nerves, smooth muscles, and radial blood vessels forming the minor circle of iris.
- ***Smooth muscles:*** They are two in number:
  - ***Sphincter pupillae:*** A circular bundle of smooth muscles running around the pupillary margin, causes constriction of the pupil.
  - ***Dilator pupillae:*** Arranged radially near the root of the iris, causes dilatation of the pupil.
- ***Posterior two-layered epithelium:*** Both layers are pigmented and developmentally derived from the retina. The anterior layer consists of flattened cells and the posterior layer consists of cuboidal cells.

Nerve Supply

- ***Sensory:*** Nasociliary nerve, branch of 1st division of 5th cranial nerve.
- ***Sphincter pupillae:*** Oculomotor nerve (3rd cranial).
- ***Dilator pupillae:*** Nerves derived from cervical sympathetic chain.

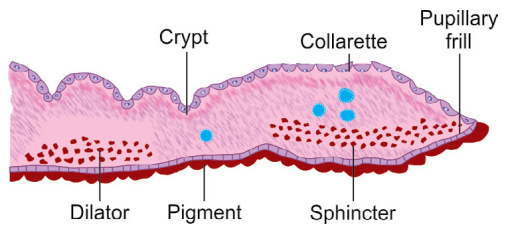


Fig. 1.7: Transverse section of the iris

THE CILIARY BODY

The ciliary body is a ring of tissue about 6 mm wide that extends from the scleral spur to the ora serrata of the retina.

Structure

In anteroposterior section it is roughly an isosceles triangle, with base forwards. Iris is attached at the middle of its base.

- The chief mass of the ciliary body is composed of unstriped muscle fibers, called ***ciliary muscle***. It has three parts, with a common origin circumferentially at the scleral spur:
  1. ***Longitudinal:*** The greater part, running antero-posteriorly.
  2. ***Circular:*** Concentrically with the root of the iris.
  3. ***Radial***.
- The inner surface of ciliary body is divided into two regions:
  1. ***Pars plicata:*** Anterior part; about 70 plications are visible. Microscopically, they have ciliary processes responsible for the production of aqueous.
  2. ***Pars plana:*** Posterior smooth part; a relatively safe and avascular zone for pars plana lensectomy and/or vitrectomy operation.
- They are covered by two layers of epithelium, continuous with the iris anteriorly, and retina posteriorly.

Ciliary body extends backward as far as the ora serrata. At this point the retina proper begins abruptly.

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Nerve Supply

- ***Sensory:*** Via nasociliary branch of 5th cranial nerve.
- ***Ciliary muscles:*** Oculomotor (3rd cranial) and sympathetic nerves.

Blood Supply

By branches of ***major circle*** of iris which is formed by two long posterior ciliary arteries and seven anterior ciliary arteries (the ***minor circle*** lies within the iris stroma).

Functions

- Formation of aqueous humor by the ciliary processes.
- Ciliary muscles help in accommodation for near work.
- Ciliary muscles also help in opening up the Schlemm's canal, and thus facilitate in aqueous outflow.

THE CHOROID

- This vascular sheet separates the sclera from the retina. It is 0.25 mm thick at the posterior pole and 0.1 mm thick anteriorly.
- It is attached firmly to the sclera around the optic nerve and at the points of exit of the vortex veins.

Structures

It consists of three layers of blood vessels, having supporting structures on either side, i.e. suprachoroid ***(lamina fusca)*** on outer side and the basal lamina ***(Bruch's membrane)*** on the inner side.

Three vessel layers are (Fig. 1.8):

1. The ***outer vessel layer (of Haller):*** It is nearest to the sclera and consists of large veins that lead to the vortex veins.
2. The ***middle vessel layer (of Sattler):*** It consists of medium-size veins and arterioles, with fibrous tissues.

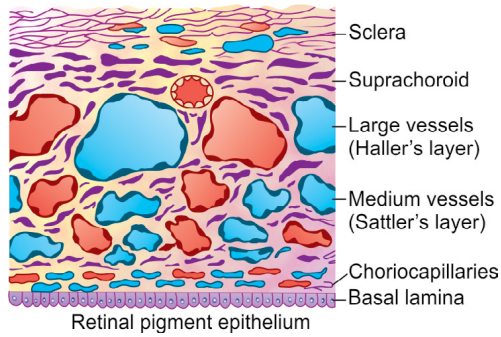


Fig. 1.8: Transverse section of the choroid

3. The ***inner choriocapillaries*** consist of large fenestrated capillaries.

Bruch's membrane: It is about 7μ thick, and separates the choriocapillaries from the retinal pigment epithelium. Electron microscopically, it consists of five layers ***(from outside to inside)***:

1. Basement membrane of the choriocapillaries
2. Outer collagen layer
3. Middle elastic layer
4. Inner collagen layer
5. Basement membrane of the retinal pigment epithelium.

Bruch's membrane is important for blood-retinal barrier function.

Blood Supply (Fig. 1.9)

- ***Short posterior ciliary arteries:*** Originate from the ophthalmic artery as 2–3 branches. These branches are subdivided into 10–20 branches which perforate the sclera around the optic nerve, and directly communicate with the choriocapillaries.
- ***Two long posterior ciliary arteries:*** Perforate the sclera on either side of the optic nerve → via suprachoroidal space → to the ciliary body. There, each divides into two branches that extends circumferentially to form 'major arterial circle' of the iris, located in the ciliary body. Branches extend anteriorly to the iris.

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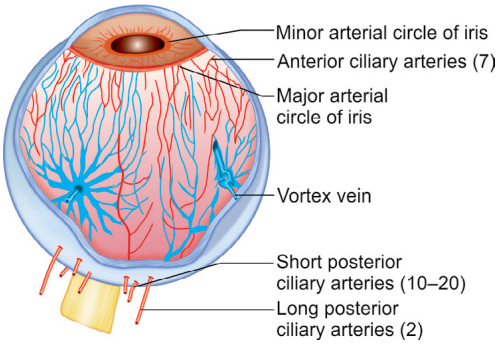


Fig. 1.9: Blood supply of the uveal tract

- ***Anterior ciliary arteries:*** They are the terminal branches of two muscular arteries of each rectus muscle (except the lateral rectus, which has one muscular artery). The vessels provide supply to the ciliary body, and send branches to the major arterial circle of iris and also to the choriocapillaries.

Venous blood is collected from the iris, ciliary body and the choroid by a series of veins. These lead of four (or more) large ***vortex veins*** located behind the equator of the globe. The vortex veins drain into superior and inferior ophthalmic veins → cavernous sinus.

THE CRYSTALLINE LENS

- The lens is a transparent bi-convex body of crystalline structure.
- It is about 9 mm in diameter and 4 mm in thickness when the suspensory ligament is relaxed.
- Radius of curvature of anterior surface is 10 mm and that of posterior surface is 6 mm.
- The lens is held in its position by the suspensory ligament, called ***zonules*** of Zinn. They arise from the sides of the ciliary processes, and the valleys between them.
- The zonular fibers insert into the anterior and the posterior lens capsule near the equator, and extend further over the anterior surface more than the posterior surface.

Structures (Fig. 1.10)

- The ***lens capsule*** that envelops the entire lens.
- An anterior ***lens epithelium***.
- A ***lens substance***, consisting of the ***cortex*** (newly formed lens fibers) and the ***nucleus*** (a dense central area of old lens fibers.)
- ***The capsule:*** It is a smooth, homogenous, acellular highly elastic envelope, (but contains no true elastic tissue).
  The anterior capsule is the basement membrane of the anterior lens epithelium, and the thickest basement membrane of the body. The lens capsule is ***thickest*** on the anterior and posterior surface just central to the insertion of zonular fibers (14 μ) (i.e. pre-equator regions). It is *thinnest* at the posterior pole (3–4 μ).
- ***The lens epithelium:*** It consists of a single layer of cuboidal cells just deep to the capsule. There is no corresponding posterior epithelium. Towards the equator the anterior cuboidal cells gradually become columnar and elongated, and eventually converted into lens fibers. At the equator, the division of lens fibers is most active and mitosis is frequently observed.
- ***Lens substance:*** It consists of elongated lens cells (fibers). Mature lens fibers are cells which have lost their nuclei, and are no longer in contact with the posterior capsule. They form the greatest portion of the lens substance.

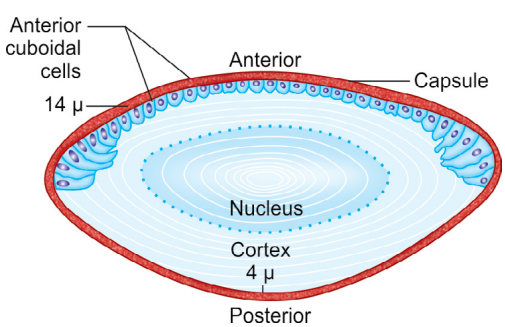


Fig. 1.10: Structure of the lens

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- In the infantile lens, each lens fiber starts and finishes on the ***anterior and posterior Y sutures respectively*** in such a way, that the nearer the axis of the lens it commences, the farther away it ends (anterior Y is straight and the posterior Y is inverted).
- Once this fetal nucleus is formed, these outlines become more irregular and more complicated.
- In the beam of slit-lamp, various layers or ***zones of discontinuity*** may be seen and these represents the boundaries as follows (Fig. 1.11):
  - Embryonic nucleus (first 3 months of fetal life)
  - Fetal nucleus (3–8 months of fetal life)
  - Infantile nucleus (upto puberty)
  - Adult nucleus (rest of life).

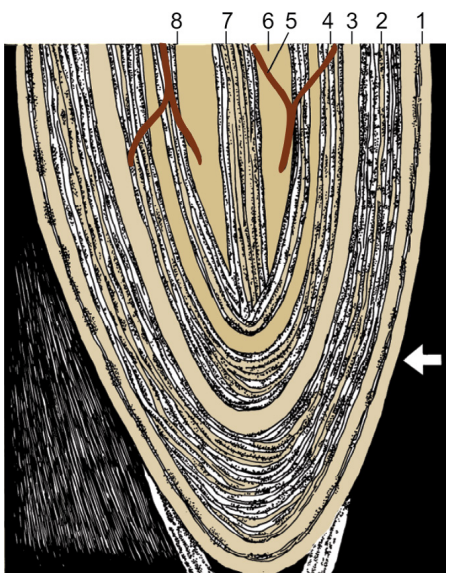


Fig. 1.11: Adult crystalline lens as seen in optical section of the slit lamp in higher magnification

1—Anterior capsule; **2**—Lens cortex; **3**—Adult nucleus; **4**—Infantile nucleus; **5**—Anterior Y suture; **6**—Fetal nucleus; **7**—Embryonic nucleus; **8**—Posterior Y suture.

CHAMBERS OF THE EYE

The eye consists of three chambers—anterior chamber, posterior chamber, and vitreous cavity.

1. ***Anterior chamber:*** It is bounded anteriorly by the cornea, posteriorly by the front surface of the iris and lens, and peripherally by the anterior chamber angle.
   It is deepest at its central portion (2.5–3.0 mm), and its ***volume*** is 0.25 mL.
2. ***Posterior chamber:*** It consists of various boundaries are as follows:
   - ***Anteriorly:*** Iris,
   - ***Laterally:*** Ciliary processes,
   - ***Medially:*** Equator of the lens and
   - ***Posteriorly:*** Anterior surface of the lens.

   Its volume in adults is 0.06 mL.
3. ***Vitreous cavity:*** It contains vitreous humor which is a transparent gel-like structure. It is composed of a network of collagen fibers suspended in a viscus liquid containing hyaluronic acid.
   It has a saucer-like depression anteriorly, for the lens, called ***patellar fossa***. Here, the vitreous humor is condensed, called ***anterior hyaloid face***. The vitreous humor adheres firmly to the:
   - Ciliary epithelium in the region of ora serrata (vitreous base),
   - Peripheral retina,
   - Margin of the optic disc, and
   - Posterior capsule of the lens (Weigert's hyaloideocapsular ligament).

Running down the center of the vitreous, from the optic disc to the posterior pole of the lens—there is a canal, called ***hyaloid canal of Cloquet***. Embryologically, the vitreous is divided into three parts:

1. ***Primary*** (mesenchymal, Cloquet's canal).
2. ***Secondary*** (most of the adult vitreous).
3. ***Tertiary*** (the lens zonules).

The vitreous contains a few cells, called ***hyalocytes,*** which are believed to be phagocytes (macrophage type).

Volume of the vitreous cavity is 4.5 mL.

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THE RETINA

The retina is the membranous light sensitive coat of eyeball. It is transparent in life, and whitish after death.

The retina is derived from the inner and outer layers of the embryological optic cup. These two primary layers are loosely adherent across the potential space (representing the primary optic vesicle,) so that they are readily separated by injury or disease. The ***outer pigmented layer*** remains as one-cell deep but the inner layer, the ***sensory retina*** becomes several layers by various visual relaying cells.

Structures

Retina apparently consists of ***ten layers*** (Fig. 1.12). From outside (choroid-side) inwards, they are:

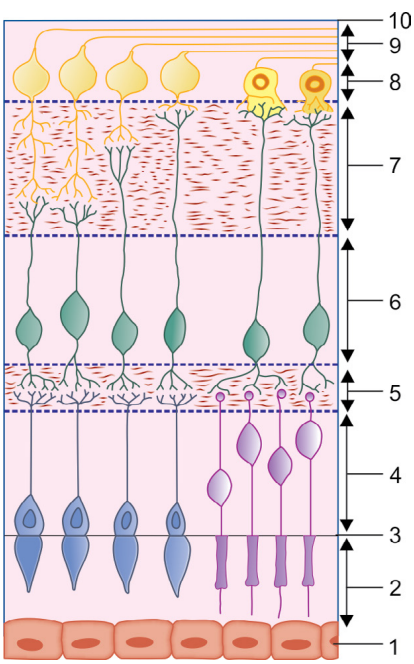


Fig. 1.12: Layers of the retina

1—Pigment epithelium; **2**—Layer of rods and cones; **3**—External limiting membrane; **4**—Outer nuclear layer; **5**—Outer plexiform layer; **6**—Inner nuclear layer; **7**—Inner plexiform layer; **8**—Ganglion cell layer; **9**—Nerve fiber layer; **10**—Internal limiting membrane

1. Retinal pigment epithelium (RPE)
2. Layer of rods and cones
3. External limiting membrane
4. Outer nuclear layer
5. Outer plexiform layer
6. Inner nuclear layer
7. Inner plexiform layer
8. Ganglion cell layer
9. Nerve-fiber layer
10. Internal limiting membrane

- ***The retinal pigment epithelium:*** It is a single layer of flattened hexagonal cells with fine cytoplasmic villi, projecting for a short distance between the bases of rods and cones.
  The cells of RPE contain varying amount of melanin. The cells are taller at the fovea and contain more pigments (hence, the darker color in this region). Around the optic disc they are heaped up as a ***choroidal ring***.
- ***Layer of rods and cones:*** They are the outer segments of photoreceptor cells, arranged in a palisade manner.
  The rods are about 125 million in number, but the cones are about 7 million.
  Each rod and cone may be divided into three parts (Fig. 1.13):
  1. ***An outer segment:*** It is cylindrical in shape, with its base related to the projecting villi of RPE. It consists of a dense vertical stack of 700 discs that originates from infolding of double layer of cell membrane. The infoldings contain the visual pigments.
  2. ***A cilium:*** It is a tubular connection with the inner segment that contains linear striations.
  3. ***An inner segment:*** It is divided into an ***outer ellipsoid*** and an ***inner myoid*** portion. The myoid part is densely packed with endoplasmic reticulum and Golgi bodies.
- ***External limiting membrane:*** It is a thin lamina, formed by the supporting fibers of Muller on which the rods and cones rest, and pierced by the fibers of these photoreceptors.

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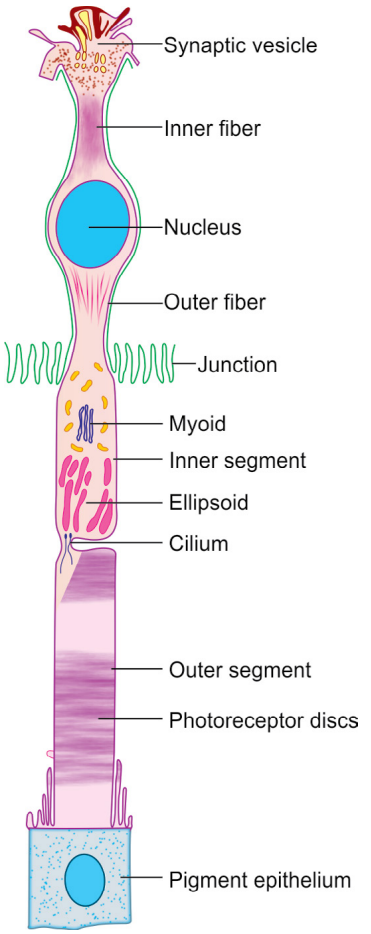


Fig. 1.13: Diagram of the vertebrate visual cell

- ***Outer nuclear layer:*** It contains the rod and cone nuclei. Cone nuclei are larger and more oval than the rod nuclei, and carry a layer of cytoplasm.
- ***Outer plexiform layer:*** It is formed by the anastomoses of the photoreceptor cells, with bipolar and horizontal cells. The innermost portion of each rod and cone cell is swollen with several lateral processes, known as ***rod spherules*** and ***cone pedicles***. Many photoreceptors converge onto one bipolar cell and interconnect with one another. But at the fovea, each 'midget' cone transmits to a single bipolar cell.
- ***Inner nuclear layer:*** It consists of the nuclei of bipolar, horizontal and amacrine cells. Amacrine cell processes pass inwards to synapse in the inner plexiform layer. The nuclei of Muller's fibers are also found here. Capillaries from the retinal vessels reach upto this layer, but the outer layers are avascular, the rods and cones being nourished by the choriocapillaries.
- ***Inner plexiform layer:*** It consists of arborizations of the bipolar cells, with the ganglion cells and amacrine cells.
- ***Ganglion cell layer:*** It consists of large multipolar nerve cells, with clear round nuclei containing nucleoli, and have Nissl's granules in their cytoplasm. In the retinal periphery, a single ganglion cell may synapse upto a hundred bipolar cells, but in the macular region, there tends to be a single connection with the 'midget' bipolar cells.
- ***Nerve fiber layer:*** It consists of bundles of ganglion cell axons, running parallel to the retinal surface. The layer increases in depth as it converges towards the optic disc. These nerve fibers are fine and ***non-medullated***. The macular fibers themselves pass directly to the disc as the ***papillomacular bundle***.
- ***Internal limiting membrane:*** It is a thin lamina separating retina from the vitreous. It is formed by the union of terminal expansions of Muller's fibers, and essentially a basement membrane.

Regions

- ***Ora serrata:*** It is the anterior termination of retina, located about 8 mm from the limbus. Here, only two layers of primitive optic vesicle fuse, and continue forward as the ciliary epithelium.
- ***Central retina (Fig. 1.14):*** It is 4.5 mm in diameter. It extends from the fovea centralis—nasally, almost to the optic disc; same distance temporarily, and a similar distance above and below the fovea centralis. In this region, ganglion cell layer

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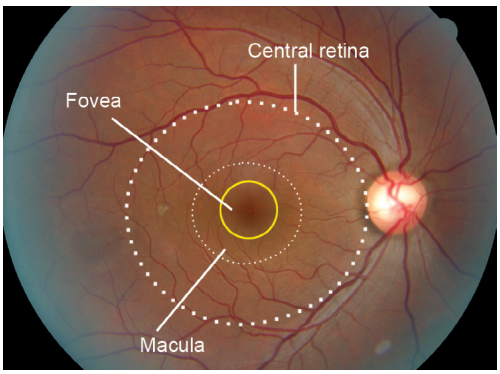


Fig. 1.14: The human retina

is more than one layer of cell bodies. The central part of this region is called ***macula leutea*** which contains a yellow pigment, xanthophyll.

- The ***fovea centralis*** is a depressed area, located in the central retina, about 3 mm (2 disc diameter) temporal to the optic disc and 0.8 mm below the horizontal meridian. It measures about 1.5 mm (1500 μm). It's central depression is called ***foveola,*** measuring about 0.5 mm (500 μm).
- The photoreceptors in the fovea are exclusively cones. All cell layers are displaced peripherally so that light falls directly on the cones' outer segment. The foveola is nourished solely by the choriocapillaries of the choroid and does not contain any vessels, hence, called ***foveolar avascular zone (FAZ)***.

- ***Peripheral retina:*** The photoreceptors are mainly rods. The ganglion cells are larger, and their cell bodies are arranged in a single layer.
- ***Functionally,*** retina is divided into ***temporal and nasal portion,*** by a line drawn vertically through the center of the fovea. Nerve fibers originating from the cells temporal to this line pass to the lateral geniculate body of the same side and from nasal side, cross in the optic chiasma to reach the lateral geniculate body of the opposite side.
- ***Ophthalmoscopically,*** the ophthalmologist uses the optic nerve as a hub to divide the retina into superior and inferior temporal portions, superior and inferior nasal portions, and a central retina.

Blood Supply

Retina gets its nourishment from two sources:

1. ***Outer portion:*** It mainly by the choriocapillaries.
2. ***Inner portion:*** It mainly by the central retinal artery, the first branch of ophthalmic artery which enters the optic nerve about 10–12 mm posterior to the globe.

THE CONJUNCTIVA

It is a mucous membrane covering the inner surface of the eyelids and reflected to cover the anterior part of the eyeball over the sclera, upto the corneal margin.

Parts (Fig. 1.15)

- ***Palpebral:*** It consists of marginal, tarsal and orbital part. It is firmly adherent to the deeper tissue.
- ***Bulbar:*** It lies over the sclera and it is freely mobile.
- ***Fornix:*** It is the cul-de-sac at the junction of palpebral and bulbar conjunctiva.
- ***Limbal:*** It is the conjunctiva at the corneal junction which is adherent.

Structures

- ***Epithelium:*** There are two layers of epithelium over the palpebral conjunctiva, and transitional stratified squamous epithelium at the intermarginal strip.
  - From fornices to the limbus the epithelium is gradually thicker (4–6 layers).
  - Again, it is stratified epithelium at the limbus.
  - Goblet cells (mucin secreting cells) are present throughout the epithelium, especially more near the fornices.

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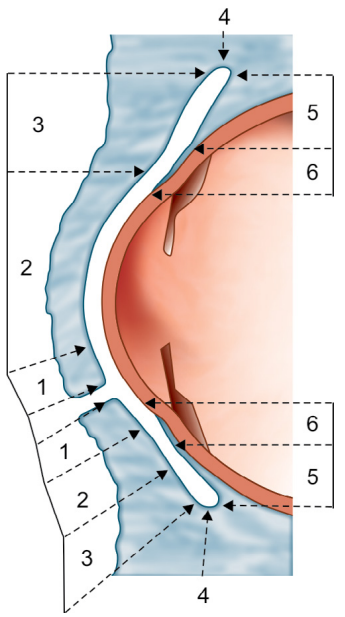


Fig. 1.15: The conjunctival areas
1—Marginal; **2**—Tarsal; **3**—Orbital; **4**—Fornicial; **5**—Bulbar; **6**—Limbal

- ***Subepithelial layer:*** It is an adenoid layer of loose connective tissue containing leukocytes.
- ***Fibrous layer:*** It is much dense, and blended with the deeper structures (e.g. episclera or tarsus).

Nerve Supply

Ophthalmic division of trigeminal nerve (5th cranial).

Arterial Supply

- Anterior conjunctival artery from the anterior ciliary artery.
- Posterior conjunctival artery from the lacrimal artery.
- Palpebral branch of nasal artery.

TENON'S CAPSULE

Tenon's capsule is a thin membrane which envelops the eyeball from the optic nerve to the limbus, separating it from the orbital fat and forming a socket in which it moves.

Its inner surface is smooth, and is separated from the outer surface of the sclera. Its anterior part (anterior Tenon's capsule) adheres with the undersurface of the conjunctiva and attaches to sclera at the limbus. Posterior Tenon's capsule is made up of the fibrous sheath of the rectus muscles together with the intermuscular membrane.

The fascia is perforated behind by the ciliary vessels and nerves, and fuses with the sheath of the optic nerve and with the sclera around the entrance of the optic nerve.

It is perforated by the tendons of the extraocular muscles, and is reflected backward on each as a tubular sheath. The expansions from the sheaths of the lateral and medial recti are strong, especially that from the latter muscle, and are attached to the zygomatic bone and lacrimal bone respectively. They also check the actions of these two recti and are called the ***medial and lateral check ligaments***.

There is thickening of the lower part of the Tenon's capsle, which is known as ***suspensory ligament of the eye (of Lockwood)***. It is slung like a hammock below the eyeball, being expanded in the center, and narrow at its extremities which are attached to the zygomatic and lacrimal bones respectively.

Sub-Tenon's block for ocular surgery: Local anesthetic may be injected into the space between Tenon's capsule and the sclera to provide anesthesia for eye surgery, principally cataract surgery. After applying topical anesthetic drops to the conjunctiva, a small fold of conjunctiva is lifted off and a small nick made. A blunt, curved cannula is passed through the incision into the sub-Tenon's space and then 1.5 mL 2% lignocaine solution is injected. The advantages are a reduced risk of bleeding and of penetration of the globe, compared to peribulbar and retrobulbar blocks. However, the akinesia of the extraocular muscles may be less complete.

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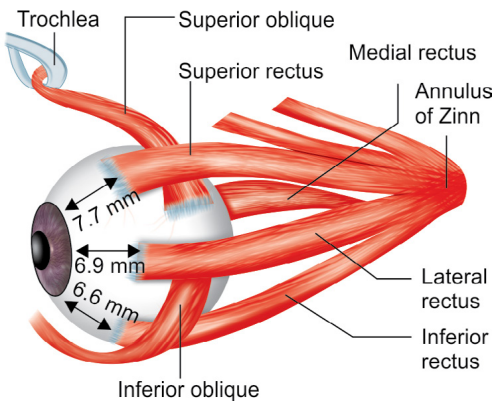


Fig. 1.16: Extraocular muscles of the eye

Note: Both oblique muscles insert behind the equator of the globe. The inferior oblique muscle passes inferior to the body of the inferior rectus muscle but beneath the lateral rectus muscle. The numbers indicate the distance of the insertion in mm from the corneoscleral limbus. Medial rectus (M) = 5.5 mm

THE EXTRAOCULAR MUSCLES (FIG. 1.16)

They are six in number. Four rectus muscles and two oblique muscles.

- ***Origin of rectus muscles:*** Common origin from annular ***tendon of Zinn*** around the optic foramen at the apex of the orbit.
  - ***Insertion:*** They are inserted to the sclera as a spiral line ***(spiral of Tillaux)*** after piercing the Tenon's capsule. The distance from limbus are as follows:
    Superior rectus (S) = 7.7 mm
    Lateral rectus (L) = 6.9 mm
    Inferior rectus (I) = 6.6 mm
    Medial rectus (M) = 5.5 mm
- ***Origin of superior oblique:*** Common origin at the apex of the orbit from annular tendon of Zinn → runs to the trochlea, at upper and inner angle of orbit → becomes tendinous → reflected backwards under the superior rectus muscle.
  - ***Insertion:*** It is inserted in the sclera at superolateral part of the posterior pole of the globe.
- ***Origin of inferior oblique:*** Anteriorly from the lower and inner orbital walls near the lacrimal fossa. It is the only muscle, which does not arise from the apex of the orbit.
  - ***Insertion:*** It is inserted in the sclera at inferolateral part of the posterior pole of the globe ***(corresponds to the area near the macula)***.
- ***Nerve supply:*** All the muscles are supplied by 3rd (oculomotor) cranial nerve except, lateral rectus—by 6th (abducens) nerve, and superior oblique—by 4th (trochlear) nerve.
- ***Actions of extraocular muscles:*** *See* Chapter 23.
  - ***Medial rectus:*** Adduction.
  - ***Lateral rectus:*** Abduction.
  - ***Superior rectus:*** Elevation on abduction and intorsion.
  - ***Inferior rectus:*** Depression on abduction and extorsion.
  - ***Superior oblique:*** Depression on adduction and intorsion.
  - ***Inferior oblique:*** Elevation on adduction and extorsion.

THE EYELIDS

The eyelids are thin curtains of skin, muscles, fibrous tissue and mucous membrane. The upper eyelid is limited above by the eyebrow, and the lower eyelid merges with the cheek. Each eyelid is divided by a horizontal furrow (sulcus) into an orbital and a tarsal part. The upper furrow is formed by skin-insertions of the levator palpebrae superioris muscle.

- ***Palpebral aperture:*** When the eyes are open, the eyelids form an elliptical opening, the ***palpebral aperture (fissure)*** which measures about 12 mm by 30 mm. The lateral canthus is about 2 mm higher than medial canthus (may be upto 5 mm in orientals). At the inner canthus, there is a small bay, the ***lacus lacrimalis,*** formed by the eminences (papillae) which bear the lacrimal puncta 6 mm lateral to the angle itself. There is a small separated

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knob of skin, ***the caruncle,*** bearing a few hairs or glands, contained in this bay. Just lateral to the edge of the caruncle, there is a crescentic fold of conjunctiva, the ***plica semilunaris,*** which is similar to the nictitating membrane in lower vertebrates.

- ***Lid margin:*** Located on the free margin of each eyelid are the openings of the lacrimal canaliculi (the puncta), the eyelashes and the openings of the glands. The medial one-sixth of the lid margin ***(the lacrimal portion)*** has no eyelash or gland openings, and are rounded. The lateral five-sixths ***(the ciliary portion)*** of the lid margin has square edges. The eyelashes on the upper eyelid margin curve upwards, and are more numerous than those of the lower eyelid margin, which curve downwards.

Structures

- ***Cutaneous layer:*** It is thin, smooth, delicate, elastic, having creases and without any long hair.
- ***Subcutaneous tissue:*** Loose areolar tissue devoid of fat.
- ***Muscular layer***
  - Orbicularis oculi
  - Levator palpebrae superioris (LPS) in the upper lid only
  - Muller's muscle.
- ***Fibrous layer***
  - Orbital septum in the upper part.
  - Tarsal plate in the lower part.

  Different glands of the eyelid (i.e. Meibomian glands, glands of Zeis, glands of Moll, glands of Krause and Woulfring) lie in this plane.
- ***Palpebral conjunctival layer:*** Inner most layer of the eyelid (Fig. 1.17).

Muscles of the Eyelid

- ***Orbicularis oculi***
  - ***Action*:** Closure of lids, blinking, winking, squeezing and helps in the drainage of tears.
  - ***Nerve supply:*** Zygomatic branch of facial (7th cranial) nerve.
  - ***In case of its paralysis:*** There will be lagophthalmos (leading to exposure keratitis due to dryness of the cornea and conjunctiva).
- ***Levator palpebrae superioris:*** It is present only in the upper lid.
  - ***Origin:*** From the apex of the orbit, above the annulus of Zinn.

Fig. 1.17: The surface anatomy of the eyelids (left eye)

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- ***Insertion:*** It can be into 5 parts:
  1. The ***main tendinous slip*** is inserted into the upper margin and the anterior surface of the tarsal plate.
  2. ***Anterior slip*** to the skin of upper lid.
  3. ***Posterior slip*** to the conjunctiva of the upper fornix along with the sheath of superior rectus muscle.
  4. ***Medial and*** (5) ***Lateral slips*** are attached to the medial and lateral palpebral ligaments respectively.
- ***Action:*** Elevates the upper eyelid including upper fornix, and helps in the formation of upper lid-fold.
- ***Nerve supply:*** Upper division of oculomotor nerve (3rd cranial nerve).
  Paralysis of LPS—causes ptosis.

- ***Muller's muscle (unstriped)***
  - ***Upper Muller's muscle:*** Arises from the stripped fibers of levator muscle, passes downwards behind it, and is inserted into upper border of the tarsus.
    ***Action*:** It elevates the upper lid.
  - ***Lower muller's muscle:*** It arises from inferior rectus muscle, lies below it, and is inserted into the lower trasus.
    Action: It elevates the lower lid to some extent.
  - ***Nerve supply:*** Cervical sympathetic nerve.
  - ***Paralysis of cervical sympathetic nerve*** will cause Horner's syndrome (ptosis, miosis, enophthalmos and anhydrosis of the face).

Glands

- ***Meibomian glands:*** They are modified sebaceous glands (tubular) of larger size and responsible for oily secretion of the tear film. They are situated within the substance of tarsal plate, arranged vertically, and each opens by a single duct on the margin of the lid.
  - ***Number:*** 30–40 in the upper lid, and 20–30 in the lower lid.

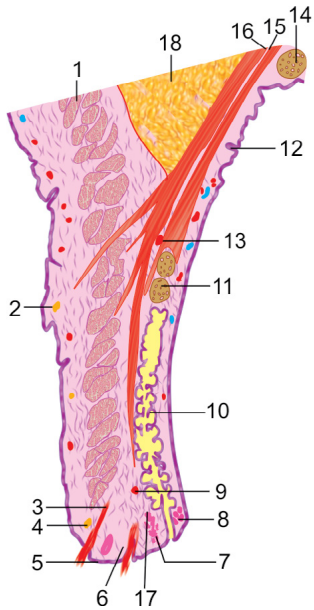


Fig. 1.18: Section through the upper eyelid
1—Orbicularis muscle; **2**—Sweat gland; **3**—Hair follicle; **4**—Gland of Zeis; **5**—Cilium; **6**—Gland of Moll; **7**—Marginal part of orbicularis muscle; **8**—Subtarsalis part of orbicularis muscle; **9**—Inferior arterial arcade, **10**—Meibomian gland; **11**—Gland of Woulfring; **12**—Conjunctival crypts; **13**—Superior arterial arcade; **14**—Gland of Krause; **15**—Muller's muscle; **16**—Levator palpebrae superioris muscle; **17**—Grey line; **18**—Fat

- ***Glands of Zeis:*** They are sebaceous glands, lie in the lid margin, and open in the follicle of eyelashes.
- ***Glands of Moll:*** Modified sweat glands, situated immediately behind the hair follicles, and their ducts open into the ducts of Zeis' gland, or into the follicle, (not directly onto the skin surface as elsewhere).
- ***Glands of Krause and Woulfring:*** They are accessory lacrimal glands situated on the palpebral conjunctival side (Fig. 1.18).

Intermarginal Strip

It is the margin or free edge of the lid. It is covered with stratified epithelium which forms a transition between the skin and conjunctiva.

- ***Structures*** (from anterior to posterior):
  - Anterior round border

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- Eyelashes
- Grey line
- Orifices of the ducts of Meibomian glands and
- Posterior sharp border.

- ***Grey line*** is an important land mark for operations in which the lid is splitted, since it indicates the position of the loose fibrous tissue between the orbicularis oculi and the tarsus.

Arterial Supply

- ***In upper lid:*** In the form of two arches:
  1. ***Superior:*** Lying between the upper border of tarsus and the orbicularis.
  2. ***Inferior:*** In front of tarsal plate, just above the hair follicle at the free edge.
- ***In lower lid:*** One arch near the free edge.

The arteries are: (i) Palpebral and lacrimal branch of ophthalmic artery, (ii) superficial temporal artery, (iii) infraorbital artery and (iv) facial artery.

Venous Drainage

Two plexuses in each lid—(i) post-tarsal, draining into the ophthalmic vein, and (ii) pretarsal, into the subcutaneous veins.

Lymphatic Drainage

- ***From inner half:*** Into the submandibular lymph node.
- ***From outer half:*** To the preauricular lymph node.

Functions

- Protection of eyeball proper, from external injuries, e.g. dust, fumes, foreign body, etc.
- Maintain the precorneal tear film (by sharp posterior borders of the lid margin).
- Interrupt and limit the amount of light entering the eye.
- Drainage of the tears by the lacrimal pump system.
- Emotional expressions.

THE LACRIMAL APPARATUS

The lacrimal apparatus consists of two parts:

1. ***Secretory portion:*** Lacrimal gland and accessory lacrimal glands of Krause and Woulfring.
2. ***Drainage portion:*** Via which the tears drain into the inferior meatus of the nose.

Secretory Portion

- ***Lacrimal gland:*** It is located in the anterolateral portion of the roof of the orbit in the lacrimal fossa. It has two parts—a large orbital portion and a small palpebral portion separated by lateral part of the aponeuroses of LPS muscle.
  It is a tubuloalveolar type of gland. Their ducts open separately onto the superior temporal fornix (Fig. 1.19).
  Nerve supply: Via facial nerve, parasympathetic from lacrimal (salivary) nucleus.
- ***Accessory lacrimal glands of Krause and Woulfring:*** They are located deep in the conjunctiva particularly in the fornices, mostly on the temporal side.

Drainage Portion

It is composed of the puncta, the canaliculi, the lacrimal sac and the nasolacrimal duct.

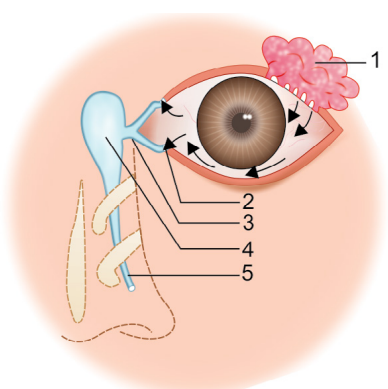


Fig. 1.19: Parts of the lacrimal apparatus
1—Lacrimal gland; **2**—Punctum; **3**—Common canaliculus; **4**— Lacrimal sac; **5**—Nasolacrimal duct

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- ***Two lacrimal puncta:*** These are two small openings, situated near the posterior border of the free margins of the lid about 6 mm from the inner canthus. The punctum is situated upon a slight elevation (large in elderly people) called ***lacrimal papilla***.
- ***Two canaliculi:*** It pass from the punctum to the lacrimal sac. They are first directed vertically for 1–2 mm, then horizontally for 6–7 mm. The canaliculi usually open separately into the outer wall of lacrimal sac. Sometimes, they join together to form a common canaliculus before opening into the sac.
- ***Lacrimal sac:*** It lies in the lacrimal fossa formed by the lacrimal bone. When distended it is about 15 mm long and 5–6 mm wide. The upper portion is called ***fundus***, which lies slightly above the level of medial palpebral ligament. Sac itself is covered by fibers of the orbicularis muscles and loose fibrous tissues.
- ***Nasolacrimal duct:*** It is the continuation of lacrimal sac. 12–24 mm long and 3–6 mm in diameter. The duct has two parts; intraosseous and intrameatal. It passes downwards, slightly outwards and backwards to open into anterior part of the outer wall of the inferior meatus of the nose.

The upper end of the nasolacrimal duct is the narrowest part. The mucous lining forms an imperfect valve at the orifice into the nose ***(valve of Hasner)***.

THE ORBIT

The orbits are pear-shaped cavities. Their medial walls are parallel, but lateral walls diverge at an angle of 45°. The orbit is roughly 40 mm in height, width and depth. Its ***volume*** is about 30 mL.

Portions of seven bones (Fig. 1.20) form the orbit are—(1) frontal, (2) maxilla, (3) zygoma, (4) sphenoid, (5) palatine, (6) ethmoid and (7) lacrimal.

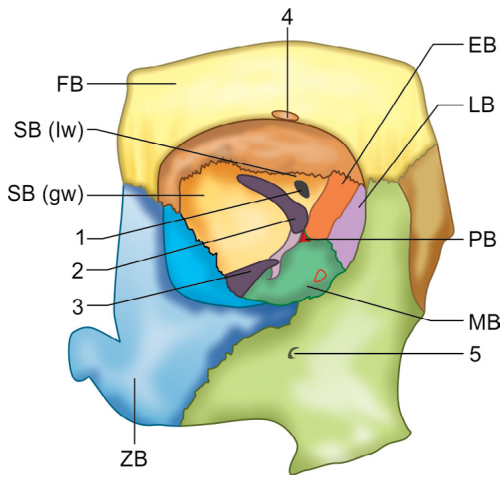


Fig. 1.20: The bony orbit

FB—Frontal bone; **MB**—Maxillary bone; **ZB**—Zygomatic bone; **SB**—Sphenoid bone-greater wing (gw); lesser wing (lw); **PB**—Palatine bone; **EB**—Ethmoid bone; **LB**—Lacrimal bone; **1**—Optic canal; **2**—Superior orbital fissure; **3**—Inferior orbital fissure; **4**—Supraorbital notch; **5**—Intraorbital foramen

Contents

- The eyeball and intraorbital part of optic nerve
- Retrobulbar fat
- Extraocular muscles
- Ophthalmic arteries and veins
- 3rd, 4th and 6th nerve and first two divisions of 5th nerve
- Ciliary ganglion
- Sympathetic plexus
- Lymphatic vessels
- Tenon's capsule and orbital fascia
- Lacrimal gland and lacrimal sac.

Optic Foramina

It is located at the posteromedial portion of the orbit in the body of the sphenoid bone. It measures 4–10 mm in diameter.

Through it passes:

- The optic nerve with its sheaths,
- Ophthalmic artery and
- Sympathetic nerve from carotid plexus.

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Superior Orbital Fissure

It is just lateral to the optic foramen, and is the gap between the greater and lesser wings of sphenoid. The fissure is divided into lateral and medial portions by the tendinous annulus of Zinn (Fig. 1.21).

- ***Passing through the annulus***
  - Two divisions of the oculomotor nerve (CN III)
  - Abducens nerve (CN VI)
  - Branches of the ophthalmic division of trigeminal nerve (CN V), except the lacrimal and frontal branches.
- ***Passing through the lateral portion***
  - Lacrimal nerve
  - Frontal nerve
  - Trochlear nerve (CN IV)
  - Superior ophthalmic veins
  - Recurrent lacrimal artery

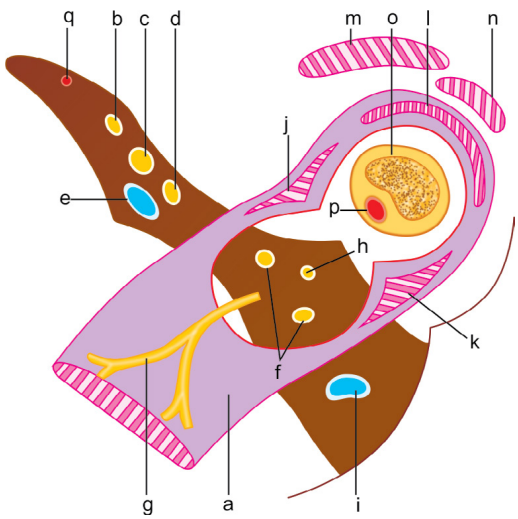


Fig. 1.21: Structures passing through the superior orbital fissure and the optic foramen

a—Lateral rectus; **b**—Lacrimal nerve; **c**—Frontal nerve; **d**—Trochlear nerve; **e**—Superior ophthalmic vein; **f**—Oculomotor nerve; **g**—Abducen nerve; **h**—Nasociliary nerve; **i**—Inferior ophthalmic vein; **j**—Superior rectus; **k**—Inferior rectus; **l**—Medial rectus; **m**—Levator palpebrae superioris; **n**—Superior oblique; **o**—Optic nerve; **p**—Ophthalmic artery; **q**—Recurrent lacrimal artery

- ***Passing through the medial portion:*** Inferior ophthalmic vein.

Inferior Orbital Fissure

It lies between the maxilla and the greater wing of sphenoid.

It transmits

- Maxillary division of trigeminal nerve
- Infraorbital artery
- Zygomatic nerve
- Branches of inferior ophthalmic vein draining into pterygoid venous plexus.

Surgical Spaces of the Orbit (Fig. 1.22)

From the surgical point of view, there are four spaces of the orbit. They are relatively self-contained, as the inflammatory processes are contained for a considerable period of time and each of which must, if necessary, be opened separately.

- ***The subperiosteal space:*** Between the bones of the orbital wall and the periorbita (periosteum).

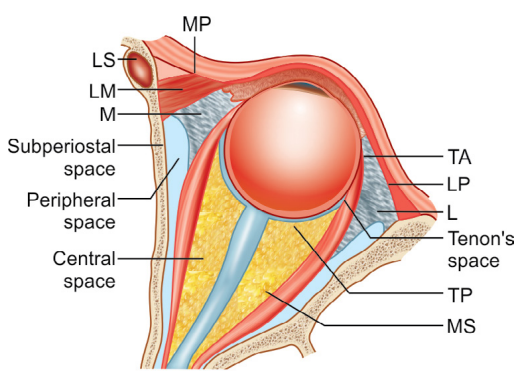


Fig. 1.22: Spaces of the orbit

L—Lateral check ligament; **LM**—Lacrimal portion of orbicularis muscle; **LP**—Lateral palpebral ligament; **LS**—Lacrimal sac; **M**—Medial check ligament; **MP**—Medial palpebral ligament dividing into a superficial and a deep band; **MS**—Muscle sheaths; **TA**—Anterior part of Tenon's capsule; **TP**—Posterior part of Tenon's capsule

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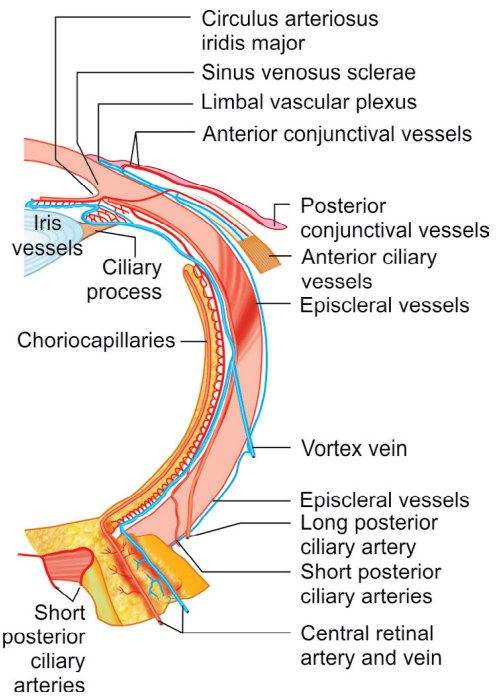


Fig. 1.23: The blood supply of the eye

- ***The peripheral orbital space:*** Between the periorbita and the extraocular muscles which are joined together by fascial connections.
- ***The central space:*** A cone-shaped area enclosed by the muscles (the ***muscle cone***).
- ***Tenon's space:*** Around the globe.

BLOOD SUPPLY OF THE EYE (FIG. 1.23)

ARTERIES (FIG. 1.24)

The eye and the orbital contents receive their main blood supply from the ophthalmic artery.

The eyelids and conjunctiva have an anastomotic supply from the branches of both external carotid and ophthalmic artery.

Ophthalmic Artery

It arises from the fifth bend of the internal carotid artery. It enters the orbit through the optic foramina, below and lateral to the optic nerve.

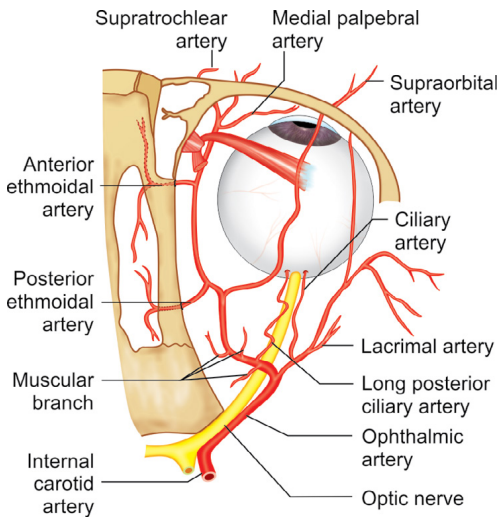


Fig. 1.24: Arterial supply of the eyeball

Branches

- The central retinal artery
- The short posterior ciliary arteries (10–20 in number)
- Two long posterior ciliary arteries
- Recurrent meningeal artery
- Lacrimal artery
- Variable number of recurrent arteries
- Mascular branches to each of the extraocular muscles.

The anterior ciliary arteries are the forward continuations of muscular arteries. Each rectus muscle has two muscular arteries ***except the lateral rectus,*** which has one.

External Carotid Artery

The blood supply to the eyelids and conjunctiva from the branches of the external carotid artery, originates from the external maxillary artery, the superficial temporal artery and the internal maxillary artery.

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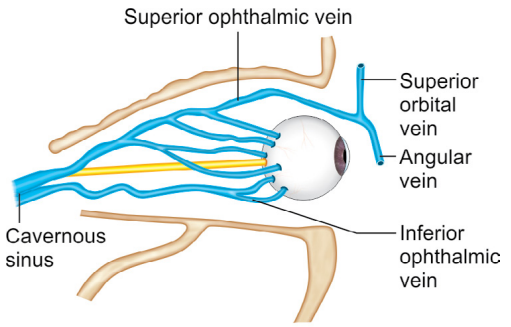


Fig. 1.25: Venous drainage of the eyeball

VEINS

Mainly through the superior and inferior orbital veins and they empty into the cavernous sinus (Fig. 1.25).

- ***Superior orbital vein*** communicates with the angular vein, and then to facial vein.
- ***Inferior ophthalmic vein*** communicates with the pterygoid venous plexus, and also to the cavernous sinus directly or via superior ophthalmic veins.
- Two or more ***superior vortex veins*** drain into the superior orbital vein and inferior vortex veins into the inferior orbital vein.
- The ***central retinal vein*** enters the cavernous sinus separately, or to the superior ophthalmic veins.

Cavernous Sinus

It is an irregular-shaped, endothelium lined venous space, situated on either side of the body of the sphenoid bone.

Connections (Figs 1.26A and B)

- The superior and inferior ophthalmic veins enter it from the front.
- The superior and inferior petrosal sinuses leave it from behind.
- It connects directly with pterygoid plexus, and indirectly via inferior ophthalmic veins.
- To the opposite sinus by two or three transverse sinuses which surround the pituitary stalk.

Structures passing through it (Fig. 1.27):

- Internal carotid artery with sympathetic plexus via medial wall.
- Abducens nerve is just lateral to the artery.
- Ophthalmic and maxillary divisions of the trigeminal nerve, are lateral and below the artery.
- Oculomotor and trochlear nerves are on its lateral wall, on the superior aspect.

A B

Figs 1.26A and B: Tributaries of the cavernous sinus. **A.** Lateral view; **B.** Above view

Ang.—Angular vein; **Cav. sinus**—Cavernous sinus; **Com.**—Communicating vein; **Fac.**—Facial vein; **Fr.**—Frontal vein; **I. oph.**—Inferior ophthalmic vein; **I. petr.,**—(infr. petr.s), Inferior petrosal sinus; **Jug. v.**—Jugular vein; **Lab.**—Labyrinthine veins; **Lat. sinus**—Lateral sinus; **Mas. em.**—Mastoid emissary vein; **Mid. men.**—Middle meningeal veins; **Nasal**—Nasal veins; **Pt. px.**—Pterygoid plexus; **S.o. (oph)**—Superior ophthalmic vein; **Supra-orb**—Supraorbital vein; **S. (Supr.) petr.**—Superior petrosal sinus; **Tr.**—Transverse sinus; **Sps. sinus**—Sphenoparietal; **C. ret. v**—Central retinal vein

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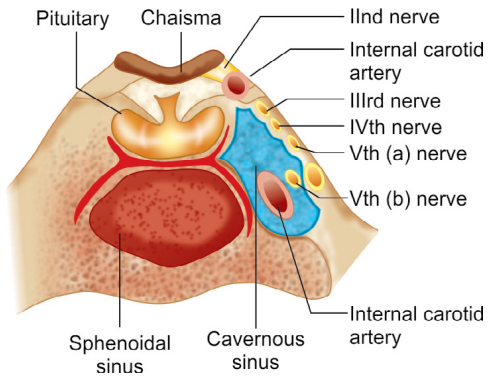


Fig. 1.27: Anatomy of the cavernous sinus and its relationship with the vital structures

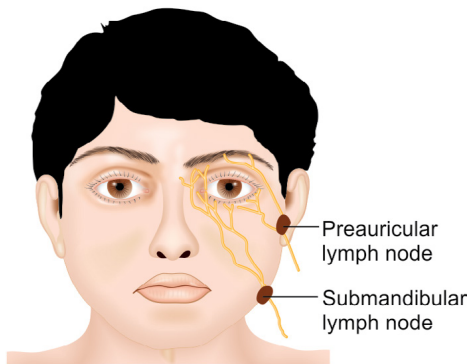


Fig. 1.28: Lymphatic drainage of the eyelids and conjunctiva

One or more nerves may be affected by disease of the cavernous sinus, e.g. thrombosis of the sinus, rupture or aneurysm of the internal carotid artery.

NERVES OF THE EYE

- ***Visual optic nerve***
  The visual pathway—optic nerve → optic chiasma → optic tract → lateral geniculate body → optic radiation → visual cortex.
- ***Motor***
  - ***Oculomotor (N III)***
    - ***Superior division*** → Superior rectus and levator palpebrae superioris.
    - ***Inferior division*** → Medial rectus, Inferior rectus, and inferior oblique.
    - Short root to ciliary ganglion → ciliary and sphincter pupillae muscle.
  - ***Trochlear (N IV)*** → Superior oblique.
  - ***Abducens (N VI)*** → Lateral rectus.
- ***Mixed motor and secretory***
  Facial (N VII) → (i) Motor to face, especially to the orbicularis oculi. (ii) Secretory to the lacrimal gland.
- ***Sensory***
  Trigeminal (NV) → Via ophthalmic and maxillary divisions.
- ***Autonomic***
  - ***Parasympathetic supply***
    - ***Oculomotor nerve*** (from the Edinger-Westphal nucleus) → inferior division → branch to inferior oblique → short root of ciliary ganglion → ciliary and sphincter pupillae muscles.
    - ***Facial nerve*** (from salivary nucleus) → lacrimal gland.
  - ***Sympathetic supply:*** Postganglionic fibers from the superior cervical ganglion → around the internal carotid artery (carotid plexus) → cavernous plexus → via the ophthalmic division of 5th nerve → nasociliary nerve → long ciliary nerve avoiding the ciliary ganglion (some via ciliary ganglion without a relay → short ciliary nerves) → along with long ciliary arteries into the suprachoriodal space → enter ciliary body and iris, to supply dilator pupillae.

LYMPHATIC DRAINAGE (FIG. 1.28)

From the eyelids, conjunctiva and from the orbital tissues.

- ***Medial group*** drains into the submandibular lymph nodes.
- ***Lateral group*** drains into the preauricular lymph nodes and sometimes, into the postauricular lymph nodes.

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2 CHAPTER Physiology of the Eye

CORNEA

The cornea is a transparent tissue, anterior surface of which is bathed with tears, and endothelial surface is bathed in aqueous humor.

The stroma consists of type I collagen fibers of uniform diameter, arranged in a regular lattice work within a ground substance.

The ground substance consists of acid mucopolysaccharides, chondroitin sulfate (type A and type C) and keratin sulfate.

Transparency

In the visible range of spectrum (380 nm to 760 nm), the cornea transmits almost 100% of the light energy.

This transparency is due to following factors:

- ***Anatomical factors***
  - Avascularity of cornea
  - Absence of pigment in the cornea
  - Demyelinated nerve supply
  - Regular arrangement of the epithelial and endothelial cells
  - Regular arrangement of stromal collagen fibrils (lattice theory)
  - Paucity of cells in the stroma
  - Epithelial cells are non-keratinized
  - Anterior surface of the tear-film helps in forming a regular refracting surface.
- ***Relative dehydration (deturgescence) of the stroma which is maintained by:***
  - Epithelium, which is largely impermeable to water.
  - ***Endothelial transport system:*** The endothelial transport system pumps fluid from the corneal stroma to the aqueous by Na-K-ATPase mechanism.
  - Special intercellular junction in the endothelium is also responsible to control the fluid traffic.
- ***Intraocular pressure (IOP):*** It must be optimum to control fluid transport. An acute rise in IOP may result in corneal edema.

Nutrition and Permeability

- ***The peripheral cornea*** receives its nutrients via the blood stream of perilimbal plexus.
- ***The central cornea*** is avascular and the nutrition depends on substances that enter either via the endothelium or the epithelium.
- ***Permeability:*** The tear film is permeable to water soluble substances.
  - The corneal epithelium is readily permeable to lipid-soluble substances, as the cell membrane are composed of lipoprotein.
  - The stroma and the endothelium are permeable to water soluble substances.

Metabolism

Cornea gets its oxygen mostly from the atmosphere. Cornea requires energy to maintain its deturgescence, and also for epithelial cell renewal.

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Energy in the form of adenosine triphosphate (ATP), is provided by the metabolism of glucose. The epithelium and the endothelium are the sites of most metabolism.

The metabolic pathways are:

- ***Glycolysis*** (Embden-Meyerhof pathway) which requires no oxygen.
- ***Tricarboxylic acid cycle*** (Kreb's cycle), in presence of oxygen, mainly in the epithelium.
- ***Hexosemonophosphate (HMP) shunt:*** About 65% of corneal metabolism occurs via glycolysis and the remainder by way of Kreb cycle and HMP shunt.

Wound Healing

- ***Epithelium*** regenerates rapidly, large abrasion is being covered within 24 hours. This involves both epithelial migration, and proliferation of the surviving epithelial cells. After 6 weeks, epithelium adheres to its underlying basement membrane firmly.
- ***Injury to the Bowman's membrane*** causes scar formation.
- ***Stromal healing:*** By multiplication of undamaged keratocytes and migrated fibroblasts. New mucopolysaccharide synthesis begins after 24–48 hours, and established by 5th day. Stromal healing is not initiated until the defect is covered by epithelium.
- ***Endothelium:*** No multiplication occurs in human beings. The cells spread to cover the defect, by enlargement and migration.

LENS

The lens is a transparent crystalline structure, covered by a homogenous capsule, and has epithelium only beneath the anterior lens capsule.

Transparency

It transmits almost 80% of light energy. Its transparency is due to:

- Sparsity of cells
- Single layer of epithelial cells, which is not thick.
- Close alignment of individual cells (the lens extracellular space is less than 5% of its total volume, so the ***zone of discontinuity*** are very small compared to the wavelength of light).
- Semipermeable character of the lens capsule.
- Avascularity
- It's index of refraction ranges from about 1.406 at the center (nucleus) to about 1.386 in outer cortical layers, making it a gradient index lens.
- Pump mechanism of the lens fiber membranes, which maintains relative dehydration of the lens.
- ***Autoxidation:*** High concentration of reduced glutathione in the lens, maintains the lens protein in a reduced state and ensures the integrity of the cell-membrane pump.

Metabolism

The lens fibers are composed entirely of soluble and insoluble proteins.

- ***Water-soluble proteins (85%)*** are α-crystalline, β heavy and β light crystalline, and γ-crystalline; and they are found mainly in the lens cortex. Human lens protein is more labile than that of other species.
- ***Water-insoluble protein (15%):*** Albuminoid fraction and found mainly in the lens nucleus. With age insoluble protein increases. In strict physiological terms, the position of the lens within the eye, is similar to a cell surrounded by extracellular fluid.
- ***The lens epithelium generates energy from carbohydrate metabolism:***
  - Anerobic glycolysis by Embden-Meyerhof pathway—85%
  - Hexose-monophosphate shunt
  - Kreb's cycle
  - Sorbitol pathway.

The lens epithelium maintains low concentration of sodium and water within the lens by active Na-K-ATPase pump system. Glycolysis provides the necessary ATP energy.

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Pathogenesis of Cataract

Any opacity in the lens, or its capsule is called a cataract. Three basic mechanisms cause a cataract:

1. Damage to the lens capsule that changes its membranous properties.
2. Change in the lens-fiber protein synthesis.
3. Increased lens hydration.

When a ***cortical cataract*** forms, the sodium-extruding mechanisms fail.

In Nuclear Cataract

TEARS

Anterior surface of the eye is moistened by tears, secreted by the lacrimal gland and basic secretors, located in the margin of the eyelid and the conjunctiva. The basic secretors of the conjunctiva contribute 10% of the total mass of lacrimal gland.

Circulation of Tears

Composition of Tears

- Very much similar to blood plasma, slightly more dilute (98.2% of water).
- Relative lack of protein (0.6%).
- pH of tear is 7.35, but it may vary with disease ***(alkaline pH is found in vernal catarrh)***.
- Osmotic pressure varies from that of blood (0.9% saline) to the equivalent of 1.4% saline (normal = 290–300 mOsmol).
- ***Protective substances*** present in tears are:
  - Immunoglobulins (IgA, IgG and IgM)

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- Lymphocytes
- Complements
- Lactoferrin
- ***Lysozyme (muramidase):*** A specific enzyme which is lethal to common bacteria, since it is mucolytic and dissolves the bacterial membrane.

Production

The average normal secretion of tears is 0.5–2.2 μl/minute. The maximum capacity of cunjunctival cul-de-sac is about 30 μl.

The lacrimal glands secrete continuously throughout the day, but not during sleep. Half of this secretion is lost from the surface by evaporation.

Reflex stimulation occurs in following situations:

- From irritative sensation of the cornea and the conjunctiva.
- In yawning, coughing, sneezing or vomiting
- After psychic stimulation, as in weeping or laughing. This curious reflex is confined to human beings, and only develops in infants after 3–4 months.
- After exposure to bright lights.
- Secretion can be stimulated by cholinergic drugs (e.g. pilocarpine) and inhibited by anti-cholinergic drugs (e.g. atropine).

Precorneal Tear Film (Figs 2.1 and 2.2) and its Functions

It is a relatively stagnant layer of tears that covers the corneal epithelium and consists of ***three layers***, each of which has separate functions.

1. ***The outer lipid layer:*** It is secreted by the Meibomian glands, glands of Zeis and glands of Moll.
   Functions
   - To retard the evaporation of the aqueous layer of the tear film.
   - To increase surface tension and vertical stability of the tear film to prevent overflow of tears.
   - To lubricate the eyelids.

Fig. 2.1: Precorneal tear film (traditional view)

Fig. 2.2: Precorneal tear film (recent concept)

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2. ***The middle aqueous layer:*** It is the main bulk of the tear film and secreted by the lacrimal and accessory lacrimal glands.
   Functions
   - To supply atmospheric oxygen to the corneal epithelium.
   - It has antibacterial action due to presence of lysozyme and lactoferrin.
   - To provide a smooth optical surface.
   - To wash away dust and debris from the conjunctiva and cornea.
3. ***The inner mucin layer:*** It is secreted by the conjunctival goblet cells, glands of Manz and crypts of Henle.
   Functions
   - To convert the corneal epithelium from a ***hydrophobic*** to a ***hydrophilic*** surface.
   - To wet the microvilli of the corneal epithelium and thereby retains the pre-corneal tear film.

According to a recent concept

- ***Outer lipid layer:*** It contains both polar and non-polar lipids. ***Polar lipids*** such as phospholipids and fatty acids lie adjacent to the aqueous-mucin layer and ***non-polar lipids*** such as cholesterol esters, and triglycerides are present at the tear-air interface.
- ***Middle aqueous-mucin layer:*** Contains soluble mucins and proteins in additions to water and ions.
- ***Inner glycocalyx layer:*** Contains membrane-adherent mucin which is anchored at the cell membrane of numerous microvilli of the corneal epithelium.
- ***Glycocalyx/MUC1:*** It is secreted by the apical conjunctival and corneal epithelial cells. It provides hydrophilic surface.
- ***Mucin (MUC4 and 5AC):*** It is secreted by the conjunctival goblet cells. It adds viscosity to tears and protect the ocular surface during blinking.

AQUEOUS HUMOR

The internal structures of the eye are bathed and surrounded by the aqueous humor, a crystal clear fluid.

Functions

- It helps to maintain IOP.
- It supplies nourishment to the cornea and the lens.
- It maintains optical transparency.
- It takes the place of the lymph that is absent within the eye.

Refractive index	= 1.336
Composition	
Water content	= 98.92%
Solid	= 1.08%

The composition of aqueous of the posterior chamber differs from the aqueous of the anterior chamber. Posterior chamber aqueous contains chloride and ascorbate in excess, than that of the anterior chamber aqueous. But there is deficiency of bicarbonates.

Solid Components

- ***Proteins:*** They are more dilute than plasma, due to blood-aqueous barrier. But when the protein concentration increased, a ***plasmoid aqueous*** results as in inflammation of anterior uvea (iridocyclitis), and also after surgery with newly-formed aqueous.
- ***Non-electrolytes:*** Examples, glucose, urea, uric acid, alcohol and creatinine (all are little less than those of plasma).
- ***Electrolytes:*** Na-ions, K-ions, chloride, and bicarbonates.
- ***Organic acids:*** Ascorbic acid is present in greater concentration than in plasma. Other acids present in aqueous are lactic acid and hyaluronic acid.
- ***Oxygen*** is present in aqueous in dissolved state.

Aqueous Humor Formation

Two mechanisms are involved: 1. Ultrafiltration and 2. Secretion.

Ultrafiltration

Capillary wall in the ciliary processes behave as a crude semi-permeable membrane. It

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retains most proteins, but allows smallest molecules to leave the blood stream, and collects in the stroma of the ciliary processes, and then into the posterior chamber across the ciliary epithelium.

Secretion

By active secretion of the ciliary processes. Two mechanisms are involved:

1. ***Bicarbonate system*** mediated by carbonic anhydrase, and
2. ***Na-K-ATPase system*** for electrolytes.

Once formed, the aqueous is modified by metabolic processes of the cornea and lens, and this chiefly results in a fall in glucose and bicarbonate concentration, but a rise in lactate levels.

Circulation of Aqueous Humor

Circulation is necessary both for metabolic process and to regulate the IOP.

Aqueous formed in the ciliary region → flows from posterior chamber through the pupil into the anterior chamber → angle of the anterior chamber → trabecular meshwork → Schlemm's canal→ aqueous veins → episcleral venous plexus.

Normal flow rate of aqueous—2μl/minute.

In addition there is a second accessory exit ***(uveoscleral outflow)*** which may sometimes be of importance (as in ***buphthalmos* or in *retinal detachment***). Aqueous formed in the ciliary body → suprachoroidal space → episcleral venous plexus.

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3 CHAPTER Physiology of Vision

EFFECTS OF LIGHT ON THE EYE

When light falls upon the retina, it acts as a stimulus to rods and cones, which serve as the visual nerve endings. As there are no rods and cones in the region of optic disc, it has no visual sensation and this is called the ***blind-spot (of Mariotte)***.

When light falls upon the retina, two essential reactions occur in the end-organs—***photochemical*** and ***electrical***.

Photochemical Changes (Fig. 3.1)

- The rods are the receptors for night (scotopic) vision and contain a pigment, visual purple or ***rhodopsin***. The maximum absorption spectrum for rhodopsin is around 500 nm.
- Rhodopsin consists of a colorless protein, called ***opsin*** and is coupled with a chromophore, ***11-cis retinal*** (aldehyde of vitamin A).
- When light falls upon the rods, ***11-cis retinal*** component is converted into ***all-trans retinal*** through various stages.
- This reaction is reversible in the dark, i.e. ***11-cis retinal*** is regenerated from all-trans retinal and vitamin A (retinol) from the blood.
- ***All-trans retinol*** (an alcohol) is vitamin A and is transported in the blood to a specific retinal binding protein.

The cones contain three different photopigments, with maximum absorption at 450, 535 and 570 nm, roughly corresponding to the blue, green and red part of the visible spectrum. These pigments are called ***iodopsin***. Cones not only respond to light of high intensities ***(photopic vision)***, but also act as receptors for color vision.

Electrical Changes

Visual transduction is the process by which light absorbed by the photoreceptors of the outer segment is converted into electrical energy. Though the process is complex, but a battery of ***electrodiagnostic tests*** are now in clinical uses for assessing the integrity of the retina and its central connections.

ELECTROOCULOGRAPHY (FIG. 3.2)

- The average electrical potential of the cornea is positive (about 6 mV) in relation to the retina.
- When electrooculography (EOG) is used, the increase in potential with light adaptation is measured, to evaluate the condition of retinal pigment epithelium (RPE).
- Electrodes are placed at each canthus and the changes in potential between these two electrodes are recorded as the eye moves.
- The average amplitude of the resting potential in light and dark adaptation is masured.

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Fig. 3.1: Visual cycle

Fig. 3.2: Electrooculography. The electrode closer to the cornea is positive, and when the eye turns, a deflection is induced in the recording system (below). The electrooculography deflection is measured from the trough of the deflection on eyes left to the height of the deflection on eyes right

- The ratio of ***light peak*** *to* ***dark trough*** is measured ***(arden ratio)***.
  (***Light peak*** = maximum amplitude obtained in light and ***dark trough*** = minimum amplitude obtained in the dark).
- ***Normal arden ratio*** is 1.80 (or 180%) or greater. 1.65 to 1.80 is borderline, and less than 1.65 is significantly subnormal.

Subnormal arden ratio implies a defect in photoreceptors or RPE. Like, Best vitelliform macular dystrophy, Stargart's macular dystrophy, retinitis pigmentosa and rod-cone dystrophies, choroideremia, gyrate atrophy, chloroquine or hydroxychloroquine toxicity, retained intraocular iron particles (siderosis bulbi), etc.

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Fig. 3.3: Electroretinography

ELECTRORETINOGRAPHY (FIG. 3.3)

Electroretinography (ERG) means a gross record of electrical potential changes in the retina after stimulation with light.

A recorder is made by placing an active electrode on the cornea (via a contact lens) and the other is on the forehead. The small voltage is amplified and usually photographed from the face of the oscilloscope.

Electroretinography Wave Forms

- An initial positive deflection $\mathbf{R}_1$ (early receptor potential—ERP) is followed by a negative deflection $\mathbf{R}_2$. These are due to photochemical reactions in the rod and cone outer segments.
- This is followed by a large negative wave called ***a wave***, and this reflects photo receptor activity.
- Then a large positive ***b wave*** follows. It is due to the response of bipolar cells. There is usually some superimposed oscillatory potentials (OPs) on the ***b wave*** as a response of the amacrine cells.
- Lastly, a small positive ***c wave*** which is generated by RPE.
- The duration of entire response is less than 250 milliseconds.

Responses

The ERG is a mass response of the outer layers of the retina. A normal ERG implies a healthy retinal and choroidal circulation, and normally functioning tissues between the RPE and the bipolar cells. The ganglion cell layer, nerve fiber layer and the optic nerve play no part in this response.

Pathologic responses may be supernormal, subnormal, negative or non-recordable.

Clinical Significances

- Electroretinography is a useful guide in diagnosis and prognosis of certain retinal disorders particularly in retinitis pigmentosa, chorioretinitis, rod-cone dystrophy, etc.
- In ***siderosis bulbi*** (earliest sign).
- Cortical blindness or hysterical blindness.
- Assessing retinal functions in presence of opacity in the media (e.g. cataract, corneal opacity, or in case of vitreous hemorrhage).
- Assessing retinal functions in babies with impaired vision.

VISUAL-EVOKED RESPONSE (FIG. 3.4)

Stimulation of the retina with light, changes the electrical activity of the cerebral cortex.

- The visual-evoked potential or response (VEP or VER) is the electroretinography (EEG), recorded at the occipital pole.
- Since the VER is very small, and often indistinguishable from the background other electrical activities, an averaging technique using a computer, is necessary to identify its waveforms.
- The only consistent recordable response is a large positive deflection occurring 120 milliseconds after stimulation when a pattern is used (pattern VER) and 100 milliseconds when a flash is used (flash VER).

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Fig. 3.4: Diagram of the system used to record the visual-evoked response (VER). The amplitude of the potential is shown to the right. In multiple sclerosis, the amplitude of the response is reduced, and there is a significant delay in the peak-time of the positive wave

- Pattern VER is related to visual acuity and is important to record the visual acuity in children.
- Flash VER reflects the transmission of light from the entire retina, including the fast conducting axons.

Clinical Significances

- ***In optic neuritis:*** VER amplitude is reduced and the latency is increased.
- It is also altered in macular disease or other optic nerve diseases.

So, VER is important in assessing the macular or optic nerve functions in presence of opacities in the media.

VISUAL PERCEPTIONS (SENSATIONS)

Visual sensations result from stimulation of the retina with light. These are four types—(1) light sense, (2) form sense, (3) sense of contrast, and (4) color sense.

Light Sense

This is the faculty which permits us to perceive light in all its gradations of intensities.

- ***Light minimum*** is the minimum amount of light energy which, when falls upon the retina, causes a visual sensation. The light minimum for fovea is higher than parafoveal region. It is increased in diseases of rods and cones, e.g. retinitis pigmentosa.
- ***Dark adaptation (Fig. 3.5):*** The increase in sensitivity of the eye for detection of light that occurs in the dark is called ***dark adaptation***.
  - In the darkness, after the exposure of bright light, there is an initial increase in sensitivity following an exponential time course that reaches a plateau

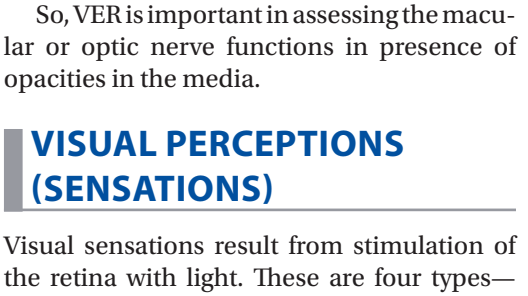


Fig. 3.5: The dark-adaptation curve. There is a plateau between 5 and 9 minutes. The initial portion of the curve indicates the smallest light intensity that will stimulate cones. Rods attain their maximum sensitivity after 30 to 45 minutes. The luminance at –7 is that of sunlight. The luminance at –5 is that of good reading luminance for white paper. The luminance at –4 is that of city street lighting. The luminance at –3 is mean ground luminance in full moon

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after 5 to 9 minutes. This initial phase is due to regeneration of photosensitive pigments in the cones.
  - Then a slower exponential time course that reaches a plateau in 30–45 minutes. This second phase is attributed to rhodopsin regeneration.
  - Dark adaptation is delayed by prolonged exposure to bright light.
  - When fully dark-adapted, the retina is about 100,000 times more sensitive to light than when it is bleached.
  - ***Delayed dark-adaptation*** occurs in diseases of rods, e.g. retinitis pigmentosa and in case of vitamin A deficiency.
- ***Light adaptation:*** It refers to the fall in the visual threshold on moving from darkness into a well lit room.

This decrease in sensitivity is involving two changes—(1) a neural process that is completed in about 0.05 seconds and (2) a slower process, apparently involving the uncoupling of retinal and opsin in rhodopsin, occurring in about 1 minute.

The rods are much more sensitive to low illumination than the cones, so that in the dusk we see with our rods ***(scotopic vision)***; in bright light the cones, come into play ***(photopic vision)***. Nocturnal animals (e.g. bat) have few or no cones, and diurnal animals (like squirrel) have no rods.

Form Sense

This is the faculty which enables us to perceive the shape of the objects.

- Cone is responsible for ***form sense*** which is most accurate the fovea, because here the cones are most closely set and highly differentiated.
- It falls off very rapidly towards the periphery.
- ***Visual acuity*** is defined as the ability to distinguish the shape of the objects and it applies to central vision.
- The form sense is not purely a retinal function for the perception of its composite form (e.g. letters)—is largely ***psychological***.
- In order to discriminate the form of an object, its several parts must be differentiated.
  So, for two points discrimination, it is necessary that two individual cones should be stimulated by them, while the one, in between two cones remains unstimulated.
- Histologically, the diameter of a cone in the macular region is 0.004 mm and this, therefore, represents the smallest distance between two cones.
- It is found that in order to produce an image of minimum size of .004 mm ***(resolving power of the eye)***, the object must subtend a visual angle of 1 minute at the nodal point. This is known as ***minimum visual angle***.
- These principles have been embodied in Snellen's test type (Fig. 3.6), where each letter is perfectly placed in a sqaure which is subdivided into small 25 squares. Each component part of the letter subtends an angle of 1 minute, and the entire letter subtends an angle of 5 minutes at the nodal point of the eye, from a given distance.

Fig. 3.6: Construction of the Snellen letter. Each part subtends an angle of 1 minute. Whole letter subtending an angle of 5 minutes at varying distance from the eye

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Fig. 3.7: Sense of contrast

Sense of Contrast (Fig. 3.7)

It is the ability to perceive slight changes in luminance between regions which are not separated by definite borders. Of all the gratings, the two main variables are the degree of blackness to whiteness, and this is the contrast.

Contrast sensitivity is reduced in many ocular diseases, e.g. in glaucoma, macular disease or refractive errors, and sometimes, it is more important than the loss of visual acuity.

It is especially important for screening the visual acuity in illiterates and young children. Contrast sensitivity is also important in old age and after cataract surgery.

Contrast Sensitivity

In its simplest terms, contrast sensitivity means the ability of the visual system to distinguish between an object and its background. It indirectly assesses the quality of vision.

Contrast sensitivity is the reciprocal value of the contrast threshold. A patient who needs a lot of contrast to see a target has low contrast sensitivity and ***vice versa***.

Blur is not the same as poor contrast sensitivity. People who have poor vision than 6/6 on Snellen's letter chart will experience blurred vision. On the other hand, a person who has poor contrast sensitivity (e.g. due to cataract) may still test well on the 6/6 letters on chart and he may still experience cloudy vision.

Fig. 3.8: Sinusoidal grating

Contrast sensitivity is expressed using sine-wave gratings (Fig. 3.8). A sine-wave grating is a repetitive number of fuzzy dark and light bars, or cycle whose luminance profiles have the shape of simple mathematical function, sine. The number of cycles over a specified visual angle determines its spatial frequency.

Contrast sensitivity tests address the weakness of Snellen's high contrast acuity by varying two parameters—***grating size*** and ***contrast level***. It tests the functional vision, or how well individuals see everyday visual objects or scene (while driving or at work and play).

Contrast sensitivity changes with the age. As we get older, we lose contrast sensitivity, first at the higher spatial frequencies and then for all the spatial frequencies.

Contrast Sensitivity Tests

Several test systems are available (both for distance and near)—the key difference is the target type:

- ***Pelli-Robson chart (Fig. 3.9):*** Low contrast letter charts with same size.
- ***Regan chart:*** Low contrast letter charts of variable size.
- ***Functional acuity contrast test (FACT):*** It measures the specific visual channel. It provides more informative ***sine-wave grating curve***.
- ***Cambridge contrast sensitivity test.***

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Fig. 3.9: Pelli-Robson contrast sensitivity test chart

Diseases and clinical applications

Contrast sensitivity testing provides early detection of eye diseases/conditions that a standard 6/6 letter acuity chart may not detect until the condition is more advanced. They are cataracts, glaucoma, amblyopia, age-related macular degeneration, optic neuritis, drug induced maculopathy or neuropathy, diabetic retinopathy, etc.

- With the advent of new technology such as ***wave-front, aspheric intraocular lens (IOLs)*** and refractive surgery—many intraocular lens ophthalmologists and ophthalmic companies are in favor of contrast sensitivity testing to monitor the progress of the patients.
- It is also recognized by the food and drug administration (FDA) as a more comprehensive and accurate method of assessing vision in ***clinical research trials***.

Color Sense

This is a faculty to distinguish between different colors as excited by light of different wavelengths. The appreciation of colors is a function of the cones and occurs only in photopic condition. In very low intensities of illumination the dark-adapted eyes see no color and all objects are seen as grey, differing somewhat in brightness.

Theories of Color Vision

This is a complicated process that involves physical, biological and psychological mechanisms.

Aristotle's Theory

Color vision derives from the intermingling of elements of light and darkness.

A more advanced eye of primates and human being can distinguish color which are just electromagnetic radiation of different wavelengths. We can see wavelengths in the range of 380–760 nm, the visible spectrum of light (Fig. 3.10). We ***can not see*** ultraviolet or infrared light.

We also do not perceive color in dim light (or in dark), because the light intensity is not enough to stimulate/bleach ***iodopsin***. The threshold frequency of cones is high and requires more light for stimulation.

Trichromatic Theory (of Young-Helmholtz) (Fig. 3.11)

Trichromatic color vision is mediated by interactions among three types of color sensing cones in the retina. Each of the three types of cone contains a different type of photosensitive pigment which is composed of transmembrane protein, called ***opsin***

Fig. 3.10: The total range of electromagnetic spectrum and visible spectrum of light

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Fig. 3.11: Trichromatic theory of color vision

and a light sensitive molecule, called ***11 cis-retinal***. The ***three types of cone*** are:

1. ***Long wavelength (L):*** Peak sensitivity at 570 nm (red-sensitive cones or ***erythrolabe***).
2. ***Medium wavelength (M):*** Peak sensitivity at 535 nm (green-sensitive cones or ***chlorolabe***).
3. ***Short wavelength (S):*** Peak sensitivity at 440 nm (blue-sensitive cones or ***cyanolabe***).

   The peak sensitivity of rods (R) is 498 nm.

   Each different pigment is especially sensitive to certain wavelength of light (photon) to produce a cellular response. This response of a given cone varies not only with the ***wavelength of light*** that hits it, but also with its ***intensity***. The brain would not able to discriminate different colors if it had input from ***only*** one type of cone. Thus interaction between two types of cone is necessary to produce ability to perceive color and trichromatic color vision is accomplished by using combination of cell responses.

   But trichromatic theory does not explain why red matched with green appears yellow, or why blue added to yellow appears white.

   It also does not explain the phenomenon of complementary afterimages (if the eye is adapted to a yellow stimulus, the removal of stimulus leaves a blue sensation or afterimage).

Opponent-process Theory (of Herring) (Fig. 3.12)

Opponent color theory states that the human visual system interprets information about color by processing signals from cones in an antagonist manner. The L, M and S cones have some overlap in the wavelengths of light. So the visual system is more efficient to record differences between the responses of cones, rather than individual cone's response.

The opponent theory suggests that there are ***three opponent channels***—red versus green, blue versus yellow, and white versus black. The last one is achromatic and detects dark-light variations, i.e. ***luminance***. The responses to one color of an opponent channel are antagonistic to those of other color.

Although, ***trichromatic*** theory allows the visual system to ***detect color*** with L, M and S cones (at the photoreceptor level); the ***opponent theory*** (Fig. 3.12) accounts for mechanisms that ***receive and process*** information from cones (inter-neurally). It is now accepted that ***both the theories are important*** to understand the mechanism of color vision (Fig. 3.12).

The recently proposed explanation seems to be more complex:

- L, M and S cones respond best to long, medium and short-wavelength of light respectively.
- Information from cones is passed to the bipolar cells which may be cells in opponent process.

Fig. 3.12: Opponent color vision theory

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- This information is then passed to ***two types*** of ganglion cells—magnocellular (large cells) and parvocellular (small cells).
  Parvocellular cells (P cells) are the major cells ***which handle information about color***. Two groups:
  1. One that processes information about difference between excitation of L and M cones and
  2. Other one that processes difference between S cones and a combined signal from both L and M cones.
     These two subtypes are responsible for ***red-green*** and ***blue-yellow*** processing respectively.

  Magnocellular cells are responsible for transmitting information about intensity of light in the receptive fields.
- Ultimately, these signals are decoded and processed in the brain. At this stage, color memory, context and other factors modify the perception of color. After interpretation of these decoded signals, we ***see*** a color of an object we are looking at.

Color Blindness

Color blinds have defective color discrimination. It is of two type—(1) congenital and (2) acquired.

- ***Congenital:*** Most common, and inherited as an X-linked recessive anomaly. Male 7% and female only 0.7%, have congenitally defective color vision.
  Visual acuity is normal and ophthalmoscopically the fundi appear normal. The defect is present at birth and stationary.
  A ***protanope*** has (***protan*** means first) the red sensation missing, a ***deuteranope*** (***deutan*** means second) has the green and ***a tritanope*** (***tritan*** means third) has the blue sensation missing.
  Red-green blindness is more common in congenital type.
  The term ***achromatopsia*** means the individual borns with severely deficient color perception, where there is absence of all three cones, and thus visual acuity is also very much reduced, and nystagmus is present.
- ***Acquired color blindness:*** Occurs as a result of diseases of the cones, macular diseases, lental sclerosis, optic nerve disease, and drug-induced (e.g. ethambutol).

Importance: Social importance of color blindness is important particularly for drivers, pilots, chemists, biochemists, and also in higher education.

Tests for Color Blindness

- ***Pseudoisochromatic (Ishihara's) test (Figs 3.13A to F)*** is used for routine screening in most ophthalmologist's office. Here,

A B

Normal and color blind should read the number 12 and illiterate can trace the line between the two Xs

Figs 3.13A and B

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C

Normal should read 74, but red-green (R-G) blind people will read the number as 21

D

Normal should see the number 8, but R-G blind people will read the number as 3

E

Normal can trace the orange line between two X's, but R-G blind people cannot follow

F

R-G blind people can trace the line between two X's but normal people cannot follow

Figs 3.13A to F: Ishihara's charts—for normal color vision and color blind people

a series of plates containing panels that are filled by colored dots in which bold numericals or different lines (for illiterates) are represented in dots of various tinted sets. This gives a fair assessment, especially for red-green (R-G) blindness.

- ***Farnsworth-Munsell 100 hue test is scientifically accurate*** and of academic interest.
- ***Edridge-Green lantern test*** is modified by various filters (e.g. to simulate mist, rain, fog, etc). Mainly important for engine drivers.
- ***Holmgren's wools test:*** Color matches are done with different shades of colored wool.
- Nagel's anamaloscope.

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4 CHAPTER Neurology of Vision

THE VISUAL PATHWAYS (FIG. 4.1)

- ***The end-organ:*** It is the neural epithelium of the rods and cones.
- ***The first-order neuron:*** It is the bipolar cell with its axons in the inner layers of the retina.
- ***The second-order neuron:*** It is the ganglion cell of the retina. Its axon passes into the nerve-fiber layer and along the optic nerve to the lateral geniculate body.
- ***The third-order neuron:*** It originates in the cells of lateral geniculate body, then travels by way of the optic radiations to the occipital cortex (visual center).

The visual pathways thus consist of:

- Two optic nerves
- An optic chiasma
- Two optic tracts
- Two lateral geniculate bodies
- Two optic radiations and
- Visual cortex on each side.

In general, the fibers from the peripheral retina enter the periphery of the optic nerve, and the fibers near the optic disc enter the central part of the nerve.

The fibers from the macular area forms the ***papillomacular bundles***, which have a separate course. Partial decussation occurs where the nasal fibers cross at the chiasma.

The fibers of the peripheral retina have two distinct groups, corresponding to the nasal and temporal halves of the retina.

The fibers of the temporal half enter the chiasma and pass to the optic tract of same side, and then to the lateral geniculate body. The fibers from the nasal half enter the chiasma, decussate, and then pass to the optic tract of opposite side, then to the lateral geniculate body.

The third-order neurons pass by the optic radiation into the corresponding occipital lobe. It follow that a lesion of the optic radiation, optic tract, or occipital lobe will cause blindness of the temporal half of the retina of the same side and nasal half of the opposite side. Projecting this outwards, such lesion will cause loss of vision in the opposite half of visual field a condition known as ***hemianopia***.

Figure 4.1 describes the sites of lesions and the corresponding visual field defects in classical cases.

PUPILLARY PATHWAYS AND REACTIONS

Functions of the Pupil

- It regulates the amount of light that enters the eye depending on the state of retinal adaptation.
- It increases the depth of focus, particularly for near objects.
- It reduces the various optical aberrations, e.g. peripheral and chromatic aberrations, and also astigmatism.

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Fig. 4.1: Optic (visual) pathways—all the right and left nasal fibers decussate at the chiasma
The sites of lesions and corresponding visual field defect are **A**—Through optic nerve—ipsilateral blindness; **B**—At the proximal part of optic nerve at chaismal junction—ipsilateral blindness with contralateral superior quadrantopia (Traquair's junctional scotoma); **C**—Middle of chiasma—bitemporal hemianopia; **D**—From lateral sides of chiasma—binasal hemianopia; **E**—Of optic tract—contralateral homonymous hemianopia; **F**—Of temporal lobe—contralateral quadrantic homonymous defects; **G**—Whole optic radiation—contralateral homonymous hemianopia; **H**—Anterior part of the optic radiations—contralateral inferior homonymous quadrantic defects; **I**—Posterior part of the optic radiations—contralateral superior homonymous quadrantic defects; **J**—Occipital cortex—contralateral homonymous hemianopia with macular sparing

The ***sphincter pupillae*** is supplied by the parasympathetic system through the third cranial nerve.

The ***dilator pupillae*** is supplied by the adrenergic fibers of the cervical sympathetic nerve.

The Pupillary Reflexes

Three reflexes are of clinical importance:

1. ***Light reflex:*** If light enters an eye, the pupil of this eye constricts (***direct light reflex***), and there is an equal constriction of the pupil of the other eye (***consensual light reflex***).
2. ***Near reflex:*** A constriction of pupil occurs on looking at a near object, a reflex largely determined by the reaction to convergence.
3. ***Psychosensory reflex:*** A dilatation of the pupil occurs on psychic or sensory stimuli.

Light Reflex

Rods and cones → optic nerve → optic chiasma (partially decussate) → optic tract → pretectal nucleus (instead of running to the lateral geniculate body) → partial decussation in the midbrain → Edinger-Westphal nucleus on each side → third nerve → inferior division → branch to inferior oblique → Short root of ciliary ganglion → ciliary ganglion → short ciliary nerves → sphincter pupillae muscle (Fig. 4.2).

This decussation is important to explain the mechanism of consensual reaction, as

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Fig. 4.2: Pathway of the light reflex

well as direct reaction to light, and also for several pathological reactions.

Near Reflex

It initiates mainly by the fibers from the medial rectus muscle, which contracts on convergence.

Medial rectus muscle → via third nerve → mesencephalic nucleus of fifth nerve → a presumptive center for convergence at pons → Edinger-Westphal nucleus → along the third nerve → accessory ciliary ganglion → sphincter pupillae muscle of the iris.

Sensory Reflex

It is a more complicated process, for both the dilator and constrictor centers play parts in its production.

COMMON ANOMALIES OF PUPILLARY REFLEXES

Hippus

- Alternate rhythmic dilatation and constriction of the pupil.
- These oscillations are very large and often independent of the light.
- It is not a peripheral phenomenon, but depends upon the rhythmic activity of the central nervous system.
- It is found in ***multiple sclerosis***.

Marcus Gunn Pupil

- It is also known as ***relative afferent pupillary defect*** (RAPD) or pupillary escape phenomenon.
- It occurs in defect of the visual pathway anterior to the chiasma.
- ***It consists of:***
  - A diminished amplitude of pupillary reaction,
  - A lengthened latent period and
  - Pupillary dilatation (escape) with continuous light stimulation.
- ***Detection:*** The test for its detection is called ***swinging flash-light test***.
  Normally, if a illuminated penlight is alternately directed to each eye, the pupils constrict and do not vary as the light alternate between the eyes.
  In afferent pupillary defect, both pupils dilate when the light is moved from the unaffected eye to the affected eye. But they constrict when the light is directed to the normal eye.
- Afferent pupillary defect is most conspicuous in unilateral optic neuritis. It distinguishes the reduction of visual acuity caused by optic neuritis, from that caused by cystoid macular edema (CME) or central serous retinopathy (CSR).

Argyll Robertson's Pupil (Fig. 4.3)

It is a bilateral abnormality characterized by failure of the pupils to constrict with light, but retention of constriction presents with accommodation.

- ***The entire syndrome includes:***
  - Absence of light reaction,
  - Presence of accommodation reaction,
  - Pupils are miotic, irregular, eccentric and unequal.

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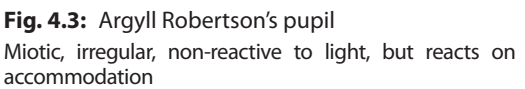


Fig. 4.3: Argyll Robertson's pupil
Miotic, irregular, non-reactive to light, but reacts on accommodation

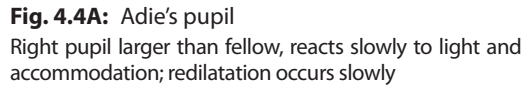


Fig. 4.4A: Adie's pupil
Right pupil larger than fellow, reacts slowly to light and accommodation; redilatation occurs slowly

- Atrophic depigmented patches on iris.
- Pupils fail to dilate with mydriatic, but constrict further with eserine.
- Presence of good vision in both eyes.

- ***Causes***
  - Tabes dorsalis (neurosyphilis), here all the signs are present.
  - ***Non-syphilitic causes:*** Diabetes, multiple sclerosis, hemorrhage, and tumors involving the pretectal region. Here, pupils are not miotic, irregular or unequal, as seen in typical case.
- ***Site of lesion:*** Internuncial neurones between pretectal nucleus and Edinger-Westphal nucleus at the level of pretectum.

Adie's Pupil (Figs 4.4A and B) (Adie's Tonic Pupil)

It is an unilateral dilated but tonic pupil of unknown etiology, a common cause of anisocoria.

- ***Etiology:*** Mostly unknown.
  It may follow a viral illness and in some cases, associated idiopathically with reduced tendon reflexes (like, ankle jerks). There is an impairment of postganglionic parasympathetic innervation of the ciliary muscle or the sphincter pupillae, or both.
- ***Clinical features***
  - Mostly women than men of 3rd to 4th decade.
  - Initially uniocular, but later in many cases, second eye is also involved.
  - Affected pupil is larger than the fellow.
  - Poor pupillary reaction to light, that may involve entire muscle or only a segment.
  - Reaction to near is slow and tonic.
  - Redilatation of pupil occurs slowly.
  - Occasional paresis of accommodation and delay in focusing near object.
  - ***Vermiform movements*** of the iris due to sector paralysis, is appreciated during slit lamp examination.
- ***Pharmacological diagnostic tests***
  - ***With 0.125% pilocarpine:*** The tonic pupil constricts rapidly, but with this concentration, the normal pupil (Fig. 4.4B) does not constrict. This is due to super-sensitivity of the cholinergic stimulation.
  - ***With mydriatic (atropine):*** The tonic pupil dilates with atropine, but the Argyll Robertson pupil does not.

    Holmes-Adie's pupil: It is the condition where Adie's pupil is associated with other signs of nervous system disease, such as absent knee jerks or ankle jerks.

    Adie's pupil is a benign condition. Patient may have headache, or blurring of vision for near.
- ***Treatment:*** There is no effective treatment. Weak miotics may be helpful, or a near correction may be given for the affected eye.

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Fig. 4.4B: Normal pupil (right eye) and Adie's pupil (left eye)

A

B

Figs 4.5A and B: A. Horner's syndrome pupil (right eye); **B.** Horner's syndrome pupil (left eye) (photograph) Right eye—Miosis, ptosis, enophthalmos and anhydrosis of the face on the side of lesion

Horner's Syndrome (Figs 4.5A and B)

It consists of ***miosis, partial ptosis*** and ***enophthalmos***, and sometimes, associated with unilateral absence of sweating of the face on the affected side (anhydrosis).

In congenital form, there may be associated heterochromia of the iris.

It arises from the damage of sympathetic innervation of the eye.

- ***Etiology***
  - In the cervical region—apical bronchial carcinoma, neck glands tumor, etc.
  - Centrally—as with multiple sclerosis, pontine tumor, syringomyelia, etc.
- ***Clinical features:*** The miosed pupil reacts normally to light and accommodation, but dilates poorly with cocaine drop.
  If the lesion lies peripherally (postganglionic)—it dilates rapidly with 1 in 1000 adrenaline drops. This is due to denervation supersensitivity, and this response enables to differentiate a pre- and postganglionic lesion of the sympathetic system.
- ***Treatment:*** If cosmetically unaccepted, ptosis may be corrected surgically (by mullerectomy).

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5 CHAPTER Ocular Pharmacology

DRUG DELIVERY SYSTEM

Therapeutic substances may be introduced into the eye by two major routes—***systemic route*** and ***local route***.

1. ***Systemic routes:*** Here, the drug can be given by mouth or by injections, but it has its obvious limitations because of ***two blood-ocular barriers—***the blood-aqueous barrier, and the blood-retinal barrier.
   i. ***The blood-aqueous barrier:*** It is formed by the non-pigmented layer of the ciliary epithelium, and by the endothelium of the iris vessels. Both of these tissues have tight junctions of the ***leaky*** type.
   ii. ***The blood-retinal barrier:*** It is located in the retinal pigment epithelium and the endothelium of the retinal blood vessels. Both of these tissues have tight junctions of ***non-leaky*** type.

   Drugs enter the eye in proportion to their lipid solubility and smaller molecule size, e.g. sulphonamides has much better penetration than penicillins.

   Severe inflammations and trauma damage the blood-ocular barriers and facilitate the penetration of the drugs in higher concentration.
2. ***Local routes:*** They are as follows:
   - Aqueous or viscous solutions, as drop
   - Ointments
   - Drug-impregnated contact lens
   - Membrane release system (ocusert)
   - Subconjunctival or sub-tenon injections
   - Retrobulbar or peribulbar injection
   - Injections directly into the eye.

- ***Corneal penetration:*** Compounds applied to the anterior globe surface, enter the anterior chamber mainly through the cornea. Epithelium is permeable to the lipid-soluble compounds and stroma to the water-soluble compounds. Thus, the highest intraocular concentrations following administration of compounds should be both water and lipid soluble. Since epithelium is the main barrier, permeability is much increased, if the epithelium is damaged or abraded.
- ***Eye drops:*** They are most popular and easy to instillate. One drop instillation is enough for each time, as the conjunctival sac can not retain more than that.

  Disadvantages
  - Shorter duration of action.
  - Dilution of the drug by tears.
  - Drainage through the nasolacrimal duct leading to systemic toxicity.
  - Difficult for use in children.
- ***Ointments:*** They are particularly useful for night application and also for children. Less systemic absorption via nasolacrimal duct. It also minimizes dilution and prolongs contact time.

  Disadvantages
  - It can not be used during day time as it causes blurring of vision.

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- Allergic reaction to ointment or its base may occur.
- ***Ocuserts*** provides continuous release of a predetermined amount of drug over a period of 5–7 days. Ocusert (pilocarpine) was available earlier for glaucoma treatment and Lacrisert (polyvinyl alcohol) for dry eye management.
- ***Drug-impregnated contact lens:*** A soft contact lens is soaked in a compound, and when the lens is placed in contact with the cornea, it gradually releases the drug, e.g. pilocarpine for glaucoma treatment.
- ***Subconjunctival or sub-Tenon injections*** are popular for administration of antibiotics, corticosteroids or atropine. This is useful at the end of intraocular operation, or treatment of corneal ulcer, or iridocyclitis. A high tissue concentration is maintained for a long time. Here, the route of penetration of drug is mainly via the cornea, though the injection sites are different (*see* Chapter 28).
- ***Injections into the eye***
  - Either into the ***anterior chamber (intracameral)*** or ***vitreous (intravitreal)*** is reserved for desperate cases, specially in endophthalmitis.
  - ***Into the anterior chamber:*** In intraocular lens implantation, e.g. ***viscoelastic substance, adrenaline*** to dilate pupil, preservativefree lignocaine for iris anesthesia in topical phaco emulsification, and ***pilocarpine*** to constrict the pupil.

ASTRINGENTS AND DECONGESTANT

Commonly used in ocular irritations or discomfort, and they are:

- ***Zinc sulfate:*** As drop, also used specifically in ***Morax-Axenfeld bacillus*** infection (angular conjunctivitis).
- ***Silver nitrate (1%):*** As antiseptic and astringent, useful in chronic conjunctivitis or to prevent ophthalmia neonatorum ***(Crede's prophylaxis)***.
- ***Boric acid:*** As an astringent.
- ***Naphazoline:*** As a decongestant.
- ***Antihistaminics:*** As a milder alternative to steroids in allergic conjunctivitis.

ANTIBIOTICS

- ***Topical antibiotics:*** They are generally required to eliminate superficial bacterial infections.
  - Chloramphenicol (0.5%), tetracycline (1%), ciprofloxacin (0.3%), moxifloxacin (0.5%) or gatifloxacin (0.3%) have broad spectrum effectivity.
  - Gentamycin (0.3%), framycetin (0.5%), tobramycin (1%) and ploymyxin-B are effective against Gram '-ve' bacilli.
  - Neomycin (0.5%) is useful only in Gram '+ve' infections and causes local allergy in many cases.
  - Penicillin drops remain stable only for a week or 10 days, and specifically used in gonococcal infections.
- ***Systemic antibiotics:*** Oral (intramuscular or intravenous) are required to eliminate intraocular infections, and in soft tissue affections. Newer penicillin preparations (like, cefazoline and other cephalosporins) are having higher penetration in intraocular tissues.
- ***Subconjunctival antibiotics:*** They are best for treating deep seated infections, as high concentrations are maintained in aqueous for a prolonged period, e.g. gentamycin, amikacin, vancomycin, etc.
- ***Intraocular injections of antibiotics:*** They either into the anterior chamber or vitreous, may be necessary for intractable ocular suppuration, as in endophthalmitis. Gentamycin, amikacin or vancomycin is usually used along with corticosteroids. But they may cause damage to the corneal endothelium or retina (macula).

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ANTIVIRALS

Chemotherapy of ocular viral infections is concerned mainly with keratitis caused by ***Herpes simplex virus (HSV)***, and also to some extent by ***Herpes zoster***.

- ***Idoxuridine (IDU) or 5-iodo-2-deoxyuridine*** inhibits the synthesis of DNA.
  - It is used as 0.1% eye drop or 0.5% ointment. Eighty percent of dendritic keratitis is cured by two weeks. ***Important side effects*** are superficial punctate keratopathy and punctal stenosis.
  - It is applied 1 as drop, or 5 times daily as ointment.
- ***Acyclovir*** is effective both topically and systemically. It is used as 3% ointment for 5 times daily, or oral tablets (200–400 mg) 4 times daily for 7–10 days in herpes simplex keratitis.
  - It is more potent (90%) than IDU and less toxic.
- ***TFT (Triflurothymidine or F_3T)*** is given as 3% drop for 5 times daily. It has less toxicity, greater potency and greater effectiveness in resistance cases.
- ***Vidarabine (Ara-A)*** as 3% ointment. Generally, it is more potent than IDU and active against IDU-resistant virus.
- ***Ganciclovir:*** The first antiviral agent for the treatment of ***cytomegalovirus (CMV)*** retinitis. It is given as 5 mg/kg I/V bid for 14 days. Weekly intravitreal injections of 400 micrograms of ganciclovir for maintenance therapy is also suggested. Myelosuppression is the main side-effect.
- ***Ganciclovir ophthalmic gel (0.15%):*** It is equally effective as acyclovir ointment in acute HSV keratitis. It is given 5 times daily for 7–14 days depending upon the response. Toxicity includes, irritation, blurring of vision, superficial punctate keratitis (SPKs), follicular reaction, etc.
- ***Valaciclovir or valacyclovir:*** It is an antiviral drug used in the management of severe HSV, herpes zoster opthalmicus (HZO) and CMV infection of the eye. It is a prodrug, being converted *in vivo* to acyclovir. It is given orally 500–1000 mg bid or tid doses.
- ***Generally oral valganciclovir*** (combination of ganciclovir and valaciclovir) is the first line of treatment in CMV endotheliitis. It is given 900 mg bid for 21 days followed by 900 mg daily as maintenance dose.
- ***Other drugs include:*** Foscarnet or cidofovir intravenous and/or intravitreal injections in severe sight-threatening HSV, CMV infections in immunocompromized patients.

Note: Not all compounds are commercially available in India.

ANTIFUNGALS

The available antifungal drugs are mainly fungistatic. They are mainly used in ***keratomycosis*** and ***fungal endophthalmitis***.

Three groups of agents are used:

1. ***Polyenes:*** Amphotericin-B, nystatin and natamycin.
2. ***Imidazoles:*** Ketoconazole, miconazole, econazole, fluconazole, itraconazole, voriconazole, etc.
3. ***Flucytosine:*** 5-fluorocytosine.

Amphotericin-B is too toxic for systemic use, but may be used locally as 0.15–0.25% solution (made in 5% dextrose solution or in distilled water)—as eye drop at 1 hour interval. As this solution is preservative-free, it should be discarded after 4 days.

Nystatin is used topically (as 100,000 units/mL) in fungal keratitis—particularly effective against *Candida* species. It is less potent.

Natamycin (pimafucin) is used topically as 5% suspension. It has fairly broad spectrum effect against *Fusarium* and *Aspergillus*, and least against *Candida*. It is the most commonly used antifungal.

Ketoconazole is a well tolerted oral antifungal drug. It is not effective against *Fusarium*.

Voriconazole has very good broad spectrum antifungal activity. It may be given orally

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(200 mg bid) or as a topical (1%) preparation. It is more effective against *Aspergillus* and *Candida* than *Fusarium*. It may be used as adjunct therapy or monotherapy. It is expensive.

Itraconazole (100 mg bid) orally and ***econazole*** as 2% topical preparation are sometimes used with other topical antifungals in resistance cases.

Fluconazole is also a well tolerated oral drug and has broader spectrum of antifungal activity. It is used as 200 mg daily for 3–4 weeks. It is also available as 0.3% solution, as 1 hourly drop in fungal corneal ulcer.

Flucytosine is a less effective agent, and used systemically. It is not much useful in oculomycosis.

AUTONOMIC DRUGS

Autonomic drugs act on sphincter and dilator pupillae muscles of the iris, and also on the ciliary muscles. They are ***miotics***, (pupil constricting), ***mydriatics*** (pupil dilating) or ***cycloplegics*** (drugs causing paralysis of accommodation).

Miotics

Miotics are the drugs which cause miosis or pupillary constriction.

Systemically used drug, e.g. morphine is a strong miotic.

Topical Miotics

- ***Cholinergic drugs*** are of three types:
  1. ***Direct stimulant*** at myoneural junction as acetylcholine, e.g. pilocarpine (1–4%), methacholine.
  2. ***Indirect stimulant*** by abolishing the effects of cholinesterase enzyme, i.e. ***anticholinesterase,*** e.g. eserine, (0.25–1%), neostigmine, demecarium bromide, diisopropyl fluorophosphate (DFP) (0.1%), phospholine iodide (0.06–0.25%), etc.
  3. ***Combination*** of direct and indirect actions—carbachol and urecholine.
- ***Sympatholytic miotics:*** They are rarely used topically, although thymoxamine (0.5%) solution is a powerful miotic, used as an antidote of phenylephrine.
- Miosis can be induced by direct stimulation with histamine.

All miotics stimulate the ciliary muscles to contract, so that the eye assumes a state of partial or complete accommodation. This is observed with long acting anticholinesterases, e.g. DFP, phospholine iodide, etc.

Uses of Miotics

- Almost in all cases of glaucoma including primary angle-closure glaucoma (PACG) or primary open-angle glaucoma (POAG).
- In the treatment of accommodative convergent squint.
- May be useful in accommodation weakness.
- During cataract surgery, after placing a posterior chamber (PC) intraocular lense (IOL) or before placement of an anterior chamber (AC) IOL (with dilute pilocarpine directly into the anterior chamber).
- May be used to counteract the effect of mydriatics, used for ophthalmic examination.

Mydriatics and Cycloplegics

Pupil dilating drugs are called ***mydriatics***. All drugs which dilate the pupil also paralyze the accommodation in greater or lesser degree, due to paralysis of the ciliary muscles (cycloplegia).

Systemically used atropine causes pupillary dilatation.

Topical Mydriatics

- ***Parasympatholytic mydriatics:*** They abolish the action of acetylcholine and, thus, cause mydriasis by making it impossible for the sphincter muscle to contract, e.g. atropine (1%), homatropine (1–2%), hyoscine (0.1–1%), cyclopentolate (0.5–1%) and tropicamide (0.5-1%).

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- ***Sympathomimetic mydriatics:*** Directly act on dilator pupillae to produce mydriasis, e.g. adrenaline (1 in 10,000) as intracameral injection (into AC) phenylephrine drop (2.5–10%), and cocaine hydrochloride.

Phenylephrine has little effect on ciliary muscles and therefore, should not be considered as a cycloplegic. But other parasympatholytic drugs are all cycloplegics.

Among these, ***atropine*** is the strongest cycloplegic agent, and the effect lasts for 10–14 days. ***Homatropine*** is moderately effective and its effect lasts for 48–72 hours.

Cyclopentolate and ***tropicamide*** are very rapidly acting drug, the effect of which lasts only for 12–14 hours.

Uses of Mydriatics

- Fundus examination.
- For preoperative assessment of cataract.
- During extracapsular cataract extraction (ECCE) or posterior segment surgery.
- To test any posterior synechia is present or not.
- To break posterior synechiae (along with cycloplegics).

Uses of Cycloplegics

- For perfect refraction in children, hypermetropic subject and in squints.
- Refraction in case where the pupil is very small.
- In cases of iridocyclitis, keratitis and endophthalmitis.
- In accommodative convergent squint.
- In case of accommodative spasm.
- With atropine as ***penalization*** treatment in amblyopia.

Indomethacin, flurbiprofen or diclofenac eye drop is used along with mydriatics for prolonging the mydriatic effect during intraocular surgery.

Combination of mydriatic cycloplegic is always better except in refraction, e.g. homatropine and phenylephrine, tropicamide and phenylephrine, etc.

Mydriatics should not be used in angle-closure glaucoma, or very shallow anterior chamber; and in case of operated anterior chamber or iris-fixation IOL. They should be used with caution.

LOCAL ANESTHETICS

The small size of the eye and the accessibility of its nerve supply permit most adult ocular surgeries to be done under local anesthesia.

They are of two types—***topical*** and ***infiltrative***.

Topical Anesthetics

They work by stabilizing the nerve membrane potential temporarily, so that it can not be depolarized. They are:

- Lignocaine hydrochloride (4%). Two percent may also be useful.
- Tetracaine (0.5%)
- Proparacaine (0.5%)
- Amethocaine (0.5%)
- Cocaine (2–4%): Also causes mydriasis.

Among these amethocaine and proparacaine, although less potent, are less damaging to the corneal epithelium. They are specially useful in tonometry or gonioscopy.

Infiltrative Anesthetics

Simultaneous motor and sensory block are obtained by infiltration of anesthetic agents in and around the eyeball.

They may be ***facial block*** (to paralyze orbicularis oculi muscle,) ***retrobulbar block, peribulbar block, regional block of the eyelids*** (to excise local lesion), etc.

For infiltration, lignocaine (2%) is normally used. ***Bupivacaine*** (0.5%), a long acting local anesthetic agent is usually mixed with lignocaine (50:50) to achieve better and prolonged anesthetic effect.

Adrenaline (1 in 100,000) prolongs the effect of anesthesia by inducing vasospasm in the injected area, but with slower action.

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It also reduces the chance of bleeding during surgery, especially in oculoplastic procedures.

Hyaluronidase enhances the dispersion and absorption of local infiltrative anesthetic agents. The increased dispersion makes possible, a more effective motor block. But it limits the duration of anesthetic effect.

OCULAR HYPOTENSIVE

The drugs causing lowering of intraocular pressure is called ***ocular hypotensives***. Two types—systemic and local.

Systemic Ocular Hypotensive

- ***Carbonic anhydrase inhibitors***, e.g. acetazolamide 250 mg tab, 1–2 tabs 4 times daily. As there is chance of hypokalemia, a potassium supplement is necessary along with it, in the form of tablet or syrup. Other carbonic anhydrase inhibitors, e.g. ethoxzolamide, methazolamide and dichlorphenamide may also be used.
- ***Hyperosmotic agents:*** (i) Inj. mannitol: 20% solution is used intravenously. (ii) Oral glycerine: 30 mL with 30 mL of lemon or orange juice is given 3 times daily (*see* also Chapter 16).

Local Ocular Hypotensive

- ***Parasympathomimetic:*** Pilocarpine (1–4%) eye drop, 3–4 times daily. Two percent solution is commonly used.
- ***Beta-blockers:*** (i) Timolol maleate (0.25–0.5%) twice daily, (ii) betaxolol (0.50%) twice daily.
- ***Sympathomimetics:*** Epinephrine (0.5– 2%) twice daily.
- ***Topical carbonic anhydrase inhibitor:*** Two percent solution of dorzolamide hydrochloride—3 times daily, in open angle glaucoma and ocular hypertension.

Ocular hypotensive may be used alone or in combination (local and systemic; or two local drops) depending upon the height of intraocular pressure (*see* also Chapter 16).

Ocular hypotensive effect can also be achieved by retrobulbar injection of 1 mL of 70% alcohol, specially in case of absolute glaucoma with pain.

CORTICOSTEROIDS

The corticosteroids reduce the inflammatory responses of the ocular tissues by:

- Decreasing capillary permeability,
- Limiting exudation and
- Inhibiting the formation of new vessels and granulation tissue.

They inhibit the cyclooxygenase and lipoxygenase pathways by inhibiting phospholipase A2, thereby inhibiting the release of arachidonic acid.

All corticosteroids are prepared as ketone-based formulation except loteprednol which is prepared as ester-based formulation. They stays within the tissue more with good therapeutic effects and the same time with more toxicity. Whereas, enzymatic degradation occurs quickly with loteprednol; and it is less potent with less toxicity.

Indications

- ***Sterile ocular inflammations***, e.g. disciform keratitis, episcleritis, scleritis, uveitis, optic neuritis, chemical injury, etc.
- ***Allergic problems***, e.g. phlycten, seasonal allergic conjunctivitis, contact dermatitis, vernal keratoconjunctivitis, allergic blepharoconjunctivitis, etc.
- ***Postoperative***, e.g. following cataract surgery, keratoplasty, vitrectomy, trabeculectomy, etc.
- ***Miscellaneous***, e.g. pseudotumor of the orbit, graft rejection, endocrine exophthalmos, temporal arteritis, post-herpetic neuralgia, etc.

Preparation and Mode of Administration

Local

Three types—topical, periocular and intraocular.

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Topical Preparation

Mostly available as drops and also as ointment, sometimes with some antibiotic combination in India.

- Hydrocortisone acetate: 0.5%
- Prednisolone acetate: 1.0%
- Dexamethasone phosphate: 0.1%
- Betamethasone: 0.5%
- Fluoromethalone: 0.1% Loteprednol etabonate: 0.5% and 0.2%
- Difluprednate: 0.05% (a precursor for prednisolone).

Periocular

- ***Subconjunctival:*** Mainly, injection dexamethasone and in combination with gentamycin—after intraocular surgeries—like cataract, keratoplasty, etc.; in anterior uveitis—only dexamethasone.
- ***Posterior sub-Tenon's:*** Inj. Triamcinolone as in postoperative cystoid macular edema (CME); chronic uveitis, etc.
- ***Peribulbar:*** As in optic neuritis; thyroid ophthalmopathy, etc.

Intraocular

- ***Intravitreal injection:*** Triamcinolone acetonide 4 mg/0.1 mL is useful in uveitic CME, exudative AMD, DME and PDR.
- ***Sustained-release intravitreal drug-delivery:*** Fluocinolone, dexamethasone, combination of dexamethasone and cyclosporine, e.g. Ozurdex 0.7 mg dexamethasone intravitreal implant.

Systemic

Two types oral and intravenous.

Oral

- Prednisone is the most commonly used oral corticosteroid.
- Initial dose is 1–1.5 mg/kg/day and maximum adult dose is 60–90 mg/kg/day. Maintenance dose is 10 mg/kg/day. Tapering is done based on the response by every 10 mg/kg/day every 1–2 weeks.

  Blood glucose level, weight and BP should be regularly monitored. Bone density should be checked within first 3 months and annually thereafter. Calcium and vitamin D supplements may be needed.
- Oral methylprednisolone or betamethasone: May be used in some cases.

Intravenous

- ***Methylprednisolone:*** If an immediate response is needed then intravenous bolus injection of methylprednisolone can be given 500 mg–1000 mg/day for 2–3 days followed by oral prednisone. This is expensive. This is given in optic neuritis, acute graft rejection, Vogt-Koyanagi-Harada syndrome, sympathetic ophthalmia, thyroid ophthalmopathy, etc.
- ***Injection dexamethasone:*** In anaphylactic shock, and also as bolus doses in graft rejection where the patient cannot afford intravenous methyl prednisolone.
  - High dose steroids should not be given for more than 1 month. Disease worsening/no response after 2–4 weeks is an indication for addition of immunosuppressive agents.
  - Side effects of systemic steroids must be kept in mind.

Complications and Side Effects

Local

- Increased risk of superinfection, either with fungus, bacteria or HSV.
- ***Steroid-induced glaucoma:*** It is due to reduced aqueous outflow and occurs more with dexamethasone followed by difluprednate. Fluromethalone and loteprednol have little effect on intraocular pressure, although they are less potent.
- ***Steroid-induced cataract:*** It is mainly posterior subcapsular cataract (more often following systemic use of steroids).
  - Delayed wound healing

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- Corneal melting due to increased collagenase activity
- Mydriasis
- Transient ptosis and occasionally transient myopia
- Papilledema due to pseudotumor cerebri.

Systemic

- Peptic ulceration
- Generalized edema due to sodium and electrolyte imbalance
- Cushing appearance
- Increased severity of diabetes
- Osteoporosis
- Mental changes
- Benign intracranial hypertension.

TRIAMCINOLONE ACETONIDE

Triamcinolone acetonide (TA) is an intermediate acting, relatively powerful steroid. It blocks the breakdown of the blood-ocular barrier by modifying the vascular endothelial growth factor (VEGF) receptor. It is used in both anterior as well as posterior segment disorders.

- ***Giant papillae and venal keratoconjunctivitis (VKC):*** A supratarsal injection of triamcinolone is useful in VKC presenting with giant cobble-stone papillae and which is refractory to conventional treatment. Usually 0.5 mL (20 mg) is injected bilaterally. It results in a dramatic improvement in the size of the papillae, limbal involvement, itching and size of shield ulcers.
- ***Sub-Tenon injection of triamcinolone:*** A posterior sub-Tenon injection of 20 mg TA is useful in resolving CME due to posterior segment inflammation, e.g. uveitis, diabetes, post cataract surgery, post venous occlusion. However, IOP must be carefully monitored post injection.
- ***Intravitreal triamcinolone acitonide:*** It is useful in the management of postuveitic macular edema, pseudophakic macular edema, post branch retinal vein occlusion central retinal vein occlusion (post-BRVO/CRVO) macular edema and in choroidal neovascularization. This injection must be preservative free TA preparation. Postinjection endophthalmitis and secondary glaucoma are serious complications.
- ***Chromovitrectomy:*** Triamcinolone acetamide has the ability to adhere to and stain the vitreous. Thus it is a helpful tool in visualizing the vitreous during vitrectomy, both in the anterior and posterior segment. This injection should also be preservative free.
- ***Triamcinolone (0.1%) eye ointment:*** Used in cases of atopic keratoconjunctivitis or other allergic disorders of conjunctiva or lids.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

As the corticosteroids have many complications, some non-steroidal anti-inflammatory agents are being used recently. They are as follows:

- Flurbiprofen sodium
- Diclofenac sodium
- Ketorolac tromethamine
- Indomethacin
- Bromfenac
- ***Nepafenac:*** It has the best corneal and intraocular penetration.

They are mainly used. Indomethacin is commonly used as oral medicine as local drops.

They are mainly anti-prostaglandins. They compete with the arachidonic acid for cyclo-oxygenase binding, thereby prevent conversion of arachidonic acid to prostaglandins.

Uses

- Prevent miosis during intraocular surgery. It is used with mydriatics in routine practice just before surgery.
- Prevent cystoid macular edema (CME).

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- Reduce ocular inflammation when steroids are contraindicated; as in vernal conjunctivitis; scleritis, allergic conjunctivitis, mild iridocyclitis and post operative cases.

VISCOELASTIC SUBSTANCES

These are tissue-protective viscoelastic liquids with molecular weights ranging from 30,000 to 4 million daltons. They are non-toxic, non-antigenic, and they do not interfere with normal wound healing.

The substances are as follows:

- Sodium hyaluronate (1%)
- Methyl cellulose (2%)
- Chondroitin sulphate
- Hydroxypropyl methylcellulose (HPMC).

Among these, methylcellulose is cheaper and most widely used, though sodium hyaluronate has the most favorable viscoelastic properties (but, the cost is much higher).

Roles of viscoelastics in intraocular surgeries:

- It creates and maintains depth of the anterior chamber during surgery.
- It ***protects corneal endothelial cells*** from mechanical trauma.
- It ***provides a highly viscous environment*** to control a circular capsulorrhexis.
- It fully ***reforms the capsular bag*** to facilitate easy 'in-the-bag' IOL fixation.
- It acts as an internal tamponade in vitreoretinal surgery.
- Overall, it ***acts as a soft instrument*** to manoeuvre intraocular tissue gently.
- It may also be used as ***an artificial tear, or for gonioscopic examination***.

Indications

- Cataract extraction and IOL implantation
- Penetrating keratoplasty
- Glaucoma filtering surgery
- Vitreoretinal surgery.

Complications

Early postoperative secondary glaucoma is common with viscoelastic substance which blocks the angle of the anterior chamber. So, after completion of surgery, it should be thoroughly aspirated from the anterior chamber by diluting with irrigating solution.

IMMUNOSUPPRESSIVE AGENTS IN OPHTHALMOLOGY

Classification

- ***Antimetabolites:*** Azathioprine, methotrexate, mycophenolate mofetil.
- ***Alkylating agents:*** Cyclophosphamide and chlorambucil.
- ***T-cell inhibitors:*** Cyclosporine A, tacrolimus, sirolimus.
- ***Biologic response modifiers:*** Infliximab, adalimumab, daclizumab, rituximab, anakinra.

Antimetabolites

- ***Azathioprine:*** It is a purine nucleoside analogue and interferes with deoxyribonucleic acid (DNA) replication and ribonucleic acid (RNA) transcription.
- ***Indications:*** Given in chronic uveitis, Behcet's disease and sarcoidosis.
- ***Dose:*** 1–3 mg/kg/day for months to years.
- ***Side effects:*** Bone marrow suppression is the most serious side effect and hence a total blood count should be done every 4–6 weeks. Liver function test (LFT) should be done every 12 weeks. The drug is stopped if total white blood cells (WBC) less than 3000/mm^3 or platelets less than 100,000/mm^3.
- ***Methotrexate:*** It is a folic acid analogue and it inhibits dihydrofolate reductase and interferes with DNA replication.
  - ***Indications***: Given in panuveitis, intermediate uveitis, vasculitis, scleritis and orbital pseudotumor.
  - ***Dose:*** 7.5–25 mg/week (single dose) and folate 1 mg/day is given concurrently.

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- ***Side effects:*** Bone marrow suppression, hepatotoxicity, nausea, anorexia are the side effects.
- Complete blood count and LFT should be done every 1–2 months.

- ***Mycophenolate mofetil:*** It metabolizes to mycophenolic acid which reversibly inhibits inosine monophosphate dehydrogenase that inhibits guanosine nucleotide synthesis without incorporating into DNA. Major effects are on T and B lymphocytes.
  - ***Indications:*** It is use in Stevens-Johnson syndrome (SJS), ocular cicatricial pemphigoid (OCP) and high-risk corneal transplants.
  - ***Dose:*** It has good oral bioavailability and should be given on an empty stomach at the dose of 1–1.5 g twice daily.
  - ***Side effects:*** Gastrointestinal distress and diarrhoea are the most common side effects. Bone marrow suppression is serious problem in some patients.
  - Complete blood counts should be performed every week for 1 month, then every 2 weeks for 2 months and then monthly.

Alkylating Agents

- ***Cyclophosphamide:*** There is alkylation of DNA and RNA resulting in cross-linking and cell death. It decreases the number of activated T lymphocytes.
  - ***Indications:*** Given in severe bilateral sight threatening uveitis, Behcet's, progressive systemic sclerosis, Wegener's granulomatosis, intermediate uveitis, sympathetic ophthalmitis and necrotizing scleritis.
  - ***Dose:*** Starting dose is 150–200 mg/day and then 2 mg/kg/day in intermittent pulses. Dose is adjusted to maintain the leukocyte counts between 3000–4000/mm^3.
  - ***Side effects:*** Increased risk of malignancy, bone marrow suppression, teratogenicity, hemorrhagic cystitis, ovarian suppression and azoospermia are the side effects.

- ***Chlorambucil:*** DNA-DNA cross-linking and DNA-protein cross-linking occurs leading to interference in DNA replication and nucleic acid function.
  - ***Indications:*** Given in Behcet's and sympathetic ophthalmitis.
  - ***Dose:*** 0.1–0.2 mg/kg/day.
  - ***Side effects:*** Reversible bone marrow suppression, opportunistic infections, permanent sterility in men, amenorrhea and teratogenicity are the side effects.

T-cell Inhibitors

- ***Cyclosporine:*** Obtained from the fungus *Beauveria nivea*. It is a calcineurin inhibitor that eliminates T-cell receptor signal transduction and down-regulate IL-2 gene transcription and receptor expression of $CD4^+$ T-lymphocytes.
  - ***Indications:*** Oral cyclosporine A is used in VKH, sympathetic ophthalmitis and high-risk cornea transplants cases.
  - ***Doses:*** It is available in two oral emulsions—neoral (microemulsion) and sandimmune. These 2 formulations are not bioequivalent. Neoral is begun at 2 mg/kg/day and sandimmune at 2.5 mg/kg/day. Dose is adjusted to 1–5 mg/kg/day.
  - ***Side effects:*** Most serious side effect is nephrotoxicity. Monthly monitoring of blood pressure, serum creatinine and complete blood counts is a must.
  - Topical cyclosporine (0.05%) is used in dry eye diseases. Topical cyclosporine (2%) may be helpful in VKC, high-risk corneal transplants where oral and topical steroids are contraindicated.
- ***Tacrolimus:*** Obtained from *Streptomyces tsukubaensis*.
  - ***Indications:*** Same as cyclsporine.
  - ***Dose:*** Given orally at 0.1-0.15 mg/kg/day.
  - ***Side effects:*** Nephrotoxicity is less than cyclosporine. Serum creatinine and complete blood counts should be monitored monthly.

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- ***Tacrolimus ointment (0.01%):*** It is used in recalcitrant cases of VKC, in various types of ocular surface inflammation (as in SJS, OCP, etc.).

Biologic Response Modifiers

- ***Infliximab:*** It is a chimeric, monoclonal IgG1κ antibody against tumor necrosis factor-α (TNF-α) and thus decreases pro-inflammatory cytokines.
  - ***Indications:*** Given in human leukocyte antigen B27 (HLA-B27) associated uveitis and Behcet's disease.
  - ***Dose:*** Initial dose—5 mg/kg. 1st dose is on 1st day of therapy; 2nd dose is at the end of 2 weeks and 3rd dose is at the end of 6 weeks.
  - ***Side effects:*** Major side effect is increased risk of infections like tuberculosis. It may enhance brain lesions associated with multiple sclerosis.

Cyclosporine in Ophthalmology

Cyclosporine A (CsA) is a neutral, hydrophobic, cyclic peptide of amino acids which can be isolated from several species of fungi. The unusual structure of CsA is responsible for very low water solubility, causing highly variable and incomplete absorption from its conventional oral or topical formulations.

- It acts as a selective inhibitor of interleukin-2 (IL-2) release during the activation of T-cells and causes cell-mediated immune response suppression.
- ***In dry eye:*** CsA's inhibits subconjunctival and lacrimal gland inflammation, resulting in an increase in tear production and conjunctival goblet-cell density in moderate-to-severe dry eye patients.

Indications and Doses

Systemic

- Along with oral corticosteroids or as corticosteroids-sparing agents in non-infectious ocular inflammations. As in autoimmune uveitis, Bechets' disease, VKH syndrome, sympathetic ophthalmia, severe scleritis, etc.
- High-risk penetrating keratoplasty: To prevent graft rejection.
- May be in Stevens, Johnson syndrome and mucous membrane pemphigoid.
- ***Dose:*** 2–3.5 mg/kg/day of cyclosporine is effective and has a low-risk of side effects.

Topical

- Topical CsA emulsion (0.05%) (Restasis) is approved by the US FDA for dry eye syndrome.
- ***Dose:*** One drop twice daily for 3–6 months or more. The drug is to be kept in refrigerator.
- ***The other indications of 0.05% emulsion:*** Superior limbic keratoconjunctivitis, adenoviral keratoconjunctivitis, Meibomian gland dysfunctions, etc.
- ***Topical CsA (2%) in various oil:*** It may be useful in severe VKC, in high-risk grafts where steroids are contraindicated.

Side Effects

- ***For systemic use:*** It include gingival hyperplasia, pancreatitis, hirsutism, hypertension, renal and hepatic dysfunctions and an increased vulnerability to opportunistic fungal and viral infections.
- ***For topical use:*** Burning and stinging sensation are most common.
- Cyclosporine A, both oral and topical preparations are very expensive.

ANTI-VASCULAR ENDOTHELIAL GROWTH FACTOR AGENTS (VEGF)

- VEGF means vascular endothelial growth factor, is a naturally occurring signal protein which is responsible for angiogenesis or growth of blood vessels.
- Besides having a role in normal vascular growth, VEGF is also responsible for new

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vessels growth in various parts of the eye especially in the retina. VEGF is primarily responsible for many retinal diseases by increasing leakage from these new vessels, causing retinal hemorrhage and macular edema.

- VEGF-A is a chemical signal that also stimulates angiogenesis in a variety of diseases, especially in carcinoma. Bevacizumab is the first clinically available angiogenesis inhibitor to treat colorectal carcinoma in the USA.
- The anti-VEGF agents block the VEGF molecules and thus benefit the patients by decreasing the abnormal and harmful new blood vessels formation, and by decreasing the leakage and macular edema. This leads to stabilization of vision and even improvement in vision in many cases.

The available anti-VEGF agents are:

- ***Monoclonal antibody:*** Bevacizumab (avastin).
- ***Antibody derivative:*** Ranibizumab (lucentis).
- ***Aptamer:*** Pegaptanib (macugen).
- ***Fusion proteins-VEGF trap:*** Aflibercept (eylea).
- ***Oral molecules:*** Lapatinib, sunitinib, sorafenib.
- ***Miscellaneous:*** Bevasiranib.

Indications

- Wet age-related macular degeneration (AMD)
- Choroidal neovascular membrane (CNM)
- CRVO/BRVO and macular edema
- Proliferative diabetic retinopathy (PDR)
- Diabetic macular edema (DME)
- Preoperative in vitreoretinal (VR) surgery for PDR and vitreous hemorrhage
- Eales disease and Coats' disease
- Refractory post-surgical CME
- Neovascular glaucoma (NVG) and/or neovascular iris (NVI)
- To treat corneal neovascularization, e.g. in lipid keratopathy or prior to penetrating keratoplasty (PK).
- In early pterygium recurrence to prevent further progression.

Contraindications

- Fibrovascular epiretinal proliferation (FVP) threatening the macula.
- Patients with active ocular or periocular inflammation.
- Uncontrolled hypertension.
- Cardiovascular diseases.

Complications

- Raise in IOP
- Cataract formation
- Infective endophthalmitis
- Risk of arterial thromboembolic events; like, cerebrovascular accidents, increasing myocardial infarction
- Rebound macular edema
- Immunoreactivity
- Retinal detachment.

Bevacizumab (avastin): Humanized monoclonal antibody that blocks VEGF having an off-label use in ophthalmology. Available as a 4 mL vial (100 mg) and dose is 1.25 mg/0.05 mL. Nonocular uses of avastin include metastatic colorectal cancer, non-small cell lung cancer, metastatic breast cancer, metastatic renal cell carcinoma and glioblastoma multiforme.

Ranibizumab (lucentis/accentrix): Binds and inactivates all isoforms of VEGF. It penetrates the internal limiting membrane (ILM) and can gain access to the subretinal space. Available as a single use glass vial (2 cc) and dose is 0.5 mg/0.05 mL.

Pegaptanib (macugen): It is a pegylated aptamer and selectively binds VEGF-165 with a high affinity. Available as a single dose prefilled syringe 0.3 mg/90 μL.

Aflibercept (eylea): It is indicated for the treatment of patients with—neovascular wet age-related macular degeneration and macular edema following CRVO. It may also be helpful in patients that do not completely respond to lucentis and avastin. It has the potential of lasting effect longer than lucentis and avastin.

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SYSTEMIC REACTIONS TO LOCAL INSTILLATION OF MEDICINES

After instillation, the drug may be absorbed through the conjunctiva or by nasal mucous membrane via lacrimal passages. Some drugs really cause systemic toxic reactions even after local instillation.

Among these, 1% atropine and 10% phenylephrine, may cause characteristic systemic reactions because a single eye drop (1 mL = 15 drops) may exceed the systemic therapeutic dose.

Drug	1 drop	Systemic dose
1% atropine	0.67 mg	0.6 mg
10% phenylephrine (i.e. adrenaline)	6.67 mg	5.0 mg

- ***Atropine intoxication:*** Dry mouth, difficulty in swallowing, the skin is dry, red and hot; the body temperature is raised; tachycardia, and there may be behavioral changes.
- ***Pilocarpine:*** Gastrointestinal overactivity, sweating, tremor, bradycardia and decreased blood pressure.
- ***Ten percent phenylephrine:*** It may increase blood pressure dangerously in old age. Cerebrovascular accidents, and cardiac arrhythmia with extrasystole may also occur.
- ***Cyclopentolate:*** It may cause hallucination, ataxia, vertigo, behavioral changes, and at times syncopal attack.
- ***Timolol maleate:*** It cause respiratory distress specially in bronchial asthma patients, and cardiac arrhythmia in patients with heart block.

Eye drops should be prevented from entering the nasolacrimal duct by maintaining pressure over the inner corners of the closed eyelids for 2 minutes after instillation. An ointment or an oily suspension is better to minimize systemic absorption.

OCULAR REACTIONS TO SYSTEMIC MEDICATION

Ocular side effects may occur from the systemic administration of the drugs. Some of them are as follows:

- ***Corticosteroids:*** Posterior subcapsular cataract and glaucoma are most important. Others are myopia, exophthalmos, papilledema (due to benign intracranial hypertension).
- ***Chloroquine:*** It is usually dose dependent (a regime of 200 mg/day for more than one year), and ***three important effects*** are:
  1. ***Keratopathy:*** Whitish dots in a whorl pattern in the cornea, usually reversible on discontinuation of the drug.
  2. ***Myopathy:*** Failure of accommodation (reversible).
  3. ***Retinopathy:*** A severe pigmentary degeneration of the retina that may progress into blindness. The typical lesion is ***Bull's eye maculopathy*** (concentric rings of pigment loss and hyperplasia around the fovea), leading to central scotoma. The first sign of retinal toxicity is the demonstration of an arcuate scotoma with a red target (decreased color sensation). Retinopathy may not be reversible at late stage.
- ***Quinine:*** Irreversible constriction of visual fields, impaired dark adaptation, and loss of vision (quinine amblyopia).
- ***Ethambutol: Optic neuritis*** in 2% of those treated, leading to reduced visual acuity and color vision, and a central scotoma. This optic neuritis is reversible.
- ***Phenothiazines (chlorpromazine):*** conjunctival pigmentation (light-brown), anterior subcapsular cataract and pigmentary retinopathy.
- ***Digitalis:*** Photopsia (flash), xanthopsia (yellow vision) and central scotoma.
- ***Oral contraceptives:*** Retinal vascular thrombosis, papilledema due to benign intracranial hypertension in susceptible

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individuals. ***Contact lens*** wearers develop an ***intolerance*** with discomfort and irritation due to corneal edema.
- ***Vitamin A:*** Papilledema due to benign intracranial hypertension, and retinal hemorrhage.
- ***Rifampicin:*** Orange-colored tears, which may stain a soft contact lens; painful purulent conjunctivitis.
- ***Sulphonamides including acetazolamide:*** They induce myopia either through spasm of accommodation, or by increased refractive index of the lens. Severe dry eye due to SJS may result blindness.

OCULAR PRESERVATIVES

Ocular medications (eye drops) are composed of unique mixtures of:

- The active drug
- A preservative
- The drug delivery system
- Viscosity-increasing agents
- An aqueous buffered vehicle.

All multi-dose topical ophthalmic preparations use preservatives. In fact, it is mandatory by drug regulation by all countries.

Preservative Benefits

- Plays the key role in maintaining the sterility of ophthalmic solutions through multiple uses.
- Protects against bacterial, fungal or viral contamination that can occur when the dropper tip touches the skin, eyelids, fingers or other non-sterile surfaces. Once the tip is contaminated, the offending agent may be aspirated back into the bottle; or it may blend into the solution with the next use as the medication collects on the tip before dropping into the eye.
- But the ophthalmic ointments are often supplied non-preserved, presumably because they are not as susceptible to contamination from retrograde flow back into the tube.
- Also prolongs the shelf life of the formulation by preventing biodegradation and maintaining drug potency.

Preservative Toxicity

- Primary concern with many preservatives is not their value or efficacy, but rather their recognized cytotoxic side effects.
- Although intermittent use of preserved multi-dose eye drops in normal individuals is probably not harmful, but high concentration (or prolonged and frequent use) of some preservatives can cause damage and irritation to the ocular tissue, particularly in patient with dry eye or glaucoma.
- Preservative-induced toxicity does not have distinct signs or symptomatology. So, damage due to ophthalmic preservatives often goes undiagnosed.
- But now-a-days, it is most often considered the culprit in damaging the corneal epithelium leading to disruption of the corneal apical glycocalyx.
- Awareness of the potential effects of preservatives on overall ocular surface health is relatively low among the physician.

Types and Mechanism of Action

Ocular preservatives can be classified into ***two main categories*** detergents and oxidative preservatives.

1. ***Detergent preservatives***
   - They act upon microorganisms by altering cell membrane permeability and lysing the cytoplasmic content, e.g. benzalkonium chloride (BAK), Polyquad, chlorobutanol and thiomersol.
   - Ocular toxicity can occur because some detergent preservatives can affect eukaryotic cells and thus, cause ocular damage.
2. ***Oxidative preservatives***
   - They are usually smaller molecules that penetrate the cell membranes and interfere with the cellular function.

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- Stabilized oxychloro complex (SOC) and sodium perborate are ***two examples*** of oxidative preservatives.

Oxidative preservatives have an advantage over detergent preservatives because they can provide enough activity against micro-organisms while having negligible toxicity on eukaryotic cells.

Common Ocular Preservatives

Benzalkonium Chloride

- Benzalkonium chloride is a quaternary ammonium compound and is often used in conjunction with disodium ethylenediaminetetraacetic acid (EDTA).
- Edetate sodium is an additive which augments the preservative activity of BAK. However, it is not a true preservative by itself. This is just to lower the concentration of primary preservatives.
- Benzalkonium chloride is the ***most common*** and ***gold standard*** preservative used in topical multidose vials for many years. It is chemically stable, does not degrade easily, even at a higher temperature. It is usually used at a concentration of 0.01% to 0.02%. BAK acts upon micro-organisms by altering cell membrane permeability and lysing cytoplasmic contents.
- Benzalkonium chloride does not appear to have significant adverse effects unless its frequency exceeds four to six times per day. This becomes a concern when patients use other drops on top of chronic medications, such as glaucoma drops or tears substitutes.
- We need to be careful with its use in patients when they are using several medications; are overdoses or have a history of compromized corneal epithelium.

Polyquad (Polyquaternium-1)

- It is a new polymeric quaternary ammonium preservative and it has less toxic effect on corneal epithelial cells than BAK.
- It does not go into deep level and only cause superficial epithelial damage compared to BAK. It is used as 0.001% in ophthalmic solution.

Chlorobutanol

- It is an alcohol-based preservative; so it does not have surfactant action. It works by disorganizing the lipid structure of the cell membrane which increases cell permeability and leads to cell lysis.
- Chlorobutanol has broad spectrum antimicrobial activity. It is used as 0.5% in ophthalmic preparation and in human corneal epithelial cells, the cytotoxic effects are less severe.

Thiomersol

It is used as 0.001–0.004% in ophthalmic drops. It causes cellular retraction, cessation of mitotic activity, and superficial corneal epithelial cell loss.

Phenylmercuric Nitrate, Methylparaben and Propylparaben

They have similar antimicrobial activities as chlorobutanol, but they are relatively ***more epitheliotoxic*** than chlorobutanol or thiomersol.

Noble Preservatives

Stabilized Oxychloro Complex or Purite

- Stabilized oxychloro complex (SOC) is a relatively new preservative and consists of an equilibrium mixture of oxychloro compounds—99.5% chlorite (ClO_2^-), 0.5% chlorate (ClO_3^-) and trace amount of chlorine dioxide (ClO_2).
- It has bactericidal, fungicidal and virucidal activity.
- SOC dissipates by converting into component normally found in tears, such as sodium-ion (Na^+), chloride-ion (Cl^-),

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oxygen and water. Due to oxidation potential of chlorite and possibly from the generation of chlorine dioxide in presence of acidic environments of the microbes, it leads to disruption of the protein synthesis and thereby kill them.

- Sodium chlorite, the key component of SOC, has been used in water purification plant since 1944. It is also used in toothpaste, mouthwash and some antacids. SOC has wide spectrum of antimicrobial activities at a low concentration of 0.005% w/v.

Sodium Perborate (Fig. 5.1)

- It works by oxidizing cell walls or membranes and thereby disrupting the cellular functions. It destroys most bacteria, and can also destroy some viruses and most of the fungi.
- When sodium perborate is combined with water it is converted into hydrogen peroxide, an effective antimicrobial agent. Once sodium perborate enters the eye, it is decomposed to water and oxygen by catalase and other enzymes present in the tear film. It is much gentler than any other preservatives.

Fig. 5.1: Sodium perborate—mechanism of action

Both sodium perborate and purite are noble preservatives and offer attractive options as multi-dose drugs for patients who either require more than 6 doses/day or for those who use more than one type of drop to treat concomitant diseases (e.g. glaucoma and dry eye).

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6 CHAPTER

Refraction of the Normal Eye

THE OPTICAL SYSTEM OF THE EYE

The Roles of the Cornea and Lens

Light that enters the eye is refracted at each of the refractive surfaces that lie in its pathway between the air and the retina. But for all practical purposes, refraction by the eye, takes place at two structures—***the anterior corneal surface*** and ***the lens***.

- ***Refraction at anterior corneal surface:*** Here the major part of ocular refraction takes place, because of:
  - Radius of curvature is 8 mm (approx).
  - Big difference in the refractive indices of the air (1) and the cornea (1.37).
  - Optical power of cornea = +43D (40–45D).
- ***Refraction by the lens:*** This is complicated by the lack of optical homogenecity of the lens substance:
  - The nucleus has a greater optical density.
  - Greater curvature of its surfaces.

Both of these effects enhance its converging power.

Optical power of the lens = +17D (16–20D) (in non-accommodative state).

SCHEMATIC EYE (FIGS 6.1A AND B)

From the above discussion it is clear, that the optical system of the eye reduces itself into two elements only. This simplified optical system in the eye is the ***schematic eye*** which has three pairs of cardinal points:

1. Two principal points (P and P^1)
2. Two nodal points (N and N^1)
3. Two focal points (F and F^1).

A

B

Figs 6.1A and B: A. Schematic eye; **B.** Reduced eye
Upper figure represents schematic eye, with the two focal points at F and F^1, the two principal points at P and P^1, and the nodal points at N and N^1. The reduced eye, drawn in scale to correspond, is shown below, with the two principal foci at F and F^1, the single refracting, surface at P corresponding to the mean of P and P^1, and the nodal point at N, corresponding to the mean of N and N^1

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REDUCED EYE

In schematic eye, the two principal points and the two nodal points are very close together, so close, indeed, it is better to consider for an intermediate point in between them.

Thus, the optical system of the eye is being treated as a single ideal refracting surface. This is the ***reduced eye (of Donder)***.
It has following features (Table 6.1):

- Radius of curvature = 5.73, which separates two media of refractive indices of 1 and 1.336.
- One principal point (P)—lies about 1.35 mm behind the anterior corneal surface.
- One nodal point (N) (optical center)—lies 7.08 mm behind the anterior corneal surface, i.e. just in front of the posterior pole of the lens.
- ***Two principal foci:***
  - Anterior = 15.7 mm in front of the cornea.
  - Posterior = 24.13 mm behind the cornea, i.e. in an average eye, on the retina.

Refractive index for substances of interest in ophthalmology is described in Table 6.1.

TABLE 6.1: Refractive index for substances of interest in ophthalmology

Substances	Refractive index
Air	1.00
Water	1.33
Aqueous humor	1.34
Vitreous humor	1.33
Cornea	1.37
Crystalline lens	1.39
PMMA (IOL)	1.49
Hydrophilic acrylic (IOL)	1.43
Hydrphobic acrylic (IOL)	1.47–1.54
Silicone (IOL)	1.41
Crown glass	1.52
Flint glass	1.65

Abbreviations: **PMMA**—Polymethyl methacrylate; **IOL**—Intraocular lense

Fig. 6.2: Formation of retinal images
The image 'ab' of an object 'AB' is formed, by drawing lines from A and B through nodal point N. 're' is the position of the refracting surface of the reduced eye. 'ANB' or 'aNb' represents the visual angle

The Formation of Retinal Images (Fig. 6.2)

Since the nodal point (N) acts as the optical center of the reduced eye, rays which pass through it, will not be appreciably refracted. It is evident that the image thus formed, is inverted and diminished (just as an image formed by a convex lens). It is reinverted psychologically at its accustomed size in the cerebral cortex.

The angle, subtended by the object at the nodal point is called ***visual angle*** and it is, of course, equal to the angle, subtended by the retinal image at the nodal point.

PHYSIOLOGICAL OPTICAL DEFECTS

There are certain inherent limitations in the eye as a perfect refracting system.

- ***Diffraction of light:*** Light brought to a focus does not come to a point, but gives rise to a blurred disc of light surrounded by several dark and light bands. In human eye, diffraction is of little consequence unless the pupil diameter is under 2 mm.
- ***Chromatic aberration (Fig. 6.3):*** There is a tendency of white light to be split up into its components after refraction, and the image will have a colored edge.

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Fig. 6.3: Chromatic aberration in the eye. The blue rays being normally focused in front of the retina and the red rays behind it

After refraction of white light:

- A yellow light is focused on the retina.
- A blue (short wavelength) light is slightly pre-retinal (eye is myopic for blue).
- A red (longer wavelength) light is slightly post-retinal (eye is hypermetropic for red).

This property is used for testing refraction by the duochrome ***(Duochrome test)***.

- Chromatic aberration can be neutralized by ***achromatic lens***. A convex lens of crown glass (refractive index = 1.52) combined with a concave lens of half the strength of flint glass (refractive index = 1.65) is used for this purpose.
- ***Spherical aberration (Fig. 6.4):*** As the periphery of a spherical lens has a higher refracting power than the central part, the peripheral rays are focused nearer to the lens than the central ones.

  This error can be eliminated by making the anterior surface greater than that of the posterior ***(aplanatic lens)***.
- ***Decentration:*** The center of curvature of the separate lens systems of the eye are never exactly placed on the optic axis (Fig. 6.5).
  - ***Optic axis (AB)*** is the axis through the centers of the various media of the eye.
  - ***Visual axis (OM)*** is the line passing through the macula and nodal point.

  The angle between these two axes is called angle alpha (α). Normally visual axis cuts the cornea on nasal side of the optic axis. It is 'positive -α' (about 5°), and causing an 'apparent squint'. It may be 'negative' when the visual axis cuts the cornea on the temporal side.
  - ***Pupillary line*** is the line which passes perpendicularly through the central point of the pupil. The optic axis is difficult to determine, but it virtually corresponds to the central pupillary line.

    Angle kappa (κ), the angle between the visual axis and the pupillary line, thus, roughly corresponds with angle alpha (α).
  - ***Fixation axis (OC)*** is the line joining the point of fixation with center of rotation of the eyeball.

    Angle gamma (γ) is the angle between optic axis and fixation axis. When optic axis of a lens does not coincide with that of the eye, the lens is not centerd and induces a prismatic effect.

    In general, each cm of decentration of 1D lens induces an image displacement equivalent to 1 prism diopter.
- ***Peripheral aberrations:*** The clarity of the image at retinal periphery is impaired by

Fig. 6.4: Spherical aberration

Fig. 6.5: Visual angles: 'Angle α'=between optical and visual axis; 'Angle κ'=between pupillary line and visual axis; 'Angle λ'=between optic and fixation axis
AB—Optical axis; **OM**—Visual axis; **OC**—Fixation axis

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a number of optical aberrations. These include ***comma, radial astigmatism, image distortion***, etc.

EXTRANEOUS IMAGERY IN NORMAL EYE

- ***Dioptric imagery:*** When light falls on the eye, the major part of it passes through the ocular media to reach the retina and the imagery that forms is called dioptric imagery.
- ***Catoptric imagery:*** A part of the light will always reflected from the media of the eye, and will be apparent to the observer as catoptric imagery.
- ***Entoptic imagery:*** Some light may reach the retina by reflection of structures within the eyeball and they produce entoptic imagery.

Catoptric Imagery

The light that is reflected by the ocular media back to the observer may be either regularly reflected light, or diffusely reflected light. They are appreciated because of the zones of optical discontinuity.

Purkinje-Sanson Images (Fig. 6.6)

There are four types of images:

- ***1st image:*** It derives from the anterior corneal surface. It is the ***brightest*** and erect image, and moves in the same direction.
- ***2nd image:*** It derives from the posterior corneal surface, it lies adjacent to 1st image. It is faint but erect image.
- ***3rd image:*** It derives form the anterior lens surface. It is ***largest***, dim and erect image.
- ***4th image:*** It derives from the posterior lens surface. It is small, dim and ***inverted***, as the posterior lens surface is concave. The image lies at the pupillary plane and moves in the ***opposite direction***.

Purkinje-Sanson images are important to diagnose corneal pathology, different types of cataract, and presence or absence of lens. If the patient is taken in a dark room, or the pupil is dilated, the images will be better appreciated.

Fig. 6.6: Purkinje-Sanson images
a—anterior corneal reflection; **b**—posterior corneal reflection; **c**—anterior lens reflection; **d**—posterior lens reflection; **e**—second order reflection

Entoptic Imagery

An entoptic image arises, when light from any structures within the eye reaches the retina, so as to excite visual sensation.

Any structure infront of rods and cones, and less transparent, can be made visible entoptically. They may be normal structures (in connection with retinal circulation), anatomic anomalies, or normal response to an abnormal stimuli.

- ***Opacities in the media*** as (1) in the cornea or lens, gives a fixed black spot. (2) in the vitreous—as floaters, like flies to a large floating black spot.
- ***Retinal vessels*** as branching black lines, or corpuscular movements ***(flying corpuscles)*** are better appreciated in blue field.
- ***Phosphene*** are the visual sensations that can result from abnormal stimuli, e.g. prolonged pressure on the globe with finger.

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7 CHAPTER

Refractive Errors

DEFINITIONS AND CLASSIFICATIONS

Emmetropia (no refractive error): It is the ideal condition in which the incident parallel rays come to a perfect focus upon the light sensitive layer of the retina, when accommodation is at rest (Fig. 7.1).

Ametropia (refractive error): It is the opposite condition, wherein the parallel rays of light are not focused exactly upon the retina, when the accommodation is at rest.

Anisometropia (unequal error): When the refractive condition of the two eyes are unequal.

Ametropia or ***refractive errors*** may be of three main types.

1. ***Hypermetropia*** principal focus is formed behind the retina (Fig. 7.1).
2. ***Myopia*** principal focus is formed infront of the retina (Fig. 7.1).
3. ***Astigmatism*** the refractive system is unequal in different meridians, and no single point focus is formed upon the retina.

Fig. 7.1: Hypermetropia (H), emmetropia (E) and myopia (M)

Ametropia may be due to one or more of the following conditions:

- ***Abnormal length of the globe (axial ametropia):*** The anteroposterior diameter of the eye is too short in hypermetropia or too long in myopia. ***1 mm shortening will cause +3.0D hypermetropia, similarly 1 mm enlargement will cause –3.0D myopia.***
- ***Abnormal curvature of the refractive surfaces of the cornea or lens (curvature ametropia):***
  - Too weak or flat curvature of the cornea or lens results in hypermetropia and too strong or steep curvature results in myopia. ***1 mm flattening will cause +6.0D hypermetropia, similarly 1 mm steepening causes –6.0D myopia.***
  - Irregular or unequal curvature, in different meridians, gives rise to astigmatism.
- ***Abnormal refractive indices of the media (index ametropia):*** This is clinically evident in case of lenticular changes.
  - If the index of the lens cortex is increased, as in early cortical cataract—there is hypermetropia.
  - Conversely, if the index of the nucleus increases, as in nuclear sclerosis (early nuclear cataract)—myopia is produced.
  - If the increase in the refractive index of the nucleus is very marked—a 'false lenticonus' may be produced. Here, the

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central part of the lens is myopic, but the peripheral part is hypermetropic.
  - If the index of any part varies irregularly (as in developing cataract)—an 'index astigmatism' is produced.
- ***Abnormal position of the lens:*** If the crystalline lens is displaced backwards—***hypermetropia*** will result, and if forwards— ***myopia***.
- ***Obliquity of the media:*** As in lenticular obliquity [subluxated lens or oblique placement of intraocular lens (IOL)]—***astigmatism*** will result.
- ***Absence of an element of the system:*** Absence of the lens (aphakia) produces high ***hypermetropia***.

HYPERMETROPIA (LONG-SIGHTEDNESS)

It is the refractive error, in which the incident parallel rays of light are brought to a focus posterior to the light-sensitive layer of the retina, when the accommodation is at rest.

Etiology

- ***Axial hypermetropia*** is by far the most common.
  - In fact, all the newborns are almost invariably hypermetropic (approx + 2.5D). This is due to shortness of the globe and is physiological.
  - It may also occur pathologically, when retina is displaced forward [retinal detachment, central serous retinopathy (CSR), orbital tumors, retinal tumor, etc.]
  - In microphthalmos or nanophthalmos ('*nano*' means '*dwarf*'—where the axial length is less than 20.0 mm), there is high hypermetropia.
- ***Curvature hypermetropia*** as in cornea plana, following a corneal injury, lens plana, etc.
- ***Index hypermetropia*** due to increase in refractive index of the lens-cortex in old age.
- ***Removal of the lens*** (aphakia).

Clinical Types

Depending upon the act of accommodation, ***total hypermetropia (tH)*** may be divided into:

- ***Latent hypermetropia (lH):*** Which is corrected physiologically by the tone of the ciliary muscle. As a rule, latent hypermetropia amounts to only one diopter. It can be revealed only after atropine cycloplegia.
- ***Manifest hypermetropia (mH):*** It is made up of two components:
  1. ***Facultative hypermetropia (fH)*** is that part of hypermetropia which can be corrected by the effort of accommodation.
  2. ***Absolute hypermetropia (aH)*** which can not be overcome by the effort of accommodation.

  tH = lH + mH (fH + aH)
     - As the tone of the ciliary muscle decreases with age, some of the lH become manifest.
     - As the range of accommodation gets reduced with age more of the fH becomes absolute.
     - In early life (unless the error is unusually large), the accommodative power can correct it all, and none of the hypermetropia is absolute.
     - But after age of 60 years, all of hypermetropia become absolute (as the accommodation is minimum).
     - Total hypermetropia is usually elicited after complete paralysis of accommodation, i.e. by atropinization.

Optics

Theoretically, far point ***(punctum remotum)*** of the emmetropic eye is at infinity. But in hypermetropia, far point of the eye is a virtual point behind the eye.

- In hypermetropia, rays coming from a point on the retina will be divergent than the corresponding rays of the emmetropic eye (Fig. 7.2A).

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- So, in hypermetropia the formation of a clear image is impossible, unless the converging power of the optical system is increased (Fig. 7.2B).
- This may be done in two ways:
  1. By the eye itself, i.e. by the effort of accommodation (Fig. 7.2C).
  2. By artificial means, i.e. by a convex lens placed in front of the eye (Fig. 7.2D).









Figs 7.2A to D: Optics of hypermetropia. **A.** Parallel rays come to a focus upon the retina in emmetropic eye; **B.** Parallel rays come to a focus 'behind' the retina (hypermetropia); **C.** Parallel rays are brought to a focus upon the retina by increasing the refractivity of the lens (a) normal lens (b) more convex in accommodation; **D.** Parallel rays are brought to a focus upon the retina by a convex spectacle lens

Symptoms

They vary with the amount of hypermetropia and the age of the patient (accommodative effort).

- Blurred vision—more for near than for the distant.
- Eye strain (accommodative asthenopia).
- Artificial myopia—due to excessive accommodation → spasm of the ciliary muscle.
- Convergent squint—due to continuous efforts of accommodation → excess of convergence → dissociation of muscle balance → a convergent squint.
- Early onset of presbyopia.

Signs

- ***Small eyeball*** (in all directions)
- ***Smaller cornea***
- ***Shallow anterior chamber:*** It predisposes to angle-closure glaucoma since the size of the lens is normal.
- ***Apparent divergent squint:*** It is due to a large positive ***angle alpha*** (α), as the macula is relatively far from the disc.
- ***Ophthalmoscopy:*** Optic disc is smaller, hyperemic, with less defined edges, even simulating a papillitis ***(pseudoneuritis)*** (Fig. 7.3).

Fig. 7.3: Hypermetropic fundus—pseudoneuritis and shot-silk appearence

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- ***Shot-silk retina:*** A peculiar sheen, is a reflex effect of the retina (Fig. 7.3).
- ***Blood vessels:*** Undue tortuosity and abnormal branching.

Problems with hypermetropic eye

- Amblyopia—more with unilateral high hypermetropia
- Accommodative convergent squint
- Angle-closure glaucoma
- Early onset of presbyopia.

Clinical test to find out different types of hypermetropia:

- The ***strongest convex*** lens with which the patient can still maintain full distant vision (6/6)—indicates manifest hypermetropia.
- While, if the patient can not normally see 6/6 without a lens—then the ***weakest convex lens*** that will allow him to read this line indicates his absolute hypermetropia.
- Facultative hypermetropia is the manifest hypermetropia minus absolute hypermetropia (fH = mH – aH).

Treatment

Mild hypermetropia without any definite symptom does not require any treatment especially for young individual.

Treatment is required in middle-aged patient, in high hypermetropia and if the patient is having symptoms. Treatment may be ***optical , surgical*** and ***laser***.

Optical

Glasses and contact lens.

- ***Glasses:*** Convex lenses are prescribed after full cycloplegic correction, particularly in children. Refraction under atropine is obligatory for small children.
  - A child with convergent squint may require ***full atropine correction***. But such glasses often make the distant vision blurred.
  - So a fair compromise ***(rule)*** may be manifest hypermetropia plus a quarter of latent hypermetropia for final prescription.
- ***Contact lens:*** The power is little more than spectacle power. It is useful for high hypermetropia.

Surgical

- Clear lens extraction with IOL implantation (monofocal or multifocal)—preferable for patients above 40 years of age.
- Intraocular lens implantation—most popular in aphakic hypermetropia.

Laser

Photorefractive keratoplasty (PRK) with excimer or hyperopic laser. Here, peripheral cornea is made thinner with laser, thereby cornea becomes more convex.

MYOPIA (SHORT-SIGHTEDNESS)

It is that dioptric condition of the eye in which, incident parallel rays come to a focus anterior to the light sensitive part of the retina, when accommodation is at rest.

Myopia means ***I shut the eye***; the term was introduced from the habit of half-shutting the eye (gaining the advantage of stenopeic slit vision) to improve distant vision.

Etiology

- ***Axial myopia:*** When the axial length is more.
- ***Curvature myopia:*** When the curvature of the cornea or lens is more, e.g. keratoconus, keratoglobus or megalocornea, and lenticonus.
- ***Index myopia:*** Nuclear sclerosis and in diabetes (as the index of nucleus is more).
- ***Forward displacement of the lens.***

The progressive myopia is largely hereditary (recessive) and frequently racial. Here, the characteristic changes of distension and degeneration at the posterior pole of the eye is genetically determined.

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There are few reports which state that myopia is aggravated by close work, watching TV, computer game or any other ***exactions*** of the civilized world.

Clinical Types

It is of three types—***congenital***, ***simple*** and ***pathological***.

1. ***Congenital or developmental myopia***
   - The child is born with elongated eyes.
   - The refraction may be upto –10D.
   - Typical fundus changes are seen.
   - Progression is rare.
2. ***Simple myopia***
   - Most common clinical type.
   - Does not progress much after the adolescence.
   - May be upto –5D or –6D.
   - No degenerative changes are seen in the fundus, although peripheral retinal degeneration may be seen in later life.
   - Associated with good vision with a good prognosis.
3. ***Pathological myopia***
   - It is also called ***progressive*** or ***degenerative myopia***.
   - Myopia appears in childhood (5–10 years of age), and increasing steadily with age upto 25 years or beyond.
   - The final amount of myopia may be –15D to –25D or more.
   - There are typical degenerative changes in the fundus.
   - Strongly hereditary and more common in female.
   - More in Japanese, Chinese or Jews.
   - Prognosis is usually poor.

If the myopia is more than –6D, it is called high myopia.

Optics

- Far point of the emmetropic eye is at infinity. But in myopia, the far point is at a finite distance in front of the eye (Figs 7.4A to C).



Figs 7.4A and D: Optics of myopia. **A.** Parallel rays are being focused upon the retina (emmetropia); **B.** Parallel rays being focused 'in front' of the retina (myopia); **C.** When looking at a near object, the divergent rays are focused upon the retina. N is the near point; **D.** Parallel rays are brought to focus upon the retina by a concave spectacle lens

- In myopia, the rays coming from a point on the retina are less divergent than the corresponding rays in emmetropic eye.
- So, in myopic eye, the clear image is only possible when the diverging power of optical system is increased, i.e. by means of a concave lens (Fig. 7.4D).
- Since the nodal point is farther away from the retina, the image is larger, but blurred.
- There may be an apparent convergent squint due to the presence of large negative ***angle alpha***.

Symptoms

- ***Impaired distant vision:*** This is the most prominent symptom. Greater the degree of myopia, greater is the blurring for distant vision.

  Serious visual loss with loss of central vision may be seen in progressive myopia.

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- ***Eye strain:*** It is only with small degree of error, but not so obvious as in case of hypermetropia.
- ***Exophoria* or *divergent squint:*** Myope exerts less accommodation → converge to a lesser extent → leading to exophoria, or even a manifest divergent squint. (Accommodation will worsen rather than to improve vision, in myopic eye).
- ***Black floaters*** (due to vitreous degeneration), and sometimes ***flashes of light*** are noticed.
- ***Delayed dark adaptation*** or even night blindness seen in progressive myopia.
- ***Sudden loss of vision*** due to retinal detachment which may lead to ultimate blindness (especially in pathological myopia).

Fig. 7.5: The myopic crescent and supertraction crescent

RPE—Retinal pigment epithelium

Note: Retina and choroid become separated from the disc on the temporal side, forming a myopic crescent. They encroach over the disc on the nasal side forming the super-traction crescent, thus blurring the disc margin

Signs

Opposite of those found in hypermetropia:

- Prominent eyeball (pseudoproptosis)
- Larger cornea, deep anterior chamber and a large pupil
- Apparent convergent squint
- Degeneration (liquefaction) of the vitreous.
- ***Ophthalmoscopically (Figs 7.5 and 7.6):***
  - ***In simple myopia:*** It is almost normal, with only a large disc. Temporal crescent may be seen. There may be peripheral retinal degenerations.
  - ***In pathological myopia***
    - ***Optic disc:*** Large disc with mild pallor. Large physiological cup. Temporal crescent (Fig. 7.6A), or sometimes annular crescent (Fig. 7.6B). There may be super-traction crescent on nasal side. Posterior staphyloma.
    - ***Macula:*** Dull, ***Foster-Fuchs fleck*** (spot) and degeneration (Fig. 7.6C).
    - ***General fundus:*** Pale and tesselated appearance. There may be choroidal sclerosis and patches of choroidal atrophy.
    - ***Peripheral retina:*** Cystoid degeneration is common, other degenerations are also seen.
    - ***Incidence of open-angle glaucoma is more*** (applanation tonometry is mandatory as the scleral rigidity is low in myopia).

A B C

Figs 7.6A to C: Fundoscopic findings in myopia. **A.** Temporal crescent; **B.** Annular crescent; **C.** Retinal degeneration

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Complications

These are mainly with progressive myopia.

- Retinal tear → vitreous hemorrhage
- Retinal detachment
- Degeneration of the vitreous
- Primary open-angle glaucoma (higher incidence).
- Posterior cortical and nuclear cataract.

Treatment

Optical

Glasses and contact lens.

- ***Glasses:*** By appropriate concave lenses. ***Myopia must never be over-corrected***
  - ***In cases of low myopia (upto –6D):*** A full correction is given for constant use, especially for children. In adults, weaker lenses for near work are essential.
  - ***In high myopia (more than –6D):*** Slight under-correction is always done, and same, or more weaker lenses are prescribed for the near work.

  Problems of high minus glasses
  - Minification of the object, and the image appears very bright and clear, which is uncomfortable.
  - Image distortion ('barrel' distortion) due to spherical aberration.
  - Reduced peripheral field of vision.
  - Cosmetically, the eyes appear smaller behind the glasses.
- ***Contact lenses:*** It have most important role in myopic correction.

Advantages

- Less magnification of image
- Image distortion can be eliminated
- Field of vision is increased
- May arrest myopia to-some-extent.

Disadvantages

- Not tolerated by all patients
- Needs extreme accuracy and hygiene
- Costly
- Corneal problems, like infection, abrasions, etc. are dangerous.

Surgery

- ***Keratorefractive surgeries***
  - ***Radial keratotomy (RK) (Figs 7.7 and 7.8):*** Here, the myopia is reduced completely or partially by flattening the cornea, by giving a series (4–16) of deep radial incisions. Obsolete procedure now-a-days.



Figs 7.7A and B: Radial keratotomy. **A.** Parallel rays in front of retina in myopia; **B.** Parallel rays being focused upon the retina after radial keratotomy

Fig. 7.8: Radial keratotomy—16 radial cuts

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Figs 7.10A to D: LASIK procedure. **A.** Lamellar corneal flap by microkeratome; **B.** Corneal flap being lifted; **C.** Excimer laser photoablation; **D.** Flap reposited. Cornea is thinner and flatter

Fig. 7.9: Myopic keratomileusis

  - ***Keratomileusis (Fig. 7.9):*** A lenticule is prepared from removed anterior part of the cornea of the patient and reshapened. This is also an obsolete procedure.
- ***Fukala's operation:*** Removal of clear crystalline lens if the myopia is –21D or near, and make the patient aphakic.
- ***Clear lens extraction (CLE):*** Extracapsular cataract extraction with posterior chamber intraocular lens (PCIOL) implantation may be a good choice in high myopia. Here, the lens may be clear without any cataractous change.

Minus phakic intraocular lens: A minus anterior chamber intraocular lens (ACIOL) or PCIOL implantation is done, even in presence of clear crystalline lens.

Among these, removal of clear crystalline lens with PCIOL implantation and phakic IOL implantation are becoming popular now-a-days throughout the world.

Intracorneal rings (ICR): Two rings are inserted intrastromal corneal ring segments (INTACS) in the paracentral area to flatten the central cornea. This is especially useful in keratoconus.

Laser

- ***Photorefractive keratoplasty:*** Here, the cornea is re-shapened by photoablation of superficial corneal tissue by excimer laser. Photorefractive keratoplasty (PRK) is now replaced by Laser-assisted stromal *in situ* keratomileusis (LASIK) procedure.
- ***LASIK***: Here, the superficial corneal flap is lifted first, and then central corneal stromal bed is ablated by excimer laser to correct myopia. It is the most popular method now-a-days. But it is a very costly procedure (Figs 7.10A to D).

General Hygiene

It is important in case of children like, good nutritional diet, exercise, fresh environment, proper position for reading in good illumination, etc. ***But exercise of the extraocular muscles can not cure myopia, or other refractive errors***.

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Genetic Counselling

There need be no restraint on marriage and procreation among simple myopes.

- A parent with degenerative myopia should be warned that any offspring may have same disability.
- Two highly myopic adults with degenerative changes should never, from the medical point of view, have children.

ASTIGMATISM

It is the type of refractive error, in which the incident parallel rays do not come to a point focus upon the retina, due to refraction varies in different meridians of the eye.

Etiology

Curvature astigmatism

- ***Corneal***
  - Astigmatism 'with the rule' (direct astigmatism)—theoretically, no eye is 'stigmatic' as the vertical meridian is more curved than horizontal by 0.25D owing to pressure of the upper lid over the globe.
  - Astigmatism 'against the rule' (indirect astigmatism) when the horizontal curvature is greater than vertical.
    - It may be seen as normal variation.
    - Post-operative following cataract surgery or trabeculectomy.
- ***Lenticular:*** As in lenticonus.

Diseases of the cornea

- Pterygium
- Keratoconus
- Marginal degeneration
- Corneal scar.

Index astigmatism: In early cataract, due to inequalities of refractive index in different sectors (cause of polyopia).

Decentration of the lens

- Subluxation of the crystalline lens
- Decentration or tilting of the IOL.

Types

Regular Astigmatism

The two principal meridians of greatest and least curvature are at right-angle to each other.

1. ***Oblique:*** The two meridians do not lie in the principal planes, but remain at right-angle to each other.
2. ***Bi-oblique:*** The axes are not right-angled but crossed obliquely.

It is of following types (Figs 7.11A to E)

- ***Simple:*** Where one of the principal meridian is emmetropic and the other is myopic or hypermetropic, ***(simple myopic or simple hypermetropic astigmatism)***.





Figs 7.11A to E: Classification of regular astigmatism (o: circle of least diffusion). **A.** Compound myopic astigmatism; **B.** Simple myopic astigmatism; **C.** Mixed astigmatism; **D.** Simple hypermetropic astigmatism; **E.** Compound hypermetropic astigmatism

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- ***Compound:*** Where both the principal meridians are either myopic or hypermetropic, ***(compound myopic or compound hypermetropic astigmatism)***.
- ***Mixed:*** Where one principal meridian is myopic, and the other one is hypermetropic. Visual acuity is relatively less impaired in mixed astigmatism.

Irregular Astigmatism

Here, the surface is so distorted that it admits no geometrical analysis, and it can not be corrected adequately with glasses.

- Scarring of the cornea
- Keratoconus
- Incipient cataract
- Lenticonus
- After penetrating keratoplasty.

Optics

A regularly astigmatic surface is said to have a ***toric curvature***.

- The more curved meridian will have more convergent power, than the less curved.
- If parallel rays fall upon such a surface, the vertical rays will come to a focus sooner than the horizontal.
- The rays after refraction will be symmetrical when referred to the vertical and horizontal planes, but they will have two foci.
  - The whole bundle of rays is called ***Strum's conoid***, and the distance between the two foci, is called ***focal interval of Strum***.
  - Refractive properties of such astigmatic surface are described in Figure 7.12.

The surface is having different curvatures in two meridians—the vertical meridians (VV) being more curved than horizontal (HH). The appearences of the bundle of rays at different points are illustrated in the Figure 7.12.

- ***Point A*** is a horizontal oval ellipse, since vertical rays are converging more rapidly than the horizontal.
  If the retina is placed at A: Compound hypermetropic astigmatism results.

Fig. 7.12: 'Strum's conoid'—refraction by an astigmatic lens

- ***Point B*** will be horizontal straight line, as the vertical rays have come to a focus, while the horizontal rays are still converging.
  If the retina is placed at B: Vertical meridian is emmetropic, whereas the horizontal meridian is still hypermetropic, resulting in simple hypermetropic astigmatism.
- ***Point C, D and E:*** The vertical meridian will be in the condition of a myopic, and the horizontal still in that of a hypermetropic eye—this is mixed astigmatism.
- ***Point D:*** The two opposing tendencies are equal and the section becomes a circle—this is called ***the circle of least diffusion***.
- ***Point F:*** The vertical meridian is still myopic (divergent) and the horizontal rays have come to a focus (emmetropic)—the section will be a vertical line. This is simple myopic astigmatism.
- ***Point G:*** Section is verically oval, since both the rays are diverging.
  If the retina is placed at G: Both meridians are myopic, and it is compound myopic astigmatism.

Importance of circle of least diffusion: Distant vision is often found to be surprisingly good with relatively high degree of mixed astigmatism. This is because, the circle of least diffusion falls upon or near the retina, and there is least amount of distortion of the images at this point.

Symptoms

- Decrease in visual acuity.
- ***Asthenopia or eye strain:*** It is often worse in low degree of astigmatism than in

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higher, because of endeavours to accommodate so as to produce a circle of least diffusion upon the retina. This is more in hypermetropic astigmatism.
- Eyeache and headache
- ***Running together*** of the letters while reading, and the eyes quickly become fatigued.

Signs

- Head tilt in children, especially in oblique astigmatism.
- Half-closure of the lids (as in myope) to achieve greater clarity of stenopeic vision.
- Signs of causative factor (e.g. scarring of the cornea, decentration of the lens, etc.).
- Ophthalmoscopically, the disc appears oval or tilted in high degree of astigmatism.

Investigations

- ***Retinoscopy:*** For determination of its power and axis.
- ***Keratomerty:*** To measure the corneal curvatures. It is useful to measure the power and axis of corneal astigmatism, and to find out irregular corneal surfaces.
- ***Jackson's cross cylinder (Fig. 7.13):*** The most convenient form is a combination of a –0.25D sphere with + 0.50D cylinder.
  It is to check the power and axis of the cylinder in the optical correction.
- ***Astigmatic fan (Fig. 7.14):*** It is also to measure the amount and axis of the astigmatism.
  The cylinder which renders the outline of the whole fan equally clear, is a measure of astigmatism. And the axis of the cylinder is at right angle to the line, which was initially most clearly defined.
- ***Astigmatic dial:*** It measures the degree of astigmatism in reation to the cornea (Fig. 7.15).
- ***Placido's disc:*** To find out irregular corneal surfaces.
- ***Photokeratoscope:*** Illuminated placido's disc with photographic facility. This is for permanent record.
- ***Computerized corneal topography.***

Fig. 7.13: Jackson's cross cylinder

Fig. 7.14: (Above) Astigmatic fan. (Below) As seen by a patient with astigmatism at a horizontal axis. The vertical lines appear most clear and the others are progressively less well-defined

Fig. 7.15: Degree of astigmatism in relation to the cornea

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Treatment

Regular Astigmatism

- ***Spectacles:*** Full cylindrical correction with perfect axis. It should be used both for the distant and near vision.
- ***Contact lenses:*** Rigid lenses are useful. Soft contact lens can correct only little astigmatism.
- ***Surgery***
  - Astigmatic correction by giving cuts in the direction of more curved or steep axis called limbal relaxing incision (LRI).
  - Removal of sutures—in astigmatism following cataract surgery or keratoplasty.
- ***Laser:*** Excimer laser (by LASIK or PRK) is used to re-shape the cornea in a particular meridian.

Irregular Astigmatism

Best treatment is by contact lens. Excimer laser may be helpful phototherapeutic keratectomy (PTK) for superficial corneal scar responsible for irregular astigmatism.

APHAKIA

Literally, 'aphakia' means absence of the crystalline lens from the eyeball. ***Optically,*** it means absence of the crystalline lens from its normal anatomical positivon in the pupillary area (patellar fossa).

Etiology

- ***Congenital:*** Rare
  - ***Primary:*** It is resulting from failure of the development of the lens in fetal life.
  - ***Secondary:*** The lens forms, but gets absorbed *in utero* due to some factors.
- ***Acquired***
  - ***Post-operative (most common):*** Following cataract surgery [needling, intracapsular cataract extraction (ICCE) or extracapsular cataract extraction (ECCE)].
  - ***Post-traumatic:*** Following blunt or penetrating injury of the eye. It includes subluxation or dislocation of the lens.
  - ***Post-inflammatory:*** Following large perforated corneal ulcer.
  - ***Couching:*** An ancient surgery (still practised by some quacks), where the lens is forcibly dislocated into the vitreous by a needle via limbus.

Optics

The optical condition of the aphakic eye is very simple. It consists of a curved surface, the cornea, separating two media of different refractive indices—air (1.00) and aqueous plus vitreous (1.33).

- As the radius of curvature of cornea is 8 mm, the:
  - Anterior focal distance—23 mm. (15 mm in the normal eye)
  - Posterior focal distance—31 mm. (24 mm in the normal eye)

  So parallel rays of light brought to a focus 31 mm behind the cornea (Fig. 7.16).
- The dioptric system must, therefore, be supplemented by a strong converging (convex) lens, and usually, if the eye were originally emmetropic, of about +10D.
- The nodal point of the eye is thus moved forward.
- If the aphakic eye is 31 mm long, the rays will focus upon the retina and no correcting lens would be required for the distant vision. The axial myopia of a phakic eye which is 31 mm long—equals to –21D.

Fig. 7.16: Hypermetropia in aphakia. Parallel rays are brought to a focus, 31 mm behind the cornea

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Optical Defects

- ***Acquired high hypermetropia:*** Crystalline lens contributes +15D to +20D in the normal eye.
- ***Astigmatism against the rule:*** Since, the cornea is flatter in vertical meridian, and it is due to contracture or fibrosis of the limbal scar. The amount of astigmatism is +1.0 to +3.0D initially, but gradually diminishes.
- ***Absence of accommodation.***
- ***Change in color vision:*** Due to increased entry of infrared or ultraviolet rays into the eye, which are normally absorbed by the crystalline lens.

Clinical Features

- ***History***
  - Blurred vision for distance and near.
  - History of cataract operation in most cases.
- ***Signs:*** Following a standard cataract surgery.
  - ***Vision*** (unaided)—only finger counting at 2–3 ft. Patient may have thick convex glasses, or if recently operated, using dark glasses.
  - ***Sutures*** may be visible at the upper limbus.
  - ***Linear scar*** at the upper limbus.
  - ***Anterior chamber:*** Deep (as the iris recedes back in absence of the lens).
  - ***Iris:*** (1) Iridodonesis or tremulousness of iris due to loss of support from the lens. (2) Peripheral button-hole iridectomy (PI) mark (Fig. 7.17).
  - ***Pupil:*** Jet black reflex (due to loss of reflection of light rays from the anterior surface of the lens).
  - Absence of 3rd and 4th Purkinje images (in ICCE) and only absence of 3rd image (in ECCE).
  - ***Retinoscopy:*** Reveals high hypermetropia and astigmatism.
  - ***Ophthalmoscopy:*** As in hypermetropic fundus with a small optic disc.

Fig. 7.17: Very good aphakia with peripheral button-hole iridectomy (PI) mark

Fig. 7.18: Correction of aphakia
a—Spectacle; **b**—Contact lens; **c**—Intraocular lens

In ***congenital aphakia*** or in ***dislocation of lens*** the clinical pictures are different, as there is:

- No history of operation
- No limbal scar mark
- No astigmatism
- Poor vision even with glasses because of amblyopia or other complications.

Treatment (Fig. 7.18)

Spectacle Correction

If the eye is previously emmetropic the power will be approximately:
Distant vision: +10.0 DSph with +2.0 DCyl x 180°
Near vision: +13.0 DSph with + 2.0 DCyl x 180°

Two separate glasses are given for distant and near vision. Bifocal glasses are usually troublesome, as most of the patients can not tolerate them.

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Fig. 7.19: Image magnification

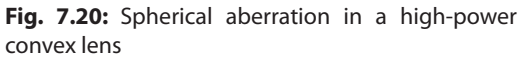


Fig. 7.20: Spherical aberration in a high-power convex lens

A +3.0 DSph addition is given for near vision, as there is loss of accommodation due to absence of lens, and the comfortable reading distance is 33 cm (i.e. 100/33 = +3.0D).

Glasses are prescribed usually 4 to 6 weeks after the surgery, as by this time wound healing is nearly completed.

Disadvantages of aphakic glasses

- ***Image magnification*** (Fig. 7.19) of about 25–30%
- ***Spherical aberration (Fig. 7.20):*** Producing 'pin-cusion distortion.' Patient finds himself in a 'parabolic world.'
- ***Lack of physical co-ordination***, particularly for finer movements.
- ***Roving-ring scotoma (Fig. 7.21)*** or ***Jack-in-the-box*** phenomenon—due to prismatic aberration at the edge of the thick lens.
  The scotoma extends from 50–65° from central fixation. It is not fixed, and its movements are initiated by the movements of the eyeball.
- Restriction of the visual field, and ***poor eccentric visual acuity***.
- In monocular cases—there will be ***high aniseikonia***, resulting in binocular diplopia. So, the normal eye must be blocked by a frosted or a balanced (+10.0D) glass.
- ***Colored vision***—due to absence of natural filter of the crystalline lens, and due to chromatic aberration.
- Inaccurate spectacle correction because of errorneous vertex distance.
- The glasses are very thick, heavy, and cumbersome.

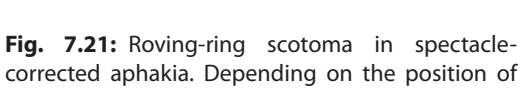


Fig. 7.21: Roving-ring scotoma in spectacle-corrected aphakia. Depending on the position of the eye, objects tend to appear and disappear

- Cosmetically embarrasing—as the eyes appear larger behind the glasses.

Contact Lens

Advantages

- Image magnification is 7–8%, and this magnification is usually tolerated by the patient.
- All the aberrations are less, so there is (1) increase in the visual field, (2) improvement of physical coordination, and (3) better eccentric visual acuity.
- In monocular cases, diplopia is usually absent with retention of binocularity.

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- ***In traumatic cases***, contact lens is the only mean to correct aphakia, where IOL implantation may not be possible.
- Cosmetically, it is well accepted.

Disadvantages

- Lack of dexterity in old patients.
- Foreign body sensation.
- Lens spoilage (due to loss, breakage, dislodgement, etc.) leading to high recurrent expenditure.
- Corneal complications—like erosions, ulceration, edema, vascularization, etc. may be troublesome.
- Light spectacles are required for reading, and/or for distant vision.

Secondary Intraocular Lens Implantation

Advantages

- Image magnification is only 0–1%.
- No spherical and prismatic aberrations.
- Minimum or no aniseikonia, with rapid return of binocularity.
- Normal peripheral field of vision and eccentric vision.
- Good hand-eye co-ordination and spatial sensation.
- Freedom from handlng of the optical devices (e.g. contact lens or heavy spectacles).
- Cosmetically, it is best accepted.

Disadvantages

- Risks and complications may be more, e.g. corneal decompensation, lens displacement, chronic iridocyclitis, posterior capsular opacification, etc.
- It needs specially trained surgeons and sophisticated instruments, like operative microscope.
- Initially, the cost is more.

An IOL implantation may be done in an aphakic eye after careful examination. This may be:

- ***Secondary anterior chamber IOL*** in aphakia following ICCE.
- ***Secondary posterior chamber IOL*** in aphakia following ECCE.
- ***Secondary scleral fixation IOL (SFIOL)*** in aphakia following ICCE.

The best optical correction is, of course, a primary PCIOL implantation, which is the ***most current concept in the surgical management of cataract***. Intraocular lens details are discussed in Chapter 25.

ANISOMETROPIA

This is a condition in which the refractions of the two eyes are unequal.

It is extremely common in small amount, and may be found in every possible variety.

Etiology

- Usually congenital
- Unequal rates of change in refraction between the two eyes.
- Corneal diseases and cataract.
- Following surgical and nonsurgical trauma.

Vision

- ***Binocular vision:*** It is the rule with smaller degrees of defect.
  Each 0.25D difference between the two eyes causes 0.5% difference in size between the two retinal images.
  A difference of 5% of the image size can be tolerated well (i.e. 2.50D difference of refraction between two eyes). If it is more, the effort of fusion may give rise the symptom of eye strain or diplopia.
- ***Alternating vision:*** Each of the two eyes is used one at a time, e.g. one eye is emmetropic or mild hypermetropic, but the other eye is myopic.
- ***Exclusively uniocular:*** If the defect in one eye is high with poor visual acuity, the other and better eye will then be used exclusively. In this case, the worse eye will soon become amblyopic.

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Problems with Anisometropia

- ***Imperfect binocular vision—*** an effort to fuse the images leads to symptom of eye strain.
- ***Amblyopia (anisometropic)***, specially in children.
- ***Development of squint***—convergent squint in children and divergent in adult.
- ***Diplopia***—due to unequal image sizes.

Treatment

- ***Contact lenses***—are most suitable.
- ***Iseikonic lenses***—correct the difference by their optical construction, but their clinical results are often disappointing.
- If the patient is amblyopic—treatment for amblyopia.
- Excimer laser PRK or LASIK.

Monovision

It is used in patients needing correction for both reading and distance. It is the practice of prescribing a corrective prescription for one eye for distant vision and the other eye for near vision.

The idea is that a person will always be able to see both near and far.

Unfortunately, acuity is only one aspect of vision. The eyes need to work together to produce a 3-dimensional image in the brain. On using this type of correction, the person disrupts this ability which interferes with depth perception.

Since one eye is corrected for near and one for far, one eye will always be in visual somatic dysfunction, causing very large and noticeable visual strains. These strains will eventually manifest as other symptoms.

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8 CHAPTER

Accommodation and its Disturbances

ACCOMMODATION (FIG. 8.1)

It is the ability to see the near object clearly, by increasing the converging power of the eye. This is by increasing the refractivity of the lens by increasing the curvature of its anterior surface.

At rest, the radius of curvature of the anterior surface of the lens is 10 mm and that of the posterior surface is 6 mm.

In accommodation, the curvature of the posterior surface remains almost the same, but the anterior surface changes, so that in strong accommodation, its radius of curvature becomes 6 mm. The refraction of the eye in this condition is called its ***dynamic refraction***.

Mechanism of Accommodation

In unaccommodating state of the eye, the suspensory ligament is in tension so the lens remains flat (Fig. 8.2).

In accommodation: There is contraction of the ciliary muscles → reduction of the circle formed by the ciliary processes → suspensory ligament relaxes → elastic capsule of the lens acts unrestrainedly to deform the lens substance → lens then alters its shape to become more spherical (perhaps conoidal).

This conoidal shape assumes by the lens, may be due to configuration of the capsule, which is thicker behind the iris than in the center.

The shape of the lens is thus the result of a balance between its own elasticity and that of its capsule ***(modern version of von Helmholtz theory)***.

Accommodation Pathway

It initiates mainly by the fibers from the medial rectus muscle, which contracts on convergence.

Fig. 8.1: Accommodation in an emmetropic eye parallel rays being focused upon the retina at R. When a near object N, is looked at, focus behind the retina at N^1. In order to bring this upon at R (N^1), the lens increases its convexity

Fig. 8.2: Mechanism of accommodation

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Medial rectus muscle → via third nerve → mesencephalic nucleus of fifth nerve → a presumptive center for convergence at pons → Edinger-Westphal nucleus → along the third nerve → accessory ciliary ganglion → sphincter pupillae muscle of the iris.

Range of Accommodation

- Far point ***(punctum remotum)*** is the farthest point at which object can be focused on the retina with ciliary muscle relaxed, and it varies with the emmetropia, myopia or hypermetropia.
- Near point ***(punctum proximum)*** is the nearest point at which small objects can be seen clearly after full accommodation.

Range of accommodation is the difference between these two distances.

Amplitude of accommodation (Fig. 8.3) is the difference in refractive power of the eye between these two points. It gradually diminishes throughout the life.

Physical and Physiological Accommodation

Two factors are responsible for the act of accommodation:

1. The ability of the lens to alter its shape ***(physical accommodation)*** and it is measured in diopter.
2. The power of the ciliary muscle to contract ***(physiological accommodation)***.

The physiological accommodation is the cause, and the physical accommodation is the effect.

There are two other phenomena which occur with accommodation—(i) pupils become smaller and (ii) convergence of the eyes.

Age changes: As the age advances, the amplitude of accommodation progressively decreases—from 14D at the age of 10, to 4D at 45 years, and by 60 years it is only 1D years (near point then being one meter away). This is due to a progressive sclerosis of the lens so that it becomes less malleable, tending to set in unaccommodative shape.

Presbyopia

It means ***the eye sight of old age***. This is a physiological aging process, in which the near point gradually recedes beyond the normal reading or working distance.

Causes

Diminution of accommodative power with age, and the factors are:

- Lens matrix is harder and less easily moulded.
- Lens capsule is less elastic.
- Progressive increase in size of the lens.
- Weakening of the ciliary muscle.

Symptoms

- Gradual difficulty in reading small prints, particularly in the evening or in dim illumination.
- Inability to perform near work meticulously, e.g. sewing, threading a needle, etc.
- Fatigue or headache while doing near work.
- ***Arms are not long enough*** is a common experience.

Fig. 8.3: Amplitude of accommodation with age

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Treatment

To provide the patient with appropriate convex lens so that his accommodation is reinforced, and his near point is brought within an useful and workable distance.

Methods of Prescription

- Knowledge of patient's ***static refraction*** (for distance).
- To know the distance at which the patient is accustomed to carry out his close work.
- Subjective test of near vision for each eye with addition of appropriate convex lenses to the distant correction.
- It is customary to start with an addition of +0.75 or +1.00 DSph.
- It is always better to under-correct than to over-correct. Since, if the lenses tend to be strong, the normal relation of accommodation and convergence will be disturbed.

Benjamin Franklin first invented the bifocal lens for his own presbyopia in 1783.

The glasses may be :

- ***Unifocal or monofocal lenses:*** It is in the form of rim (half-eye spectacles).
- ***Bifocals lenses (Fig. 8.4)***
  - ***Kryptok bifocals (Fig. 8.4A):*** These are crescent-shaped lenses for near vision. In kryptok bifocal, the near-field becomes smaller.
  - ***Executive bifocals (Fig. 8.4B):*** It has wider field, but cosmetically does not look nice and heavier.

Usual rule (*though not always fixed*)

Age	Near addition
40 years	+ 1.0 DSph
45 years	+ 1.5 DSph
50 years	+ 2.0 DSph
55 years	+ 2.5 DSph
60 years	+ 3.0 DSph
Above 60 years	+ 3.0 to + 3.5 DSph





Fig. 8.4: Bifocal lenses. **A.** Kryptok; **B.** Executive **DVC**—Distant visual center; **NVC**—Near visual center

- ***Trifocal*** or multifocal (omnifocal) lenses may also be given.
- ***Progressive multifocal lenses (Fig. 8.5) (no-line bifocals)*** correct vision for all distances—far, intermediate and near.

Unlike bifocal lenses that have one clearly visible segment for near vision and one segment for distant vision; progressive lenses offer a continuous, gradual change in prescription strength from the lower ***(reading correction)*** to the upper ***(distance correction)*** portion of the spectacles lens.

- ***Prisms:*** Sometimes patient may experience a difficulty in convergence (as all of his accommodative efforts have now been neutralised), then a prismatic addition is helpful.
- ***Surgery:*** Various surgical methods have been proposed to treat presbyopia. These include—monovision LASIK, conductive

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Fig. 8.5: Schematic representation of progressive lens demonstrating different zones of focus

keratoplasty (CK), presbyopic LASIK, and more recently, the corneal inlay. Beside these, presbyopic clear lens extraction with multifocal or accommodative intraocular lense (IOL) implantation. Anterior ciliary sclerotomy is another procedure proposed earlier.

Insufficiency of Accommodation

In this condition, the accommodative power is below the lower limit of normal for the patient's age.

- ***The causes are as follows:***
  - Early presbyopia.
  - Weakness of the ciliary muscle due to general debility, anemia or toxemia.
  - Open-angle glaucoma due to impairment of the effectivity of the ciliary muscle by the increased intraocular pressure.
- ***Symptoms***
  - Eye strain
  - Difficulty with near work.
- ***Treatment***
  - Treatment of the cause
  - Reading spectacles (near addition)
  - Accommodation exercises.

Paralysis of Accommodation

- ***Causes***
  - ***Unilateral***
    - Cycloplegics (e.g. atropine)
    - Contusion of the eye
    - Paralysis of the third cranial nerve.
  - ***Bilateral:*** (Paresis is more common)
    - Diphtheria
    - Syphilis
    - Diabetes
    - Alcoholism.

Mydriasis usually accompanies the paralysis of accommodation. The prognosis is good in cases due to drugs or diphtheria. In trauma the condition may be permanent.

- ***Treatment***
  - Treatment of the cause
  - If the paralysis is permanent—suitable convex glasses may be prescribed
  - Miotics are seldom useful.

Spasm of Accommodation

- ***Causes***
  - Found mainly in children, who attempt to compensate his refractive error.
  - Myopes are more affected than hypermetropes.
  - May occur artificially by instillation of miotics (e.g. with pilocarpine in young glaucoma patients).
  - Neurotic individuals who converge excessively.
- ***Symptoms***
  - Asthenopia
  - Blurring for distant vision (due to variable degrees of artificial myopia).
- ***Treatment***
  - Atropinization for a few days or weeks.
  - Assurance; if necessary, psychotherapy.

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9 CHAPTER

Estimation and Correction of Refractive Errors

In estimating the refraction of the eye the best routine is, first to estimate the condition ***objectively***, and then to verify and adjust this finding by ***subjective tests***.

Objective methods are:

- Retinoscopy (skiascopy or shadow test)
- Refractometry
- Keratometry.

OBJECTIVE METHODS

RETINOSCOPY

It is the most practical method of estimating the condition of refraction objectively, ***with accommodation at rest***.

Optics: It entails a study of the movements of the retinal image produced by a beam of light that sweeps across the pupil.

The observer then watches this illuminated retinal image by looking down the path of incident light, through a hole in the center of a mirror (retinoscope) (Figs 9.1A and B).

- ***If the eye is emmetropic:*** Parallel rays of light come to a point focus on retina, so they are emerging in the same pathway.
- ***If the eye is hypermetropic:*** Parallel rays converge behind the retina and hence, the emerging rays are divergent.

The principle of retinoscopy is to make every observing eye emmetropic, so that the emerging rays should form a parallel beam.

A

B

Figs 9.1A and B: Retinoscopic mirror. **A.** Double mirror; **B.** Single plane mirror

Procedure

Cycloplegia

It is required in children and young patients. In adults and old patients, it is usually not necessary.

- ***If the patient is less than 5 years:*** Atropine eye ointment (1%) is to be applied three times daily for 3 days.
- ***If the patient is between 5 and 15 years:*** 1% cyclopentolate, or 2% homatropine eye drop is instilled for 3 times, about 1 hour before examination.
- ***If the patient is between 15 and 20 years:*** The same procedure may be undertaken.

The refraction under cycloplegia is always pathological because the shape of the lens has been altered. A ***post-cycloplegic test (PCT)*** is therefore advisable.

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Dark-room Test

Retinoscopy should preferably be conducted in a dark room.

- Examiner sits at 1 meter away from the patient (point of reversal will then be 1.5D). It is even more convenient to sit at arm's length i.e. two-third meter away, so that the trial lenses can be held in the other hand while the light beam is passed. The point of reversal is then be 1.5D.
- The patient is normally seated and looking toward the far end of the room (relaxed eye).
- Source of light is from behind the patient.
- The surgeon looks through a ***plane mirror*** with central perforation, and light is reflected into the patient's eye.
- The mirror is slowly moved from side to side in different meridians, and movement of the shadow is noted.
- In hypermetropia, emmetropia and myopia <1.0D = the reflex moves in the same direction, in myopia of 1.0D = there is no shadow, in myopia of >1D = the shadow moves in the opposite direction.
- Increasing convex (if the movement is on the same side) or concave (if on the opposite side) lenses are placed before the eye until the ***point of reversal*** is reached. ***At this point there will be no movement of the shadow, and pupil will be brightly illuminated.***
- The procedure is done for each meridians separately.
  - In simple spherical refractive error—the movement and the point of reversal will be same in both meridians.
  - In astigmatism, they are different. If the axes are oblique, the shadow themselves will seem to move obliquely and the mirror is then tilted accordingly.

Calculation

Refraction of patient's eye = lens required to reach end point = –1D (myopia).

Since the surgeon is sitting at 1 meter distance, and if he is at two-third meter, it will be –1.5D (myopic).

So the refraction of the eye = –1.0D + lens.

Examples

- If the end point is with + 4.0D lens: ***Refraction*** = –1.0D + 4.0D = +3.0D.
- Similarly with –4.0D lens: ***Refraction*** = –1.0D – 4.0D = –5.0D
- If the end point is with + 1.0D lens: ***Refraction*** = –1.0D + 1.0D = 0, i.e. the patient is emmetropic.
- In case of astigmatism, each meridian is to be calculated separately.

Streak retinoscopy (Figs 9.2A and B): Instead of circular light as obtained by a plain mirror, a self-illuminated streak of light is used. Here, the appearances of the shadow are more dramatic. Axis of the astigmatism is easily determined. It has certain other advantages:

- It can be done in any position of the patient.
- It can be done in difficult patients, e.g. in children or non cooperative patient or for the patient under general anesthesia.
- It can be used peroperatively.

REFRACTOMETRY (OBJECTIVE OPTOMETRY)

It is a method to determine the degree of ametropia by special instrument called ***refractometer***.

In it, a clear retinal image of a test object is formed by an optical system, and the degree of adjustment required, gives a measurement of the ametropia.

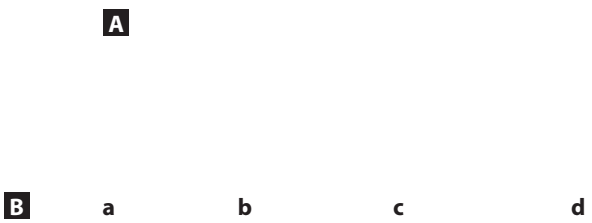
Recently, electronic refractometry (auto-refractometer) (Fig. 9.3) is popularly used in clinical practice. Apart from regular use, this instrument is excellent for quick screening of refractive errors in a given population.

KERATOMETRY (FIG. 9.4)

It measures the astigmatism of the anterior surface of the cornea at two points about 1.25 mm on either side of the center.

Since lenticular astigmatism may coexist, ***this technique is not reliable except in***

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Figs 9.2A and B: **A.** Streak retinoscope; **B.** Reflexes taken by streak retinoscope at different point (**a.** Reflex parallel to retinoscopic streak; **b.** Reflex and streak in a "with" movement in hypermetropia; **c.** Reflex and streak in an against movement in myopia; **d.** Reflex at the point of neutralization)

Fig. 9.3: Auto refractometer

aphakia. It is useful in measuring the curv ture of the cornea:

- In contact lens practice.
- For diagnosis of certain corneal conditions, e.g. keratoconus, pellucid marginal degeneration, etc.
- Before cataract operation for measuring intraocular lens (IOL) power (biometry) and for planning of incision in cataract surgery to reduce astigmatism.

Fig. 9.4: Keratometer

SUBJECTIVE METHODS

SUBJECTIVE VERIFICATION OF REFRACTION

After objective test, it should always be verified subjectively by testing the visual acuity. If a cycloplegic has been used, the process should be repeated in a ***post-cycloplegic test***.

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Procedures

- Each eye is to be tested separately, the other eye being blocked, and then finally tested together.
- Appropriate lenses as found by objective test are inserted in the trial frame.
- Slight modification of the inserted lens gives a maximum visual acuity.
- Verification may be needed with a ***cross cylinder***, or ***astigmatic fan*** in case of astigmatism.
- Sometimes, ***fogging method*** is necessary to induce a relaxation of accommodation especially in hypermetropia.

Here, the eyes are made artificially myopic by addition of convex lenses (e.g. + 4.0D). This is then gradually lessened by a small fraction (0.5D) until the maximum acuity is just reached. The first lens is not removed until the next is in position, to prevent from accommodation becoming active.

As a rule, the patient is given the strongest hypermetropic, or weakest myopic correction with normal visual acuity.

- The addition of the correction for near work (if necessary), and testing of the acuity with near-types, uniocularly and then binocularly.
- Then the spectacles are ordered with necessary comments (e.g. for constant wear, or near works only, etc).

EXAMINATION OF THE FUNDUS

Adequate ophthalmoscopic examination of the ocular fundus requires dilation of the pupil. ***Caution is required in dilating the pupils of patients with shallow anterior chambers to avoid precipitation of an acute attack of angle closure glaucoma***. The methods of fundus examination should be as follows:

- ***With a retinoscopic plain mirror from 1 meter distance:*** This is to know the fundal glow and refractive status.
- ***With an ophthalmoscope or a plain mirror at a distance of 22 cm (distant direct ophthalmoscopy):*** This is—
  - To know the opacities in the media.
  - To discover the edge of a subluxated or dislocated lens.
  - To recognize a retinal detachment or a tumor.
  - To confirm the results found by external examination.
- ***By direct ophthalmoscopy:*** The details of central retina upto the equator is examined uniocularly. The image is erect and virtual. The magnification is about 15 times.
- ***By indirect ophthalmoscopy:*** The details of the retina upto the periphery (ora serrata) is examined binocularly.
- ***By slit lamp with special lenses*** (by eliminating refractive influence of the corneal curvature): (i) Hruby lens (–58.6D), (ii) fundus contact lens, (iii) Goldmann three-mirror contact lens and (iv) +78D or +90D lens.

Fundus examination includes (Fig. 9.5):

- ***Fundal glow:*** Good, poor or absent.
- ***Optic disc:*** Margin, color, shape, cup/disc ratio, neural rim, venous pulsation and any abnormal vessels.
- ***Retinal vessels:*** Vascular reflexes, arteriovenous (A/V) crossing, any abnormal or new vessels.
- ***General fundus:*** Any abnormality, e.g. exudates, hemorrhage, pigment patch, tumor, new vessels, etc.
- ***Macula and foveal reflex:*** The fovea centralis is situated about 2 dd temporal to the

Fig. 9.5: Normal fundus

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optic disc (dd = disc diameter; 1 dd = 1.5 mm). Any abnormality, e.g. cyst, hole, scar, edema, hemorrhage, etc. should be looked for.

- ***Choroidal blood vessels:*** Normally they are not visible, but in case of sclerosis they appear as ribbons.

METHODS OF OPHTHALMOSCOPY

There are two forms of ophthalmoscopy—direct and indirect.

Direct Ophthalmoscopy (Figs 9.6A and B)

The hand-held direct ophthalmoscope is designed to provide a direct magnified (X 15) view of the fundus. The source of illumination is projected by means of a mirror or prism coinciding with the observer's line of vision through the aperture.

Procedure

- The ophthalmoscope is held close to the observer's eye and approximately 15 cm from the patient's eye.
- It is held in the right hand and observer uses his/her right eye to examine patient's right eye. Similarly to examine the patient's left eye, it is held in the left hand and the observer uses his/her left eye.
- The patient should fix on a distant target with the eye as steady as possible.
- The observer has to adjust ophthalmoscope power setting to accommodate for the patient's refractive error and/or his/her own.
- If both patient and observer are emmetropic, the lens is set at 0. A red reflex will be seen and is considered normal.
- Moving the ophthalmoscope as close to the patient's eye as possible and using the 'plus' or 'minus' lens, the observer will able to see central retina in details upto the equator.
- Lens setting at more 'plus' power will focus the ophthalmoscope in the vitreous or more anterior.

Indirect Ophthalmoscopy (Figs 9.7A and B)

This technique is generally used by retina specialist and involves the use of a head mounted, prism directed light source coupled with use of a high condensing lens (+14, +20 or +30D) to see the retinal image. A +20D condensing lens is most commonly used.

The retinal image formed by the indirect ophthalmoscopy ***is inverted, real and capable of being seen at the focal plane of the lens***.

The image is brilliantly illuminated, binocular stereoscopic one that covers approxi-

A B

Figs 9.6A and B: A. Direct ophthalmoscope; **B.** Procedure of direct ophthalmoscopy

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Figs 9.7A and B: A. Indirect ophthalmoscope with lens; **B.** Procedure of indirect ophthalmoscopy

mately 10 times of the area compare to direct ophthalmoscope. The image is, however, smaller (X 3 times). It is essential to see retinal periphery to locate different types of lesions. Because of its stronger illumination, it allows light to pass through moderate opacities in the media which are obstructive to direct ophthalmoscope.

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10 CHAPTER

History-taking and Examination of an Eye Case

HISTORY-TAKING

Before examining the patient's eye, a careful history of ocular complaints should be taken.

- Name, age, gender, address and occupation of the patient.
- Date of admission and/or date of examination.
- Chief presenting complaints of the patient, its duration and mode of presentation
  Common eye complaints are as follows:
  - Painless progressive dimness of vision
  - Pain } may be together as acute pain, redness and watering
  - Redness }
  - Watering and discharge
  - Itchiness of the eyes
  - Foreign body sensation
  - Sudden loss of vision
  - Dimness of vision at night
  - Stickiness of the eyelids
  - Small swelling on the eyelids
  - Abnormal fleshly mass in the eye
  - Disfigurement of the eyeball
  - White opacity over the black of the eye
  - Smallness of the eyeball
  - Abnormal deviation of the eyeball
  - Headache
  - History of any injury.
- ***Past history***
  - Acute pain, redness and watering in the same eye or other eye
  - Ocular trauma
  - Wearing of spectacle
  - Acute infectious fever with rash
  - Similar problem in the other eye.
- ***Operative history***
  - Any operation on the same eye or opposite eye with date of operation
  - Duration of hospital stay at that time
  - Any known operative complication.
- ***Medical history***
  - Diabetes, hypertension
  - Bronchial asthma
  - Tuberculosis
  - Tobacco, alcohol
  - Acidity, heart burn
  - Drug history including drug allergy
  - Straining factors (e.g. chronic cough, enlarged prostate or bleeding piles, etc.).

EXAMINATION OF AN EYE CASE

- ***General examination:*** Pallor, jaundice, pulse, respiration, blood pressure (BP), any obvious abnormalities (e.g. malnutrition in case of Bitot's spot or keratomalacia; Rickets in case of congenital cataract, pock marks on the face in leucoma, etc.).
  Heart, lungs, central nervous system (CNS) gastrointestinal track (GIT):
- ***Ocular examination:*** Examination of the anterior segment of the eye (upto the lens-iris diaphragm) can be done by three methods:

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1. Diffuse illumination with a good torch light
2. Focal/oblique illumination using a pencil-torch and loupe (uniocular or binocular)
3. Focal/oblique illumination with the help of a slit lamp biomicroscope.

METHOD OF EXAMINATION BY TORCH AND UNIOCULAR LOUPE

Patient is placed in a dark room and examined with a pencil torch light focussed about 2 feet away from the patient's side.

The surgeon holds the loupe by thumb and index finger while lifting the upper lid by middle finger and two other fingers rest on eyebrow. Surgeon moves very close to the loupe to see the enlarged image of the cornea, iris or lens in details.

It is useful to search for ***superficial punctate keratitis (SPK), keratic precipitates (KPs), minute foreign body, caterpillar hairs,*** etc.

Uniocular Loupe

Its power is +40.0D) (Fig. 10.1).

- ***Advantages:***
  - 10 times magnified image
  - Small and handy instrument.

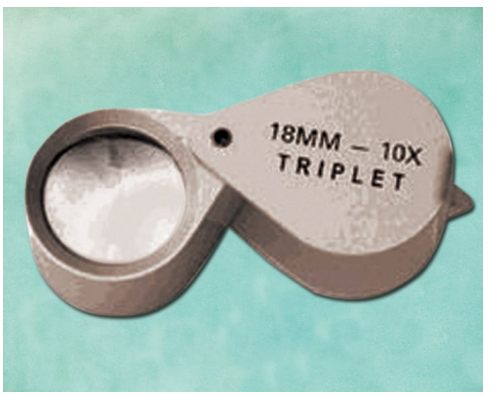


Fig. 10.1 Uniocular loupe



Fig. 10.2: Binocular loupe

- ***Disadvantages:***
  - Both hands are engaged during examination
  - Inconvenience due to closeness to the patient
  - No depth perception.

Binocular Loupe (Fig. 10.2)

It is kept fixed before the surgeon's eyes by an elastic band or a belt at forehead.

- ***Advantages***
  - Both hands remain free during ocular examination
  - Depth of lesion is better judged
  - Convenient, as surgeon is away from the patient.
- ***Disadvantages***
  - Low magnification (3 to 4 times)
  - Interpupillary distance may be difficult to adjust.

EXAMINATION WITH A SLIT LAMP

It is the best method as bright illumination and various grades of magnification are used.

A slit lamp biomicroscope has three parts (Fig. 10.3):

1. Illumination system,
2. Viewing system, and
3. Mechanical devices to adjust the slit lamp.

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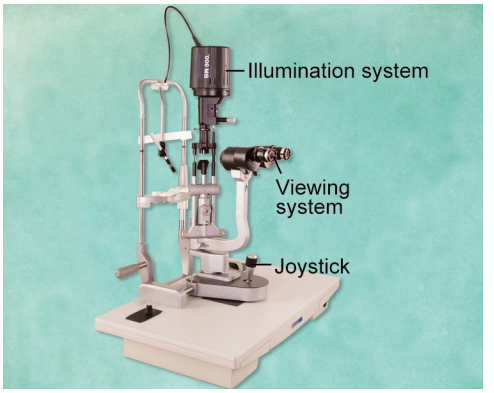


Fig. 10.3: Slit lamp biomicroscope



Fig. 10.4: Slit-lamp examination

It is binocular, so depth perception is accurate. In fact, one can cut a cross section (optical section) of the anterior segment of the living eye for detailed microscopic examination (Fig. 10.4).

Uses

- Detailed microscopic examination of the anterior segment of the eye layer by layer.
- For fundus examination by Hruby (spell as Ruby) lens (–58.6D) or +90D lens.
- For examination of angle of the anterior chamber by gonioscope.
- To measure intraocular pressure (IOP) by applanation tonometer.
- For fluorescein staining with blue filter.
- For anterior segment photography.
- As a delivery system for argon and YAG (yttrium aluminium garnet) laser.

Slit Lamp Examination of the Anterior Segment (Fig. 10.4)

Slit lamp offers a variety of illuminating and observing methods:

- Diffuse illumination
- Direct (focal) illumination
  - Broad beam (parallelepiped)
  - Narrow beam (optical section)
- Indirect illumination
- Retroillumination
  - Direct
  - Indirect
- Specular reflection
- Sclerotic scatter
- Oscillatory illumination.

Direct Diffuse Illumination (Figs 10.5A to C)

It is a good method of observing the eye and adnexa in general. Diffusers are generally ground glass plates that cover the light source. The slit should be opened wide and the magnification should be set as low as possible to enable a large field of view.

Direct Focal Illumination

This is the most common method of viewing all tissues of the anterior eye, the focused slit is viewed directly by the observer through the microscope. The magnification can be increased quite markedly (10x to 40x or more) to view any areas of interest in greater detail.

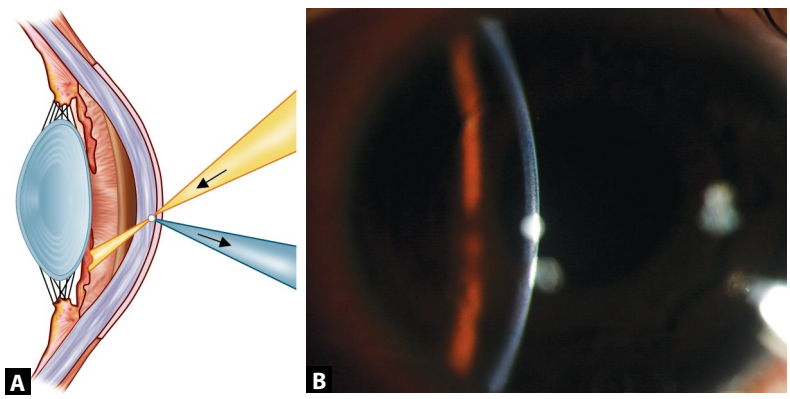
Generally a very wide beam is used for surface study, whilst a very narrow one is used for sections.

- ***Narrow beam (optical section) (Figs 10.6A and B):*** Once an abnormality has been found it is easier to determine the precise depth using an optical section. Generally the angle between the illuminating and observation systems should be set around 45–60°.

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A B C

Figs 10.5A to C: Direct diffuse illumination. **A.** Schematic representation; **B and C.** Photograph



Figs 10.6A and B: Direct focal (narrow beam). **A.** Schematic representation; **B.** Photograph

A good corneal section will allow at least four layers to be seen—tears (outer), epithelium (and Bowman's membrane), stroma seen as the central grey granular area and the fainter back line which is the endothelium (and Descemet's membrane).

- ***Broad beam (parallelepiped) (Figs 10.7A and B):*** A useful combination of the two is the parallelepiped section of the cornea, which uses a 2 mm slit width enabling corneal surface as well as stroma to be studied. This allows us to ascertain the depth of any interesting feature, e.g. foreign body, corneal abrasion. Direct illumination on the front surface of the crystalline lens reveals the ***orange peel effect*** and on the iris allows observation of iris pattern.

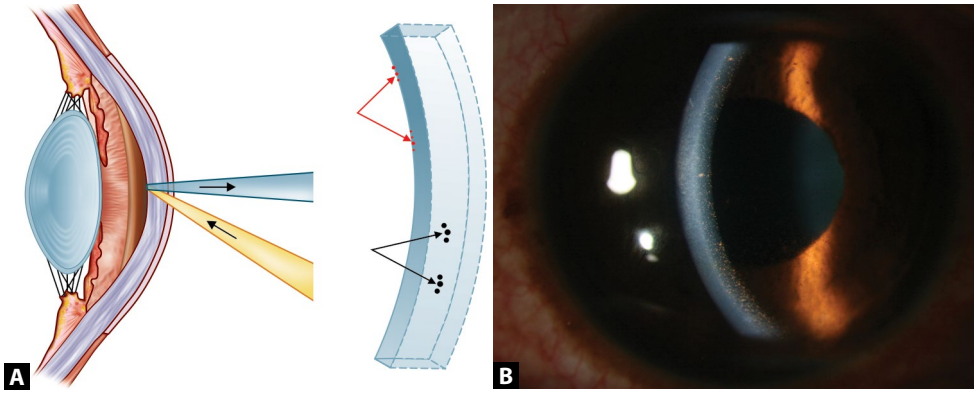
Indirect Illumination (Figs 10.8A and B)

Structures are often easier to see under indirect illumination as glare is reduced, e.g. opacities, corneal nerves and limbal vessels.

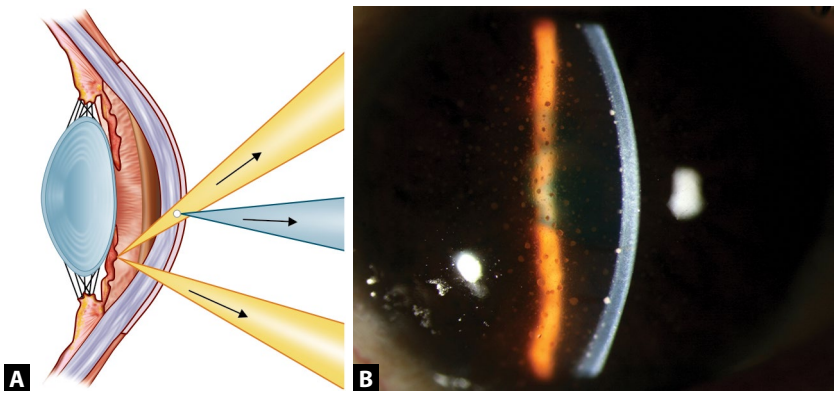
When using the slit lamp direct and indirect illumination are viewed simultaneously, structures viewed in the illuminated field are seen under direct illumination, but as this does not fill the whole of the field of view, anything which reflects or scatters light from outside the illuminated area is being viewed by indirect.

To view certain features by indirect illumination, first locate it by direct illumination and keeping the viewing system unchanged swing the lamp to one side.

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Figs 10.7A and B: Direct focal (broad beam illumination). **A.** Schematic representation; **B.** Photograph



Figs 10.8A and B: Indirect illumination. **A.** Schematic representation; **B.** Photograph

Retroillumination

The light is reflected off the deeper structures, such as the iris or retina, while the microscope is focused to study the cornea in the reflected light. Features that are opaque to light appear dark against a light background (e.g. scars, pigment, and vessels containing blood).

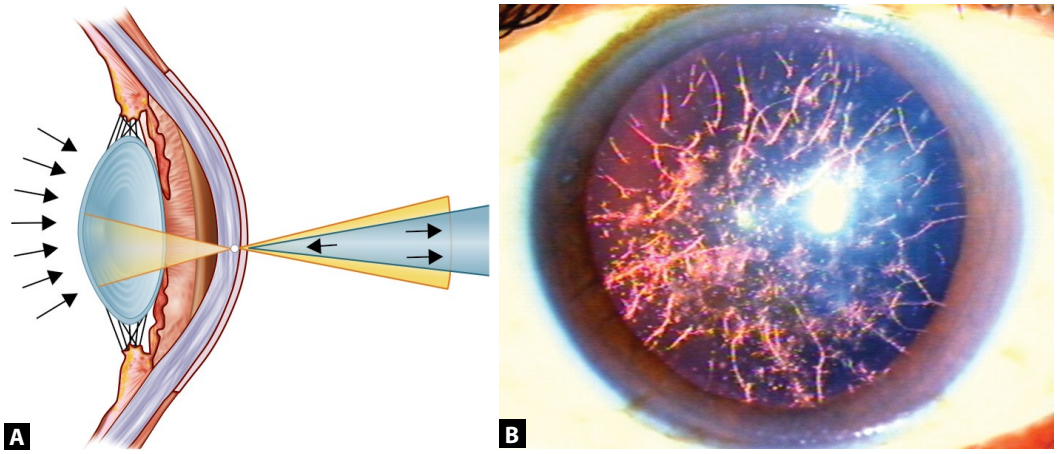
- ***Direct retroillumination (Figs 10.9A and B):*** The observed feature on the cornea is viewed in the direct pathway of reflected light. The angle between the microscope and the illuminating arm is about 60°.
- ***Indirect retroillumination (Figs 10.10A and B):*** The angle between the microscope and slit lamp arms is greatly reduced or increased so that the feature on the cornea is viewed against a dark background.

Specular Reflection (Figs 10.11A to C)

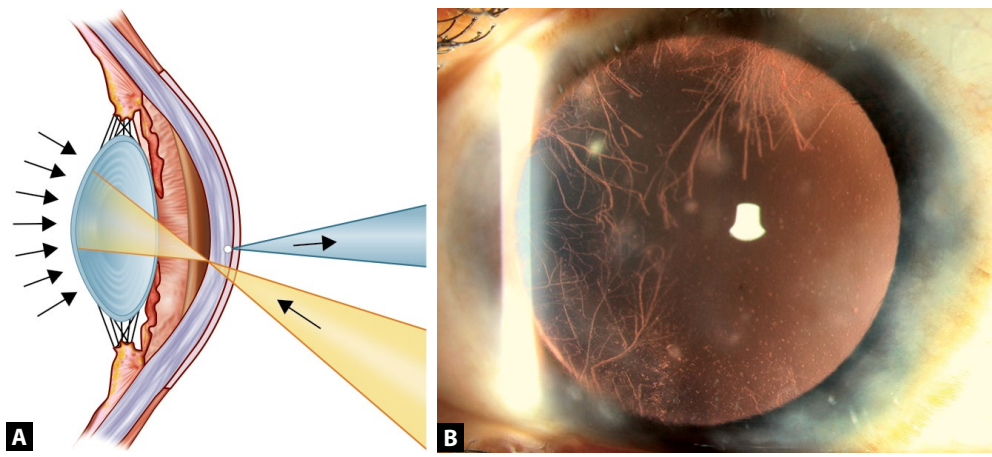
This type of viewing is achieved by positioning the beam of light and microscope such that the angle of incidence is equal to the angle of reflection. The light can be reflected from either the anterior (i.e. tears and epithelium) or posterior (i.e. endothelium) corneal surface.

Note that the reflected light should pass through only one eyepiece and therefore this method is monocular.

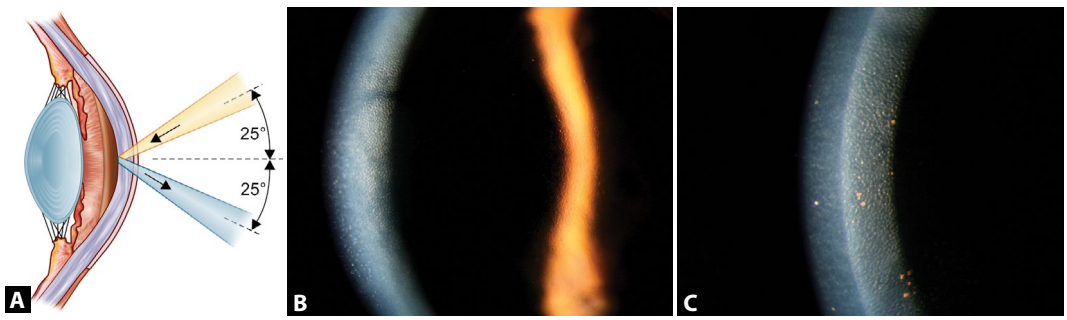
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Figs 10.9A and B: Direct retroillumination. **A.** Schematic representation; **B.** Photograph



Figs 10.10A and B: Indirect retroillumination. **A.** Schematic representation; **B.** Photograph



Figs 10.11A to C: Specular reflection. **A.** Schematic representation; **B and C.** Photograph

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- ***Method for viewing the posterior surface:*** The angle between the light and microscope arms should be about 50–60°.

A 2 mm wide parallelepiped and magnification of 20x–25x is used.

Find the image of the illuminating bulb, then move the light beam until the image of the bulb is just behind the posterior surface of the parallelepiped (Incidence = reflection when the dazzle from the precorneal fluid is seen).

Focus on the back of the parallelepiped. A mosaic of hexagonal endothelial cells will appear. The posterior endothelium and keratic precipitates may thus be studied.

This method of illumination is particularly useful to examine the endothelium layer of the cornea (e.g. blebs, polymegathism), although very high magnification is necessary, at least 40x is required and to see individual cells at 80x.

Sclerotic Scatter (Figs 10.12A and B)

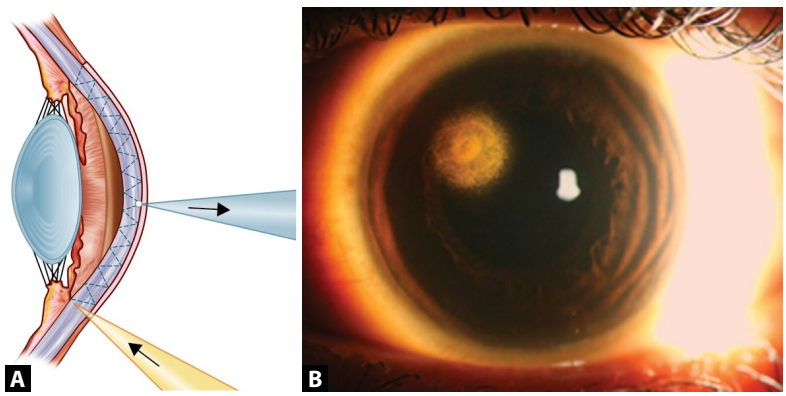
This method uses the principle of total internal reflection. A narrow vertical slit (1–1.5 mm in width) is directed in line with the temporal (or nasal) limbus. A halo of light will be observed around the limbus as light is internally reflected within the cornea, but scattered by the sclera. Any corneal opacities, edema or foreign bodies will be made visible by the scattering light, appearing as bright patches against the dark background of the iris and pupil. It is important that the room illumination is as dark as possible.

Oscillatory Illumination

A beam of light is rocked back and forth by moving the illuminating arm or rotating the prism or mirror. Occasional aqueous floaters and glass foreign body in the anterior chamber are easier to observe.

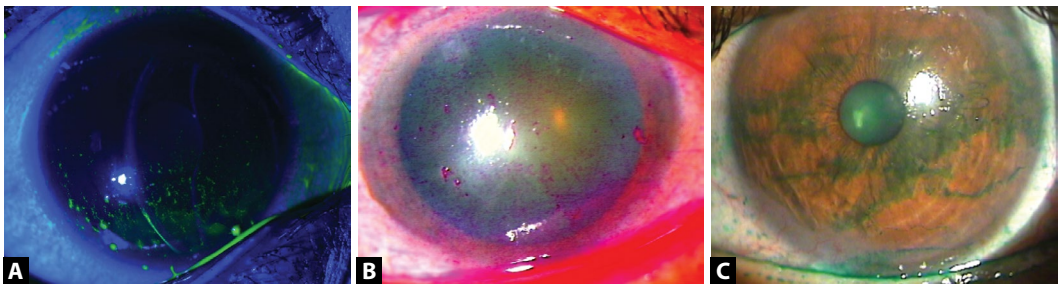
Special Stains for Epithelial Lesions

- ***Fluorescein staining (Fig. 10.13A):*** To stain various corneal pathologies in the epithelium level, e.g. corneal abrasion, erosions, filaments, epithelial defects, dendrite in herpes simplex virus (HSV) keratitis, superficial punctate keratopathies (SPKs), Seidel's test, tear film break up time (TFBUT), etc.
- ***Rose Bengal staining (Fig. 10.13B):*** It is useful for devitalized tissue, e.g. dendrite in HSV keratitis, punctate epithelial erosions (PEE) in dry eye and other lesions, conjunctival stain in dry eyes. It causes mild to moderate irritations of the eyes.
- ***Lissamine green staining (Fig. 10.13C):*** It is same as rose Bengal, except it does not cause much irritation.



Figs 10.12A and B: Sclerotic scatter. **A.** Schematic representation; **B.** Photograph

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Figs 10.13A to C: Special stains. **A.** Fluorescein staining; **B.** Rose Bengal staining; **C.** Lissamine green staining

Ocular examination proforma
At the undergraduate level only anterior segment of the eye is to be examined. A good torch and loupe are necessary for this. Examination of each eye should be done separately. Always examine the right eye first.

Right eye Left eye

- Visual acuity
- Ocular movement
- *Lids:* Lid margins, eyelashes, lid proper, palpebral aperture
- Conjunctiva
- Sclera
- Cornea
- Anterior chamber
- Iris
- *Pupil:* Size, shape, appearence
- *Light reflex:* Direct and consensual
- Lens
- Lacrimal apparatus
- Digital intraocular tension
- *Palpation:* Preauricular and submandibular lymph nodes, Any swelling related to the eye
- Transillumination (only if there is a large mass)
- Head posture, visual axis, facial symmetry

In undergraduate examination, in some centers, examiner may ask to examine one eye only. In that case write your findings for that eye only. For the other eye, just have a look with the torch light, and keep the findings in your mind. This is for better correlation with your diagnosis and management during crossing by the examiners.

CLINICAL EXAMINATION OF THE EYE

Visual Acuity

It is the ability or power of the eye by which objects are distinguished one from the other. It also measures the smallest retinal image, formed at the foveal region which can be appreciated regarding shape and size (central vision).

Visual acuity (VA) is tested for distant and near objects called ***distant vision*** and ***near vision***. Each eye is to be tested separately without spectacle ***(unaided)***, with spectacle ***(aided)*** and with pin-hole if the visual acuity is tested for distant vision only.

Principle of Normal Visual Acuity

It means when two distinct points can be only recognizable as separate when they subtend an angle of 1 minute of an arc at the nodal point of the eye.

Nodal point is an imaginary optical center or point which lies just infront of the posterior pole of the crystalline lens in the schematic eye.

Distant Vision

In case of Snellen's chart, each letter is perfectly placed in a square which is divided into 25 small squares. Each single letter subtends an angle of 5 minute and each component part of the letter subtends an angle of 1 minute at the nodal point of the eye, from a given distance in meters.

Snellen's Distant Vision Chart (Fig. 10.14)

Various charts are available in different languages, for illiterate (E chart or Landolt's broken ring) and for children (toys or picture chart).

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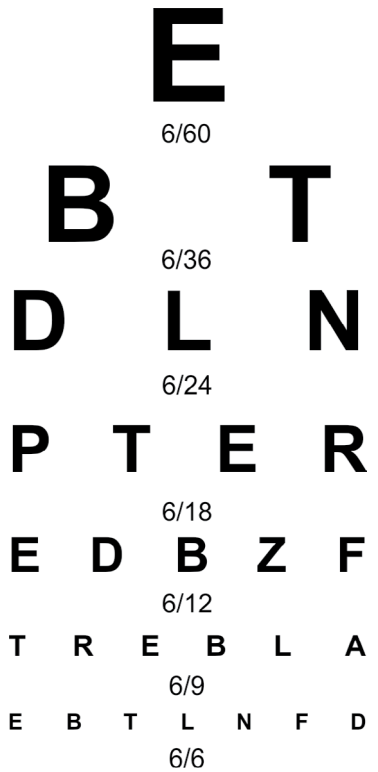


Fig. 10.14: Snellen's distant vision chart

The Snellen's chart should be read at a distance of 6 meters or 20 feet. If the room is 3 meters, with the help of a plane mirror and reverse chart this 6 meter distance is achieved. Rays coming from 6 meters or more are parallel for all practical purposes, hence accommodation does not come to play.

Visual acuity is written as numerator/denominator. ***Numerator*** is the distance of the patient from the chart and usually it is 6 meters or 20 feet. ***Denominator*** is the distance at which a normal person or the distance at which patient should be able to read.

As for example, 6/36 means that the patient reads from a distance of 6 meters what a normal person can read from a distance of 36 meters, or the patient reads from a distance of 6 meters what the patient should read from 36 meters. The normal distant visual acuity is recorded as 6/6.



Fig. 10.15: Visual acuity testing

Procedures of Recording Distant Vision (Fig. 10.15)

The patient is asked to sit or stand at 6 meters distance (i.e. 20 feet). Patient is asked to close his left eye with the cup of the palm, little bit obliquely to cross the opposite forehead. Patient is asked to read with his right eye from the top line to downward. The last line that patient reads, is recorded as the visual acuity of the right eye, e.g. top letter is 6/60, 2nd line is 6/36, 3rd line is 6/24 and so on. If patient can read some letters (not all letters) of a line, in that case visual acuity is recorded as part (e.g. 6/9p, 6/12p, etc.).

- If the patient can not read the first line (i.e. her visual acuity is less than 6/60), then she is brought nearer to the chart at a distance of 5 meters, 4 meters, 3 meters, and so on, till he is able to read the top letter of the chart. The vision is then recorded as 5/60, 4/60, 3/60 respectively.
- If the patient can not read the top letter at 1 meter distance, he is then asked to count the examiner's finger against an illuminated background. The rough distance at which he can count finger is recorded, e.g. VA FC (finger counting) at ½ meter.
- If vision is still less, the examiner will move his hand infront of the patient's eye. If he can appreciate the movements of the hand,

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then vision is recorded as hand movement (close to face).

- If the patient can not perceive hand movements—he is then taken to a dark room (ideally) and asked to close one eye firmly with the palm and look straight or look at his thumb of the other hand held in front of the eye to be tested and advised not to move that eye.
  The light is thrown on the open eye from all direction, i.e. up, down, nasal and temporal. If the patient can recognize the light and indicate its direction correctly, his visual acuity is perception of light (PL) and projection of Rays (PR) present.
  This vision is recorded as PL + PR in the right eye or left eye.
- If the patient is not able to perceive light from a particular quadrant, then a negative sign is put against that quadrant, e.g. VA = PL + PR , i.e. patient is having inaccurate PR.
- If the patient can see the glow of light only, but can not indicate the side of the projected rays, his VA is recorded as ***only PL*** with ***inaccurate PR***.

When light is thrown from one quadrant—the retina of the opposite quadrant is stimulated. "PL present" indicates the optic nerve is healthy with normal functioning nerve fiber layer of retina. PL is absent in optic atrophy.

PR accurate indicates the normal function of peripheral four quadrants of the retina. PR may be defective in detachment of retina, big patch of chorioretinal atrophy, advanced open angle glaucoma, etc.

- Last of all, if the patient cannot see or perceive the glow of the torch light—his vision is recorded as VA = No PL.

(Ideally, the illumination in the Snellen's chart is 100 foot candles, but it should not be less than 20 foot candles.)

The other eye, i.e. left eye is to be tested in a similar manner.

Lastly, one should record the distant visual acuity with both eyes open, i.e. binocular distant visual acuity.

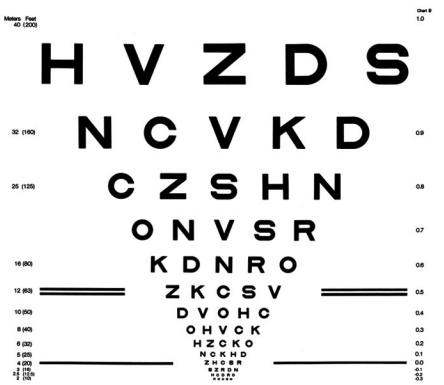


Fig. 10.16: LogMAR visual acuity

LogMAR Visual Acuity (Fig. 10.16)

LogMAR stands for logarithm of the minimum angle of resolution.

The chart was designed by Bailey and Lovie for the early treatment diabetic retinopathy study (ETDRS) which is designed to be used at a distance of 4 meters.

At this distance the top lines will give a score of 1.0. Each line below will give a score 0.1 less than the line above.

Five letters are chosen for each line (using a balanced distribution). Each of the five letters, in each line, count for a score of 0.1/5 = 0.02.

Therefore, if a patient reads the 0.4 line in its entirety, he will have a score of 0.4.

If patient reads the 0.4 line plus three letters of the 0.3 line, then he will have a score of 0.34, which results from the five letters of line 0.4 minus the score for each letter read from the 0.3 line, i.e. 0.40 – (0.02 × 3) = 0.34.

Principle of LogMAR Chart

Ten Sloan/Snellen letters have been chosen for their equality of readability and the letters are S, D, K, H, N, O, C, V, R and Z. All of these letters have been allocated at a difficulty level. These have been arranged on the charts in equal lines of 5.

The progression of letter height is that any line is 1.2589 times greater than the line

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below. This multiplier is the root of ten or 0.1 log unit.

Therefore, a three line worsening of visual acuity is equal to a doubling of the visual angle regardless of the initial acuity.

The charts are designed for use at 4 meters which helps with smaller examining rooms and, of course, the size of the chart.

Steps of visual acuity testing with LogMAR chart:

- Set the patient/chart distance to 4 metres.
- Cover one eye. If the patient cannot read any letters, move the chart to a distance of 1 meter from the patient and add 0.6 to the LogMAR score for the for each line.
- Repeat for the second eye.
- If the patient is still unable to read any letters proceed to another means of assessment of their acuity.

LogMAR scale is more important than the traditional Snellen's notation:

- Use of LogMAR scale allows analysis of visual acuity scores more effectively and comparisons of results more precisely.
- It offers this because the equal linear steps of the LogMAR scale represent equal ratios in the standard size sequence.

Visual Acuity Expression

Visual acuity depends upon—(1) how accurately light is focused on the macula, (2) the integrity of neural elements of the eye, and (3) the interpretative faculty of the brain.

It is often measured according to the size of the letters viewed in a Snellen's chart. In some countries, VA is expressed as a ***LogMAR fraction*** and in some as a ***decimal notation*** (Table 10.1).

Using the ***meter*** as a unit of measurement, VA is expressed relative to 6/6. Otherwise, using the foot, visual acuity is expressed relative to 20/20. For all practical purposes 6/6 vision is equivalent to 20/20. In the decimal system a value of 1.0 is equal to 6/6 or 20/20.

LogMAR scale is rarely used clinically. It is more frequently used in statistical calculations because it provides more scientific equivalent for the traditional clinical statement of ***line lost*** or ***line gained***.

TABLE 10.1: Visual acuity notation in different scales

Meter	Foot	Decimal	LogMAR
6/60	20/200	0.10	1.0
6/48	20/160	0.13	0.9
6/37	20/125	0.16	0.8
6/30	20/100	0.20	0.7
6/24	20/80	0.25	0.6
6/18	20/63	0.32	0.5
6/15	20/50	0.40	0.4
6/12	20/40	0.50	0.3
6/9	20/32	0.63	0.2
6/7	20/25	0.80	0.1
6/6	20/20	1.00	0.0
6/4	20/16	1.25	-0.1
6/3.75	20/12.5	1.60	-0.2
6/3	20/10	2.00	-0.3

Normal VA is frequently considered to be what was defined by Snellen as—***the ability to recognize a letter when it subtends 5 minute of an arc***. That is Snellen's chart 6/6 meter, 20/20 feet, 1.00 decimal or 0.0 logMAR. It is possible to have vision superior to 6/6 or 20/20.

Recent developments in refractive technology (surgery or corrective lens) have resulted in conferring a vision upto 6/3 or 20/10. Some birds (e.g. Hawk) are believed to have an acuity of around 6/1 or 20/3, which is much better than human eyesight.

Near Vision

It is always tested and corrected after correcting the distant vision. Different charts are used to record near vision.

- Jaeger's chart—J_1, J_2, J_3, J_4, etc.
- The 'N' chart—N_6, N_8, N_{10}, N_{12}, N_{18}, etc. (Fig. 10.17).

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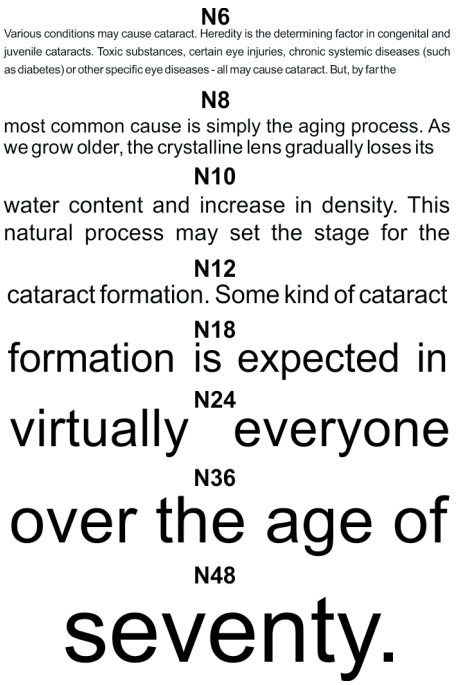


Fig. 10.17: Near vision 'N' chart

- Snellen's near chart—It is 1/17th times photographic reduction of the original Snellen's chart for distance.

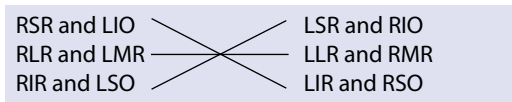
Procedure

The patient is asked to sit in a brilliantly illuminated place. Near vision chart is held at a distance of 25–33 cm depending upon the patient's nature of near work. The patient is asked to read the chart from bigger print size to smaller prints. The line that he reads upto, is recorded, e.g. N_{12}, N_6, etc. Each eye should be tested separately first, and then binocularly. Normal near acuity is N_6 in 'N' chart.

Ocular Movements

Any imbalance, uniocular and binocular is to be noted.

The sign used for ocular movement is ***union Jack position***, i.e. in 6 cardinal gazes.



RMR: Right medial rectus
RSO: Right superior oblique
RIR: Right inferior rectus
LMR: Left medial rectus
LSR: Left superior rectus
LSO: Left superior oblique
RLR: Right lateral rectus
RSR: Right superior rectus
RIO: Right inferior oblique
LLR: Left lateral rectus
LIR: Left inferior rectus
LIO: Left inferior oblique

Any under-action or over-action is noted with '–' (minus) or '+' (plus) sign. Normally, a person has orthophoria. Any abnormality in the ocular movements is noted down.

Lids

Normally upper lid covers about 1–2 mm of cornea at 12 o'clock position and the lower lid just touches the limbus at 6 o'clock position. Eyelids are examined for congenital or acquired lesion.

Lid margin

- Thickened—multiple chalazion, blepharitis.
- Inverted (entropion) or everted (ectropion).
- Any ulcer or scales (blepharitis).

Eyelashes

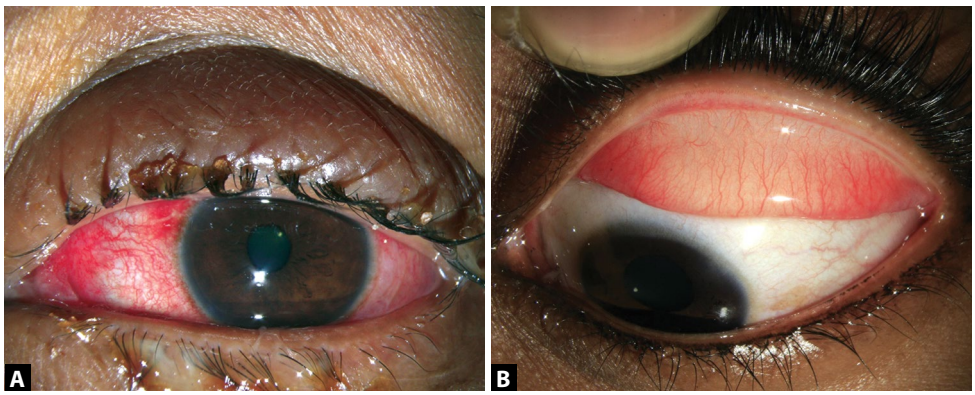
- Misdirected and touching the globe (trichiasis).
- Loss or scantiness of eyelashes—(madarosis) in blepharitis.
- ***Matting of the eyelashes:*** Due to mucopurulent discharge (Fig. 10.18A).

Lid Proper

- Thickened—multiple chalazion, blepharitis
- Redness —blepharitis, Stye
- Edema—hordeolum, allergic conjunctivitis, insect bite, corneal ulcer, lid abscess
- Localized lid—chalazion, cyst, swelling growth

Palpebral aperture: 8–9 mm vertically and 20–25 mm horizontally.

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Figs 10.18A and B: A. Lid edema with matting of eyelashes; **B.** Eversion of the upper eyelid

- Narrow—phthisis bulbi, ptosis, lid edema, Horner's syndrome, enophthalmos, microphthalmos
- Wide—Lid retraction (thyroid ophthalmopathy), proptosis, other eye in unilateral ptosis

Any other specific lid sign: Lid lag, lagophthalmos, black eye, coloboma or pigmentation is to be noted.

Conjunctiva

Conjunctiva is a mucous membrane covering the sclera and the back of the eyelids. It is to be examined for any type of congestion, inflammatory or allergic reactions, degenerative lesions and foreign bodies.

Bulbar conjunctiva, palpebral conjunctiva (lower and upper), limbal conjunctiva and fornix—all are to be examined.

- ***Examination of the lower palpebral conjunctiva, lower fornix and lower part of bulbar conjunctiva*** can be easily done by pulling down the lower lid by the thumb or index finger, while the patient is asked to look upwards.
- ***Examination of upper palpebral conjunctiva is*** done by everting the upper lid (Fig. 10.18B).
- ***Examination of upper fornix*** requires double eversion, with the help of a lid retractor.
- ***Examination of bulbar conjunctiva and limbal conjunctiva*** are done by separating both lids while the patient is asked to move his eyeball in different directions.

Bulbar conjunctiva:

- Lustre
- Congestion
- Discharge
- Edema or chemosis
- Subconjunctival hemorrhage
- Pterygium
- Pinguecula
- Bitot's spot
- Symblepharon and
- Cyst, nodule, nevus or growth

Palpebral conjunctiva:

- Papillae
- Follicles
- Concretions
- Scarring (as in trachoma)
- Foreign body
- Any tumor mass and
- Arrangement of blood vessels

Limbal conjunctiva:

- Circumcorneal congestion or ciliary congestion (CCC)
- Phlycten
- Nodules
- Follicles and
- Scar or bleb of previous surgery.

Differences between conjunctival congestion and ciliary congestion (*see* page 135).

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Sclera

It is dense tough fibrous envelope that covers 5/6th of the eyeball. Normally, the sclera is whitish in adult and bluish in children, and is covered by the conjunctiva.

- Nodule—episcleritis, scleritis
- Congestion—dusky ciliary congestion in scleritis
- Ectasia—ciliary or equatorial staphyloma
- Thinning—long standing scleritis
- Blue sclera—buphthalmos, osteogenesis imperfecta.

Cornea

It is avascular, transparent structure forming the anterior 1/6th of the eyeball, It is examined for its size, shape, surface, curvature, transparency, opacity, staining, vascularization, sensation and keratic precipitate (KPs).

Size

Horizontally 12 mm, vertically 10–11 mm.

- ***Size increased:*** Megalocornea, buphthalmos.
- ***Size decreased:*** Microcornea, microphthalmos.

Shape

Normally, it is like a part of a sphere. It may be flat, conical or globular.

- Flat—cornea plana, phthisis bulbi
- Conical—keratoconus
- Globular—keratoglobus, buphthalmos.

Surface

Normally, it is smooth and regularly curved. Surface and curvature are tested with:

- Window reflex
- Slit lamp
- Placido's disc
- Photokeratoscope
- Keratometer
- Corneal topography.

There will be distortion of the image or window reflex in case of keratoconus, corneal edema, corneal ulcer or opacity, etc.

Corneal Staining

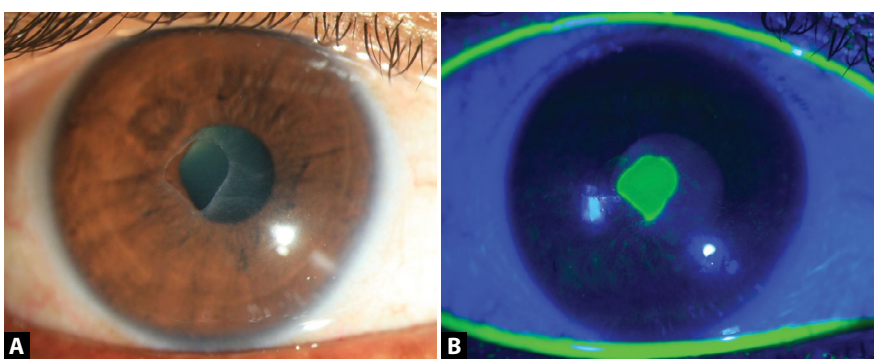
Not necessary in all cases.

- ***Fluorescein staining:*** It is used for denuded epithelium (corneal abrasion, erosions or ulcers) (Figs 10.19A and B).
- ***Rose Bengal staining:*** It is used for devitalized cells. Useful in xerosis of the conjunctiva and cornea (dry eye).
- ***Alcian blue:*** It stains mucus selectively.

Transparency

The cornea is optically transparent. This transparency is due to certain anatomical and physiological factors. They are as follows:

- Avascularity of the cornea
- Demyelinated nerve supply
- Regular arrangement of the stromal collagen fibrils (lattice theory)



Figs 10.19A and B: Corneal abrasion. **A.** Normal view; **B.** Fluorescein staining

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- Active endothelial pump mechanism
- Optimum intraocular pressure.

Any interference with these factors affects the corneal transparency. Thus the cornea becomes hazy in corneal edema, ulcers, scars, xerosis, vascularization, mucopolysaccharidosis (MPS), acute attack of angle-closure glaucoma, absolute glaucoma, etc.

Opacity

For development of a corneal opacity at least the Bowman's membrane must have to be damaged.

Grades of Opacity (Figs 10.20A to D)

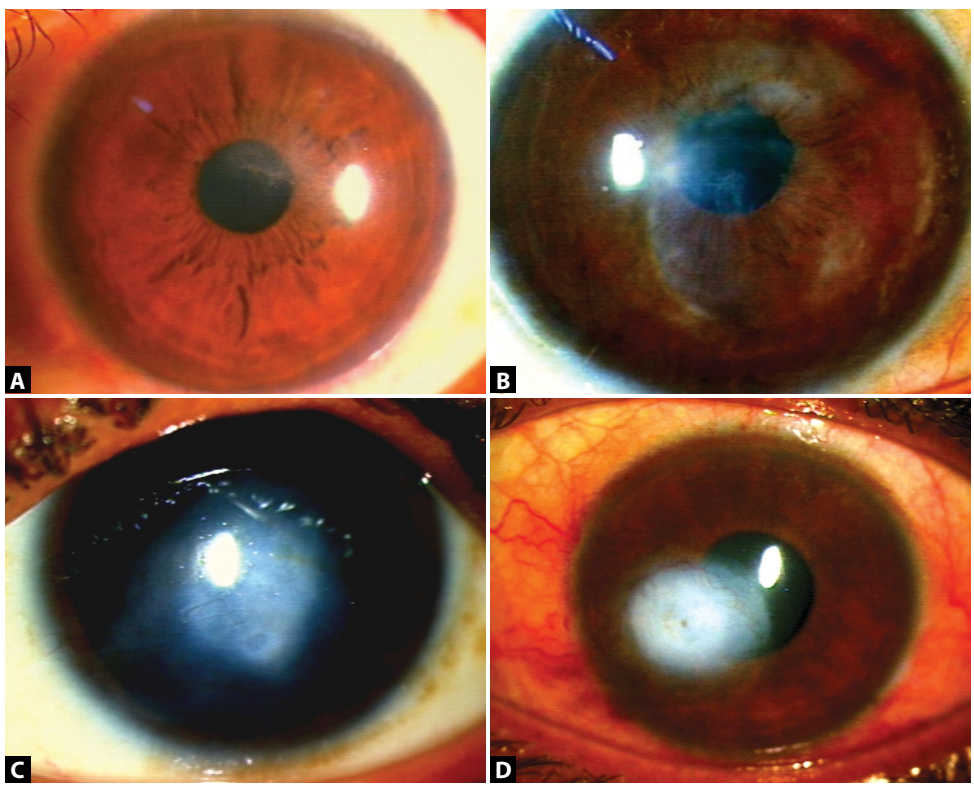
- ***Nebula:*** Only Bowman's membrane is involved.
- ***Macula:*** Bowman's membrane and part of the anterior stroma are involved.
- ***Leuroma:*** Full thickness cornea is involved.
- ***Adherent leuroma:*** A full thickness corneal opacity with iris inclusion. It indicates corneal perforation or a penetrating injury in the past.

In case of corneal opacity look for:

- Its density (grade)
- Situation and extent in relation to the pupillary axis and limbus
- Any pigmentation
- Any vascularization—superficial or deep
- Adherent or not
- Its sensation.

Causes of corneal opacity

- Degeneration
- Dystrophy



Figs 10.20A to D: Grades of opacity. **A.** Nebular corneal opacity; **B.** Macular corneal opacity; **C.** Leukomatous corneal opacity; **D.** Adherent leukoma

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- Trauma (surgical/nonsurgical)
- Healed infection (keratitis or corneal ulcer) and
- Congenital (MPS)
- Corneal sensation.

Method of examination: Patient is asked to look straight with both eyes wide open. A wisp of cotton is brought close to the patient's eye from the temporal side (to avoid optical blinking reflex) and the lower part of cornea is touched with it. The blinking reflex of the lids is observed. Avoid any accidental touch to the eyelashes (a false blinking will result).

Cornea is supplied by ophthalmic division of 5th cranial nerve and it has no kinesthetic sensation.

Causes of loss of corneal sensation:

- Herpetic keratitis
- Acute attack of angle closure glaucoma
- Keratomalacia
- Leprosy
- 5th nerve damage
- After corneal surgery
- Neuroparalytic keratitis
- Long standing corneal edema, etc.

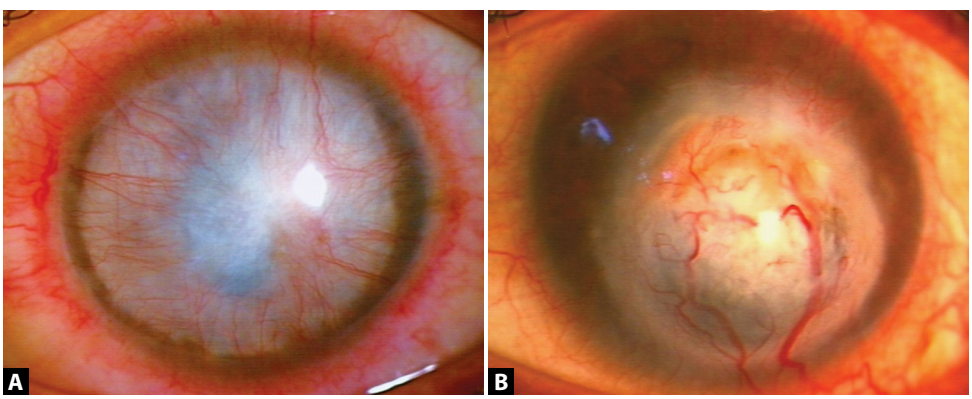
Vascularization (Fig. 10.21 and Table 10.2)

- Is it localized or circumferential?
- How much area is involved?
- Is it superficial or deep?

Causes of corneal vascularization:

- Corneal ulcers
- Trachoma
- Interstitial keratitis
- Leprosy
- Diabetes
- After penetrating keratoplasty, etc.

Keratic precipitates (KPs): These are deposits of inflammatory cells on the lower part of



Figs 10.21A and B: Vascularization. **A.** Superficial; **B.** Deep

TABLE 10.2: Differences between superficial vascularization and deep vascularization

Superficial vascularization	Deep vascularization
• It can be traced over the limbus into the conjunctiva • Bright red and well-defined • Branches in an arborescent fashion, and fans dichotomously. • It may raise the epithelium over them and corneal surface is uneven	• Its seem to come from an abrupt end at the limbus • Greyish red (red blush) and ill-defined • Branches at acute angles and runs in a radial fashion • Deep inside the stroma and corneal surface remains smooth

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corneal endothelium and best seen by a slit lamp. They may be: Fine, Medium, Mutton fat, Pigmented, and Fresh or old.

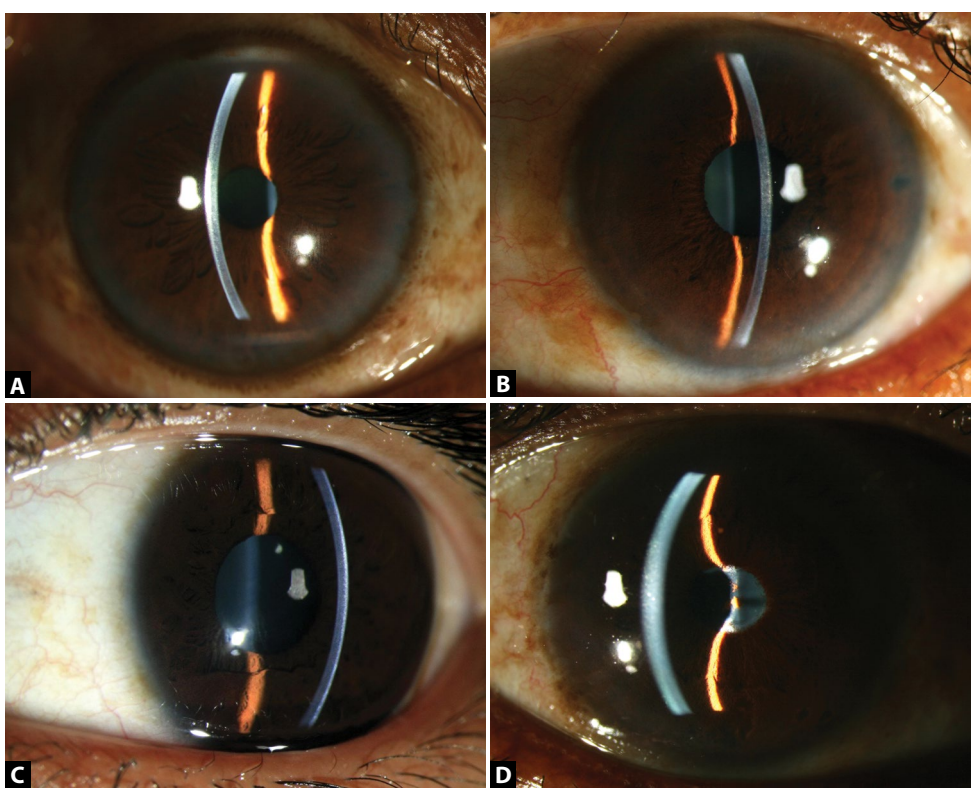
This is due to inflammation of the anterior uveal tract (anterior uveitis or iridocyclitis).

Ulcer or abrasions: Details of ulcer e.g. size, shape, extent, margin, floor, central or peripheral, etc. are to be noted. Also note the staining pattern with fluorescein dye if necessary.

Anterior Chamber

It is the space between the cornea and the iris. Normally, it contains aqueous humor. It is approximately 25 mm in depth at its center.

- ***Depth:*** Normal, shallow, deep and irregular (Figs 10.22 and 10.23).
- ***Causes of shallow anterior chamber***
  - Hypermetropia
  - Hypermature cataract (Morgagnian)
  - Intumescent cataract
  - Angle closure glaucoma
  - Choroidal detachment
  - Pupillary block
  - Wound leak after intraocular surgery
  - Perforating injury or perforating corneal ulcer, etc.
- ***Causes of deep anterior chamber***
  - Myopia
  - Aphakia
  - Keratoconus/keratoglobus
  - Buphthalmos
  - Posterior dislocation of the lens, etc.
- ***Causes of irregular anterior chamber***
  - Subluxation of the lens
  - Iris bombe (funnel-shaped anterior chamber)



Figs 10.22A to D: Anterior chamber depth. **A.** Normal depth; **B.** Shallow; **C.** Deep; **D.** Funnel-shaped

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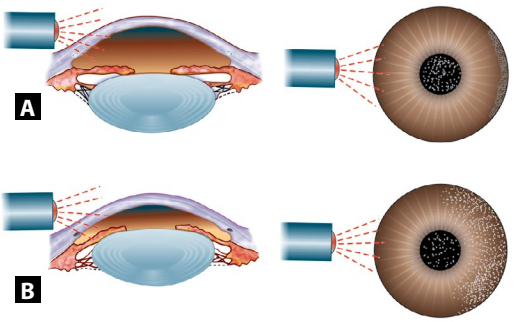
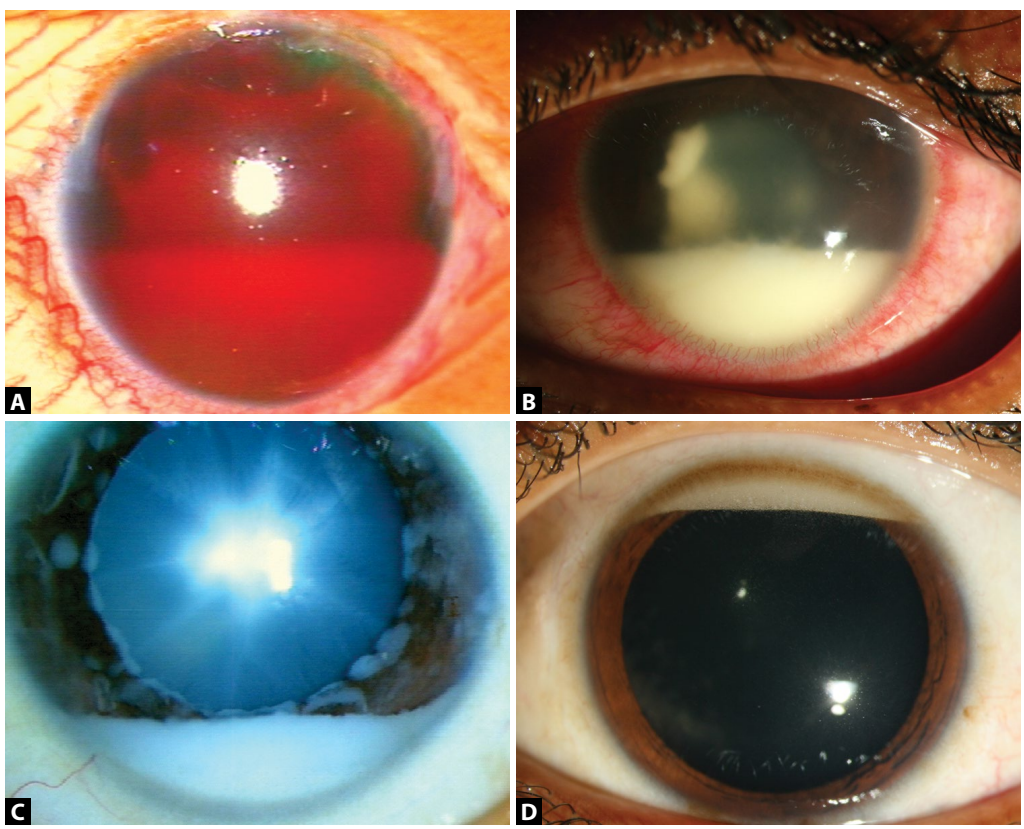


Fig. 10.23: Oblique illumination by torch light: A screening measure to estimate the anterior chamber depth; **A.** With a deep chamber, nearly the entire iris is illuminated; **B.** When the iris is bowed forward, only the proximal portion is illuminated, but a shadow is seen in the distal half

- Adherent leucoma
- Iris cyst
- Angle recession, etc.

- ***Abnormal contents of the anterior chamber (Figs 10.24A to D)***
  - Blood (***hyphema***), e.g. traumatic, postoperative, herpetic iridocyclitis, spontaneous.
  - Pus (***hypopyon***), e.g. in corneal ulcer, acute iridocyclitis, endophthalmitis.
  - Malignant cells ***(pseudohypopyon)***, e.g. in retinoblastoma.
  - Lens matter [after extracapsular cataract extraction (ECCE)] (Fig. 10.25).
  - Albuminous material (aqueous flare) as in iridocyclitis.



Figs 10.24A to D: Abnormal contents of anterior chamber. **A.** Hyphema-blood; **B.** Hypopyon-pus; **C.** Pseudohypopyon in retinoblastoma; **D.** Inverse hypopyon—emulsified silicone oil

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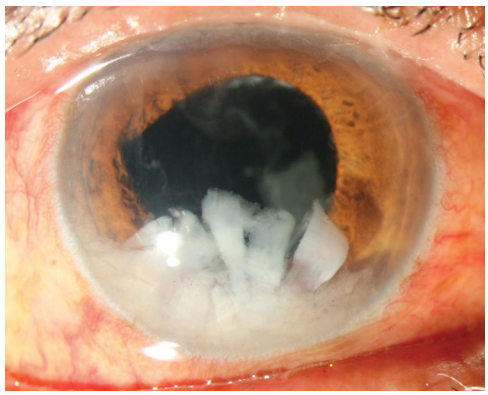


Fig. 10.25: Lens fragment in anterior chamber

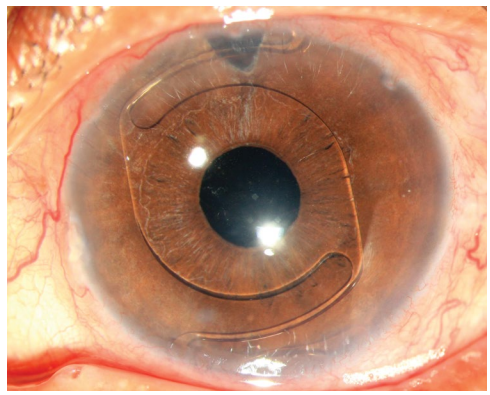


Fig. 10.26: Anterior chamber intraocular lense

- Intraocular lense (IOL) (anterior chamber IOL or iris-claw IOL) (Fig. 10.26).
- Vitreous [after intracapsular cataract extraction (ICCE) or after accidental rupture of posterior capsule in ECCE].

Angle of the anterior chamber is examined by ***gonioscope***. Normally, it is not possible to see angle structure due to high corneal refractive power and iris-corneal interface.

Iris

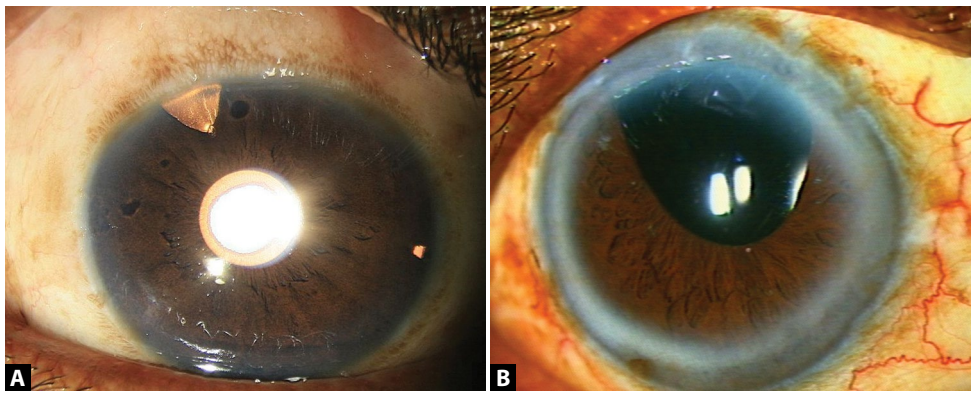
It is a brown or black diaphragm hanging infront of the lens, and is perforated centrally which is known as ***pupil***.

- ***Color:*** Difference in color of iris between two eyes is called ***heterochromia***.
- ***Causes of heterochromia***
  - Congenital
  - Iris atrophy
  - Heterochromic cyclitis of Fuchs
  - Pigmented tumour of the iris
  - Siderosis bulbi, etc.
- ***Pattern:*** 'Muddy iris' in iridocyclitis where the pattern is lost. Here, the iris becomes edematous, swollen, water-logged and shows impaired mobility.
- ***Iridodonesis:*** Tremulousness of iris (due to loss of support of the lens). This is elicited by asking the patient to move the eyeball in different directions.
  - ***Causes of iridodonesis***
    - Aphakia
    - Dislocation or subluxation of lens
    - Sometimes in pseudophakia and
    - Buphthalmos.
- ***Synechia:*** Abnormal adhesions of the iris are called ***synechiae***. Adhesion of iris with the cornea is called anterior synechia, and adhesion of iris with the lens capsule, or vitreous face is called ***posterior synechia***.
  - ***Causes of anterior synechia***
    - Perforated corneal ulcer
    - Penetrating injury
    - Angle closure glaucoma and
    - Iris bombe (iridocyclitis).

    In case of small central perforation of cornea, one may not find anterior synechia, instead anterior polar cataract is found.
  - ***Causes of posterior synechia:*** Iridocyclitis.
- ***Vascularization*** of iris is called ***rubeosis iridis***, found in diabetes, central retinal venous thrombosis, heterochromic cyclitis of Fuchs, etc.
- ***Any gap in iris:*** Congenital gap (coloboma of iris found at the inferonasal region) or marks of iridectomy (peripheral or complete) is to be noted (Figs 10.27A and B).

Peripheral button hole iridectomy is done in cataract surgery (11 and/or 1 o'clock position), in angle closure glaucoma or trabeculectomy operation.

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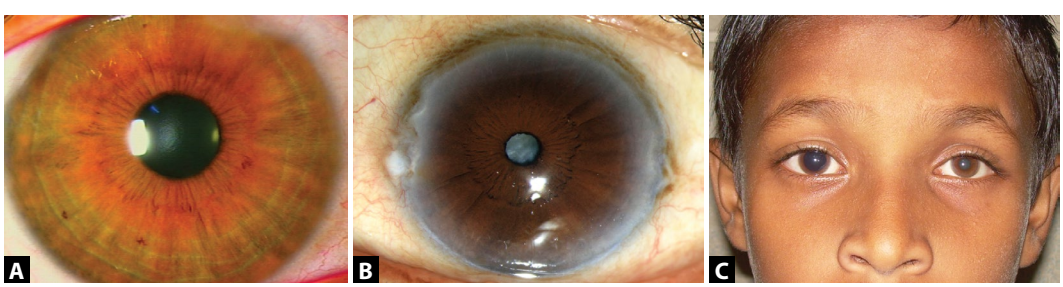
Figs 10.27A and B: A. Peripheral iridectomy mark; **B.** Pupil after complete iridectomy in aphakia

Complete iridectomy marking may be found in aphakic patient (if there is any vitreous loss during ICCE or in presence of extensive posterior synechiae).

Pupil

It is a circular aperture at the center of the iris diaphragm (Fig. 10.28A). It regulates the amount of light entering the eye, and helps in maintaining the depth of focus. Pupil is examined for its position, size, shape, reactions and color of its reflex.

- ***Position:*** The pupil is situated just inferior and nasal to the center of the iris. It may be eccentric, known as corectopia, as seen in congenital corectopia, after penetrating injury or after vitreous loss in cataract surgery (updrawn pupil).
- ***Size:*** Normally, it varies between 2 and 4 mm. It may be smaller (miosis) or larger (mydriasis).
- ***Causes of Miosis (pupil size less than 2 mm) (Figs 10.28B)***
  - Extreme of ages
  - In bright light
  - Opium addict
  - Morphine intoxication
  - Pontine hemorrhage
  - Acute iritis
  - During sleep and
  - Use of miotics (e.g. pilocarpine).
- ***Causes of mydriasis (pupil size more than 6 mm)***
  - In dark
  - Optic atrophy (Fig. 10.28C)
  - Acute attack in angle-closure glaucoma
  - Absolute glaucoma
  - Comatose patient
  - Head injury
  - 3rd nerve palsy and



Figs 10.28A to C: A. Nomal pupil; **B.** Miotic pupil in aging; **C.** Mydriasis in optic atrophy right eye

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Fig. 10.29: Pupil in different shapes

- Use of mydriatics (e.g. atropine, homatropine, phenylephrine, etc.).

- ***Shape (Fig. 10.29):*** Normally, it is perfectly circular. It may be of following types:
  - ***Irregular:*** Iritis, post-traumatic
  - ***D-shaped:*** Iridodialysis
  - ***Boat or hammock-shaped:*** Vitreous loss in cataract surgery
  - ***Pear-shaped and updrawn:*** Incarceration of iris with corneal wound
  - ***Festooned:*** Iridocyclitis
  - ***Mid-dilated and oval:*** Acute attack in angle-closure glaucoma
  - ***Oval and inferonasal:*** Coloboma of the iris
  - ***Key-hole appearance:*** After optical iridectomy.
- ***Light reactions:*** The pupil constricts briskly on exposure to bright light and dilates in the dark. The afferent pathway is via the optic nerve and the efferent, via the third cranial nerve. Light reactions are direct and consensual.
  - ***Direct light reaction:*** The patient is asked to cover one eye with his own palm, and the beam of pencil light is thrown on the pupil of uncovered eye from one side, noting the nature of pupillary constriction. Normally, the reaction is brisk and sustained.
  - ***Consensual light reaction:*** Pupil of the contralateral eye constricts when the light beam is thrown on the ipsilateral eye.

    Ill sustained pupillary reaction is a sign of optic neuritis. In ***optic atrophy,*** direct light reaction is absent but consensual reaction is present.

    In relative afferent pupillary defect (RAPD) or Marcus-Gunn pupil, both pupils dilate when the light is moved from the unaffected eye to the affected eye. RAPD is mostly seen in unilateral optic neuritis.
- ***Color reflex of the pupillary area:***
  - Jet black pupillary reflex—aphakia
  - White pupillary reflex—cataract
  - Glassy pupillary reflex—pseudophakia
  - Amaurotic cat's eye reflex—retinoblastoma, congenital cataract, persistent hyperplastic primary vitreous (PHPV) endophthalmitis, coloboma of choroid, etc. (*see* page 368).

Lens

The crystalline lens is a transparent biconvex structure with a nucleus, cortex and the capsule.

It is supported by suspensory ligaments, called zonules, attached to the ciliary processes and the valleys between them.

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Color of the Lens

Normally, it is transparent.

- ***Greyish-white:*** Immature cortical cataract
- ***White or pearly white:*** Mature cataract
- ***Milky white:*** Hypermature cataract
- ***Shrunken and white with calcified spots on the anterior capsule:*** Hypermature sclerotic cataract
- ***Amber:*** Early nuclear cataract
- ***Brown:*** Cataracta brunescens (nuclear cataract)
- ***Black:*** Cataracta nigra (nuclear cataract).

Position

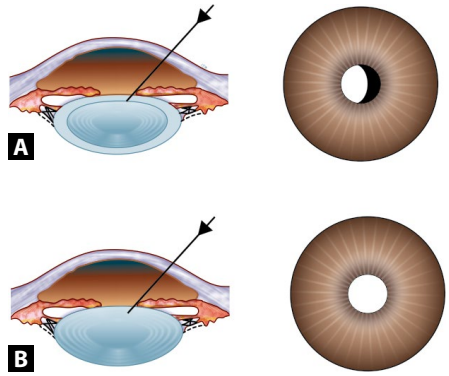
Normally, it is in the pupillary area. Note for any subluxation or dislocation.

- ***Phakodonesis*** or ***tremulousness*** of the crystalline lens is a sign of lens subluxation.
- ***Opacity*** or any subluxation is better judged with dilated pupil.
- ***Aphakia*** means absence of crystalline lens from its normal anatomical position (i.e. from the pupillary area).
- ***Ectopia lentis*** is the congenital malposition of the lens, due to faulty development of the lens zonules, as seen in Marfan's syndrome, homocystinuria, Weill Marchesani's syndrome, etc.
- ***Pseudophakia*** means the presence of an IOL in the eye after cataract operation with IOL implantation.

Iris Shadow (Figs 10.30A and B)

It is a concave shadow of the pupillary margin of the iris, cast upon the lens when light is thrown obliquely.

It signifies that some clear cortical fibers are still present beneath the lens capsule (i.e. between the iris margin and the actual opacity of the lens). It is present in immature cataract. But it is absent in mature or hypermature cataract. In case of mature cataract all the substance of lens becomes opaque, so iris margin lies almost in contact with the opaque lens surface—so no iris shadow will form if light is thrown obliquely.



Figs 10.30A and B: Iris shadow. **A.** Present—immature cataract; **B.** Absent—mature or hypermature cataract

Pigmentation on the Anterior Lens Surface

- A sign of posterior synechiae in old iridocyclitis
- In traumatic cataract (Vossius's ring)
- Sometimes, in case of persistent pupillary membrane.

Purkinje's images: Note the 3rd and 4th Purkinje's images in a dark room. 3rd image is formed by the anterior convex surface of the lens and it moves in the same direction, and is erect. 4th images is formed by the posterior concave lens surface. It is inverted and moves in the opposite direction.

In ***aphakia***; both the 3rd and 4th images are absent.

In ***mature or hypermature cataract***; 3rd image is present but the 4th image is absent.

In ***immature cataract***; 3rd image is present, but 4th image may be absent or distorted.

The structures beyond the lens like the vitreous, retina and optic nerve are not possible to examine with a torch and loupe.

Lacrimal Apparatus

Lacrimal Puncta

They are situated on the medial side of the lid margins on the papilla. They are always in contact with the eyeball. Note:

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- They are open or stenosed.
- Any eversion or non-contact with globe
- Any inflammation.

Lacrimal Sac

- ***Skin over the sac area:*** Swollen, inflamed, any excoriation, fistula or scar mark.
- ***Pressure over the sac region*** (by pressing with the thumb just below the medial canthus):
  - Regurgitation of mucoid material (mucocele), pus (pyocele) through the puncta.
  - Swelling is present but no regurgitation via puncta—encysted mucocele.
  - Sometimes regurgitates may go into the nasopharynx, i.e. there is block in common canaliculus.
- ***Acute signs of inflammation*** (i.e. swelling, redness, raised temperature and tenderness)—acute dacryocystitis.

Note: Nasal cavity is better examined grossly for DNS, polyps, hypertrophied inferior turbinate, etc. in case of chronic dacryocystitis.

Digital Tonometry

Technique

Rough assessment of IOP by eliciting the fluctuation of the eyeball. The patient is asked to look downwards at his feet. Examiner places both index fingers side by side above the upper border of the tarsal plate on the upper lid, resting the other fingers lightly on the forehead (Figs 10.31A and B). One index finger is stationary and the other one presses the globe, thereby conveying the amount of fluctuation to the stationary finger. Fluctuation can be appreciated well by regular practice. The intraocular tension is graded as normal, high or low.

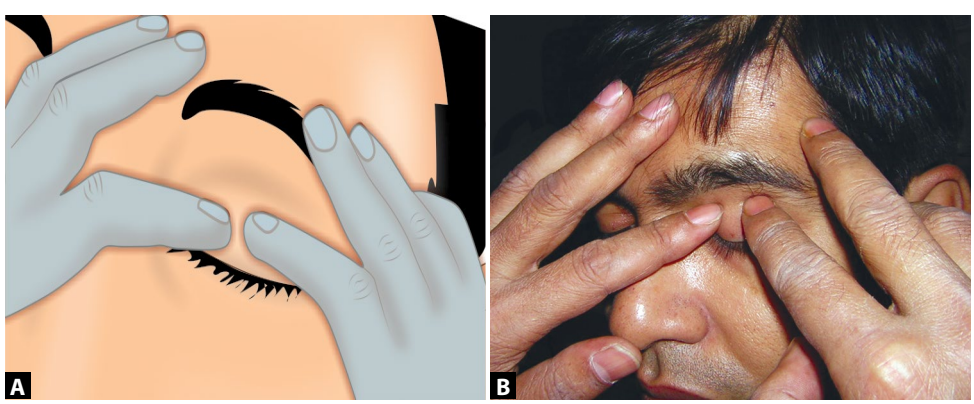
The eyeball is hard in all primary and secondary glaucomas. It is ***stony hard*** (the fluctuation is absent) in absolute glaucoma.

The eyeball is soft in chronic uveitis, recent penetrating injury, wound leak, choroidal detachment and retinal detachment. In phthisis bulbi, it is soft as water bag.

Other means of recording IOP:

- ***Schiotz tonometer:*** By indentation of the cornea with this instrument, IOP can be recorded from a chart after getting the scale reading.
- ***Applanation tonometer:*** Greatest accuracy of IOP is determined by this method by applanating the central portion of the cornea. This instrument is an optional attachment to the slit lamp.
- Non contact air-puff tonometer (NCT): is popular as a screening device for IOP measurement in busy clinic or OPD.

Normal IOP is about 14 to 21 mm of Hg.



Figs 10.31A and B: Digital tonometry (finger tension); **A.** Schematic representation; **B.** Photograph

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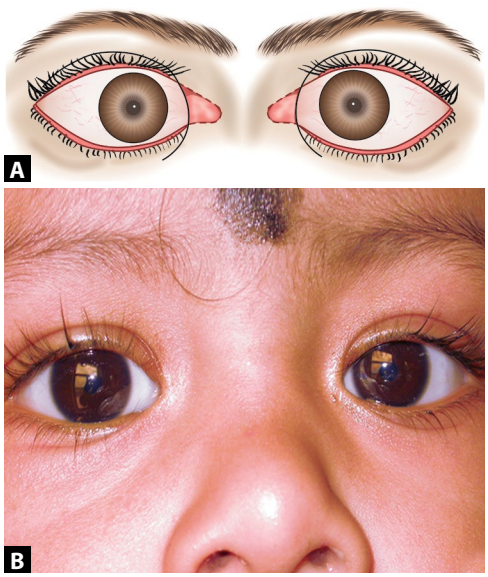
11 CHAPTER Diseases of the Eyelids

SOME CONGENITAL ABNORMALITIES

Symblepharon, ankyloblepharon, ectropion, entropion, trichiasis, coloboma, epicanthus, blepharophimosis, ptosis—all may occur as congenital malformation (Table 11.1).

Epicanthus (Figs 11.1A and B)

- Most common congenital condition.
- A semilunar skin fold, situated above and sometimes covering the inner canthus.



Figs 11.1A and B: Epicanthus, note the appearance of pseudo-convergent squint. **A.** Schematic representation; **B.** Photograph

TABLE 11.1: Some abnormalities of the eyelids

Ankyloblepharon	Adhesion between upper and lower eyelid margins
Ablepharon	Absence of the eyelid
Blepharophimosis	Decreased dimensions of the palpebral fissure
Blepharospasm	Tonic spasm of orbicularis oculi muscle
Coloboma	Absence of some ocular tissue
Distichiasis	Accessory row of eyelashes
Ectropion	Outward turning of the lid margin
Entropion	Inward turning of the lid margin
Epiblepharon	Extra fold of skin in the lower lid
Epicanthus	Extra fold of skin on medial side of the lower lid (palpebronasal fold)
Euryblepharon	Enlarged palpebral aperture
Floppy eyelid syndrome	Floppy, easily eversible upper lid with papillary conjunctivitis at the upper tarsus
Lagophthalmos	Inability to close the eyelid completely
Myokymia	Fascicular tremor of the orbicularis oculi muscle
Ptosis	Drooping of upper eyelid
Symblepharon	Adhesion between the palpebral and bulbar conjunctiva
Telecanthus	Widely separated nose and eye (medial canthi)
Trichiasis	Misdirected eyelashes

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A B

Figs 11.2A and B: Coloboma of the upper eyelid. **A.** Schematic representation; **B.** Photograph (arrow)

- Usually bilateral, and simulate the appearance of pseudoconvergent squint.
- It is normal in Mongolian races.
- Easily treated by plastic repair.

Coloboma of the Eyelid

- A notch at the edge of the lid.
- Most commonly in the middle of the upper lid (Figs 11.2A and B).
- Often associated with underlying dermoid cyst.
- ***Treatment*** is by plastic repair at a very early age to prevent exposure keratitis.

Blepharophimosis (Fig. 11.3)

- ***It is a syndrome consists of following clinical features:***
  - Narrowing of the vertical and horizontal palpebral apertures
  - Telecanthus
  - Inverse epicanthus fold and
  - Ptosis.
- Autosomal dominant inheritance.
- ***Treatment:*** Plastic reconstruction of the lids, along with bilateral brow suspension for the ptosis in stages.

Distichiasis (Figs 11.4A and B)

Extra posterior row of cilia, occasionally present in all four lids:



Fig. 11.3: Blepharophimosis syndrome

- This posterior row occupies the position of meibomian glands.
- Meibomian glands change to ordinary sebaceous glands in this condition.
- The eyelashes may irritate the cornea.
- ***Treatment*** is done by cryotherapy or excision with grafting.

ABNORMALITIES OF SHAPE AND POSITION (FIG. 11.5)

Entropion

Entropion is an inward turning of the eyelid with rubbing of the eyelashes on the conjunctiva and/or cornea.

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A B

Figs 11.4A and B: Distichiasis of the upper lid. **A.** Schematic representation; **B.** Photograph

Fig. 11.5: Abnormalities of the lid margin

A B

Figs 11.6A and B: A. Entropion of the lower eyelid (schematic representation); **B.** Senile entropion—left lower lid (photograph)

Classification

Involutional (Figs 11.6A and B)

Most common type and affects the lower lid only.

Etiopathology: Due to four changes:

1. Upward movement of preseptal part of orbicularis oculi of the lower lid.
2. A thinning of the tarsal plate with subsequent atrophy—leading to horizontal lid laxity.
3. Thinning of the orbital septum and weakening of the lower lid retractors—lead to decrease in vertical lid stability.
4. A relative disparity between lid and globe (enophthalmos) from atrophy of the adipose tissue.

Symptoms

- Foreign body sensation
- Pain
- Lacrimation and discharge.

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Signs
- Inturning of the lower eyelid
- Conjunctival congestion
- Discharge with matting of the eyelashes
- Blepharospasm
- Superficial corneal opacities and distortion of the window reflex
- Sometimes, corneal ulceration.

Treatment: Two types of procedures are:
1. ***Temporary procedures***
   - ***Adhesive tape***—pulling the skin outwards with a strip of adhesive tape.
   - ***Cautery***—over the skin below the lashes.
   - Transverse lid everting suture.
   - ***Alcohol injection***—along the edge of the lid.
2. ***Permanent procedures***
   - ***Weis' procedure:*** A full-thickness horizontal lid splitting with marginal rotation.
   - ***Horizontal lid shortening:*** An excision of full-thickness trapezoid-shaped area of the lid at the lateral canthus and then suturing the margins, to treat horizontal lid laxity.
   - ***Tucking of inferior lid retractors (Fig. 11.7):*** It may be done as a primary procedure, or in recurrent cases.
   - ***Fox procedure (Fig. 11.8):*** Excising a base-down triangle of the tarsus and conjunctiva, and then sutured together.

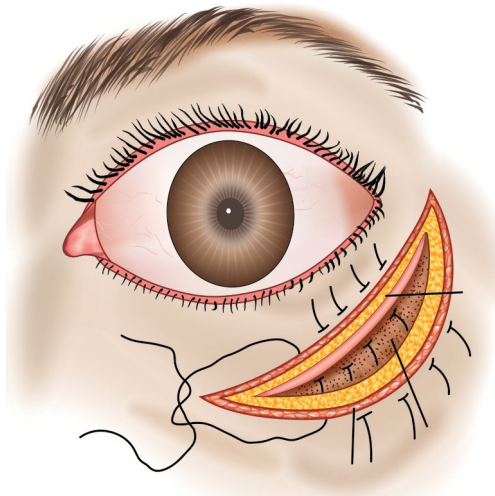


Fig. 11.7: Tucking of inferior lid retractors

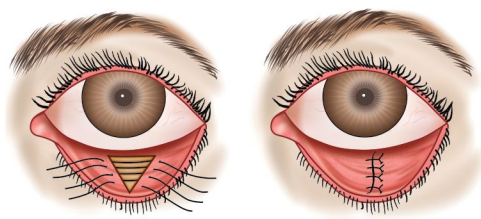


Fig. 11.8: Fox procedure

Cicatricial

It is due to scarring of the palpebral conjunctiva. It may involve both the upper and lower lids. Frequently, the tarsus is deformed and thickened.

Causes
- Chemical injuries
- Lacerated injuries
- Trachoma
- Radiation
- Stevens-Johnson syndrome
- Ocular cicatricial pemphigoid.

Treatment: Aim of the treatment, is to keep the eyelashes away from the globe.
- Soft contact lens.
- Various plastic operations, and the ideas are:
  - To alter the direction of the lashes,
  - To transplant the lashes and
  - To straighten the distorted tarsus.
- Mucous membrane grafting (from buccal mucosa) to replace the scarred conjunctiva.

Acute Spastic

It results from excessive contraction of the orbicularis oculi (blepharospasm). It affects mainly the lower lid.

Causes
- Chronic conjunctivitis
- Keratitis
- Postoperative.

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Fig. 11.9: Congenital entropion—right lower lid

Treatment

- Removal of the cause and it resolves spontaneously.
- Removal of bandage in postoperative cases.
- ***Temporary relief by***—
  - Lid everting suture
  - Adhesive tape (lid taping).

Congenital (Fig. 11.9)

It is rare and usually caused by deformity of the tarsal plate. It may be associated with microphthalmos or anophthalmos.

Sometimes, epiblepharon may occur with congenital entropion. It is an abnormal skin-fold usually at the medial one-third of lower lid, and turns the lid margin inward.

Treatment

- Resection of the abnormal portion of the tarsus.
- Excess skin may be excised if there is epiblepharon.

Ectropion

It is an outward turning of the eyelid away from the globe.

Clinical Features

- In case of lower lid involvement → inferior punctum is not in contact with the globe → epiphora and excoriation of the skin around the lid.
- Chronic exposure of the conjunctiva → secondary infection and keratinization → keratitis or frank corneal ulcer.

Classification

Involutional (Senile) (Figs 11.10A and B)

It is the most common form, which affects the lower lid in elderly. It is due to excessive horizontal lid-length with weakness of the preseptal portion of the orbicularis.

The lid laxity can be assessed with the ***snap test***. Pull the lower eyelid downward and assess how long it takes to return to a normal position. A return of less than 1 second (without blinking) is normal.

Treatment: It is corrected by reducing the horizontal lid laxity:

A **B**

Figs 11.10A and B: A. Ectropion of the lower eyelid (schematic representation); **B.** Senile ectropion—left lower lid (photograph)

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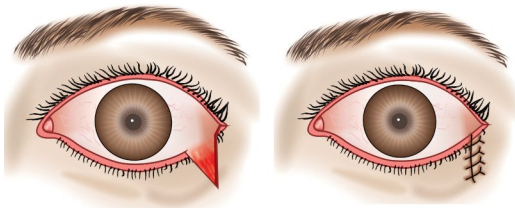


Fig. 11.11: Bick's procedure

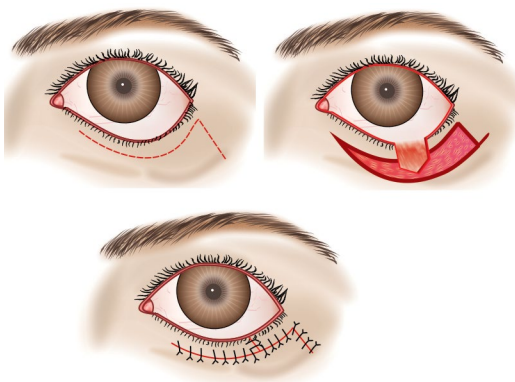


Fig. 11.12: Modified Kuhnt-Szymanowski's procedure

- ***Zeigler's cautery*** to correct the medial lid laxity with punctal eversion.
- ***Medial conjunctivoplasty*** for mild cases of medial ectropion.
- ***Horizontal lid shortening*** to correct ectropion involving the whole lid.
- ***Bick's procedure (Fig. 11.11)*** excision of a full-thickness triangular wedge of lid at the outer canthus and then suture vertically.
- ***Byron-Smith modification of Kuhnt-Szymanowski procedure (Fig. 11.12):*** Pentagonal wedge resection of the lid margin, along with excision of a triangular skin flap laterally.

Cicatricial

It is caused by contracture of the skin and underlying tissues.



Fig. 11.13: Cicatricial ectropion of the right lower lid after chemical injury (arrow)

Causes

- Burns (chemical/thermal) (Fig. 11.13)
- Trauma
- Inflammation.

It affects either the upper or lower lid.

Treatment

- Excision of the scar with a skin-graft to the raw area. Skin of the opposite upper eyelid is ideal for this purpose.
- Lengthening of the vertical-shortening of the lid—by Z-plasty.

Paralytic

It follows the paralysis of the orbicularis and is also associated lagophthalmos.

Treatment: The main aim is to prevent exposure keratitis.

- ***In mild cases***
  - Frequent instillation of artificial tears—to prevent corneal drying.
  - Antibiotic ointment, or an adhesive tape to close the lid at night to prevent corneal exposure.
- ***In severe cases***
  - ***Tarsorrhaphy:*** Shortening of the palpebral aperture by lateral tarsorrhaphy, while awaiting any spontaneous recovery.
  - ***Lateral canthoplasty*** is more acceptable cosmetically.
  - Correction by silicone-slings.

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Congenital

Rare, and may be associated with blepharophimosis. In severe cases—surgery is necessary.

Mechanical

Just the sequel to a swelling of the lower lid, e.g. a large chalazion, a tumor, or even lid edema. It can be easily rectified.

Trichiasis (Figs 11.14A and B)

Trichiasis is the inward misdirection of the eyelash(es) which irritate the cornea and/or the conjunctiva.

Pseudotrichiasis: When the misdirection is secondary to entropion.

Etiology

- ***Congenital***—known as ***distichiasis***.
- ***Acquired***—due to diseases those cause scarring of the eyelid margin, e.g. stye, ulcerative blepharitis, membranous conjunctivitis, trachoma and post-traumatic.

Symptoms

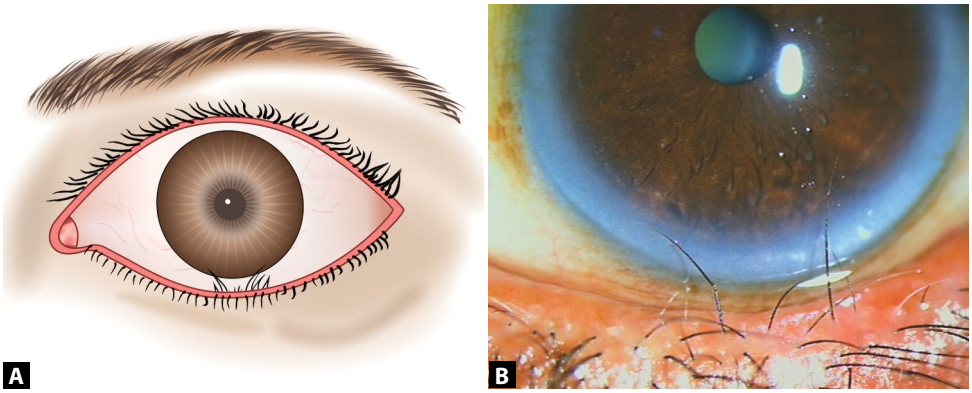
- Foreign body sensation
- Lacrimation and pain.

Signs

- Conjunctival congestion
- Reflex blepharospasm
- Ciliary congestion
- Recurrent erosions of the cornea
- Superficial corneal opacities
- Vascularization of the cornea.

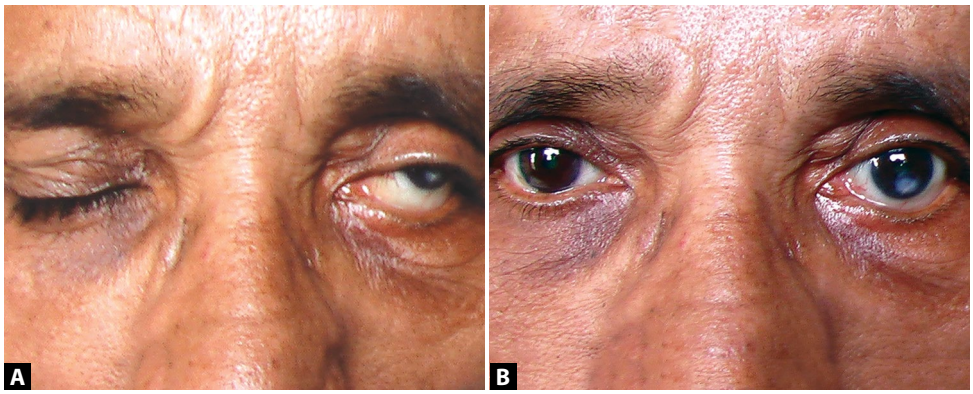
Treatment

- ***Epilation*****:** Removal of the offending eyelashes with cilia forceps. This must be repeated every 6–8 weeks.
- ***Soft bandage contact lens:*** To protect the cornea temporarily.
- ***Permanent procedures:*** By destroying the hair roots.
  - ***Electrolysis*** (under local anesthesia): A fine needle (negative pole) is introduced into the hair follicle, and a current of 2 mAmp is passed. End point is judged by the appearance of foam, and the eyelash with bulbus root can be easily lifted out.
  - ***Electrodiathermy:*** A current of 30 mAmp is used for 10 seconds.
  - ***Cryotherapy:*** Very effective for a row of ingrowing eyelashes. Under local anesthesia, the cryoprobe is applied to lid margin and then freeze at –20°C → prolonged thawing → re-freeze. Depigmentation of the skin is a problem.
  - ***Irradiation*** is effective in severe cases, but it produces conjunctival keratinization.
  - ***Argon laser cilia ablation*** is also effective.



Figs 11.14A and B: Trichiasis. **A.** Schematic representation; **B.** Photograph

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Figs 11.15A and B: Lagophthalmos with healed exposure keratitis in the left eye

- ***Plastic repair:*** If many cilia are misdirected, operative procedure as entropion is most effective.

Lagophthalmos (Figs 11.15A and B)

This is the condition of inadequate closure of the eyelids, resulting in exposure of the eye.

Literally *lagos* is a Greek word for hare, an animal which always sleeps with its eyes open.

Etiology nocturnal lagophthalmos: It is found in children, in Mongolian races, and terminal ill patient. It causes no trouble, since the eyes roll upwards (Bell's phenomenon) spontaneously during sleep.

Pathological

- Facial palsy
- Proptosis and thyroid exophthalmos
- Comatose patient
- Cicatricial deformity of the upper lid.

Sequelae

- Eye is red, irritable and watery
- Dryness of the lower part of the bulbar conjunctiva and cornea.
- Exposure keratitis → corneal ulceration → corneal perforation.

Treatment

- ***Nocturnal lagophthalmos*** does not require any treatment.
- Instillation of artificial tear at day time, and antibiotic ointment at night are required to prevent corneal drying.
- Closure of the lids by adhesive tapes.
- Soft bandage contact lens along with artificial tears to prevent exposure keratitis.
- ***Tarsorrhaphy:*** A temporary or permanent adhesion is created between upper and lower lids which may be lateral or paracentral.
- ***Lid (upper) load operation*** with gold plate—useful in facial palsy.
- Treatment of the cause—as in proptosis due to orbital tumor or thyroid exophthalmos.

Symblepharon (Figs 11.16A to C)

This is a condition of adhesion of the lid to the globe, as a result of adhesion between the bulbar and palpebral conjunctiva.

Etiology: Any cause which produces raw surfaces upon two opposed areas of bulbar and palpebral conjunctiva will lead to adhesion during the healing process.

- Chemical burns (mostly alkali)
- Thermal burns
- Membranous conjunctivitis
- Ocular pemphigoid
- Stevens-Johnson syndrome
- Postoperative
- Trachoma.

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Figs 11.16A to C: Symblepharon. **A.** Schematic representation; **B and C.** Photographs (arrows)

Pathology

- Bands of fibrous tissue are formed, and stretching between the lid and the globe.
- The bands may be narrow or broad.
- Cornea is also involved in severe cases.

Types: There are three types of symblepharon are as follows:

1. ***Anterior symblepharon:*** Bands are limited to the anterior parts and not involving the fornix.
2. ***Posterior symblepharon:*** Bands are obliterating the fornix only.
3. ***Total symblepharon:*** The lids are completely plastered against the globe and leaving a small fixed palpebral aperture.

Symptoms

- ***Pain and redness***—due to exposure.
- ***Watering***—due to inadequate lacrimal drainage.
- ***Diplopia***—due to limitation of the ocular movements resulting from pronounced adhesion.
- Cosmetic disfigurement.

Signs

- Signs of exposure
- Restriction of ocular movement
- Visible fibrotic band
- Obliteration of the fornix at places.

Treatment

- ***Prevention***—most important
  - ***Sweeping a glass rod***—well coated with ointment, around the upper and lower fornices, so that they are well packed with ointment. This procedure is to be repeated several times each day according to severity.
  - ***Scleral contact shell fitting***—to separate the two mucosal surfaces to prevent their adhesions.
- ***When established***
  - ***If there is a small band***—just excise the band.
  - ***If it is extensive***
    - Radical excision of the scarred conjunctival tissues, and
    - Mucous membrane graft to cover the bare area (mucous membrane is taken from upper fornix of opposite eye, or from the buccal mucosa).
- ***Prevention of recurrence of adhesion***
  - By therapeutic contact lens
  - By scleral shell at least for 6 weeks.
  - High dose of steroids (local and systemic) is helpful to prevent formation of excessive granulation tissues).

Blepharochalasis and dermatochalasis

Blepharochalasis (Fig. 11.17A)

- Usually bilateral
- May be unilateral or individuals (around 20 years).
- Both sexes are equally affected
- Usually starts at puberty with an intermittent, painless angioneurotic edema and redness of the lid
- Aggravated by crying and menstruation

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- Initially, there is loss of skin elasticity with subcutaneous atrophy
- Ultimately, skin hangs down over the lid margin
- Orbital fat and lacrimal gland may prolapse
- Cosmetic surgery may be helpful.

Dermatochalasis (Figs 11.17B and C)

- Usually bilateral
- Mostly senile; in younger individual a familial tendency is noted
- Loss of skin elasticity
- Skin atrophy is seen with prolapse of fat, mainly nasally in the upper lid
- Later, lower lid is also involved with bagginess and sac-like bulging of fat (herniation) in the whole lid
- This is due to relaxation and defect in orbital fascia
- Cosmetic surgery is useful, but a recurrence is common.

Floppy Eyelid Syndrome

It is a condition characterized by floppy, easily eversible upper eyelid with papillary conjunctivitis at the upper tarsus.

Symptoms

- Chronically red and irritable eye, often worse on awaking from sleep.
- A mild mucus discharge.
- The patients are typically obese and often with sleep apnea syndrome.

The symptoms are thought to be result from spontaneous eversion of upper eyelid during sleep. This allows the superior palpebral conjunctiva to rub against a pillow or bedsheet.

Signs

- Upper eye lid can be easily everted without an accessory device (finger or cotton tip applicator).
- Soft and rubbery superior tarsal plate.
- Papillary reaction of superior tarsal conjunctiva.
- Superficial punctate keratitis (SPKs).

Differential Diagnosis

The following conditions may produce superior tarsal papillary conjunctivitis, but in all cases, the upper lid is not easily eversible.

- ***Vernal conjunctivitis*** seasonal, itching, ropy discharge and papillary reaction of upper tarsal conjunctiva.
- ***Giant papillary conjunctivitis*** most often related to soft contact lens wear and exposed nylon suture.
- ***Superior limbic keratoconjunctivitis*** hyperemia and thickening of upper bulbar conjunctiva, often with filaments and corneal pannus.
- ***Toxic keratoconjunctivitis*** follicles and/or papillae are more prominent in lower tarsal conjunctiva in a patient using eye drops.



Figs 11.17A to C: A. Blepharochalasis in left eye; **B and C.** Dermatochalasis in both eyes

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Treatment

- Topical antibiotics, e.g. ciprofloxacin eye drop—4 times daily.
- Artificial tears—1–2 hourly in presence of SPKs.
- Antibiotic ointment at bed time.
- The eyelids are taped during sleep, or alternately an eye shield is worn to protect eyelids from rubbing against a pillow/bedsheet. The patient is advised to refrain from sleeping face down.
- An eyelid tightening surgical procedure is often helpful.

Ptosis (Blepharoptosis) (Fig. 11.18)

It signifies a drooping of the upper eyelid, which may be unilateral or bilateral, and partial or complete.



Fig. 11.18: Right sided ptosis

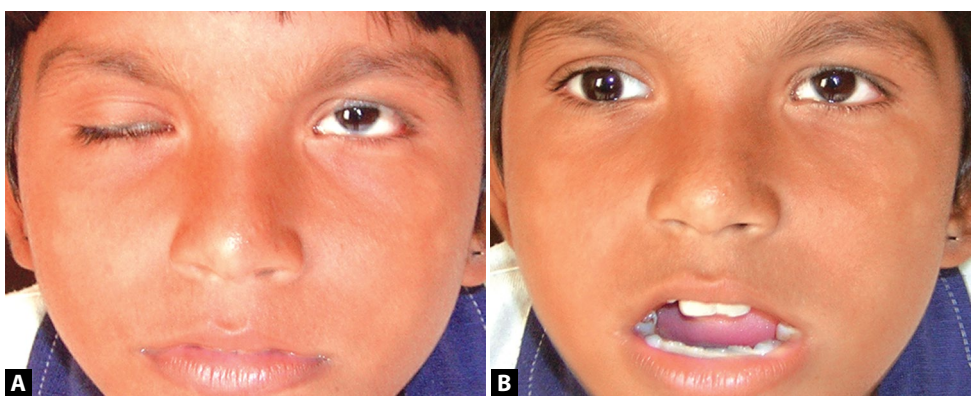
Etiology

Congenital Ptosis

- Due to imperfect differentiation of the levator muscle.
- Often associated with weakness of underlying superior rectus muscle (as levator muscle together with superior rectus, is the last extraocular muscle to be differentiated).
- Often hereditary (dominant).
- May be associated with epicanthus or blepharophimosis.
- May be associated with synkinesis (synkinetic ptosis).
- ***Marcus-Gunn jaw-winking phenomenon (Figs 11.19A and B):*** There is retraction of the ptotic eyelid, with stimulation of ipsilateral pterygoid muscle (jaw-movement).
- ***Misdirected third nerve:*** Retraction of the upper lid with various ocular movements.

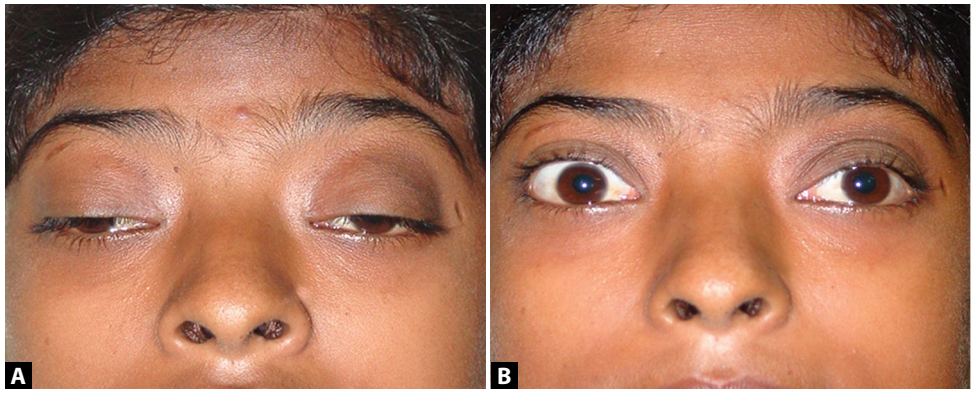
Acquired Ptosis

- ***Neurogenic***
  - Partial or complete third nerve palsy
  - Horner's syndrome.
- ***Myogenic***
  - Myasthenia gravis (Figs 11.20A and B)
  - Ocular myopathy
  - Bilateral senile ptosis (Fig. 11.21).



Figs 11.19A and B: Ptosis with Marcus-Gunn jaw-winking phenomenon

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Figs 11.20A and B: Myasthania gravis. **A.** Before prostigmin test; **B.** After prostigmin test



Fig. 11.21: Bilateral senile ptosis

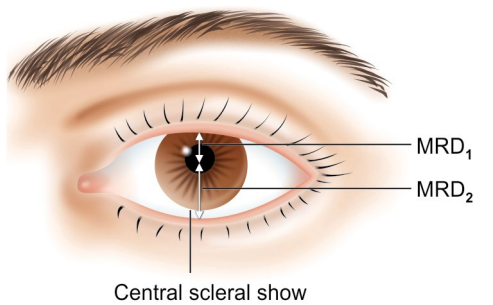


Fig. 11.22: Measurement of ptosis
MRD—Marginal reflex distance

- ***Mechanical***
  - Excess of weight due to edema, tumors, large chalazion, etc.
  - Conjunctival scarring
  - Symblepharon of the upper lid.
- ***Traumatic:***
  - Trauma to the levator muscle
  - Post-surgical (e.g. after cataract surgery).

Pseudoptosis

- Due to surgical anophthalmos, microphthalmos and phthisis bulbi
- Due to hypotropia
- Due to dermatochalasis.

Clinical Evaluation of Ptosis

History

- Age of onset
- Family history
- Presence of diplopia
- Variability of ptosis
- Symptoms of systemic problems
- Any contributing factor.

Examination

- ***Amount of ptosis:*** By noting the ptotic lid margin with respect to the limbus and pupil (Fig. 11.22).
  - Mild ptosis = 2 mm
  - Moderate ptosis = 3 mm
  - Severe ptosis = 4 mm or more.
- ***Ptosis measurements***
  - ***Margin reflex distance (Fig. 11.23):*** Margin reflex distance 1 (MRD 1) is the distance in mm from the light reflex on the patient's cornea to the level of the

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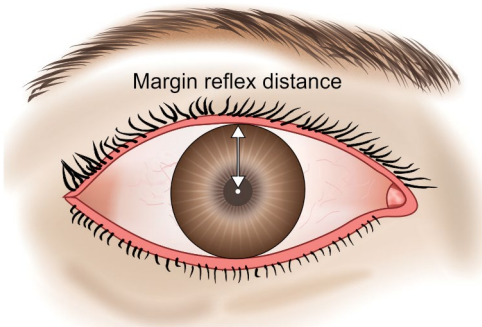


Fig. 11.23: Measurement of margin reflex distance

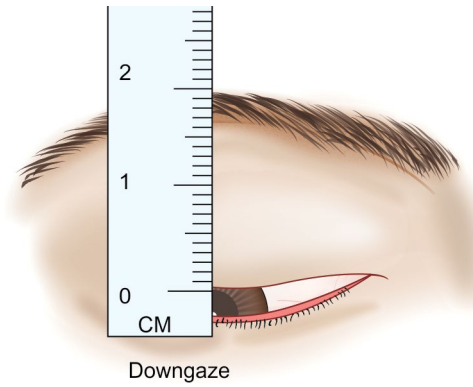


Fig. 11.24: Margin crease distance (MCD)

center of the upper lid margin with the patient seeing in primary gaze.
- Eyes of examiner and patient are at the same level. Light held in examiner's hand is directed towards the glabella of the patient
- If the reflex is not seen, then the number of mm of eyelid raised is then recorded as MRD 1 (negative value)
- The amount of ptosis in unilateral cases is the difference between MRD 1 on ptotic side and that of normal side.
- In bilateral cases, the MRD1 is subtracted from normal MRD 1 of 4–4.5.

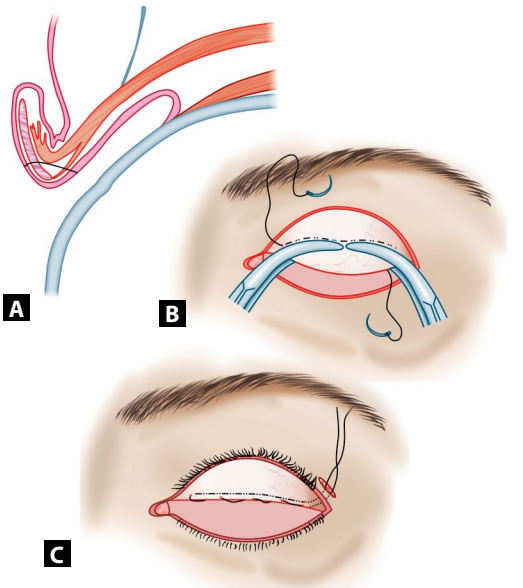
- ***MRD 2*** is the measurement of the corneal light reflex to the center of the lower lid with the patient seeing in primary gaze. A measurement greater than 5 mm is considered normal. Sum of MRD 1 and MRD 2 is equal to the palpebral fissure width.
- ***MRD 3*** is the distance of the ocular light reflex to the center of the upper lid margin when the patient looks in extreme up gaze. Generally MRD 3 is equal to MRD 1. But in associated superior rectus weakness/palsy—MRD 3 is more than MRD 1.
  - ***Margin limbal distance or MLD*** is the distance from the 6 o'clock limbus to the center of the upper lid margin when the patient looks in extreme up gaze. It is about 9.0 mm. The difference in MLD between two sides in unilateral cases, or the difference with normal in bilateral cases multiplied by 3 would give the amount of levator resection required.
- ***Margin crease distance or MCD*** is the distance from the upper eyelid margin to the lid crease measured in downgaze. In women, a normal central MCD measurement is about 8–10 mm, and in men it is about 5–7 mm. It helps in planning the surgical incision. When more than one lid creases are present, the most prominent one is to be considered (Fig. 11.24).
- ***Palpebral fissure height*** or ***PFH***—the distance between the upper and lower eyelid in vertical alignment with the pupillary center.
  - Normal = women: 9–10 mm and in men: 7–10 mm in primary gaze
  - Upper lid: 2 mm below superior limbus. Lower lid: 1 mm above the inferior limbus
  - Should be examined in primary gaze, up gaze and downgaze
  - Amount of ptosis = difference in palpebral apertures in unilateral ptosis

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or difference from normal in bilateral ptosis
  - Ptotic lid in unilateral ptosis is usually higher in downgaze due to failure of levator to relax
  - Ptotic lid in acquired ptosis is invariably lower than normal lid in downgaze.
- ***Assessment of levator function***
  - The brow is immobilized by pressure with the thumb (to negate the action of frontalis).
  - Patient is asked to look down and then to look up.
  - Amount of excursion of the upper lid margin is then measured with a ruler (2 mm of movement is contributed by the superior rectus muscle).
    - Normal = 15 mm
    - Good = 8 mm or more
    - Fair = 5–7 mm
    - Poor = 4 mm or less
- ***Ocular motility testing***
- ***Jaw-winking phenomenon***
- ***Bell's phenomenon***
- ***Corneal sensitivity in neurogenic ptosis***
  - ***Photograph:*** As pre-operative record
  - ***Tensilon test*** is to exclude ***myasthenia gravis***. Improvement of ptosis with intravenous injection of edrophonium (Tensilon), or prostigmin if the ptosis is due to myasthenia (Fig. 11.20)
  - ***Neurological evaluation*** if the ptosis is neurogenic.

Treatment

- ***Fasanella-Servat operation (Figs 11.25A to C)***
  - It is simple tarsoconjunctival resection.
  - Useful in mild ptosis with good levator function (e.g. Horner's syndrome).
- ***Levator resection***
  - Useful in congenital unilateral ptosis with fair to good levator function.



Figs 11.25A to C: Fasanella-Servat operation. **A.** Resection of upper tarsal border with its attached Muller's muscle and conjunctiva; **B.** Upper tarsal border is caught between two mosquito forceps; **C.** Wound closure after resection of lid tissues within the forceps

  - It may be via
    - ***Skin approach (Everbusch's)*** especially where larger resection is necessary
    - ***Conjunctival approach (Blaskowics')*** particularly useful for moderate resection of superioris (LPS).
- ***Brow (frontalis) suspension (Fig. 11.26)***
  - In bilateral cases where the levator action is poor.
  - Here, the tarsus is fixed to the frontalis muscle via a sling of fascia lata or non-absorbable materials.
- ***Aponeurosis strengthening***
  - Useful for acquired ptosis with good levator function.
  - Performed either by advancement or by tucking.
  - Advancement may be combined with levator resection in severe ptosis.

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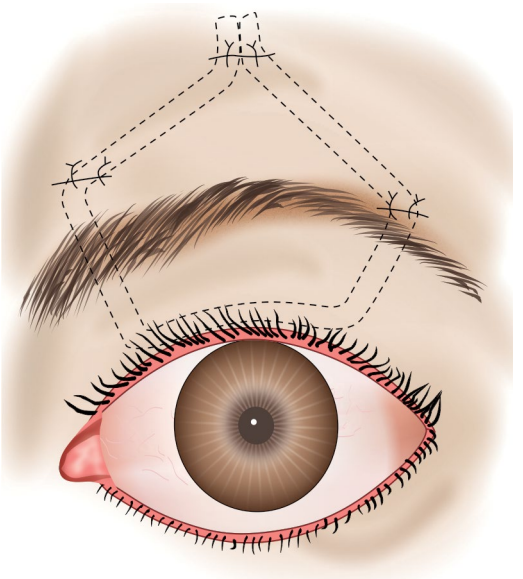


Fig. 11.26: Frontalis (brow) suspension

Timing of surgery in congenital ptosis

- ***Severe ptosis:*** Early intervention is necessary due to danger of stimulus deprivation amblyopia.
- ***Mild to moderate ptosis:*** Surgical correction is done between 3 and 4 years of age, when accurate measurement can be obtained.

Blinking

Blinking spreads tears over the surface of the eye, and limits the amount of light entering the eye. It may be:

- ***Involuntary:*** Once in every 5 seconds and lasts about 0.3 second. It is absent in neonates.
- ***Voluntary (winking):*** It is generally unioocular, and used to illustrate a variety of emotions.
- ***Reflex:*** It may follow with the peripheral stimulation of trigeminal (sensory blinking), optic (optical blinking) or auditory nerve.

Blepharospasm

It is an involuntary, tonic, spasmodic, bilateral contraction of the orbicularis oculi muscles. It lasts for few seconds to several minutes.



Fig. 11.27: Reflex blepharospasm (left eye)

Causes

- ***Essential blepharospasm:*** It may occur spontaneously in old age. Other muscles of the face may be simultaneously involved.
- ***Reflex blepharospasm (Fig. 11.27)*** is precipitated by sensory stimuli, e.g.
  - Corneal abrasions
  - Keratitis or corneal ulcer
  - Dust, fumes, or chemical irritation.

  Reflex blepharospasm can be abolished by topical anesthesia.

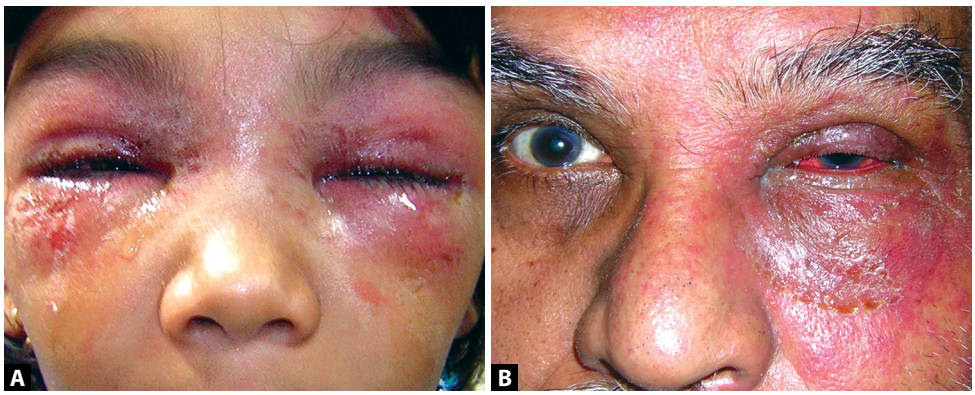
Treatment

- ***Essential blepharospasm*** is difficult to treat.
  - Alcohol injection to the facial nerve may be helpful.
  - Botulinum toxin (Botox) injection into the orbicularis muscle around the eyelid in severe cases.
- ***Reflex blepharospasm:*** Treatment of the cause.

Contact (Irritant) Dermatitis (Figs 11.28A and B)

It is characterized by a frequently recurrent, weeping eczematous lesion of the skin, associated with severe itching and swelling of the lids

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Figs 11.28A and B: A. Contact dermatitis-atropine induced; **B.** Left-sided contact dermatitis

Mostly allergic in nature, and principally dependent on external irritants, e.g.

- Cosmetics (most important), hair dye
- Topical drugs, e.g. neomycin, penicillin, and atropine are most important (Fig. 11.28A and B)
- Industrial chemicals
- ***Spectacle dermatitis*** caused by nickel or plastic frame.

Treatment

- Removal of all possible external allergens.
- Antihistamine tablet.
- Local application of corticosteroids ointment.

Blepharitis

Blepharitis is the subacute or chronic inflammation of the eyelid.

Etiological factors are complex:

- Age—mostly in children
- Usually bilateral
- Irritation—from cosmetics, dust, smoke
- Uncorrected refractive errors (rubbing of the eyes to gain clear vision → chronic infection)
- Seborrhea (dandruff) of the scalp—mainly in squamous variety
- Chronic conjunctivitis
- ***Parasitic infestation:*** 'Blepharitis acarica' due to *Demodex folliculorum,* and 'phthiriasis palpebrum' (Fig. 11.29) due to crablouse, *Phthirus pubis.*

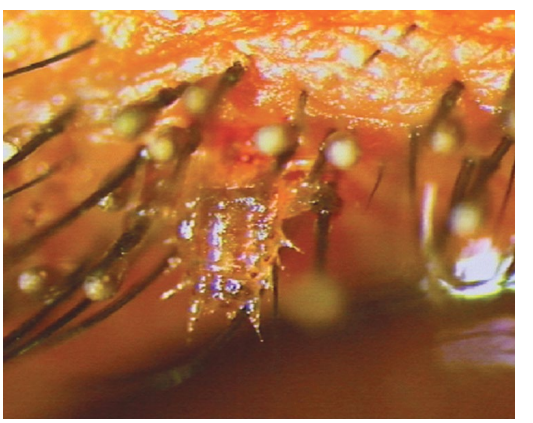


Fig. 11.29: Phthiriasis palpebrum

Types of Blepharitis

Two types of blepharitis are as follows:

1. ***Squamous blepharitis (Fig. 11.30):*** It is characterized by hyperemia, usually limited to the eyelid margins. It is not an infective condition.
   Symptoms
   - Redness of the eyelid margins
   - Burning and discomfort of the eyes
   - Epiphora.

   Signs
   - White dandruff-like scales on the lid margins

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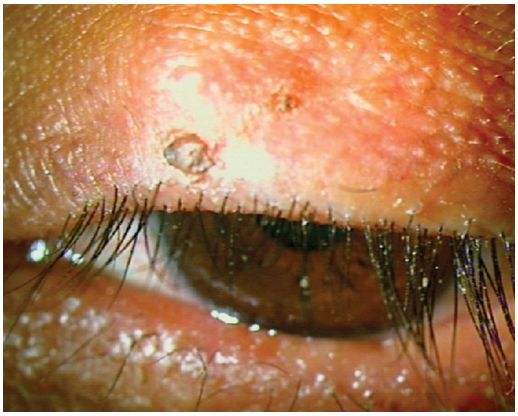


Fig. 11.30: Squamous blepharitis

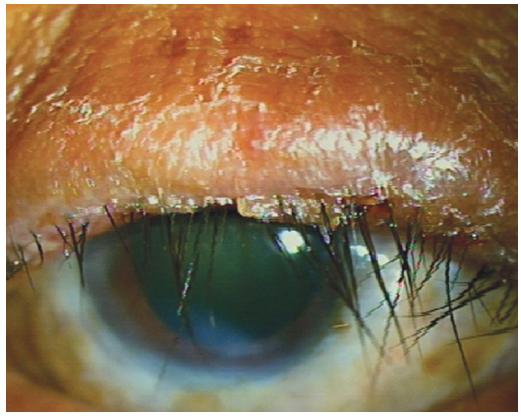


Fig. 11.31: Ulcerative blepharitis

- On removal of scales, underlying surface is found hyperemic, but no ulceration
- Falling of the eyelashes (madarosis)
- Thickening of the lid margins (tylosis)
- Associated seborrheic dermatitis of the scalp.

2. ***Ulcerative blepharitis (Fig. 11.31):*** It is caused by acute or chronic suppurative inflammation of the follicles of the eyelashes and glands of Zeis and Moll.
   Stahylococcus aureus is usually the causative agent superimposed on predisposing conditions.
   Symptoms
   - Itching, lacrimation and redness
   - Soreness of the lid margins
   - Loss of eyelashes.

   Signs
   - Yellow crusts are deposited at the roots of the eyelashes with matting.
   - The crusts are removed with difficulty, and bring eyelashes with it. On removing the crusts, small ulcers are seen around the bases of the eyelash, which bleed easily.
   - Loss of cilia with distortion of the eyelid margins, or misdirected cilia.

Sequelae of blepharitis: These are mainly seen in ulcerative form of blepharitis, and often serious.

- Chronic conjunctivitis
- ***Madarosis:*** Loss of eyelashes due to destruction of the follicles by deep ulceration.
- ***Trichiasis:*** When the ulcer heals, the cicatricial tissue contracts with misdirection of the remaining cilia.
- ***Tylosis:*** Hypertrophy of the lid margins with consequent drooping.
- ***Ectropion*** of the lower lid with epiphora.
- ***Marginal keratitis***—particularly seen in lower third of the cornea.
- ***Instability of the tear-film*** with consequent dry eye.

Treatment

Squamous blepharitis

- Mainly, treatment of seborrheic dermatitis of the scalp by means of specific shampoo
- ***Lid hygiene:*** Scales on eyelid margins is removed by a moistened cotton-tip applicator twice daily
- Antibiotic-steroid eye ointment is applied locally, twice daily for 2–4 weeks.

Ulcerative blepharitis

- ***Local treatment***
  - ***Lid scrub:*** Warm sodium bicarbonate lotion (3%) is applied to the lid margin to soak the crusts. Alternately, a baby shampoo or 0.1% selenium sulphide solution may be used. Then removal of the crusts by a cotton-tip applicator.

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- Epilation of loose and diseased eyelashes.
- Antibiotic-steroid ointment is to be applied by rubbing the lid margins 3 times daily.
- Antibiotic drop or ointment is to be added. Chloramphenicol and ciprofloxacin are most effective against *Staphylococcus aureus*. Neomycin is best avoided because of high incidence of allergic reaction. If associated with dry eyes, use artificial tears 4 to 8 times daily.

- ***Systemic therapy:*** Systemic tetracycline or doxycycline for two weeks is useful in severe form of ulcerative blepharitis. It is better avoided in children due to dental complications.
- ***General treatment:*** Ocular hygiene and handwashing
  - Avoid hair-oil, kajal or other ocular cosmetics
  - Correction of refractive errors
  - Treatment of seborrheic capitis by anti-dandruff shampoo
  - Treatment of louse infestation.

Meibomianitis (Figs 11.32A and B)

It is a chronic infection of the meibomian glands, especially occurs in the middle-age.

It is mostly due to Staphylococcal infection, but may also occur in trachoma.

Symptoms

- Watering
- Frothy discharge—mainly at the canthi
- Foreign body sensation.

Signs

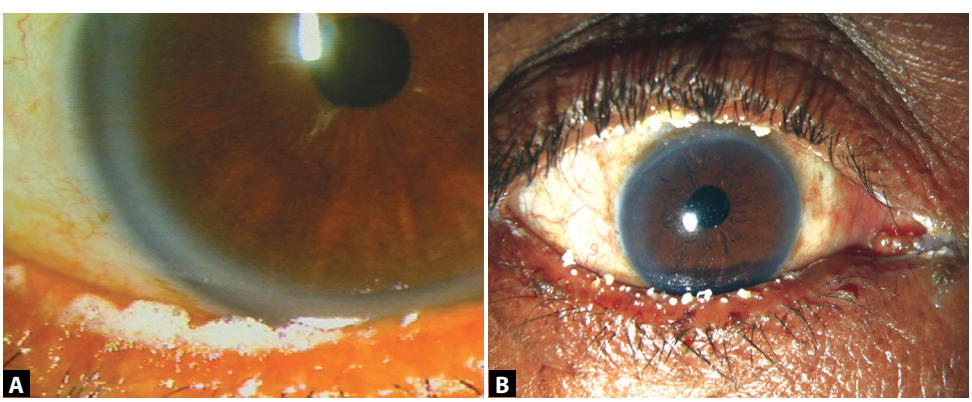
- A white, frothy secretion (seborrhea) (Fig. 11.32A) on the eyelid margins and at the canthi.
- On pressing the lid margin—meibomian secretion is expressed as tooth paste (tooth paste sign) (Fig. 11.32B).
- On eversion of the eyelids—vertical yellowish streaks shining through the conjunctiva are seen.
- Associated features of blepharitis or chalazion may be seen.

Treatment

- Hot compress
- Tarsal massage (vertical lid-massage), and removal of secretion with a moist cotton applicator
- Systemic doxycycline; and steroid-antibiotic ointment 2–4 times daily.

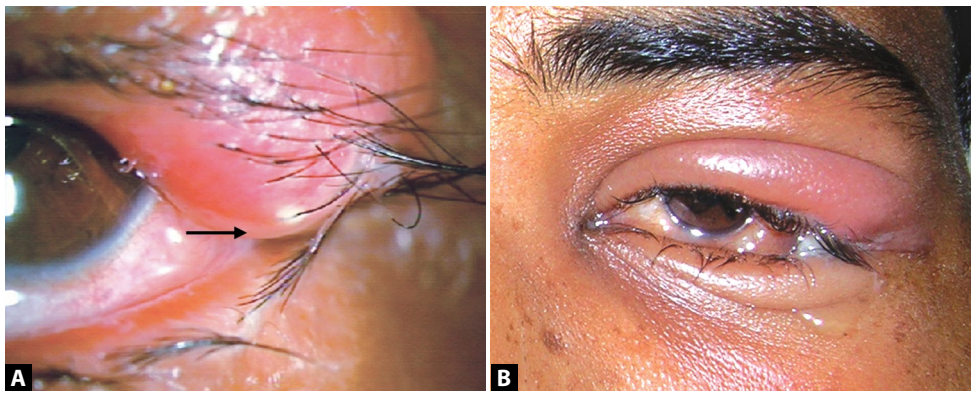
External Hordeolum (Stye) (Figs 11.33A and B)

It is an acute suppurative inflammation of the follicle of an eyelash, or associated gland of Zeis or Moll.



Figs 11.32A and B: A. Meibomian seborrhea; **B.** Meibomianitis (tooth paste sign)

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Figs 11.33A and B: External hordeolum. **A.** Stye (arrow); **B.** Preseptal cellulitis

Etiology

- Causative organism is usually *Staphylococcus aureus.*
- Common in children and young adults, but may occur at any age.
- Low general resistance as in debility or diabetes.
- Uncorrected refractive errors.
- Associated with boils, acne of the face or neck (stye in crops).

Symptoms

- Acute pain and swelling of the lid margin.
- Sense of heaviness and discharge.

Signs

- Redness and edema of the affected lid.
- Local temperature is raised.
- A swollen area at the lid margin, and it has a whitish, round, raised pus point in relation to the root of a cilium.
- The swelling is tender.
- Matting of eyelashes may be present.
- Enlargement of the pre-auricular or submandibular lymph node.

Complications

- Ulcerative blepharitis
- Cellulitis and lid abscess
- Rarely orbital cellulitis
- Very rarely cavernous sinus thrombosis.

Treatment

- Hot compress—3–4 times daily
- Evacuation of pus—by pulling out the affected eyelash (epilation)
- Alternately, a tiny horizontal incision may be given to drain out the pus
- Continue hot compress—3–4 times daily
- Systemic analgesics (like, lbuprofen or Aspirin) 2–3 times daily with antacids
- Local antibiotic drop (chloramphenicol or ciprofloxacin) 4–6 times daily and ointment at night
- In case of 'stye in crops'
  - Postprandial blood sugar to exclude diabetes
  - In addition to local treatment, a course of systemic tetracycline or doxycycline is needed for 7–14 days
- Correction of refractive errors if any
- Improvement of nutrition and general hygiene, especially in children.

Chalazion (Figs 11.34A and B)

It means a ***hailstone***. It is a chronic non-specific inflammatory granuloma of the meibomian gland.

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A B

Figs 11.34A and B: Chalazion. **A.** Right lower lid; **B.** Upper lid (magnified view)

Etiology

- Children and young adults
- Blepharitis
- Chronic conjunctivitis, e.g. trachoma
- Diabetes mellitus in adults
- ***Errors of refraction*** (excessive rubbing of eyes to gain clear vision → chronic inflammation → obstruction of the meibomian ducts).

Pathogenesis

Chronic inflammation of the gland by a low-virulence organism → blockage of the duct of the meibomian gland → accumulation of lipid secretion → break down of lipids into components of oleic acid → mucosal irritation → cellular infiltration with formation of giant cells → proliferation of fibroblasts → 'granuloma' formation.

Histology

- Centrally, cheesy sebaceous material.
- Surrounded by granulation tissue—having lymphocytes, epitheloid cells and foreign body type of giant cells, and fine blood vessels.
- Whole thing is enclosed by a fibrous capsule.

Symptoms

- Painless nodular swelling of the eyelid
- Drooping of the eyelid (in case of a large, or multiple chalazion).

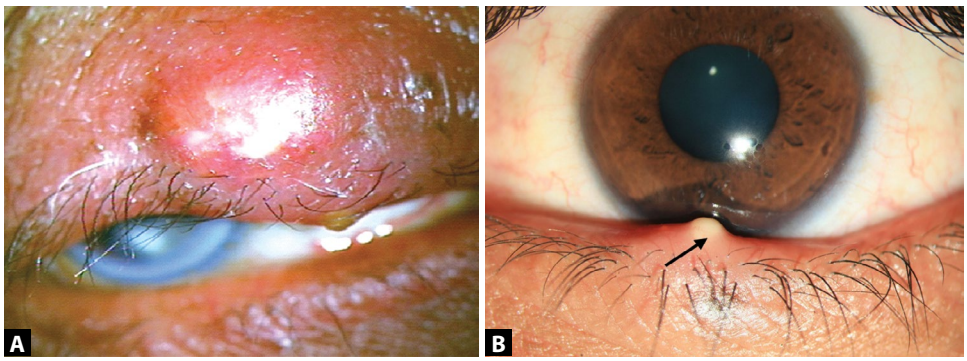
Signs

- A small pea-size nodular swelling, away from the lid margin.
- It is firm, tense and non-tender.
- No signs of inflammation.
- Skin over the swelling is normal and free from it.
- On eversion, the tarsal conjunctiva underneath the nodule is velvety-red or purple, and slightly elevated.
- Regional lymph nodes are not involved.

Course (fate) of a chalazion

- Spontaneous resolution (if it is small)
- May remain as such
- Increases in size leading to mechanical ptosis
- Secondarily infected, called ***internal hordeolum (Fig. 11.35A)***
- It can turn into ***marginal chalazion (Fig. 11.35B)***—the granulation tissue formed in the duct of the gland, projects as a reddish-grey nodule on the intermarginal strip
- It may burst either through the conjunctiva (as a fungating mass of granulation tissue), or through the skin
- It may be calcified
- Very rarely, a malignant change may occur (meibomian carcinoma), especially in old age with a history of recurrence. So, a histopathological examination is a must in such cases.

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Figs 11.35A and B: **A.** Internal hordeolum; **B.** Marginal chalazion (arrow)

Treatment

- ***In case of small chalazion***
  - Hot fomentation
  - Steroid-antibiotic ointment with lid massage for a few days.
  - Intralesional (intrachalazion) injection of depot steroids (e.g. 0.1–0.2 mL of triamcinolone, 40 mg/mL) may be helpful, especially if the chalazion is near the lacrimal punctum. A steroid injection may lead to permanent depigmentation of the skin at the injection site.
- ***In moderate to large chalazion:*** Incision and thorough curettage under local anesthesia is done as an OPD procedure.
- ***In marginal chalazion (Fig. 11.35B):*** Under local anesthesia
  - Press out the material with thumb and index finger
  - Electrocoagulation by passing 20–30 mAmp current for few seconds.
- ***In case of internal hordeolum (Fig. 11.35A):*** First, treat the acute inflammation by
  - Hot fomentation 3–4 times daily
  - Systemic analgesics with antacids
  - Local antibiotic like chloramphenicol or ciprofloxacin drop or ointment
  - Rarely, a systemic antibiotic. After the acute phase subsides, treatment is done by incision and curettage.
- ***Correction of refractive errors (if any).***

IMPORTANT LID TUMORS

Xanthelesma

- This is slightly raised, yellow plaque, most commonly found at the inner portion of the upper or lower eyelid (Fig. 11.36)
- They are often symmetrical in the two lids, and on both sides
- Most common in elderly female
- They grow slowly, and produce only a cosmetic defect
- ***Predisposing factors***
  - More commonly, it is spontaneous
  - Sometimes, associated with diabetes and excess serum cholesterol



Fig. 11.36: Xanthelesma of all four lids

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Fig. 11.37: Capillary (strawberry) hemangioma



Fig. 11.38: Port wine stain—Sturge-Weber syndrome

- ***Histologically***, it is the cutaneous deposition of lipid material, being engulfed by histocytes (foam cells).

Treatment

- Surgical excision
- Destruction by chemicals (trichloroacetic acid), diathermy or photocoagulation
- Recurrence is very common.

Hemangiomas

Cavernous hemangiomas are common in children (Fig. 11.37).

- Hemangioma often follows the distribution of the first and second divisions of the fifth nerve.
- Sometimes, it is associated with hemangioma of the choroid (may be with glaucoma), and hemangioma of the leptomeninges, called Sturge-Weber syndrome (Fig. 11.38).
- Hemangiomas appear bluish when seen through skin, and form swellings which increase in size on crying, or lowering the head.

Treatment

- Small hemangiomas may well be left alone.
- But, large hemangiomas require treatment by intralesional steroid injection, or by superficial radiotherapy.



Fig. 11.39: Basal cell carcinoma of the lower lid

Basal Cell Carcinoma (Rodent Ulcer)

Basal cell carcinoma is the most common malignant tumor of the eyelid, occuring almost twenty times more often than squamous cell carcinoma

- Lower lid is more commonly involved, specially near the inner canthus (Fig. 11.39).
- It starts as a small pimple which ulcerates, and if the scab is removed, it is found that the edges are raised (rolled out edges) and indurated.
- The ulcer spreads very slowly, and the growth extends under the skin in all directions and penetrates deeply.

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- It does not metastasize.
- The tumor arises from the basal cell of the epidermis and consists of islands of neoplastic cells (cell nests).

Treatment

- ***Surgical excision*** with a 3 mm margin outside the obvious tumor
- ***Radiotherapy:*** It is radiosensitive
- ***Cryotherapy***—for small and superficial tumors
- ***Exenteration***—if the tumor invades the globe or orbit in neglected cases.

Squamous Cell Carcinoma (Fig. 11.40)

It is the second most common malignancy of the eyelid, usually arising from pre-existing senile keratosis

- It appears as a nodule, an ulcerative lesion, or a papilloma.
- The growth rate is faster than the rodent ulcer, and it metastasizes into the regional lymph nodes.
- The diagnosis is confirmed by biopsy.

Treatment

Radical surgery with post-operative radiotherapy.

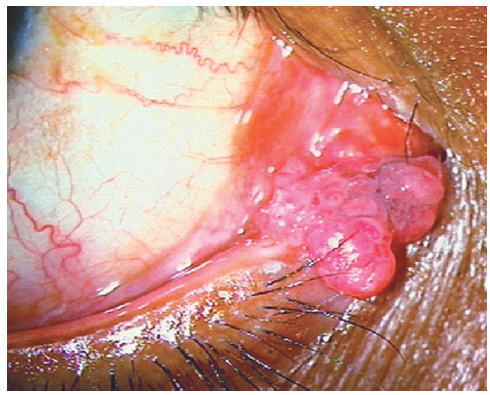


Fig. 11.40: Squamous cell carcinoma

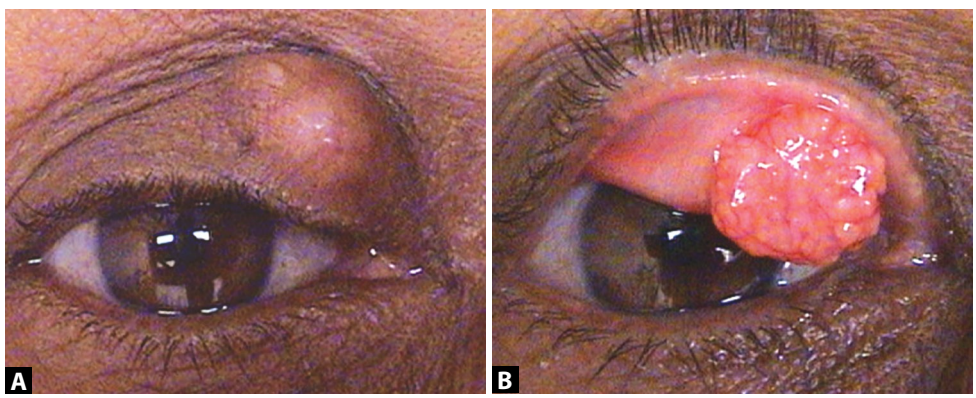
Meibomian Gland Carcinoma (Figs 11.41A and B)

It is a rare tumor arising from the Meibomian glands

- The tumor appears as a discrete, yellow, firm nodule which is sometimes incorrectly diagnosed as ***recurrent chalazion***
- Sometimes, diffuse tumor along the lid margin may be mistaken as 'chronic blepharitis,
- Widespread metastasis is commom
- Prognosis is poor in comparison to previous two tumors.

Treatment

- Radical excision with reconstruction of the lid
- Recurrence after radiotherapy is common.



Figs 11.41A and B: **A.** Meibomian carcinoma—mimicking a chalazion; **B.** After everting the lid (same patient)

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12 CHAPTER

Diseases of the Conjunctiva

CONJUNCTIVAL DISEASES

Symptoms

- Discomfort or burning sensation
- Discharge from the eyes
- Stickiness of the lids during sleep
- Itching
- Blurring of vision, or colored halos as the mucus flakes floats across the cornea
- Severe pain suggests corneal involvement
- Dryness of the eyes
- Abnormal mass or pigmentation.

Signs

- Abnormality in appearence
- Variety of discharge
- Vascular changes
- Edema (chemosis)
- Pigmentation
- Keratinization
- Hemorrhage
- Follicle formation
- Papillary hyperplasia
- Conjunctival scarring
- Associated involvement of the lids or cornea.

HYPEREMIA OF THE CONJUNCTIVA

It is the passive dilatation of the conjunctival blood vessels that occur without exudation or cellular infiltration.

Causes

- Conjunctival irritation by smoke, dust or fumes
- Exposure to wind and sun
- Following bath or swimming
- Prolonged wakefulness, and is aggravated by near work.

Symptoms

- May be absent
- A gritty foreign body sensation
- Many patients are distressed because of the redness only.

Signs

- Only redness; otherwise conjunctiva is normal.
- Temporary blanching is noted with 1 in 1000 epinephrine solution.

Treatment

- No treatment is necessary except the removal of the cause.
- Temporary relief is obtained by topical instillation of decongestant drop, containing either ***phenylephrine*** or ***naphazoline***.

CONJUNCTIVITIS

Conjunctivitis is an inflammation of the conjunctiva characterized by cellular infiltration and exudation.

Classification

Conjunctivitis can be classified as infective and allergic.

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Infective Conjunctivitis

Acute

- Catarrhal
- Serous
- Mucopurulent
- Purulent
- Membranous.

Subacute or chronic

- ***Non-specific***
  - Simple chronic
  - Angular
  - Follicular
- ***Specific***
  - Trachoma
  - Tuberculosis
  - Syphilis
  - Tularemia.

Allergic Conjunctivitis

- Simple allergic
- Phlyctenular
- Vernal (spring catarrh)
- Giant papillary.

Diagnosis

It is based on—history, clinical examination and laboratory investigations.

History

- Unilateral or bilateral
- Involvement of the other members of the family or community
- Exposure to toxins, or chemicals
- Associated symptoms—e.g. fever, pharyngitis, rhinitis, urethritis, etc.

Clinical Features

Three main features which should be considered are as follows:

1. Type of discharge,
2. Characteristics of conjunctival reaction, and
3. Preauricular lymphadenopathy.

Types of Discharge

- ***Watery discharge:*** Viral and toxic inflammation.
- ***Mucinous discharge:*** Vernal conjunctivitis and keratoconjunctivitis sicca.
- ***Purulent discharge:*** Severe acute bacterial infection.
- ***Mucopurulent discharge:*** Mild bacterial as well as chlamydial infection. It typically, gives rise to glueing up of the eyelids in the morning.

Conjunctival Reaction

It requires a good illumination and magnification. A slit-lamp examination is better.

- ***Congestion (injection):*** It is conjunctival congestion (Figs 12.1A and B) is characterized by bright-red superficial vessels which are most intense in the fornices and least at the limbus. It should be differentiated from the ciliary congestion or injection (Figs 12.2A and B) (circumcorneal) which is due to deeper diseases of the anterior segment (Table 12.1).
- ***Subconjunctival hemorrhage*** occurs in:
  - Acute hemorrhagic conjunctivitis (picornavirus) (Fig. 12.3)
  - Adenoviral conjunctivitis
  - Bacterial conjunctivitis—especially in *Pneumococcus* and *Hemophilus species*.
- ***Edema (chemosis):*** The exudation of fibrin and protein-rich fluid, through the permeable capillaries produces a translucent swelling of the conjunctiva.
  In the bulbar conjunctiva, where the attachments to the globe are lax, large quantity of the exudates cause ballooning of the conjunctiva. The palpebral conjunctiva is little affected, but the lids are often edematous.
  Chemosis (Fig. 12.4) is mainly seen in severe bacterial infection (e.g. *Gonococcus*), allergic conjunctivitis and associated orbital inflammations.
- ***Follicles (Fig. 12.5):*** They are round swellings, (0.5–2 mm in diameter and are due to localized aggregations of lymphocytes in the subepithelial adenoid layer. Each follicle

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Figs 12.1A and B: Conjunctival congestion. **A.** Schematic representation; **B.** Photograph



Figs 12.2A and B: Ciliary congestion—acute attack in angle closure glaucoma. **A.** Schematic representation; **B.** Photograph

TABLE 12.1: Difference between conjunctival and ciliary congestions

Features	Conjunctival congestion	Ciliary congestion
Appearence	Vessels superficial; bright red; mostly in the fornices and fade towards the limbus; branched dichotomously.	Vessels deep: violet or dusky-red mostly at the limbus, and fade towards the fornices; branched radially
Blood vessels	• Vessels fill up from the fornix • Branches of posterior conjunctival vessels	• Vessels fill up from the limbus • Branches of anterior ciliary vessels
Epinephrine test (1:1000)	Constricts vessels, 'whitens' the conjunctiva	No effect
Disease	Conjunctivitis	Keratitis, iridocyclitis or angle closure glaucoma

is encircled by tiny blood vessels. ***Conjunctiva of the new born is unable to produce follicles before 2–3 months of age, due to lack of adenoid layer.***

Follicles are seen in follicular conjunctivitis, trachoma, toxins or with drugs (brimonidine instead of eserine or pilocarpine).

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Fig. 12.3: Acute hemorrhagic conjunctivitis



Fig. 12.4: Chemosis of the conjunctiva

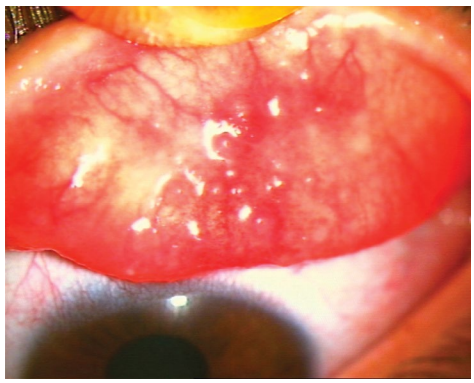


Fig. 12.5: Conjunctival follicles

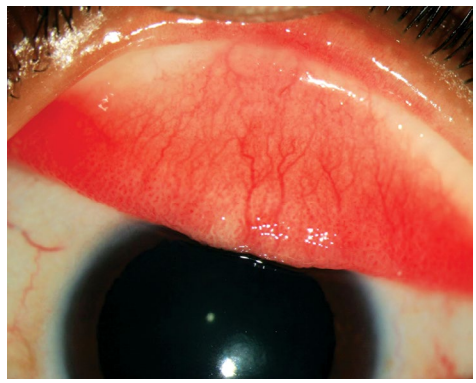


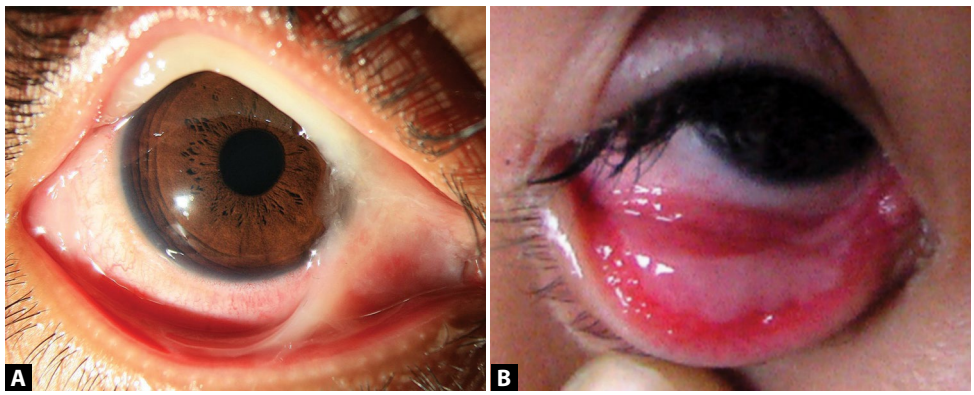
Fig. 12.6: Conjunctival papillae

- ***Papillae:*** These are essentially vascular structures invaded by inflammatory cells. In papilla, there is hyperplasia of the normal system of vascularization with glomerulous-like bunches of new capillaries, growing into the epithelium (Fig. 12.6).
  A papillary reaction is more non-specific, being of less diagnostic importance than a follicle.
  It is mostly seen in vernal conjunctivitis, trachoma and giant papillary conjunctivitis.
- ***Membranes***
  - ***True membranes (Fig. 12.7A):*** They are form when the inflammatory exudates permeate the superficial layers of the conjunctival epithelium. Attempts to remove the membrane may be accompanied by tearing of the epithelium and bleeding. True membrane forms in certain bacterial infections, ***especially in diphtheria.***
  - ***Pseudomembranes (Fig. 12.7B):*** They consist of coagulated exudates loosely adherent to the inflammed conjunctival epithelium. They can be easily peeled off, leaving the epithelium intact without any bleeding. Pseudomembrane forms in adenoviral, vernal, pneumococcal and gonococcal conjunctivitis.

Preauricular Lymphadenopathy

Preauricular lymph node enlargement is a feature of viral and chlamydial infections, but

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Figs 12.7A and B: A. True membrane; **B.** Pseudomembrane

is seldom found in bacterial conjunctivitis except in *Neisseria gonorrhoeae.*

Laboratory Investigations

The main indications are as follows:

- Severe purulent conjunctivitis—to identify the pathogens and sensitivity pattern
- Follicular conjunctivitis—to differentiate viral from chlamydial infection
- Chronic or recurrent conjunctivitis—to find out etiological diagnosis
- Ophthalmia neonatorum.

Conjunctival Scraping and Staining

Scraping is taken from the site of maximal disease with a sterile spatula. The materials are then spreaded on a glass slide for Gram and Giemsa stains.

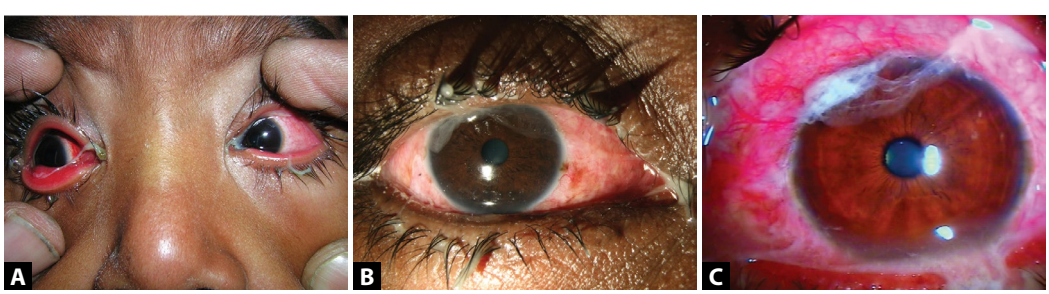
- ***Gram staining:*** It is used to identify the bacteria and to some extent fungus. It is not useful to detect the cellular response.
- ***Giemsa staining***: It is used to identify the cellular response. It also demonstrates the inclusion bodies.

Culture

The materials taken from exudates and lid margins with sterile cotton swab are placed in blood agar or chocolate agar medium. Later, sensitivity pattern is to be determined.

ACUTE MUCOPURULENT CONJUNCTIVITIS (FIGS 12.8A TO C)

It is a common type of acute conjunctival inflammation, characterized by marked hyperemia and a mucopurulent discharge.



Figs 12.8A to C: Acute mucopurulent conjunctivitis

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Etiology

- Occurs at any age
- Poor personal hygiene
- Involvement of the other members of the family or schoolmates.
- ***Causative organisms***
  - *Staphylococcus aureus* (most common),
  - *Hemophilus aegypticus* (Koch-Week's bacillus) - a Gram'–ve' bacillus
  - *Pneumococcus*
  - *Streptococcus*
  - *Adenovirus*, etc.

Clinical Features

The disease is usually bilateral, but one eye may be affected earlier, or more severe, than the other.

Symptoms

- Redness of the eyes
- Mucopurulent discharge
- Grittiness or foreign body sensation
- Stickiness of the eyelids
- Photophobia
- Colored halos around the light.

Signs

- One eye may be more affected than the other
- Lid edema
- Matting of the eyelashes
- Conjunctival congestion and chemosis
- Mucopurulent discharge or flakes of mucopus.
- Subconjunctival petechial hemorrhage (Fig. 12.9).

Composition of the discharge: Tears, mucus, epithelial cells, bacteria, leukocytes, fibrin and rarely red blood cells.

Complications if not treated properly:

- It may subside spontaneously by 10–15 days.
- It may pass into less intense, chronic conjunctivitis.
- Abrasion of the cornea → corneal ulcer.

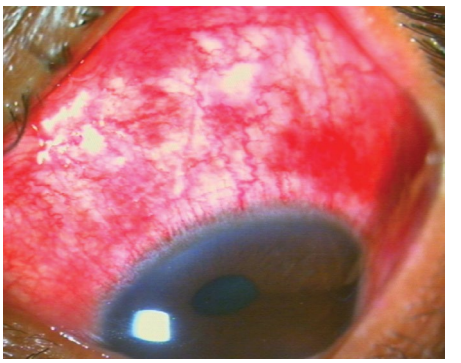


Fig. 12.9: Subconjunctival petechial hemorrhage

  - Marginal corneal ulcer → pseudopterygium formation.
  - Chronic dacryocystitis (very rarely).

Treatment

- Frequent eye wash with lukewarm saline solution to clean crusts and discharge.
- Use dark glasses to prevent photophobia.
- A broad-spectrum antibiotic eye drop, e.g. chloramphenicol or ciprofloxacin is used frequently. Depending upon the severity, it may be four times daily to 1 hourly.
- An antibiotic eye ointment like tetracycline, gentamycin or chloramphenicol at bed time.
- Neomycin is better avoided, as it causes ocular allergy in 8% of cases.
- Other family members are to be treated simultaneously.
- ***To prevent contamination***
  - The patient must keep his hands clean.
  - The patient should lie on the affected side (to prevent its spread to the unaffected eye).
  - Personal belongings of the patient like towel, handkerchief, pillow, etc. should be kept separately.

PURULENT CONJUNCTIVITIS

This is a severe, acute conjunctivitis with purulent discharge mainly caused by *Neisseria gonorrhoeae.*

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Fig. 12.10: Purulent conjunctivitis

- Sometimes, the same clinical picture may be found in staphylococcal, streptococcal and with mixed infections.
- This is rare now-a-days.
- ***Types:*** It occurs in two forms—(1) ***purulent conjunctivitis*** in adults, (2) ***ophthalmia neonatorum*** in newborns.

Adult Purulent Conjunctivitis (Fig. 12.10)

- ***Incubation period:*** few hours to 3 days.
- Males are usually affected.
- Right eye is more commonly involved.

Clinical Features

- Massive swelling of the lids.
- Copious purulent discharge.
- Conjunctival chemosis with or without membrane formation.
  - Corneal involvement → leading to central corneal ulcer → perforation.
  - Preauricular lymphadenopathy with tenderness.
  - Rise in body temperature and mental depression.

Diagnosis

- The most important diagnostic point is the ***coincidence of urethritis***.
- Conjunctival scraping and exudate show Gram '–ve' intracellular organisms.

Complications

- Corneal edema
- Central corneal ulceration
- Perforated corneal ulcer and its sequelae
- Iridocyclitis (may be with hypopyon)
- Ultimately, leading to blindness.

Treatment

Patient should be kept in isolation.

- Frequent irrigation of the eyes with warm normal saline.
- Penicillin drop (freshly prepared in distilled water 1 in 10,000) every minute for half an hour, then every 5 minutes for 1 hour, then hourly for 3–5 days.
- Alternately, newer antibiotics like—norfloxacin or ciprofloxacin eye drop may be used in the same doses.
- Tetracycline eye ointment at bed time.
- Systemic antibiotic—like crystalline penicillin or ciprofloxacin should be given intravenously for 3–5 days. Intramuscular injection of ceftriaxone—1.0 gm/daily for 5 days, if the patient is sensitive to penicillin.
- Atropine (1%) eye ointment is added, if there is corneal involvement.
- Every precaution is to be taken to prevent spread of infection.

Ophthalmia Neonatorum (Fig. 12.11)

It is a conjunctivitis occurring during the first month of life as a result of carelessness at the time of birth.

Etiology (Table 12.2)

- Gonococcal infection is rare now-a-days, but it is the most serious.
- ***Chlamydia oculogenitalis*** is the most common cause of ophthalmia neonatorum now-a-days.

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Fig. 12.11: Ophthalmia neonatorum (gonococcal)

Clinical Features

- In gonococcus, usual presentation is between 1 and 3 days after birth with a ***hyperacute purulent conjunctivitis***.
- In other cases, it is a catarrhal or mucopurulent conjunctivitis.
- In chlamydial infection, the conjunctival reaction is ***papillary only***, without any follicular response.
- Cornea is frequently involved in gonococcal infection and may be perforated with its sequelae.

Treatment

Curative

- Staining of smear and culture of the exudate to know the sensitivity pattern.
- Penicillin drop (freshly prepared, 1 in 5000 in distilled water)—is instilled as 1 minute interval for half an hour, then 5 minutes interval for 1 hour, and then hourly 3–5 days.
- Ciprofloxacin or norfloxacin eye drop may be given in same manner.
- ***For chlamydia***
  - 10% sulphacetamide drop—4 times daily.
  - 1% tetracycline ointment—2 times daily.
  - Systemic erythromycin 50 mg/kg in four divided doses for 3 weeks.
- Systemic penicillin, 50,000 units/kg intramuscularly in two divided doses daily for 7 days.

TABLE 12.2: Etiology and manifestations of ophthalmia neonatorum

Etiology	Manifestations
Chemicals	In hours (antibiotic, detergent)
Gonococcal	1–3 days
(Other) Bacterial	4–5 days
Chlamydial	5–10 days

- Atropine (1%) eye ointment, if there is any corneal involvement.

Prophylaxis

Proper antenatal care of the mother, and any vaginal discharge should be treated meticulously.

- Asepsis and care are to be maintained to protect the eyes during delivery.
- Sulphacetamide (10%), ciprofloxacin or norfloxacin eye drop—four times daily for 7–10 days, is advised following birth.

Alternately tetracycline (1%) eye ointment twice daily for few days.

Crede's prophylaxis: One drop of 1% silver nitrate is instilled into the eyes of the baby, just after the birth. It should never be stronger than 1%—otherwise corneal opacity may result.

Crede's prophylaxis was used earlier to prevent gonococcal ophthalmia, which is very rare now-a-days.

MEMBRANOUS CONJUNCTIVITIS (FIG. 12.12)

It is a type of acute conjunctivitis, associated with membrane formation on the inflamed conjunctiva.

Etiology

- Membranous conjunctivitis is mostly caused by *Corynebacterium diphtheriae*.
- Sometimes, it is caused by *Pneumococcus* or *Streptococcus*, especially in weak children with eruptive fever.

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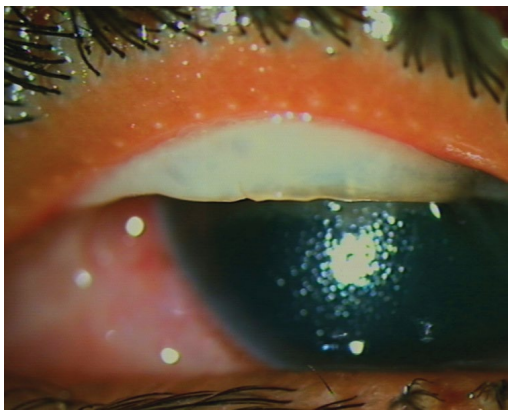


Fig. 12.12: Membranous conjunctivitis

- Chemical irritants (e.g. alkali) may be the cause in adults.

Clinical Features

Usually in children between 2 and 8 years. It occurs in two forms—(1) mild and (2) severe.

1. ***In mild cases***
   - Edema of the lids
   - Mucopurulent or sanious discharge
   - Conjunctival congestion
   - White membrane on the palpebral conjunctiva
   - This membrane peels off rapidly without much bleeding.
2. ***In severe cases***
   - Lids are more edematous, red, hot and tense
   - Scanty, thick conjunctival discharge
   - Pain and tenderness
   - Thick white or greyish-yellow membrane on the palpebral conjunctiva.
   - This membrane is difficult to separate, and ***after removal, bleeding is very common***.
   - Preauricular lymphadenopathy
   - Later, the membrane sloughs off, leaving a red, raw granulating surface
   - Then, there is danger of adhesions, forming between the bulbar and palpebral parts of conjunctiva (symblepharon).

Ligneous conjunctivitis: It is a type of membranous conjunctivitis where the woody-type of membrane is cast off but recurs again and again.

Complications

- Corneal ulcer, which may slough out
- Symblepharon
- Trichiasis and entropion
- Xerosis of the conjunctiva.

Treatment

Every case should be treated as diphtherial, unless there is good negative evidence.

- Isolation of the patient.
- Intramuscular injection of crystalline penicillin 50,000 units/kg, twice daily for 7 days.
- Antidiphtheric serum (40,000–60,000 units) intravenously, and to be repeated after 24 hours if necessary.
- Penicillin drop (freshly prepared 1 in 10,000) every half an hour.
- Erythromycin eye ointment at bed time.
- Atropine (1%) eye ointment if the cornea is involved.
- Prevention of symblepharon formation.

SIMPLE CHRONIC CONJUNCTIVITIS

It is a chronic simple or catarrhal inflammation of the conjunctiva.

Etiology

- As a continuation of simple acute conjunctivitis.
- Continuous irritation of the eyes, due to smoke, dust, heat, bad air, late hours, abuse of alcohol, etc.
- Permanent local irritation by trichiasis, concretions in the palpebral conjunctiva, retained foreign body, chronic dacryocystitis, chronic rhinitis, etc.
- Seborrhea of the scalp (dandruff) is a common factor.

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Symptoms

- Burning and grittiness of the eyes
- Redness more in the evening
- Difficulty in keeping the eyes open
- Slight frothy discharge
- Lids may or may not be stuck together.

Signs

- Apparently, the eyes may look normal
- Lower fornix is congested
- Upper and lower palpebral conjunctiva may be congested with a velvety papilliform roughness
- Thin sticky discharge.

Treatment

- Elimination of the cause of irritation.
- Culture and sensitivity of the conjunctival swab and accordingly local antibiotic drop and ointment are given.
- Astringent and decongestant eye drop, e.g. zinc sulfate, boric acid, naphazoline, etc. four times daily.
- In difficult cases, conjunctival sac is painted with 1% solution of silver nitrate.
- Repeated lid massage, if there is excessive Meibomian gland activity.

ANGULAR CONJUNCTIVITIS (FIGS 12.13A AND B)

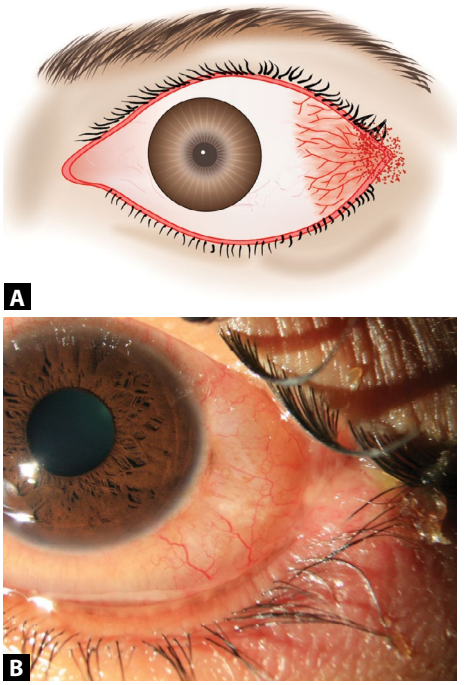
It is a chronic conjunctivitis, where the conjunctival inflammation is limited to intermarginal strip, especially at the outer and inner canthi.

Etiology

- Typically, caused by *Moraxella*, a Gram '–ve' diplobacillus (thick rods placed end-to-end). They produce a proteolytic ferment which acts by maceration of the epithelium.
- Rarely by *Staphylococcus*.

Symptoms

- Discomfort, with slight mucopurulent discharge.



Figs 12.13A and B: Angular conjunctivitis. **A.** Schematic representation; **B.** Photograph

- Itching and excoriation of the skin near the outer and inner angles of eyes.
- Frequent blinking.

Signs

- Congestion of the conjunctiva, limited to the intermarginal strip, near the outer and inner canthi.
- Congestion of the adjacent bulbar conjunctiva.
- Excoriation of the skin at the outer and inner canthi.
- If untreated, the condition becomes chronic and may give rise to blepharitis. Sometimes, a shallow marginal corneal ulcer or central ulcer with hypopyon may form.

Treatment

- Oxytetracycline (1%) eye ointment, 2–3 times daily.

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- Zinc oxide containing eye drop, 4–6 times daily (this acts by inhibiting the proteolytic ferment of the organism).
- Zinc oxide ointment to the lid-margins at bed time.

FOLLICULAR CONJUNCTIVITIS

These are acute, subacute or chronic inflammation of the conjunctiva with the appearence of the follicles.

Causes

- ***Acute follicular conjunctivitis:***
  - Inclusion conjunctivitis
  - Epidemic keratoconjunctivitis
  - Pharyngoconjunctival fever
  - Newcastle conjunctivitis
  - Herpetic keratoconjunctivitis
- Trachoma (chlamydial)
- Due to chemicals and toxins
- Drug toxicity, e.g. pilocarpine, eserine, IDU (iodoxyuridine), DFP (diisopropyl-fluorophosphate), etc.
- Associated with tonsillitis or adenoids in children.

Acute Follicular Conjunctivitis (Figs 12.14A to C)

There is always a tendency of corneal involvement in acute follicular conjunctivitis. So, the typical lesion is most of the time a keratoconjunctivitis.

Inclusion Conjunctivitis

Causative agent: *Chlamydia oculogenitalis.* The primary source of infection is mild urethritis in male and cervicitis in female.

Mode of infection

- ***In neonates***—during birth.
- ***In adults***—by the fingers and through the water of the swimming pool ***(swimming-bath conjunctivitis)***.

Though, the most common cause of swimming-bath conjunctivitis is adenoviral infection.

- Incubation period: 5–10 days.
- Follicular hypertrophy more in lower lid.
- Papillary hypertrophy may be present.
- Superficial punctate keratitis and pannus formation.

Epidemic Keratoconjunctivitis

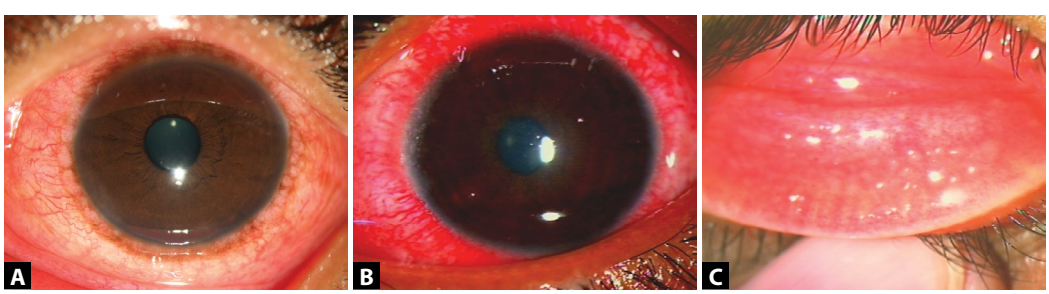
Causative agent: Adenovirus (type 8 and 19).

Clinical features

- Very much contagious and often occurs in epidemic form.
- Marked conjunctival inflammation with scanty discharge.
- After 7–10 days—superficial punctate keratitis (Fig. 12.15) and subepithelial infiltrates develop with photophobia.
- Preauricular lymphadenopathy
- Corneal opacities may persist for months.

Pharyngoconjunctival Fever

Causative agent: Adenovirus (type 3 and 7).



Figs 12.14A to C: Acute follicular conjunctivitis—note the limbal follicles in figure 'A'

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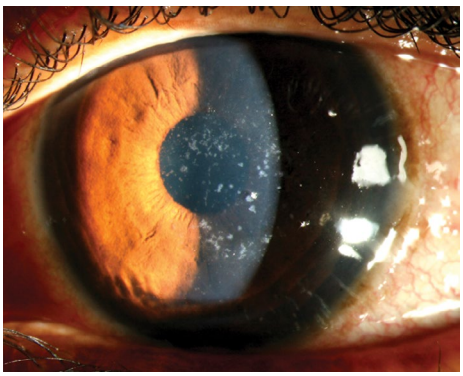


Fig. 12.15: Superficial punctate keratitis

Clinical features

- Usually children are affected
- Acute follicular conjunctivitis
- Pharyngitis and fever
- Preauricular lymphadenopathy
- Superficial punctate keratitis is rare.
- It subsides quickly.

Newcastle Conjunctivitis

Causative agent: Newcastle virus derived from contact with diseased fowls.

Clinically, it is similar to pharyngoconjunctival fever.

Acute Herpetic Keratoconjunctivitis

Causative agent: Herpes simplex virus.

Clinical features

- Usually seen in young children
- Follicles are usually large
- Small dendritic lesion on the cornea
- Pannus formation
- Corneal sensation is reduced
- Preauricular lymphadenopathy.

Treatment

- ***In inclusion conjunctivitis:*** 10–20% sulphacetamide drop and systemic tetracycline (250 mg 4 times for 14 days) are useful.
- ***In other cases***
  - Mild astringent and decongestant drop.
  - Antivirals like IDU or acyclovir may be helpful in herpetic lesions.
- Antibiotic drop may be added to prevent secondary infection, especially in children.
- When the cornea is involved—mild cycloplegic (like tropicamide or cyclopentolate) is to be added.

Trachoma

Trachoma means ***rough*** and ***swelling***. It is a chronic inflammation of the conjunctiva and the cornea characterized by the presence of follicles and papillary hypertrophy of the conjunctiva, and by growth of blood vessels over the cornea.

The specific agent is *C. trachomatis*, a Bedsonian group of organism. Trachoma was previously known as ***Egyptian ophthalmia***.

Etiological Factors

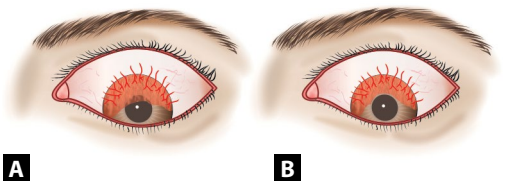
- Any age
- Agent: *C. trachomatis* and other pathogenic microorganisms also aggravate the disease process
- Dry, dirty and sandy weather
- Poor, unhygienic conditions
- ***Eye-seeking flies***
- Use of kajal or surma by the same family members from the same container
- Close person-to-person contacts.

Symptoms

- Foreign body sensation or grittiness
- Itching
- Watering, photophobia and redness
- Discharge is usually scanty, but may be more due to secondary infections.

Acute trachoma: When a secondary infection like mucopurulent or purulent conjunctivitis is superimposed on a relatively mild chronic trachoma it is called acute trachoma.

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Figs 12.16A and B: Trachomatous pannus. **A.** Progressive; **B.** Regressive

Signs

- Bulbar congestion
- Velvety papillary hypertrophy
- ***Follicles:*** Mostly seen in upper tarsal conjunctiva; on the limbus (leading to ***Herbert's pit-pathognomonic***) or on the bulbar conjunctiva.
- ***Pannus:*** It is a characteristic sign. It is defined as fine subepithelial neovascularization, arranged vertically with round cell infiltration mainly seen at the upper limbus and upper part of cornea.

  It is of two types—progressive and regressive (Figs 12.16 and 12.17). In ***progressive pannus,*** the cellular infiltration extends beyond the terminal ends of neovascularization. In ***regressive pannus***, the vessels extend a short distance beyond and area of cellular infiltration.

The above mentioned signs are seen in active trachoma. Further signs are described later in the healed stage or stage of sequelae.

Classifications

- McCallan's classification
- Jone's classification
- WHO classification

McCallan's Classification

Stage-I : ***Incipient trachoma:*** Immature follicles on upper palpebral conjunctiva with no scarring.

Stage-II : ***Established trachoma***

IIA : ***Follicular hypertrophy predominent (Fig. 12.18A)***

IIB : ***Papillary hypertrophy predominent (Fig. 12.18B)***

Stage-III : ***Cicatrizing trachoma:*** Follicles and scarring at upper tarsal conjunctiva.

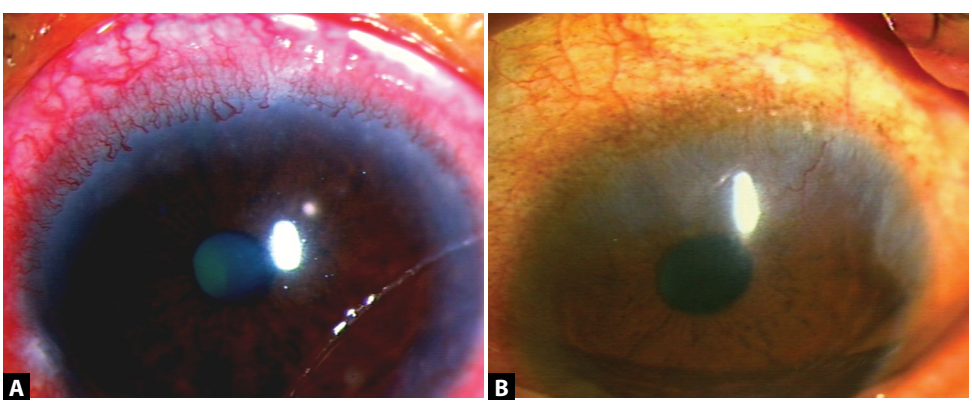
Stage-IV : ***Healed trachoma***

Pitfalls of MacCallan's classification

- It is only based on conjunctival findings. Degrees of corneal involvement are not mentioned.
- It fails to grade the severity of the disease and visual disability.
- It only tells about the evolution of the disease, but not the prognosis.

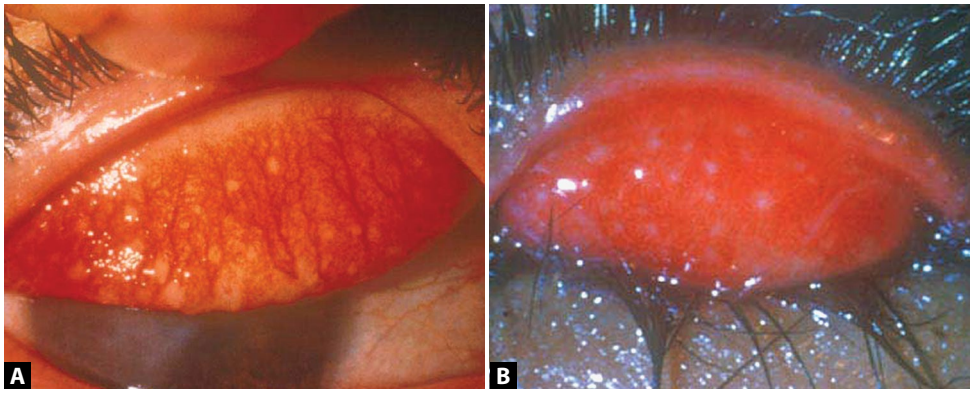
Jone's Classification

Class 1: Blinding trachoma: Hyperendemic (or endemic) trachoma caused by



Figs 12.17A and B: **A.** Trachomatous pannus; **B.** Healed pannus

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Figs 12.18A and B: Trachoma: **A.** Follicular hypertrophy; **B.** Papillary hypertrophy

C. trachomatis—serotypes A, B, Ba and C. It spreads from eye-to-eye by transfer of ocular discharge by eye-seeking' flies and is associated with secondary bacterial infections.

Class 2: Non-blinding trachoma: It is by serotypes, A, B, Ba or C in mesoendemic or hypoendemic area, with better socioeconomic conditions of the victims. It is generally mild with limited transmission because of improved hygiene. Very low incidence of secondary bacterial infection.

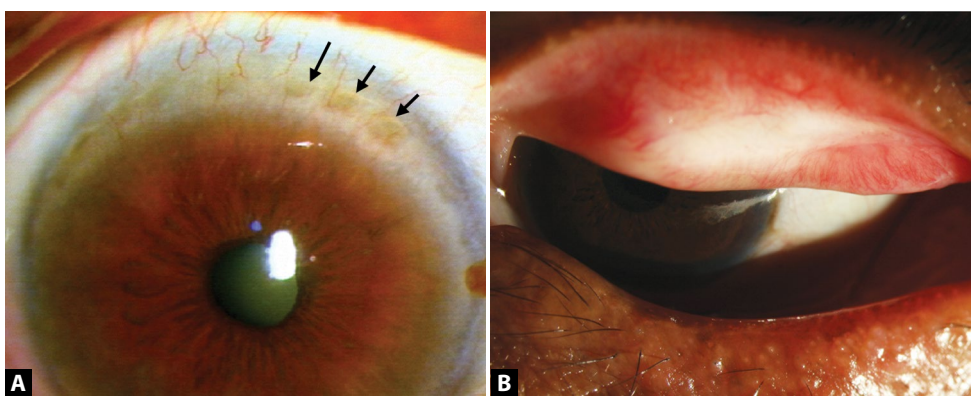
Class 3: Para-trachoma: It is caused by serotypes—D, E, F, G, H, I, J, or K mostly seen in urban population.

It is an eye disease by *C. trachomatis*, where the organisms spread via sexual transmission from a genital reservoir with sporadic transfer to the eye, e.g. inclusion conjunctivitis in adult or ophthalmia neonatorum.

Diagnostic criteria in field study: Individual cases must have at least two of the following signs:

- Follicles at the upper tarsal conjunctiva
- Limbal follicles or their sequelae, ***Herbert's pits*** (Fig. 12.19A)
- Typical conjunctival scarring (stellate-shaped scar) (Fig. 12.19B)
- Vascular pannus, mostly at the upper limbus.

Herbert's pits are the only clinical signs unique to trachoma but they do not occur in every case.



Figs 12.19A and B: **A.** Herbert's pits at the limbus (black arrows); **B.** Trachoma scar

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TABLE 12.3: WHO classifications of trachoma

Recent WHO classification of trachoma (FISTO)		
Trachomatous **F**ollicles	Implies active disease which needs treatment.	***Trachomatous inflammation, follicular:*** Five or more follicles of at least 0.5 mm diameter on the upper tarsal plate should be present. • Some papillae may be present in addition but the palpebral conjunctival blood vessels are visible. • This stage implies that the patient, if properly treated, should recover with no scarring or minimal scarring.
Trachoma **I**ntense	Severe disease which needs urgent treatment.	***Trachomatous inflammation, intense*** • The follicles and papillae are so numerous and inflamed that more than 50% of the palpebral conjunctival blood vessels cannot be seen clearly. • This stage indicates a severe infection with high risk of serious complications.
Trachomatous **S**carring	Old, now inactive infection.	***Trachomatous scarring:*** Tarsal conjunctival cicatrization with white fibrous bands.
Trachomatous **T**richiasis	Needs corrective surgery.	Presence of at least one trichiatic eyelash.
Trachomatous **O**pacities	Corneal opacities from previous trachoma cause visual loss.	Presence of a corneal opacity covering part of the papillary region.

WHO Classification

Refer Table 12.3

Sequelae of Trachoma

Eyelids
- Ptosis (sleepy eyes)
- Scaphoid or boat-shaped lid
- Entropion and trichiasis
- Tylosis (rounding of the lid borders)
- Madarosis (loss of eyelashes)
- Chalazion.

Conjunctiva
- Loss of fornices
- Parenchymatous xerosis
- Symblepharon
- Pigmentation
- Pseudopterygium.

Cornea
- Herbert's pits (Fig. 12.19A)
- Healed pannus leading to hazy cornea
- Different grades of corneal opacity—nebula, macula or leukoma
- Trachomatous nodular keratopathy (Salzmann's nodular degeneration)
- Loss of sensation.

Lacrimal sac: Chronic dacryocystitis.

Treatment

Therapeutic

Chlamydiae are sensitive to erythromycin, tetracycline, sulphonamides and rifampicin.
- ***Systemic:*** (1) Oral suphonamides in full therapeutic doses for 3 weeks or, (2) tetracycline—4 times daily or doxycycline—once daily for 3–4 weeks. (3) Oral azithromycin and erythromycin are also effective.
- ***Local:*** (1) Sulphacetamide (20% or 30%) drop—4 times daily for continuous 6 weeks (2) tetracycline ointment (1%)—twice daily for 6 weeks.

This is followed by ***intermittent treatment*** with tetracycline eye ointment—twice daily for 5 consecutive days, or once daily for

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10 consecutive days in each month for 6 months. This is specially important in epidemic or hyperendemic zone.

Topical erythromycin or rifampicin is also helpful but not available widely.

Prophylactic

- Improvement of personal hygiene and environmental sanitation.
- Avoid kajal, surma, etc.
- Avoid person-to-person close contacts.
- Periodic treatment with sulphacetamide (20%) drop or tetracycline (1%) eye ointment as ***intermittent therapy***.
- Health education.

Treatment of Complications

- Excision of the fornix
- Tarsectomy
- Surgery for the trichiasis and entropion
- Pannus may be treated by cryoapplication or peritomy
- Corneal ulcers—on general line
- Mechanical expression of the follicles by roller (Knaopp's) forceps, silver nitrate painting or diathermy (may cause more scar formation).

SAFE strategy for trachoma in 'vision-2020 programme': (*see* pages 495–496).

S = Surgery for trichiasis/entropion
A = Antibiotics
F = Facial cleanliness
E = Environmental sanitation

ALLERGIC CONJUNCTIVITIS

Simple Allergic Conjunctivitis

This is an acute or subacute non-specific urticarial reaction, due to a large amount allergen reaching the conjunctival sac.

Causes

- Hay fever
- Pollens of certain flowers
- Dust, cosmetics, chemicals, etc.
- Contact with pet animals
- Certain local drugs, e.g. neomycin, atropine, IDU, etc.

Clinical Features

- ***Symptoms*:** Itching, redness and lacrimation
- ***Signs:*** Swelling of the eyelids, mild chemosis with diffuse papillary responses. Cornea is rarely involved.

Treatment

- Removal of the allergens.
- Combination of a vasoconstrictor (naphazoline) with an antihistamine (antazoline or xylometazoline)—four times daily.
- Corticosteroids drop in severe cases.
- Antihistamine tablets are often helpful to suppress the other symptoms like running nose.
- Topical use of 2–4% sodium chromoglycate to prevent remissions.

PHLYCTENULAR CONJUNCTIVITIS

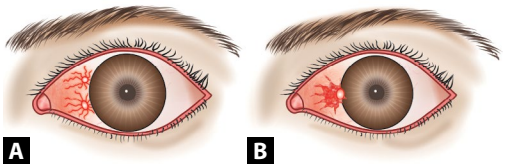
It is an allergic reaction of the conjunctiva caused by endogenous bacterial toxins and characterized by bleb or nodule formation near the limbus.

- Phlycten means a ***bleb***.
- Histopathologically, the bleb is composed of compact mass of mononuclear lymphocytes and polymorphs underneath the epithelium.

Etiology

- ***Age:*** Children of 4–14 years
- Unhygienic condition and malnutrition
- ***Endogenous toxins***, like
  - Tuberculoprotein
  - Toxins from *Staphylococcus*/*Streptococcus* (from tonsillitis or adenoids)
  - Toxins from intestinal parasites.

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Figs 12.20A and B: Phlyctenular; **A.** Conjunctivitis and **B.** Keratoconjunctivitis

Clinical Types

- ***Phlyctenular conjunctivitis (Figs 12.20A and 12.21A):*** When the conjunctiva alone is involved.
- ***Phlyctenular keratoconjunctivitis (Figs 12.20B and 12.21B):*** When at the limbus, and involves both the conjunctiva and cornea.
- ***Phlyctenular keratitis:*** When cornea alone is involved (rare).

Symptoms

- Redness with formation of bleb
- Irritation and lacrimation
- Pain and photophobia in presence of corneal involvement.

Signs

- One or more, small, round and raised nodule at, or near the limbus. They are grey or pinkish-white in color and 1–3 mm in diameter.
- Localized bulbar congestion surrounding the nodule.
- No conjunctival discharge.
- In presence of secondary infection, whole conjunctiva is congested with mucopurulent discharge.
- May be associated with enlarged neck glands, tonsillitis or adenoids.

Course

Firstly, there is a true vesicular stage → epithelium becomes necrotic → small ulcers are formed → healing occurs without any scar.

Complications

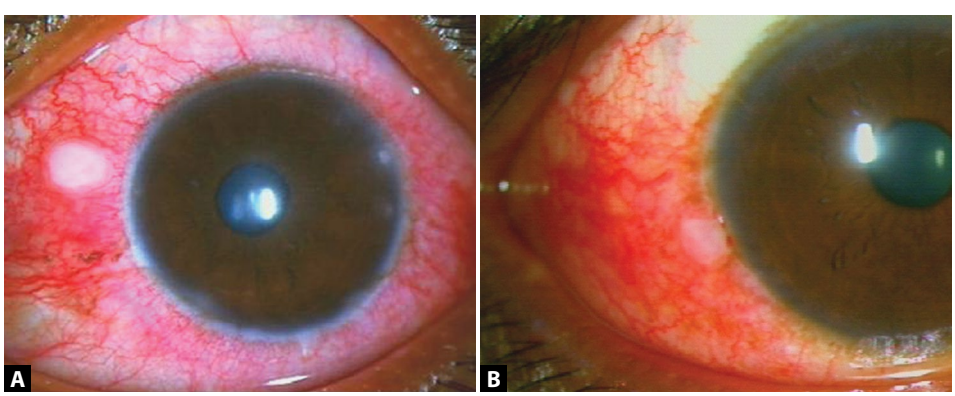
They are mainly due to involvement of the cornea which is rather infrequent.

- Phlyctenular keratitis
- Fascicular ulcer
- Superficial phlyctenular pannus
- Ring ulcer (an allergic ulcer usually does not perforate, unless secondarily infected).

Investigations

They are only indicated when the phlyctens are multiple or recurrent.

- ***To detect tuberculosis***
  - Sputum for acid-fast bacilli (AFB)
  - Blood for total leukocyte count (TLC) differential leukocyte count (DLC), erythrocyte sedimentation rate (ESR)



Figs 12.21A and B: **A.** Phlyctenular conjunctivitis; **B.** Limbal phlycten

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  - X-ray chest
  - Mantoux test.
- ENT consultation to exclude chronic tonsillitis or adenoids.
- Stool for ova, parasite and cysts (OPC).
- Conjunctival swab and corneal scraping when the cornea is involved.

Treatment

- Corticosteroid eye drops (like, dexamethasone or betamethasone) 4–6 times daily.
- In case of secondary infection, first treat bacterial conjunctivitis by local antibiotic drops (like, chloramphenicol or ciprofloxacin) and then treat with local corticosteroid drops.
- When cornea is involved—atropine (1%) eye ointment, or homatropine eye drop 2–3 times daily.
- Improvement of the nutritional status.
- Treatment of the causal factor, e.g.
  - Treatment of tuberculosis
  - Treatment of tonsillitis or adenoids
  - Antihelmintics for intestinal parasites.

VERNAL CONJUNCTIVITIS (FIG. 12.22)

Vernal conjunctivitis (***spring catarrh***) is a bilateral, recurrent, seasonal allergic conjunctivitis in children caused by exogenous allergens.

Etiological Factors

- ***Age:*** 5–14 years.
- Boys are more affected than girls.

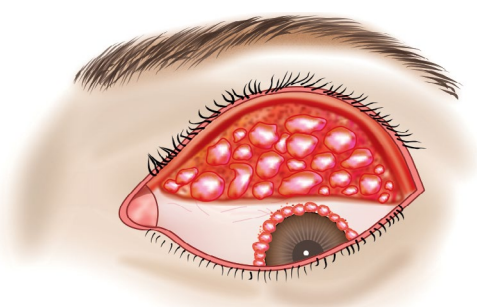


Fig. 12.22: Vernal conjunctivitis

- Prevalent in summer months in India and subsides during winter.
- External allergens like dust, pollens and moulds in presence of dry, hot weather.
- Family history of allergy.
- Self-limiting course (around 11–14 years of age).
- Immunoglobulin E (lgE) mediated mechanism plays an important role.

Symptoms

- ***Intense itching*** (due to alkaline nature of the discharge).
- ***Ropy discharge*** (due to fibrinous nature of the discharge).
- ***Photophobia*** burning and foreign body sensation.

Signs

Three types of vernal conjunctivitis—(1) palpebral, (2) bulbar and (3) mixed.

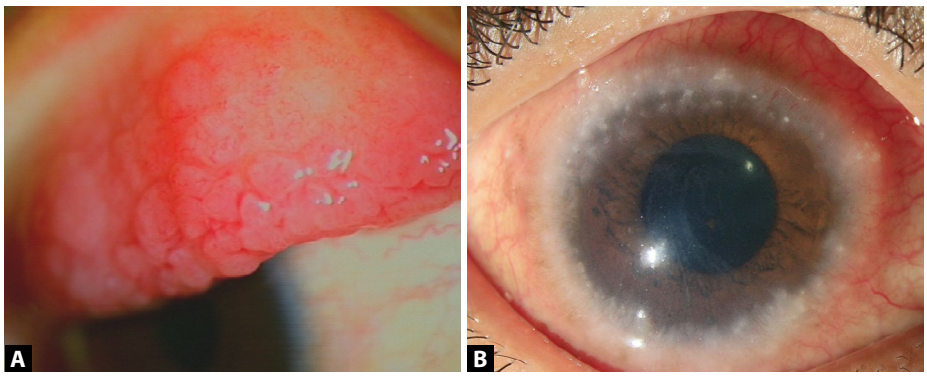
Palpebral type is the most common but bulbar type is more common among blacks.

- ***Palpebral type (Fig. 12.23A)***
  - Cobble-stone appearence of papillary hypertrophy of the palpebral conjunctiva (due to dense fibrous tissue and pressure exerted by the adjacent hard papillae).
  - The color of the papillae is bluish-white (milky-hue) due to hyaline degeneration.
  - In severe cases, the connective tissue septae rupture, giving rise to giant papillae.
- ***Bulbar type***
  - Multiple, small, nodule-like gelationous thickening around the limbus, mostly at the upper.
  - Discrete superficial spots (***Horner-Tranta's dots***) are found scattered around the limbus (Fig. 12.23B).
- ***Mixed type:*** It has the picture of both types.

Histopathology of Papillae

- Tufts of capillaries
- Dense fibrous tissue

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Figs 12.23A and B: Vernal conjunctivitis. **A.** Palpebral type; **B.** Bulbar type with Horner-Tranta's dots

- Large numbers of eosinophils with plasma cells and histocytes
- Covering epithelium is hypertrophied and at places there are hyaline degenerations.

Course and Complications

Seasonal recurrences with exacerbation and remission are common. Ultimately, the disease is self-limiting around the puberty. Complications are mainly due to corneal involvement (vernal keratoconjunctivitis).

The keratopathy changes are as follows:

- Punctate epithelial keratitis.
- Epithelial microerosions—leading to corneal ulceration (shield ulcer) (Fig. 12.24).



Fig. 12.24: Vernal keratoconjunctivitis—shield ulcer

- Corneal plaque.
- Subepithelial scarring—usually in the form of a ring scar.
- ***Pseudogerontoxon*** resembles an arcus senilis with appearence of ***cupid's bow***.
- Patients with vernal catarrh also have a ***higher incidence of keratoconus***.

Treatment

- Cold compress which is soothing.
- ***Topical steroids***, like dexamethasone or betamethasone, 4–8 times daily depending upon the severity. Steroid-antibiotic mixture is not preferred.
- Long-term use of topical steroids has problems like cataract and glaucoma. ***Dilute steroid preparations*** (1:10) can be used rather safely.
- ***Disodium chromoglycate*** (2–4%)—four times daily as drops. It can be used safely for a prolonged period. It mainly, prevents the fresh attack, as it is a mast-cell stabilizer.
- ***Topical antihistaminics*** like azelastine, epinastine, olopatadine, are helpful to give relief from severe itching.
- ***Topical nonsteroidal anti-inflammatory drugs***—like ketorolac, flurbiprofen, or diclofenac may be useful.
- For giant papillae—cryoapplication, β-irradiation or excision may be done.

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- Acetylcysteine (10–20%) drop—to prevent excessive mucus production.
- Protection from external allergens.

Giant papillary conjunctivitis (Fig. 12.25)

It is a foreign body associated allergic conjunctivitis characterized by formation of giant papillae on the palpebral conjunctiva.

Originally, the papillae were thought to be at least 1 mm to diagnose giant papillary conjunctivitis. Now, it is accepted that the papillae are at least 0.3 mm in diameter on the upper palpebral conjunctiva in association with classic symptoms.

Three groups of patients are affected:

1. Soft contact lens wearer
2. Artificial eye wearer
3. Postoperative patients with protruding ends of monofilament nylon sutures.

Clinical features are almost similar to palpebral type of vernal catarrh except the presence of giant papillae which are characteristic.

Treatment

- Discontinuation of contact lenses for 3 months and then re-fitted with rigid gas permeable contact lenses.
- Removal of the sutures.
- Topical steroids and sodium chromoglycate as in vernal catarrh.

CONJUNCTIVAL DEGENERATIONS

Concretions (Lithiasis)

- These are minute hard yellow/white spots in the palpebral conjunctiva.
- They are due to the accumulation of degenerated epithelial cells and inspissated mucus in the depression, called ***Henle's glands***.
- They never become calcareous, so the term is a misnomer.
- They are common in elderly and usually symptomless. But sometimes, they may project and irritate the cornea.
- They may be easily evacuated with a sharp needle.

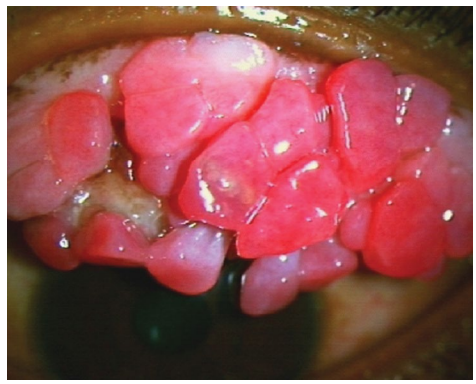


Fig. 12.25: Giant papillary conjunctivitis

Pinguecula (Fig. 12.26)

- This is a yellowish, triangular deposit on the conjunctiva near the limbus at the palpebral aperture.
- They are found in elderly people who are exposed to strong sunlight, dust and wind.
- The apex of the triangle is away from the cornea, and it affects the nasal side first, then the temporal.
- Though it looks like fat (***Pinguecula*** means ***fat***), but is due to hyaline infiltration and elastotic degeneration of the submucosal conjunctival tissue.
- Normally, it is symptomless and does not require any treatment. But when inflammed, it is treated with topical steroids.

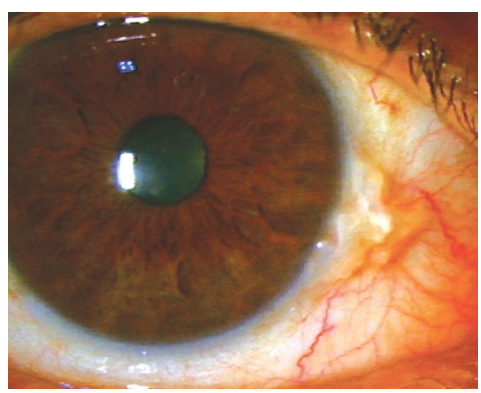


Fig. 12.26: Pinguecula

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Pterygium

It is a degenerative condition of the subconjunctival tissue which proliferates as a triangular fold of tissue-mass to invade the cornea, involving the Bowman's membrane and the superficial stroma, the whole thing being covered by conjunctival epithelium.

Literally, ***pterygium*** means a ***wing***.

Etiology

Not precisely known, but a few factors are responsible:

- Ultraviolet irradiation (more among farmers and outdoor workers).
- Exposure to hot, sandy and dusty weather.
- Pinguecula may act as a precursor.

Stages

There are two types of stages—progressive and atrophic stages (Figs 12.27 and 12.28).

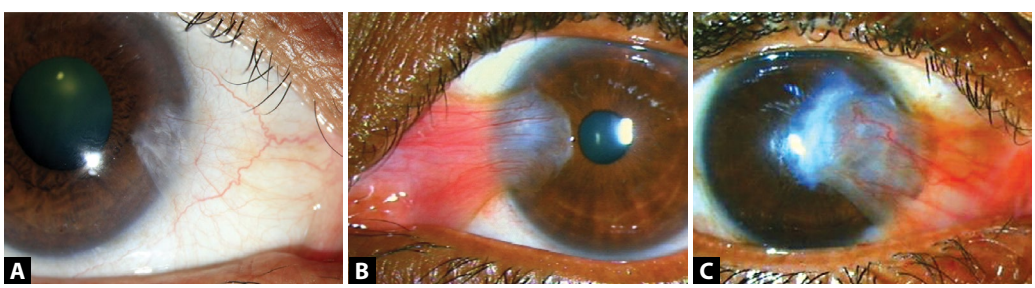
1. ***Progressive***
   - It is thick, fleshy with prominent vascularity.
   - It is gradually increasing in size and encroaching towards the center of cornea.
   - Opaque infiltrative spot (cap) is seen just in front of the apex of the pterygium.
   - Deposition of iron as a line (Stocker's line) is seen in corneal epithelium infront of the apex.
2. ***Atrophic (stationary)***
   - It is thin, attenuated with poor vascularity.
   - No opaque spot (cap) is seen.
   - It is stationary.

Parts (Fig. 12.29)

- ***Apex or head:*** It is the apex of the triangular mass.
- ***Neck:*** It is the constricted portion at the limbus.
- ***Body:*** It is the remaining bulky part.
- ***Cap:*** It is a semilunar infiltrative opaque spot just in front of the apex. This is due to cellular infiltration.

A B C

Figs 12.27A to C: Pterygium (schematic representation). **A.** Atrophic; **B.** Progressive; **C.** Crossing the papillary area



Figs 12.28A to C: Types of pterygium (photographs); **A.** Atrophic; **B.** Progressive; **C.** Covering the pupillary area

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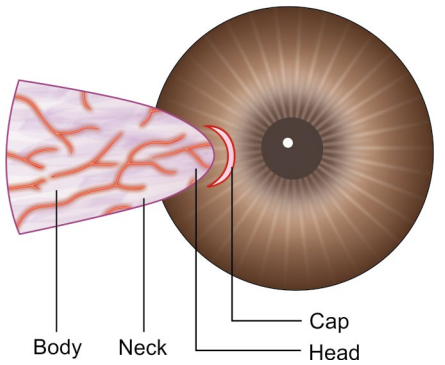


Fig. 12.29: Parts of a pterygium

Symptoms

- ***Appearence of a mass*** on nasal, or rarely on temporal side of the cornea. Sometimes, both nasal and temporal sides are involved (double-headed pterygium).
- ***Dimness of vision*** due to corneal astigmatism or obstruction of the visual axis.
- Rarely, ***diplopia*** due to limitation of ocular movements, especially when associated with symblepharon.

Signs

- Decreased visual acuity.
- Triangular fold of conjunctival mass encroaching upon the cornea in a variable degree.
- It is usually bilateral, and mainly on the nasal side at the palpebral aperture.
- There are folds at the upper and lower borders of the mass.
- Limitation of movements in few cases.

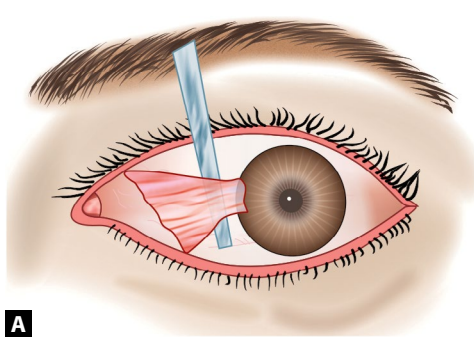
Pseudopterygium (Figs 12.30A and B)

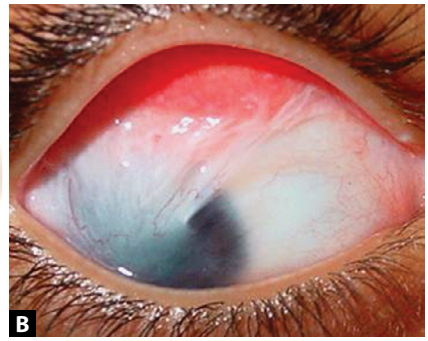
- It is the adhesion of a fold of conjunctiva to a peripheral cornea, due to some inflammation.
- It is seen at any age; usually unilateral, stationary, and at any meridian.
- Causes are chemical burns, peripheral corneal ulcer, foreign body, etc.
- As it is fixed to the cornea only at its apex, a probe can be passed easily beneath the neck of the pterygium (probe test).
- Treatment is only by excision.

Treatment

Treatment is done on the basis of types of pterygium.

- ***Atrophic pterygium, and if just on the cornea*:** It is best left alone with periodic follow-up.
- ***Progressive pterygium:***
  - Excision of the pterygium with the conjunctiva, and keeping the limbus and adjacent scleral area bare (bare-sclera technique).
  - Subconjunctival dissection of the pterygium and then excision with bare sclera. (*see* Chapter 25, page 467).





Figs 12.30A and B: Pseudopterygium. A probe can be passed beneath the neck of the pseudopterygium. **A.** Schematic representation; **B.** Photograph

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- ***Transposition operation (Mc Reynolds):*** The apex is freed and turned down under the bulbar conjunctiva, and sutured in this position.
  A pterygium cannot be removed without leaving a scar on the cornea as it involves the Bowman's membrane and superficial stroma.

- ***Recurrent pterygium (and also to prevent recurrence):***
  - After excision, the bare scleral area is treated with:
    - ***Beta-irradiation,*** (by strontium-90), not more than 2000 rads during the first week of surgery.
    - Thio-TEPA solution as drop (1 in 2000)—4 times daily for 6 weeks.
    - Mitomycin-C (0.02%) solution—locally during operation or as drop postoperatively.
  - ***Conjunctival limbal autograft:*** Cover the bare scleral area by conjunctival limbal autograft (CLAU). The recurrence rate is lowest in this procedure.
  - ***Lamellar keratoplasty*** of the affected part, along with excision of pterygium.
- ***Pterygium involving the pupillary area:*** Resection of the ptertygium with anterior lamellar keratoplasty is the treatment of choice (Figs 12.27C and 12.28C).

CONJUNCTIVAL CYSTS

- ***Lymphangiectasis (lymphatic cysts):*** They are very common, and arise as the dilatation of the lymph spaces.
- ***Retention cysts:*** They occur due to the obstruction of the ducts of accessory lacrimal gland of Krause in the upper fornix.
- ***Implantation cyst:*** This occurs due to implantation of conjunctival epithelial cells, following an injury or after an operation (e.g. squint, pterygium, etc.) (Fig. 12.31).
- ***Parasitic cyst:*** This is due to sub conjunctival ***cysticercus*** or ***hydatid*** cyst. This is rare.

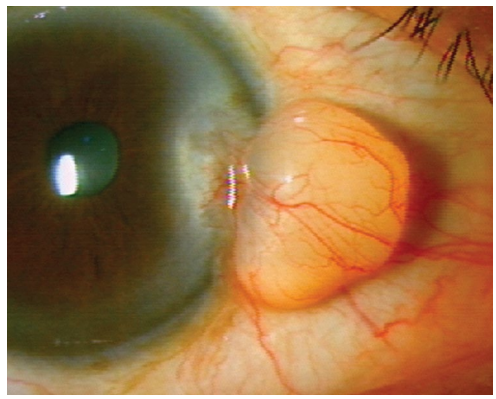


Fig. 12.31: Conjunctival implantation cyst

CONJUNCTIVAL TUMORS

Classification

1. Nonpigmented tumors

- ***Congenital (choristomas)***

Dermolipoma	Epibulbar dermoid

- ***Acquired***

Benign	*Malignant*
Papilloma	Carcinoma-in-situ
Angioma	Squamous cell carcinoma

2. Pigmented tumors

- ***Simple nevus***
- ***Precancerous***
  - Junctional nevus
  - Precancerous melanosis
  - Lentigo malignum
- ***Malignant melanoma***

Dermoids

They appear as solid white masses, most frequently at the limbus, especially in lower-outer quadrant (Fig. 12.32).

- They consist of skin with sebaceous glands and hair.
- Surgical excision with a lamellar sclerocorneal patch graft is the treatment.

Dermolipomas (Lipodermoids)

- They appear as soft, yellow, movable subconjunctival masses, located at the outer canthus or at the limbus.

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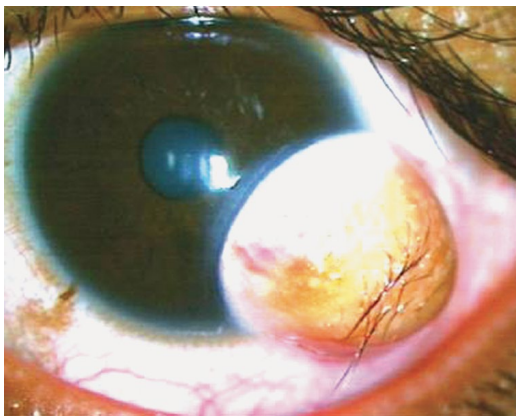


Fig. 12.32: Limbal dermoid

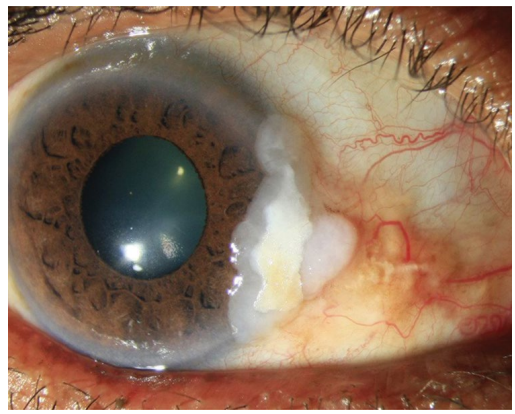


Fig. 12.34: Intraepithelial epithelioma

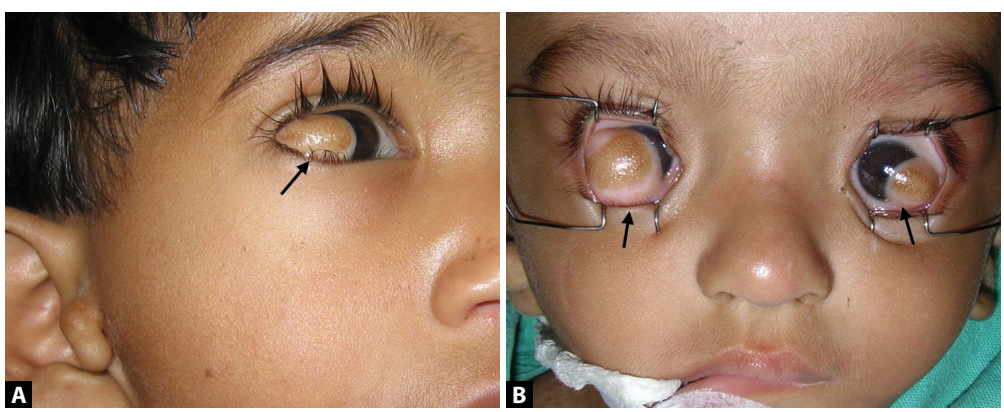
- They consist of fibrous tissue and fat, and sometimes, with dermoid tissue on their surface.
- In some cases, they are associated with ***Goldenhar's syndrome (Figs 12.33A and B)*** (preauricular tags, vertebral anomalies and hemifacial hypoplasia).
- ***Treatment:*** Surgical excision; though sometimes, it is seen that the fat is continuous with that of the orbit.

Carcinoma in situ (Intraepithelial Epithelioma) (Fig. 12.34)

- It is sometimes called ***Bowen's disease***.
- It appears as a slightly elevated, fleshy mass with tuft of blood vessels at the limbus in an elderly subject.
- The lesion is superficial to the basement membrane and conjunctiva is freely mobile.
- Treatment is by surgical local excision followed by cryoapplication.

Squamous Cell Carcinoma (Fig. 12.35)

- It appears as a reddish-grey fleshy mass with broad base and characterized by deep invasion into the stroma with fixation to the underlying structure.



Figs 12.33A and B: Goldenhar's syndrome. **A.** Unilateral; **B.** Bilateral dermolipoma (arrows)

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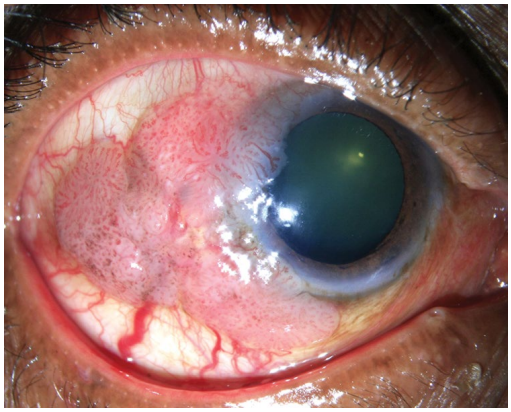


Fig. 12.35: Squamous cell carcinoma

- It is mainly seen at the limbus.
- It arises from a papilloma or carcinoma *in situ.*
- Distant metastasis occurs rapidly.
- Treatment is by radical excision, enucleation or exenteration of the orbit.

Simple Nevus (Fig. 12.36)

- This appears as a single, sharply demarcated, flat or slightly elevated lesion, most commonly near the limbus.
- It is congenital and tends to enlarge or darken during pregnancy or puberty.
- Excision is only indicated for cosmetic reason.

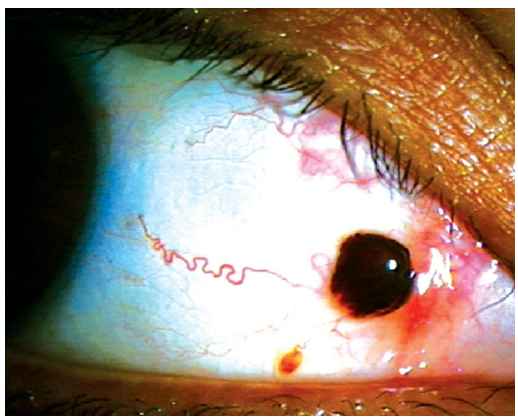


Fig. 12.36: Conjunctival simple nevus



Fig. 12.37: Precancerous melanosis

Precancerous Melanosis (Fig. 12.37)

- This is a small pigmented tumor of the conjunctiva which spreads as a diffuse patch of pigmented lesion.
- It occurs in elderly people.
- In 20% cases, it proceeds to frank malignant changes.
- It is radiosensitive at the precancerous stage.
- Local excision with post-operative radiotherapy may limit the disease.

Malignant Melanoma (Fig. 12.38)

- It is typically seen at the limbus in elderly patient.

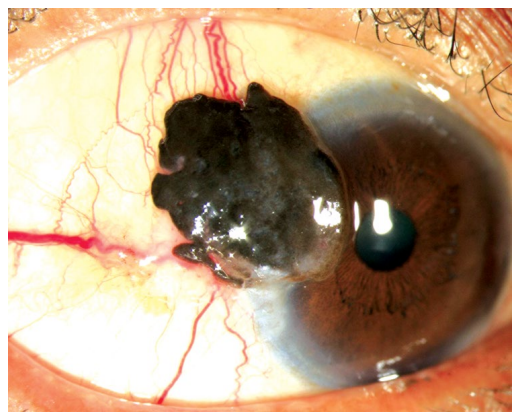


Fig. 12.38: Malignant melanoma of the conjunctiva

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- Pigmented melanomas are more common than nonpigmented melanomas.
- It may arise *de novo,* or from a precancerous lesion, e.g. junctional nevus, precancerous melanosis or lentigo malignum.
- It spreads over the surface of the globe, but rarely penetrates it.
- Metastasis is common.
- ***Treatment*** is by radical excision of the mass, enucleation or exenteration of the orbit.

SYMPTOMATIC CONDITIONS OF THE CONJUNCTIVA

Subconjunctival Hemorrhage (Figs 12.39A and B)

Subconjunctival hemorrhage is common, since the vessels are loosely supported. Rupture of a conjunctival blood vessel causes a bright-red, sharply delineated area surrounded by the normal appearing conjunctiva.

Etiology

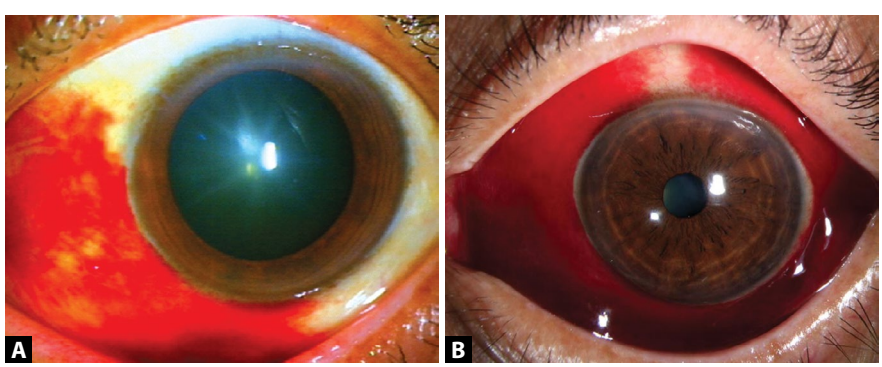
- ***Traumatic***
  - ***Direct trauma to the eyeball:*** Posterior limit of the hemorrhage is visible.
  - ***Head injury or injury to the orbit:*** Blood usually seeps under the conjunctiva 12–24 hours after the head injury. Posterior limit of the hemorrhage is not visible.
- ***Infective:*** In some conjunctivitis, e.g. due to *Pneumococcus*, Koch-Week's bacillus, adenovirus or picornavirus.
- ***Mechanical:*** Due to venous congestion and consequent rupture of the small vessels, e.g. bronchitis, whooping cough, compression of the neck or chest, etc. (Fig. 12.40A). May be associated with retrobular hemorrhage and ecchymosis of lids called ***Panda bear*** or ***Raccoon eyes sign*** (Fig. 12.40B).
- ***Arteriosclerosis and hypertension.***
- ***Blood dyscrasias:*** Leukemia, purpura, hemophilia, etc.
- ***Vicarious menstruation:*** Leading to periodic hemorrhage.
- ***Idiopathic.***

Fate of Subconjunctival Hemorrhage

- At first, it is bright red in color. This is due to ***oxyhemoglobin,*** as it is in constant contact with the atmospheric oxygen.
- Subsequently, it changes to orange-yellow or blackish-red, then to yellow discoloration; this is due to breakdown of oxyhemoglobin.
- Ultimately, it gets absorbed within 2–3 weeks, depending upon the amount of hemorrhage.

Treatment

- No treatment is necessary in most of the cases, as it is absorbed automatically.



Figs 12.39A and B: Subconjunctival hemorrhage

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Figs 12.40A and B: A. Bilateral subconjunctival hemorrhage in a child with whooping cough; **B.** With ecchymosis of lids—(Panda bear or Raccoon eyes sign)

- Initially, cold compress is helpful as it constricts the blood vessels.
- In severe cases, if the extravasated blood causes prolapse of the conjunctiva through the interpalpebral fissure, subconjunctival space may be punctured to drain the blood.
- Treatment of the cause.

Conjunctival Xerosis (Figs 12.41A and B)

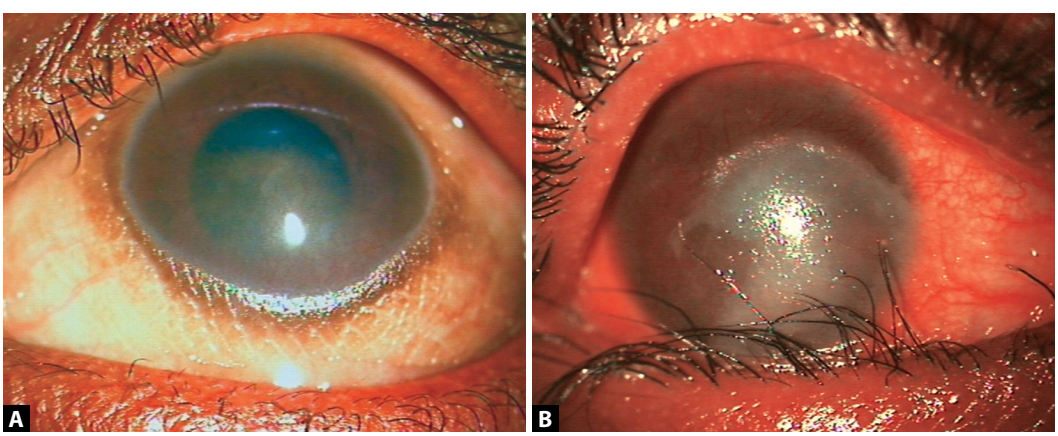
This is a dry, lustreless condition of the conjunctiva.

Types

Two types of xerosis are described as follows:

1. ***Xerosis epithelialis:*** Due to vitamin-A deficiency with associated protein-energy malnutrition (PEM).
2. ***Xerosis perenchymatous:*** Following cicatricial changes of the conjunctiva, e.g. trachoma, chemical burns, pemphigoid, diphtheria, prolong use of β-blocker, etc.

   Following prolonged exposure of the eye, e.g. proptosis, ectropion, lagophthalmos, comatose patients, etc.



Figs 12.41A and B: A. Severe conjunctival xerosis; **B.** Severe xerosis in ocular pemphigus

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Pathology

- Epithelium becomes epidermoid, like that of the skin with granular and horny layer.
- There is destruction of goblet cells of the conjunctiva.
- Profuse growth of *Corynebacterium xerosis*.

Pathogenesis

Goblet cells cease to secrete mucus → vicarious activity of the Meibomian glands → fatty secretion covers the dry surface → watery tears fail to moisten the conjunctiva.

It must be noted that xerosis has nothing to do with any failure of function on the part of the lacrimal gland. The conjunctiva can be quite efficiently moistened by its own secretion alone, and if the lacrimal gland is removed, xerosis usually does not follow.

On the other hand, xerosis may follow in spite of normal or increased lacrimal secretion, if the secretory activity of the conjunctiva itself is impaired.

Treatment

- ***Local treatment***
  - Use dark glasses.
  - Artificial tears eye drop to relief dryness, like methylcellulose, hypermellose, polyvinyl alcohol, etc. 1–2 hourly depending upon the severity. Tear-gel may be used at night.
  - Treatment of the ocular conditions responsible for dry eye, e.g. treatment of trachoma, ectropion correction, tarsorrhaphy, etc.
- ***General treatment*** (*see* Chapter 26)
  - Administration of vitamin A [in case of vitamin A deficiency showing Bitot's spot (Fig. 12.42)].
  - Correction of nutritional status.

Stevens-Johnson Syndrome (Fig. 12.43)

It is a mucocutaneous vesiculobullous disease caused by a hypersensitivity to antibiotics (penicillins), sulphonamides (including acetazolamide), phenylbutazone, allopurinol, phenytoin sodium, etc.

The basic lesion is thought to be an acute vasculitis affecting the conjunctiva and other mucous membranes.

Clinical Features

- ***Systemic***
  - Fever, sore throat, cough
  - Symmetrical erythematous and vesiculobullous reaction affecting the skin (scalp is spared) and mucous membrane of the body.

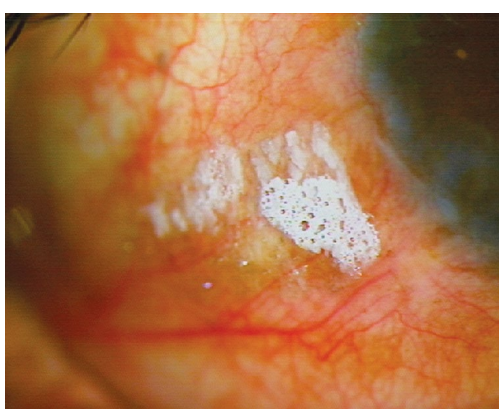


Fig. 12.42: Bitot's spot



Fig. 12.43: Stevens-Johnson syndrome—conjunctival and oral lesion

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- ***Ocular***
  - Conjunctiva is involved in 50% of cases.
  - Mucopurulent conjunctivitis with membrane or pseudomembrane formation.
  - Scarring of the conjunctiva and lid margins—with trichiasis (acquired distichiasis), symblepharon and obliteration of the fornices.

Treatment

- Discontinuation of the offending drugs.
- Systemic antibiotics and corticosteroids.
- Topical antibiotics, topical steroids and artificial tears as long-term treatment.
- Surgical correction of entropion, symblepharon, trichiasis, etc.

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13 CHAPTER

Diseases of the Cornea and Sclera

EVALUATION OF CORNEAL DISEASES

Symptoms

- ***Pain:*** The cornea is richly supplied by sensory nerve endings. In corneal abrasions or bullous keratopathy, the direct stimulation of bare nerve endings causes severe pain
- ***Decreased visual acuity:*** It is caused by loss of central corneal transparency
- ***Halos:*** They are due to diffraction of light by epithelial and subepithelial edema
- ***Photophobia***
- ***Lacrimation:*** It is due to reflex stimulation of the corneal nerve.

Signs

The precise localization of corneal defects is best done by slit-lamp biomicroscopy.

- ***Epithelium:*** Punctate erosions, edema, filaments, superficial punctate keratitis, and staining with fluorescein and Rose-Bengal.
- ***Stroma:*** Infiltrates, edema, vascularization, deposits, and scarring (opacities).
- ***Descemet's membrane:*** Breaks, folds, localized thickening (excrescences), etc.
- ***Deposition of pigments:*** Corneal pigment deposition is associated with a variety of disorders (Table 13.1).
- ***Corneal thickness (by pachymetry)*** either by the optical or ultrasonic pachymeter.
  - It indicates the functional integrity of the corneal endothelium.

TABLE 13.1: Pigment deposition in the cornea

Types of pigment	Sign (Disorders)	Corneal location
Iron	• Fleischer's ring (keratoconus) • Hudson-Stahli's line (old opacity) (Fig. 13.1) • Stocker's line (pterygium) • Ferry's line (filtering bleb) • Siderosis • Blood staining of the cornea	• Epithelium • Epithelium • Epithelium • Epithelium • Mainly stroma • Mainly stroma
Copper	Kayser-Fleischer's ring (Fig. 13.2) • Wilson's disease • Chalcosis	Descemet's membrane
Melanin	Krukenberg's spindle (pigment dispersion syndrome)	Endothelium

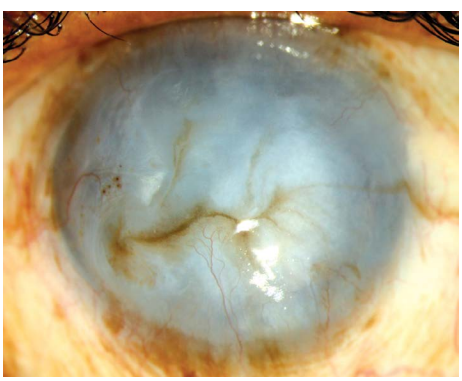


Fig. 13.1: Hudson-Stahli's line

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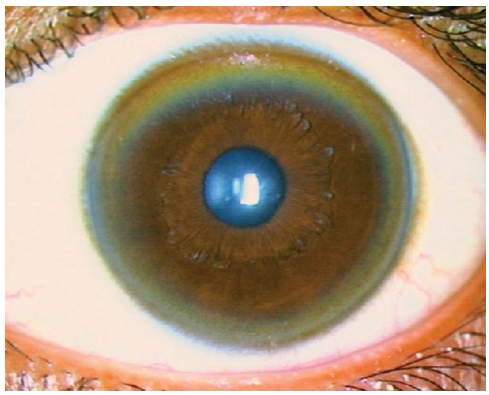


Fig. 13.2: Kayser-Fleischer's ring



Fig. 13.3: Microcornea with microphthalmos (right eye)

- Ultrasonic pachymetry is useful before laser assisted in situ keratomileusis (LASIK) and other keratorefractive operations.

- ***Corneal sensation:*** Loss of corneal sensation is an important sign to diagnose viral keratitis and neuroparalytic keratitis.

Laboratory diagnosis: Material for laboratory analysis is obtained by scraping of the ulcer, from its base and margin.

It is then plated onto the glass slides for Gram-staining and potassium hydroxide (KOH) preparation to identify the bacteria and fungus respectively. Part of the material is inoculated onto the following culture media.

- ***Blood agar*** mainly for aerobic bacteria.
- ***Sabouraud's media*** for fungus.

CONGENITAL ANOMALIES

Microcornea (Fig. 13.3)

- Corneal diameter is less than 10 mm with decreased radius of curvature.
- There is hypermetropia, and chance of narrow angle glaucoma in later years.
- This term is reserved for corneal affection alone. But when the entire eyeball is small, it is called ***microphthalmos*** which is more common.
- ***Systemic association*****:** Ehler-Danlos syndrome, Weill-Marchesani syndrome, Waardenburg's syndrome.

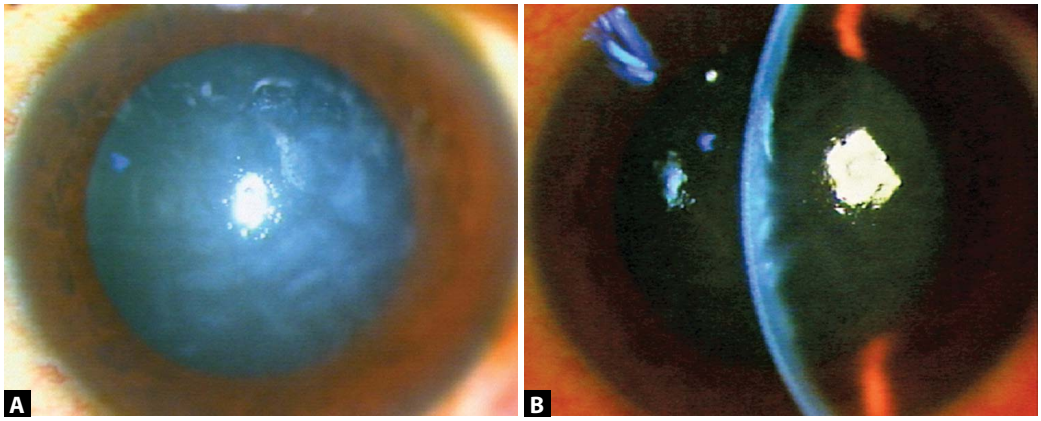
Megalocornea (Fig. 13.4)

- It is a bilateral condition, in which the corneal diameter is more than 14 mm.
- The patient is myopic.
- There are deep anterior chamber, tremulousness of iris and subluxation of the lens with or without cataract formation.
- Megalocornea must be differentiated from congenital glaucoma by tonometry and gonioscopy.



Fig. 13.4: Megalocornea (right eye more than left eye)

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Figs 13.5A and B: Corneal edema. **A.** Diffuse illumination; **B.** Slit section

Corneal Opacities

- Central corneal opacity (Peter's anomaly) is due to defect in embryogenesis, due to incomplete separation of the lens from the surface ectoderm.
- It is usually associated with other anomalies in the iris or angle of the anterior chamber, with glaucoma.

Posterior Embryotoxon

- An unusual prominence of Schwalbe's line which is the peripheral termination of Descemet's membrane.
- It appears as a ring opacity in the deeper layer of the cornea.

CORNEAL EDEMA (FIGS 13.5A AND B)

- The integrity of both epithelium and the endothelium is necessary to maintain cornea in its relatively dehydrated state.
- It is associated with increased corneal thickness, and a variable decrease in corneal transparency.
- Source of fluid is either the aqueous humor (via endothelium) or the tears (via epithelium).

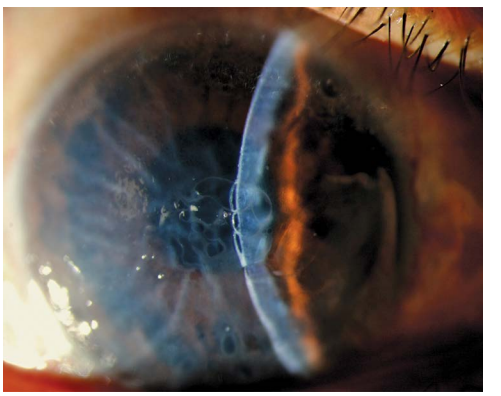


Fig. 13.6: Bullous keratopathy in pseudophakic eye in slit section

Pathology

- Fluid accumulates between the basal epithelial cells → then, in between the lamella and around the nerve fibers of the stroma → total haziness.
- In long standing cases, the epithelium tends to be raised into large vesicles or bullae, leading to ***bullous keratopathy*** (Fig. 13.6).

Etiology

- ***Inflammatory:*** Corneal ulcer, erosions, acute iridocyclitis (due to endothelial damage).

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- ***Traumatic:*** Mechanical trauma and post-surgical—due to endothelial damage, particularly when vitreous remains adherent to it.
- ***Increased intraocular pressure:*** Acute edema in angle-closure glaucoma and epidemic dropsy glaucoma. Chronic edema in long standing cases—as in absolute glaucoma and buphthalmos.
- ***Dystrophic condition of the cornea,*** e.g. Fuchs' endothelial dystrophy.
- ***Hypoxia of the cornea:*** As in contact lens wearer due to epithelial edema, as a result of prolonged deprivation of atmospheric oxygen.

Symptoms

- ***Colored halos*** around the light with red color being outside, and blue inside.
- ***Decreased visual acuity***—due to corneal haziness and irregular astigmatism.

Treatment

- Use of hot air (by hairdryer).
- Frequent instillation of concentrated sodium chloride (5%) solution, or ointment containing 6% or 15% sodium chloride.
- Glycerine drop (to clear edema quickly and temporarily), but it is irritant.
- Bandage soft contact lenses, specially in case of ruptured bullae.
- Epithelium is stripped off, and is to be replaced with a thin conjunctival flap.
- Full-thickness penetrating keratoplasty to improve visual status.

INFLAMMATION OF THE CORNEA (KERATITIS)

Modes of Infection

- ***Exogenous infections:*** Most common, and cornea is primarily affected by virulent organisms.
- ***From the ocular tissues:*** Owing to direct anatomical continuity, the diseases of—
  - Conjunctiva—spread to the corneal epithelium.
  - Sclera—to the stroma.
  - Uveal tract—to the endothelium.
- ***Endogenous infections:*** Rare, as the cornea is avascular. They are typically allergic in nature.

Keratitis or inflammation of the cornea may be classified as follows:

Superficial keratitis

- Infective keratitis
  - Bacterial
  - Fungal
  - Viral
  - Acanthamoebal.
- Non-infective keratitis
  - Central
    - Exposure keratitis
    - Neurotrophic keratitis
    - Atheromatous keratitis.
  - Peripheral
    - Marginal keratitis
    - Phlyctenular keratitis
    - Mooren's ulcer
    - Terrien's degeneration
    - Rosacea keratitis
    - Keratitis associated with collagen diseases.

Deep keratitis

- Interstitial keratitis
- Disciform keratitis
- Sclerosing keratitis.

In strict sense, keratitis and corneal ulcer are not always synonymous.

A ***corneal ulcer is defined*** as a loss of corneal epithelium with underlying stromal infiltration and suppuration associated with signs of inflammation with or without hypopyon.

BACTERIAL KERATITIS

Predisposing Factors

Intact corneal epithelium cannot be penetrated by any organism except, *Neisseria gonorrhea, Neisseria meningitidis* and *Corynebacterium diphtheriae.* In these cases, suppurative kera-

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titis is always associated with a purulent conjunctivitis.

With the other bacteria, keratitis is invariably associated with one or more of the following predisposing factors:

- ***Trauma to the corneal epithelium:*** By injury, foreign body, trichiasis, entropion, contact lens wear, etc.
- ***Underlying corneal diseases:*** Herpetic keratitis, corneal erosions, bullous keratopathy, keratomalacia, etc.
- Neurotrophic or exposure keratopathy
- Dry eyes
- Chronic dacryocystitis or blepharitis
- Use of topical steroids
- Lowering of general body resistance, e.g. malnutrition, diabetes, systemic immunosuppressive therapy, etc.

Pathology

Uncomplicated, localized corneal ulcer is having four stages—stages of infiltration, progression, regression and cicatrization (Fig. 13.7).

Stage of Infiltration and Progression

- Edema and localized necrosis of the anterior part of the corneal stroma.
- Necrotic tissue is cast off leaving a saucer shaped defect, known as ulcer.
- Epithelium is sloughed off simultaneously with an involvement of larger area.
- Dense infiltration of polymorphs followed by macrophages. This infiltration extends for some distance around the ulcer.
- There is an associated iritis due to toxin being absorbed into the anterior chamber.
- This reaction is so toxic that leucocytosis takes place, and polymorphs poured out of the blood vessels pass into the aqueous, and gravitate at the bottom of anterior chamber. This is hypopyon and this pus remains sterile, so long as the Descemet's membrane is intact.

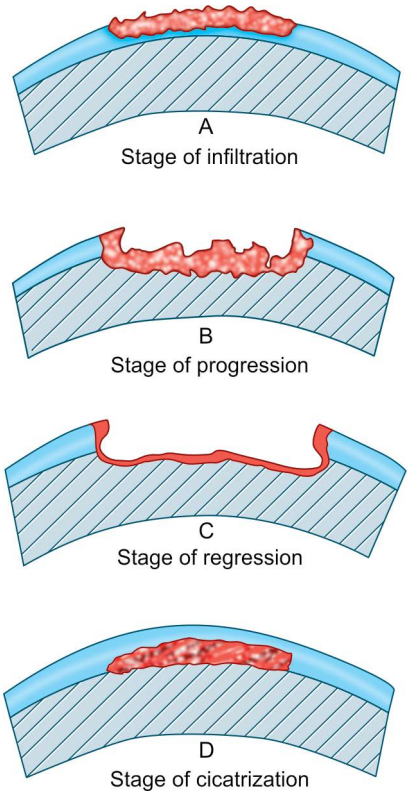


Fig. 13.7: Pathological stages of corneal ulcer

Stage of Regression

- The defect becomes larger, as there is digestion of the necrotic tissue by the polymorphs.
- Line of demarcation appears surrounding the ulcer.
- Infiltrations at the edges and the base start to disappear, and polymorphs are replaced by mononuclear cells.
- The ulcerated area becomes smooth and relatively clear.

Stage of Cicatrization

- Healing of the defect occurs by formation of the fibrous tissue.
- The fibrous tissue is derived from:
  - Invading mononuclear cells
  - Keratocytes and
  - Endothelial cells of the new vessels.

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- The epithelium grows from the edges of the defect to form a permanent covering.

Clinical Features

Symptoms

- Acute pain, redness and lacrimation
- Photophobia
- Decreased visual acuity
- White spot on the cornea.

Signs

- Marked blepharospasm
- Lid edema
- Ciliary congestion of the conjunctiva
- Ulcer usually starts as a grayish-white, circumscribed infiltration, with edema of the surrounding tissue. There is absence of window reflex. The margins of the ulcer are overhanging, and the floor is covered by necrotic material. The extent of the ulcer may be detected by fluorescein staining and seen with blue filter. Ulcer area stains as brilliant green.
  Later, the ulcer becomes gradually smooth in the regressive stage, and ultimately, scar tissue begins to appear the edges and the floor of the ulcer.
- Some degree of features of iritis.
- Hypopyon may be present (Fig. 13.8).
- Intraocular pressure (IOP) may be raised in presence of hypopyon. But if the ulcer perforates, IOP decreases.

Certain bacteria produces characteristic response:
- *Staphylococcus aureus* and *Streptococcus pneumonae*—produce oval, yellowish-white, dense, opaque, stromal ulcer surrounded by relatively clear cornea.
- *Pseudomonas*—mucopurulent sticky and greenish exudate, irregular deep ulcer with 'ground-glass' appearance of surrounding stroma. Ulcer progresses very rapidly, and results in corneal perforation within 48 hours.
- *Enterobacteriaceae (E. coli, Proteus, Klebsiella*, etc.) produce a shallow ulcer with pleomorphic grayish-white necrotic area. Sometimes, they produce ring-shaped corneal infiltrates.

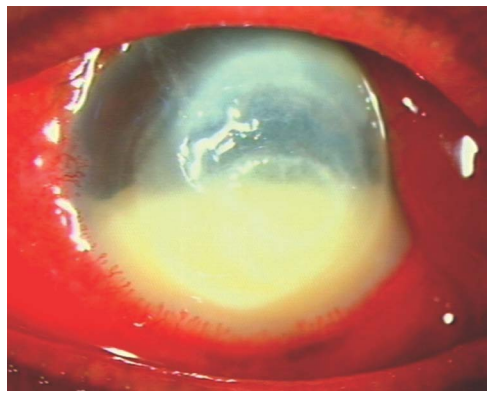
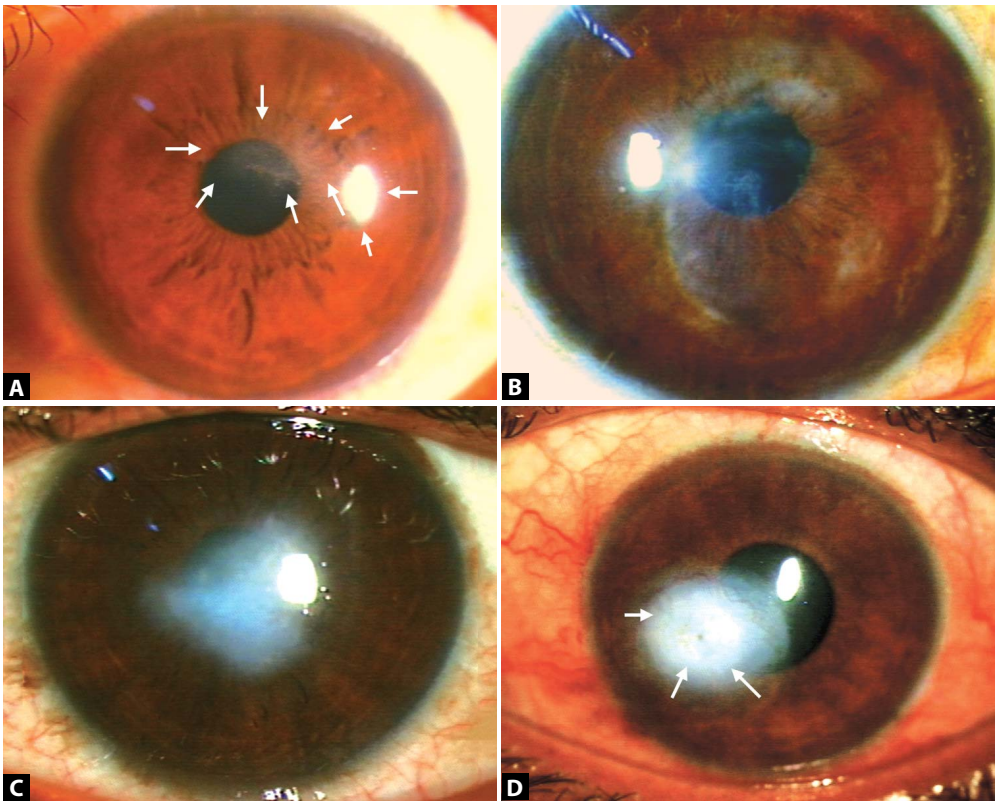


Fig. 13.8: Bacterial corneal ulcer with hypopyon

Healing of a Corneal Ulcer

- Healing of the corneal ulcer is taken place by the formation of fibrous tissue.
- The new fibers are not arranged regularly as in normal lamellae, and they refract the light irregularly. Therefore, the scar is more or less opaque.
- Bowman's membrane is never regenerated, and once it has been damaged, it leaves some degree of permanent corneal opacity.
- Scar tissue may not fill the gap exactly as level of surface, which is then little flattened—leading to the formation of ***corneal facets***.
- If the corneal scar is thin, the resulting opacity is slight, it is called ***nebular***. If rather more dense—it is called ***macular***, and if still more dense and white—it is called a ***leucomatous*** (Figs 13.9A to C).
  Old central leucoma, sometimes shows a horizontal pigmented line (Hudson-Stahli's line) at the palpebral aperture.
- A thin, diffuse nebula on the pupillary area, interferes more with the vision than a strictly localized dense leucoma, so long as the latter does not block the whole pupillary area. The reason is, leucoma stops all the light which fall upon it, whereas the nebula refracts the light irregularly causing an irregular astigmatism.

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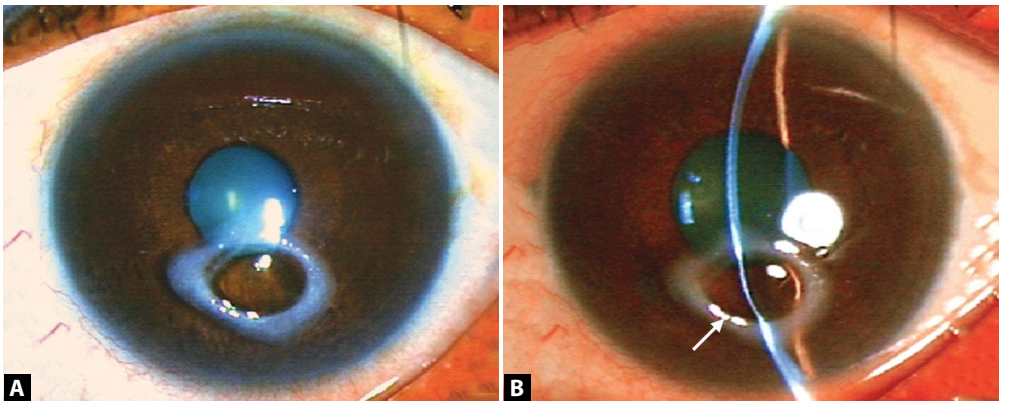


Figs 13.9A to D: Corneal opacity. **A.** Nebular (arrows); **B.** Macular; **C.** Leucomatous; **D.** Adherent leucoma (arrows)

Complications

- ***Ectatic cicatrix:*** Thinned, scarred cornea bulges under the influence of normal IOP.
- ***Descemetocele (Figs 13.10A and B):*** Herniation of the elastic Descemet's membrane through the ulcer as a transparent vesicle. It may persist, surrounded by a white cicatricial ring, or it may eventually rupture.
- ***Perforation and its effects***
  - It is caused by sudden exertion (sneezing, coughing, etc.) → acute rise in IOP → weak floor of the ulcer is unable to support this pressure → gives way.
  - Once the ulcer perforates → sudden escape of aqueous → sudden fall in IOP → iris-lens diaphragm moves forwards and comes in contact with the back of the cornea.
  - ***The advantages of perforation***
    - Pain is alleviated due to lowering of IOP.
    - Better nutrition, and more antibody or antibiotics will reach the ulcer area due to diffusion of fluid.
    - Rapid healing of the ulcer.
  - ***Complications of perforation:*** They vary according to the location and the size of the perforation.
    - ***Anterior synechia:*** If the perforation is very small, the iris becomes gummed down the opening, and the adhesion organizes.

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Figs 13.10A and B: Descemetocele (arrow)

- ***Iris prolapse:*** If the perforation is large and peripheral or paracentral (Figs 13.11 and 13.12A and B).
- ***Adherent leucoma:*** Due to healing of the perforated ulcer with prolapsed iris (Fig. 13.9D).
- ***Anterior staphyloma (Figs 13.13 and 13.14):*** In case of large perforation → total prolapse of the iris → pseudocornea formation → organization to form cicatrix in which the iris remains incarcerated → whole thing bulge out due to high IOP.
- ***Phthisis bulbi (Fig. 13.15):*** In case of large perforation → extrusion of the

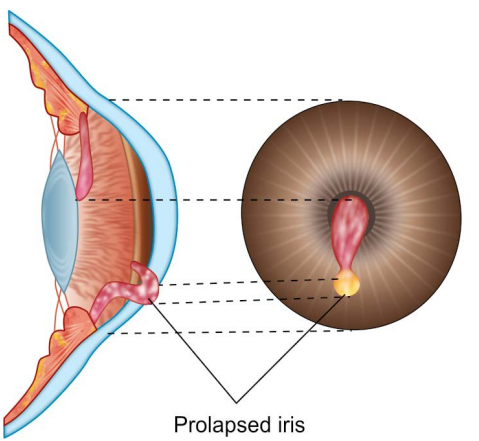
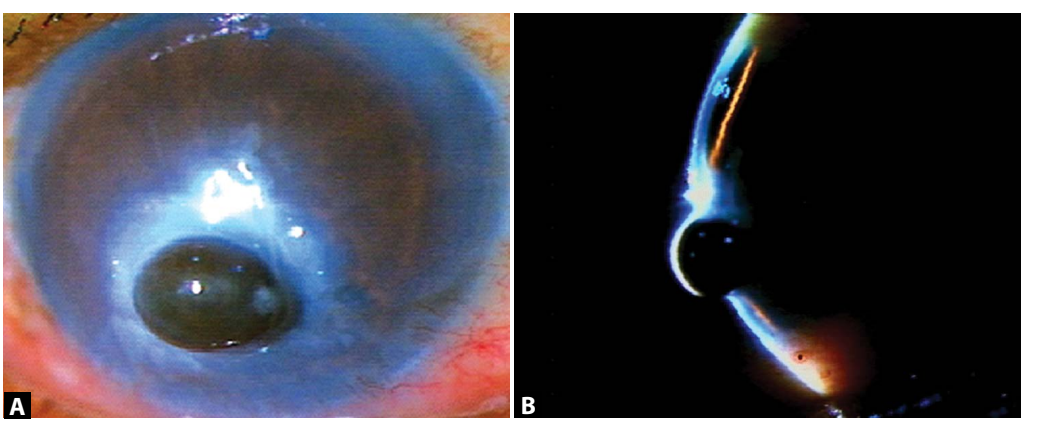


Fig. 13.11: Corneal ulcer with perforation



Figs 13.12A and B: Perforated corneal ulcer with iris prolapse

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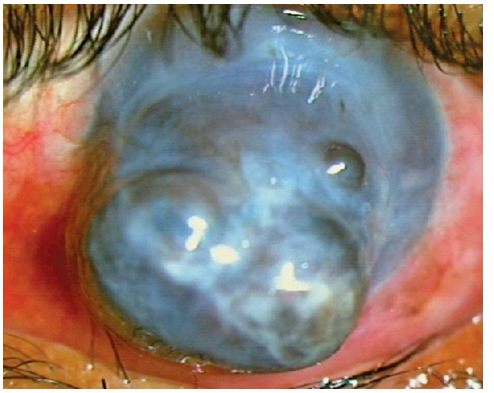


Fig. 13.13: Partial anterior staphyloma



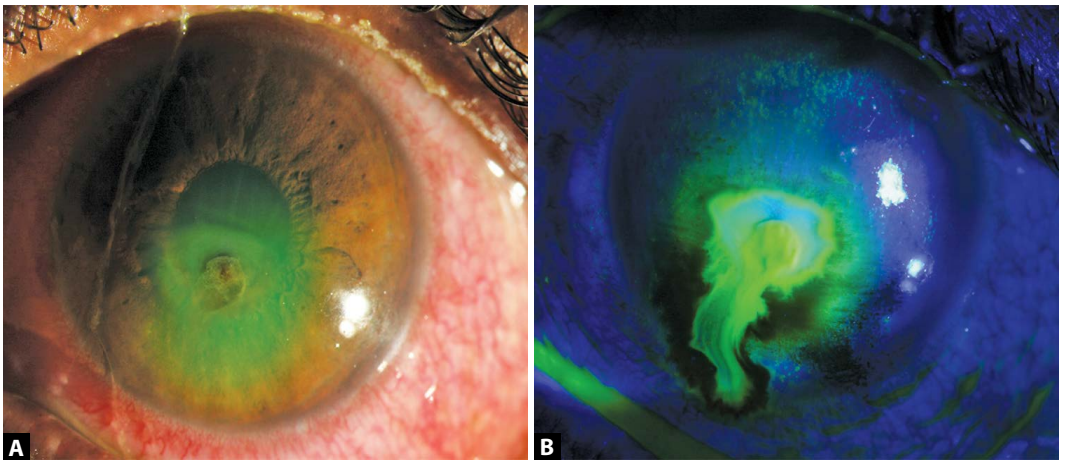
Fig. 13.14: Total anterior staphyloma



Fig. 13.15: Phthisis bulbi

contents of the eyeball → shrinkage of the globe with low IOP.

- ***Subluxation or dislocation of the lens:*** Due to sudden forward movement of the lens.
- ***Anterior capsular cataract:*** When the perforation is at the center and small, the lens remains long in contact with the ulcer.
- ***Corneal fistula (Figs 13.16A and B):*** Due to repeated perforation → sealing of perforation by the exudate → re-opening due to strain → fistula formation.



Figs13.16A and B: A. Corneal fistula; **B.** With positive Seidel's test

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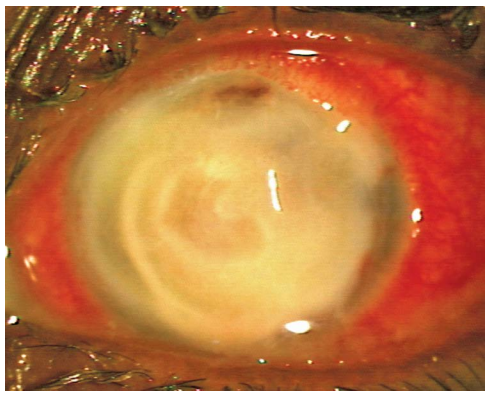


Fig. 13.17: Corneal ulcer—panophthalmitis

- ***Expulsive hemorrhage:*** Due to sudden lowering of IOP → sudden dilatation of the choroidal vessels.
- ***Purulent acute iridocyclitis*** and its complications.
- ***Panophthalmitis (Fig. 13.17):*** Due to spread of infection into the interior of the eye.

Management

A bacterial ulcer is a sight-threatening disease that demands urgent identification and eradication of the causative organism.

This is best performed with hospitalization of the patient.

- ***Identification of the organisms:*** This is done by smear preparation, and culture and sensitivity test of the scrapings, taken from the base and margin of the ulcer as described earlier.

  Meanwhile, the aggressive treatment with antibiotics is started, till the reports are available, when it may be changed accordingly.
- ***Antibiotics***
  - Gram '+ve' organisms usually respond to cephazoline, ciprofloxacin, vancomycin, gatifloxacin and moxifloxacin.
  - Gram '–ve' organisms are sensitive to gentamycin, tobramycin, polymyxin B, amikacin, ciprofloxacin, etc.
  - ***Route of administration***
    - ***Topical commercial preparation:*** The drops are instilled at half hourly intervals round the clock for the first few days. Then, depending upon the response, the frequency of instillation may be reduced.
    - ***Topical fortified drop (concentrated) preparation:*** It is the most effective way to maintain a high and sustained level of antibiotics at the site of infection, e.g. fortified cefazolin, fortified tobramycin. Fortified tobramycin = 2 mL of parenteral tobramycin (80 mg) + 5 mL bottle of commercially available tobramycin eye drop. The resultant solution contains 14 mg/mL, and is stable for upto 30 days.
    - ***Subconjunctival injections*** are for moderate to severe cases. It may be mixed with injection atropine. Gentamycin (20–40 mg) is usually preferred.
    - ***Systemic antibiotics:*** It is usually not necessary except, marginal bacterial ulcer, perforating corneal ulcer, and if the sclera is involved.

There is no need to change the initial antibiotics, if the response is good. But, if it is not so, the subsequent therapy is depended upon the sensitivity pattern.

- ***Atropine sulfate (1%)*** eye drops, 3 times daily to prevent ciliary spasm, and to control iritis.
- ***Hot fomentation*** is to improve circulation and to relieve pain.
- ***Analgesics with antacids*** for pain.
- ***Removal of local predisposing factor*** if any, like trichiasis, foreign body, chronic dacryocystitis, etc.
- ***Tab acetazolamide*** or ***timolol maleate eye drop (0.5%)*** to control IOP in selected cases.
- If the ulcer does not respond with these treatment, ***debridement*** and ***cauterization***

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of the ulcer under topical anesthesia may be done.
  - ***Debridement*** means removal of necrotic material by scraping the floor with a spatula or no. 15 Bard-Parker blade.
  - ***Cauterization*** is done by pure carbolic acid or by iodine (1% iodine in potassium iodide solution).
  - Carbolic acid penetrates more deeply, and extends its antibacterial property more widely.
- If the ulcer still does not respond, and if there is a threat of perforation—***therapeutic penetrating keratoplasty*** is the best choice.
- ***Treatment of complications***
  - ***Descemetocele:*** Rest and pressure bandage, followed by therapeutic keratoplasty.
  - ***If the ulcer perforates:*** Alternate pressure bandage and medical treatment, or temporary tarsorrhaphy.
    In case of small or impending perforation—tissue adhesive with bandage contact lens (BCL).
  - ***In leucoma or adherent leucoma:*** Penetrating keratoplasty.
  - ***A leucoma with no personalized lamellar (PL):*** Treated either by tattooing or cosmetic contact lens.
  - ***Anterior staphyloma:*** Staphylectomy or enucleation, then artificial eye.
  - ***Panophthalmitis:*** Evisceration and then artificial eye.
  - ***Corneal fistula:*** (i) Cyanoacrylate glue (tissue adhesive) application with BCL; (ii) Penetrating keratoplasty.

Corneal abscess (Fig. 13.18): It is localized collection of pus in the cornea. Here, the epithelium is usually intact.

It is similar to corneal ulcer. But in the treatment—evacuation of pus is done first, by a fine needle or knife, before starting the topical antibiotic therapy. The response of treatment will then be appreciated.

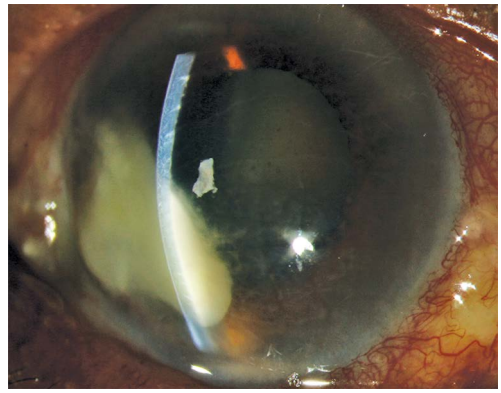


Fig. 13.18: Corneal abscess

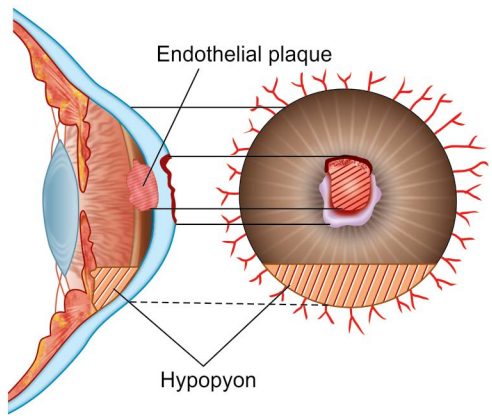


Fig. 13.19: Hypopyon corneal ulcer

HYPOPYON CORNEAL ULCER (ULCUS SERPENS) (FIG. 13.19)

It is a typical bacterial ulcer which has a tendency to creep over the cornea in a serpiginous fashion, and associated with hypopyon and violent iridocyclitis.

Etiology

- It occurs commonly in old debilitated persons or the alcoholics.
- ***Causative agent***—*Pneumococcus* in most of the cases.
- Source of infection—mostly chronic dacryocystitis.

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Symptoms

Same as the bacterial corneal ulcer, but there is remarkably little pain during the initial stage, hence, the treatment is often unduly delayed.

Signs

- Grayish-white, disc-shaped ulcer near the center of the cornea.
- The ulcer is more at the edges than at the center, and is particularly well-marked in one specific direction.
- A cloudy-gray area surrounds the ulcer area and again, more marked in the same direction.
- On one side the ulcer spreads, while on the other side it may be undergo simultaneous cicatrization, and the edges may be covered by fresh epithelium.
- The whole cornea may become hazy.
- Violent iridocyclitis with a definite hypopyon.
- Conjunctival and ciliary congestion.
- Lids are edematous.

It must be noted that any corneal ulcer may be associated with hypopyon. But a corneal ulcer with hypopyon (due to other cause) is not equivalent to hypopyon corneal ulcer or ulcus serpens which has the above typical features (Fig. 13.20).

Corneal ulcer with hypopyon is found in:

- Pneumococcal infection
- *Pseudomonas pyocyanea*
- Gonococcal, Staphylococcal, Streptococcal, *Moraxella*, etc. infection
- Mycotic hypopyon corneal ulcer, as with *Aspergillus, Fusarium* or *Candida* species.

Course

- Great tendency for early perforation of the cornea with its sequelae.
- Hypopyon increases in size very rapidly. leading to secondary glaucoma.
- Ultimately, panophthalmitis may occur.

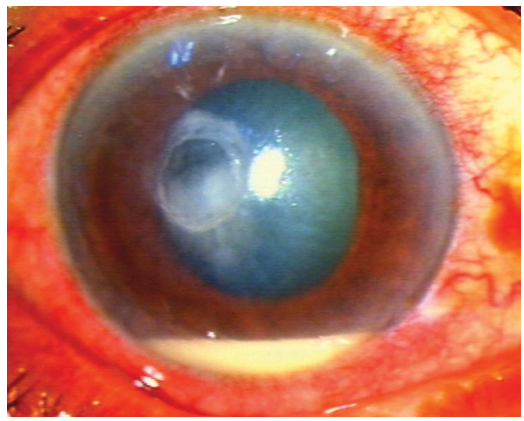


Fig. 13.20: Hypopyon corneal ulcer—bacterial

Treatment

Treatment is almost same as the treatment of a corneal ulcer. Special points are:

- Overhanging edges of the ulcer are due to be excised, and the floor is to be scraped with a spatula.
- Paracentesis to evacuate the pus if the hypopyon is massive.
- Secondary glaucoma is treated with anti-glaucoma agent like Tab. acetazolamide and timolol maleate (0.5%) eye drop.
- If there is any evidence of chronic dacryocystitis:
  - Temporary punctal cautery
  - Dacryocystectomy.

MYCOTIC KERATITIS, KERATOMYCOSIS OR FUNGAL CORNEAL ULCER

Causative Agents

- ***Filamentous fungus:*** *Aspergillus* and *Fusarium* are common in our country. They are most prevalent among agricultural workers.
- ***Yeast:*** *Candida albicans*, frequently affects the immunocompromized host.

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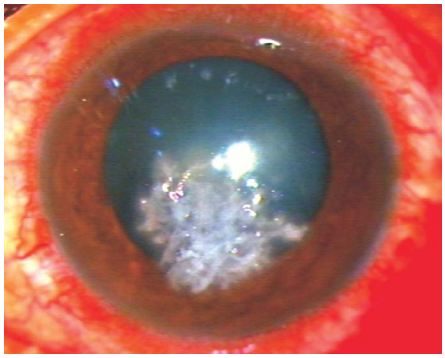


Fig. 13.21: Fungal keratitis by filamentous fungus

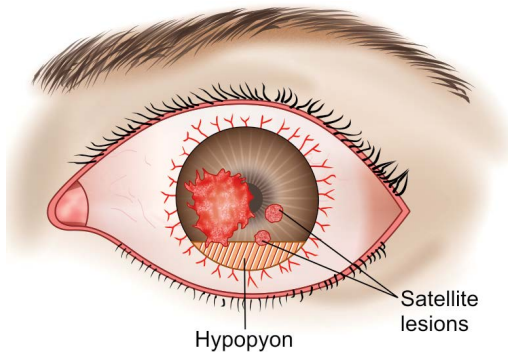


Fig. 13.22: Fungal corneal ulcer

Predisposing Factors

- Same as bacterial keratitis.
- Indiscriminate use of topical steroids.
- Immunocompromized subjects.

Mode of Infection

Mycotic keratitis is typically preceded by ocular trauma (often trivial in nature), mainly by agricultural and vegetable matters.

Symptoms

Same as for the bacterial ulcer, but the symptoms are less prominent than an equal-size bacterial ulcer.

Signs

- Dry looking, yellowish-white lesion with indistinct margin.
- Filamentous fungus keratitis has delicate, feathery, finger-like projection into the adjacent stroma (Fig. 13.21). It may be surrounded by grayish-halo and mutiple satellite lesions (Fig. 13.22). Sometimes, it may be ring-shaped (Fig. 13.23).
- Candida keratitis—typically appears as collar-button abscess without any feathery edge (Fig. 13.24)
- The overlying epithelium is elevated and may be intact.

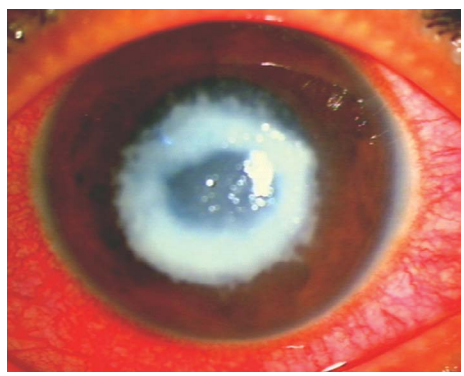


Fig. 13.23: Ring-shaped fungal corneal ulcer

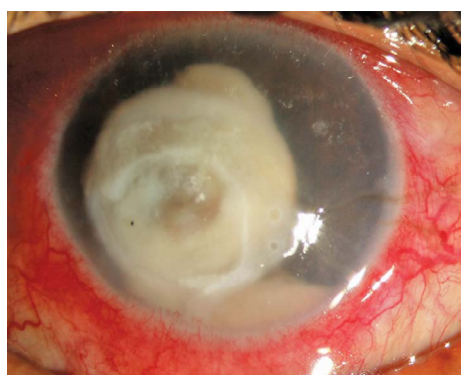


Fig. 13.24: Fungal keratitis by *Candida*

- Some degree of iridocyclitis and massive dense hypopyon are common. This hypopyon is immobile with upper convex border (Fig. 13.25).

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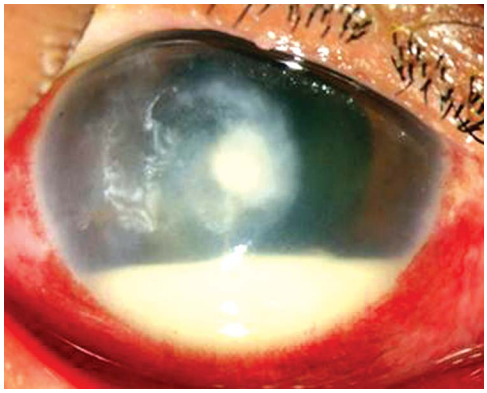


Fig. 13.25: Fungal keratitis—convex hypopyon

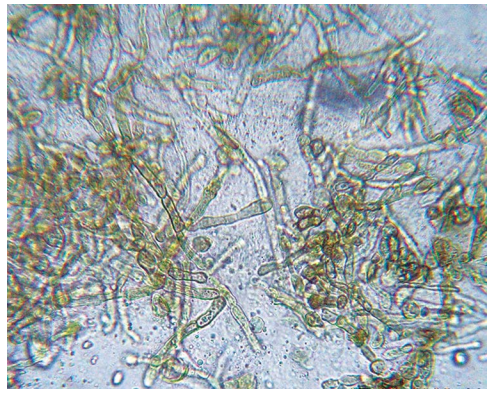


Fig. 13.26: Filamentous fungus in KOH mount preparation

Unlike bacterial ulcer, the hypopyon may ot be sterile, as the fungi can penetrate the Descemet's membrane into the anterior chamber even without perforation.
- Vascularization usually does not occur.

Investigations

- Ten percent potassium hydroxide (KOH) mount preparation of the smear, to demonstrate causative fungus microscopically (Fig. 13.26).
- Culture in Sabouraud's media.

Treatment

- Scraping and debridement of the ulcer.
- Atropine eye ointment—3 times daily.
- ***Antifungal drugs***
  - ***Topical***
    - Natamycin (5%) eye drop, 1 hourly. It is effective against most common fungi mainly *Fusarium* and *Aspergillus spp*.
    - Topical amphotericin B (0.15–0.25%), 1 hourly, effective against *Aspergillus* and *Candida*.
    - Voriconazole eye drop—1–2 hourly. It is more effective against *Aspergillus.*
    - Nystatin eye ointment, 5 times daily. It is only effective against *Candida*, and less potent.

    Topical antifungals are to be instilled for a long time, as the response is often delayed.
  - ***Systemic:*** If the ulcer is deep, or marginal, or if it is perforated, Tab. ketoconazole or Tab. fluconazole may also be tried for 2–3 weeks.
- Cauterization of the ulcer may be done in non-responsive cases.
- Therapeutic full-thickness keratoplasty is much better solution in fungal keratitis.

ACANTHAMOEBA KERATITIS (FIG. 13.27)

Acanthamoeba is a free-living protozoa which has a trophozoite and a cystic form. It is found in stagnant water (pond, swimming pool, bath tub, etc.) and contact lens solution and contact lens cases.

Modes of Infection

- ***Contact lens wearer***—via contaminated solution.
- ***Non-contact lens wearer***—direct contamination after a minor trauma.

Clinical Features

- Frequently misdiagnosed as herpetic or fungal keratitis for many weeks.

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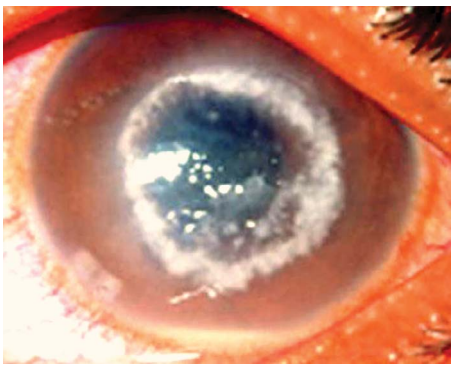


Fig. 13.27: Acanthamoeba keratitis

- Severe pain, and it is out of proportion to the degree of inflammation (radial keratoneuritis).
- Progressive chronic stromal keratitis with recurrent breakdown of corneal epithelium.
- Frequent development of paracentral ring-shaped ulcer or abscess which may perforate.
- A nodular scleritis is a frequent finding.
- It may be associated with limbal inflammation (limbitis).
- It does not respond to conventional antimicrobial treatment.

Diagnosis by calcofluor white staining of the smear for amoebic cyst. Acanthamoeba is cultured in special media—*E. coli* enriched nutrient agar plate.

Treatment

- Propamidine isethionate (Brolene) (0.02%) drops and ointment
- Neomycin drops and ointment
- Polyhexamethylene biguanide (PHMB) (0.001%) drops is also be helpful
- Chlorhexidine (0.02%) eye drop (prepared from mouthwash)—1 hourly
- In resistant cases, a therapeutic penetrating keratoplasty may be required.

VIRAL KERATITIS

Herpes Simplex Viral Keratitis

Infection with herpes simplex virus (HSV) is extremely common, and in the majority of the cases, it is subclinical.

Herper simplex virus (HSV) is of two types:

1. ***HSV-1*** causes infection above the waist (lips and eyes), and is acquired by kissing.
2. ***HSV–2*** causes infection below the waist (genital herpes), and is acquired venereally.

Primary infection may be subclinical or may cause mild ocular problems.

Following the primary infection, the virus travels upto the trigeminal ganglion, and lies in a latent state.

During recurrences, the virus travels, down along the sensory nerve to its target tissues, and causes recurrent lesions.

Predisposing factors for recurrences

- Poor general health
- Common cold and fever
- Menstruation
- Mild trauma
- Topical and systemic corticosteroids
- Immunosuppressive agents, etc.

Primary Herpetic Keratitis

- Typically it occurs between 6 months to 5 years of age.
- The typical lesion is an acute follicular conjunctivitis.
- Fine epithelial keratitis may be present, which sometimes progresses into dendritic figure.
- Vesicular eruptions and edema of the lids.
- It seldom causes serious problem, and is treated by acyclovir eye ointment, five times a day for 3 weeks.

RECURRENT HSV KERATITIS

Dendritic Keratitis (Fig. 13.28)

Symptoms

- Acute pain, redness and lacrimation.
- Photophobia and blurring of vision.

Signs

- Circumciliary congestion.
- Initially, numerous whitish plaques of epithelial keratitis appear on the cornea,

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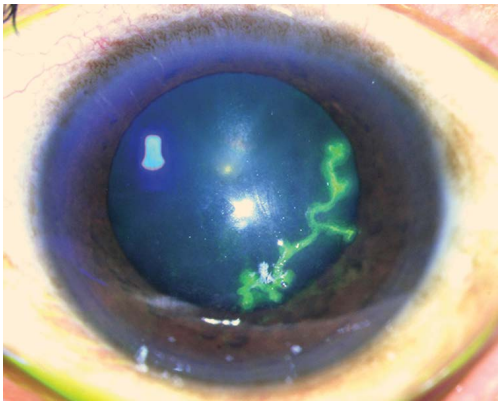


Fig. 13.28: Herpes simplex virus dendritic keratitis

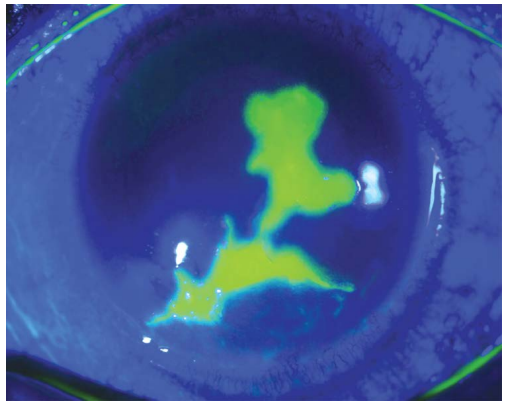


Fig. 13.29: Herpes simplex virus geographical keratitis

known as superficial punctate keratitis (SPKs), and they quickly desquamate to form erosions.

- These erosions coalesce with each other and spread in all directions, and send out lateral branches with knobbed ends, to form 'dendritic' or 'tree-like' figure and this is pathognomonic.
- The bed of the ulcer stains with fluorescein, and diseased cell at the margin takes up Rose-Bengal stain.
- Corneal sensation is diminished or absent.

Geographical Keratitis (Ulcer) (Fig. 13.29)

This is much larger epithelial lesion with typical 'geographical' or 'amoeboid' configuration which occurs as a continued enlargement of dendritic ulcer.

This is likely to occur following inadvertent use of topical steroids.

Treatment

- Debridement of the ulcer.
- ***Antiviral agents (anyone)***
  - Acyclovir (3%) eye ointment, 5 times daily for 14 days.
    - It is the drug of choice.
    - Prolong use of acyclovir ointment causes superficial punctate keratopathy.
  - IDU (idoxuridine) 0.1% drop, every hour during day time, and every 2 hours (or ointment) during night.

    or

    IDU (0.5%) ointment, 5 times a day.
  - Trifluorothymidine (TFT) (1%) drop, every 2 hours during the day. It is more toxic than acyclovir.
  - Adenine arabinoside (Ara-A) (3%) ointment, 5 times daily. It is as potent as IDU but less toxic.
  - Oral acyclovir (200–400 mg), 1 tab 4 times daily for 5 days may be added.
- Atropine (1%) or homatropine (2%) eye drop, 2–3 times daily.
- Improvement of general health.
- Cryocautery or iodine cautery of the ulcer in resistance cases.

Stromal Necrotic Keratitis

- Caused by active viral invasion and destruction.
- Cheesy and necrotic appearance of the stroma.
- Associated with anterior uveitis, called herpetic keratouveitis.

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- Vascularization, scarring and even perforation may occur.
- ***Treatment*** by oral acyclovir, topical antiviral, atropine and judicious use of corticosteroids.

Metaherpetic Keratitis (Trophic Ulcer)

- It is due to persistent defects in the basement membrane of the corneal epithelium.
- It is not an active viral disease.
- The margin is gray and thickened due to heaped-up epithelium.
- ***Treatment*** by artificial tears, patching or bandage contact lens.

Disciform Keratitis (Figs 13.30A and B)

Disciform keratitis is a deep keratitis with disc-like edema, mainly caused by herpes virus (and sometimes due to vaccinia and herpes zoster virus).

Pathogenesis

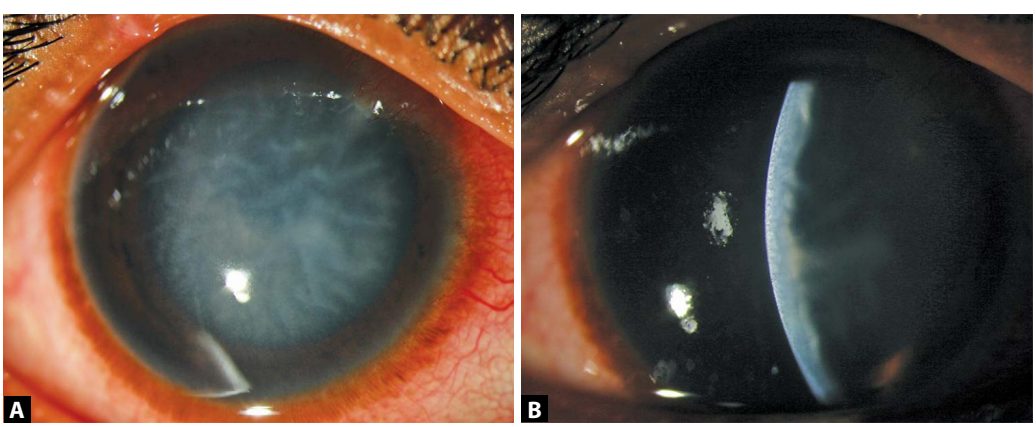
A delayed type of hypersensitivity reaction to HSV-antigen → low grade stromal inflammation with damage to the underlying endothelium → passage of aqueous into the corneal stroma.

Clinical Features

- ***In mild form***, only focal disc-shaped stromal edema with fine keratic precipitates (KPs). There is no necrosis or no neovascularization.
- ***In severe form***
  - Stromal edema is more diffuse
  - Presence of Descemet's folds
  - Deep vascularization
  - Focal bullous keratopathy
  - Wessely's immune ring surrounding the edema
  - Marked anterior uveitis.
  - Absent or diminished corneal sensation.

Treatment

- The first aim is to heal any associated epithelial lesion.
- Topical corticosteroid drops (4–5 times daily) under antiviral cover (acyclovir ointment 2–5 times daily) are given. The use of diluted steroid drops reduces the incidence of any steroid-related complication.
- Cycloplegic, like atropine eye ointment.
- In majority of the cases, it resolves over a period of several weeks.



Figs 13.30A and B: Herpes simplex virus disciform keratitis (slit section)

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HERPES ZOSTER OPHTHALMICUS

- Herpes zoster ophthalmicus (HZO) is a common infection caused by the varicella-zoster virus.
- The infection mainly affects the elderly patients.
- It is more common in immunocompromised hosts (as in HIV).

Pathogenesis

- After initial exposure, the virus remains latent in the trigeminal ganglion.
- Under the stress, it becomes reactivated, replicates, and migrates down the ophthalmic division of the trigeminal nerve to develop ocular complications.

Hutchinson's sign (rule) (Fig. 13.31): When the tip of the nose is involved, the eye will also be involved, since both are supplied by the nasociliary nerve.

Symptoms

- Vesicular eruptions around the eye, forehead and scalp.
- Severe pain along the distribution of the ophthalmic division of 5th nerve.
- Photophobia and lacrimation.

Signs

There are three stages:
Stage I: Acute lesions—which develops within 3 weeks.
Stage II: Chronic lesions—may persist for upto 10 years.
Stage III: Recurrent lesions—which may reappear after 10 years.

Stage I: Acute Ocular Lesions

- ***Lids*:** Redness, edema and vesicular eruptions.
- ***Conjunctiva:*** Acute mucopurulent conjunctivitis.
- ***Sclera:*** Episcleritis and scleritis.
- ***Cornea***
  - Punctate epithelial keratitis.
  - ***Microdendrites:*** Small, fine, multiple dendritic or stellate lesions

D/D from HSV dendrite—in herpes zoster, the dendrites are peripheral, broader, plaque-like, raised from the surface, more frequently stellate-shaped, and without any terminal knobs.

  - ***Nummular keratitis (Fig. 13.32):*** Multiple granular lesions, surrounded by a halo of stromal haze.
  - ***Disciform keratitis.***
  - Sensation is diminished or absent.



Fig. 13.31: Herpes zoster ophthalmicus—Hutchinson's sign

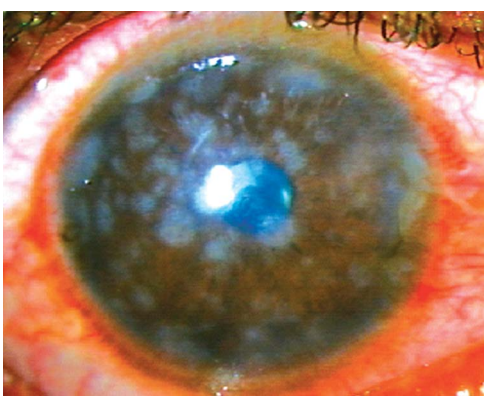


Fig. 13.32: Nummular keratitis

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- ***Iris:*** Acute iridocyclitis with hyphema (hyperacute iritis) and patches of iris atrophy.
- ***Intraocular pressure may be raised.***
- ***Retina:*** Acute retinal necrosis.
- ***Neuro-ophthalmological:*** Optic neuritis and cranial nerve palsies—affecting the 3rd (most common), 4th and 6th nerves.

Stage II: Chronic Ocular Lesions

- Ptosis, due to scarring of the lid
- Trichiasis, entropion and lid notching may also occur
- Mucus-secreting conjunctivitis
- Scleritis
- Nummular and disciform keratitis
- Seven percent of the patients present with postherpetic neuralgia, which is worse at night and aggravated by touch and heat.

Stage III: Recurrent Ocular Lesions

Like episcleritis, scleritis, mucus-plaque keratitis, nummular keratitis, iritis and secondary glaucoma.

Treatment

- ***Oral acyclovir:*** 800 mg 5 times daily for 7 days. It reduces pain, accelerates healing, and curtails vesiculation. It is also effective in anterior uveitis. It has no benefit on postherpetic neuralgia.
- Pain is relieved by ***strong analgesics***, or even by injection pethidine.
- ***Antibiotic-corticosteroid preparation*** for skin lesions. Calamine lotion promotes crust formation and is better avoided.
- ***Topical steroids*** in presence of corneal involvement, and anterior uveitis or scleritis.
- ***Topical antibiotics*** to prevent secondary infection.
- ***Cycloplegic for iritis or severe keratitis.***
- ***Systemic steroids***—indications:
  - To reduce the severity of postherpetic neuralgia
  - Optic neuritis
  - Cranial nerve palsy.
- ***Artificial tears and bandage soft contact lens*** for persistent epithelial defects.
- ***Penetrating keratoplasty*** in case of dense scarring of the cornea.

LAGOPHTHALMIC KERATITIS EXPOSURE KERATITIS

This is due to exposure of the cornea when it remains insufficiently covered by the lids.

- The epithelium of the exposed cornea becomes dessicated and the stroma becomes hazy.
- Finally, owing to drying, the epithelium is cast off, and the raw area may be invaded by the micro-organisms.

Clinical Features

- It ranges from minimum epithelial erosions to severe ulceration, secondary infection and even perforation.
- The lower-third of the cornea is commonly affected, as this part remains more exposed (Figs 13.33A and B).

Other features and treatment have already been described earlier (*see* page 122).

NEUROTROPHIC KERATITIS

- It occurs in an anesthetic cornea.
- It appears that corneal sensation is very important to maintain the health of the corneal epithelium.
- The loss of sensory influence alters the metabolic activity of the epithelium, thereby causes edema and exfoliation of the epithelial cells.

Etiology

- ***Congenital:*** Very rare. Riley-Day syndrome, anhidrotic ectodermal dysplasia, congenital insensitivity to pain may be the cause.
- ***Acquired***
  - Section or affection of the 5th nerve
  - Herpes simplex virus and herpes zoster virus keratitis

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Figs 13.33A and B: Bell's palsy with corneal ulcer—right eye

- Diabetes mellitus
- Leprosy.

Neuroparalytic keratitis: It is due to paralysis of the motor nerve that closes the eyelids, and is normally a sequel to facial palsy. The picture is similar to 'exposure keratitis'.

Clinical Features

- Punctate epithelial erosions involving the interpalpebral area.
- Exfoliation of the epithelial cells followed by central ulceration.
- Corneal sensation is absent.

Treatment

- Routine treatment of corneal ulcer
- Ointments and patching
- Amniotic membrane transplantation
- Tarsorrhaphy for several months. No anesthesia is required during tarsorrhaphy, as the sensation of the lids and conjunctiva are absent.

ATHEROMATOUS ULCER

- It develops over an old leucoma with degenerative changes.
- It may start spontaneously or after a minor trauma.
- The degenerated tissue breaks away from the surface, and an ulcer develops.
- The ulcer progresses rapidly with a little tendency to heal.
- It gets easily infected and perforation may occur.

Treatment

- Ointments and patching
- Bandage contact lens and tear substitutes
- If the eye is blind and painful—enucleation or evisceration is better.

PERIPHERAL KERATITIS (THINNING)

This is a group of diseases, characterized by keratitis and/or melting of the corneal periphery.

Marginal Keratitis (Catarrhal Ulcer) (Fig. 13.34)

It is caused by hypersensitivity reaction to staphylococcal exotoxins, and prevalent in patients having chronic staphylococcal blepharitis.

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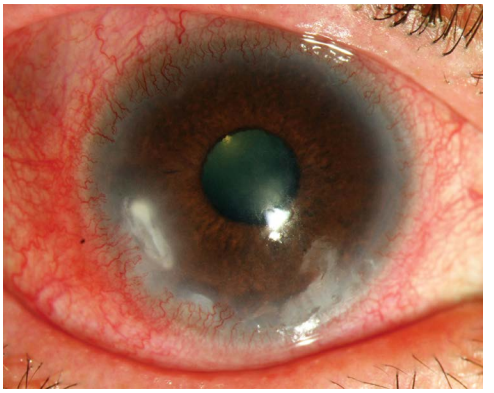


Fig. 13.34: Marginal keratitis

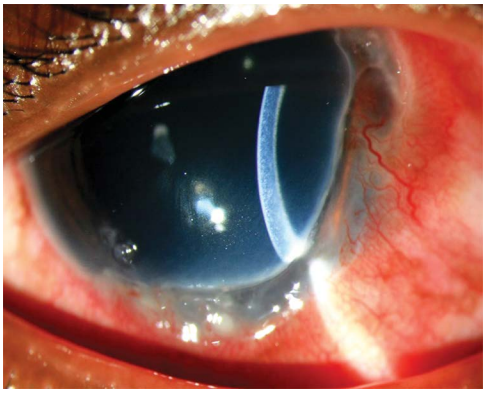


Fig. 13.35: Mooren's ulcer

Symptoms

- Mild ocular irritation
- Lacrimation and photophobia.

Signs

- Subepithelial infiltrates at the periphery, mostly at 4–8 o'clock position, or at 10–2 o'clock position.
- They are separated from the limbus by a clear zone of cornea.
- The lesions spread circumferentially and form a fluorescein staining ulcer.
- Corneal sensation is unaffected.

Treatment

- Topical corticosteroids, 3–4 times daily for a few days.
- Mild cycloplegic, like cyclopentolate or tropicamide.
- Simultaneous treatment of blepharitis to prevent recurrence.

Mooren's Ulcer (Chronic Serpiginous Ulcer) (Fig. 13.35)

It is a chronic progressive peripheral ulcer of unknown etiology.

It may be due to an ischemic necrosis resulting from vasculitis of the perilimbal vessels. Very rarely, it occurs following cataract surgery and hookworm infestation.

There are two types of Mooren's ulcer.

1. ***A limited form:*** Unilateral and usually affects the elderly people.
2. ***A progressive form:*** Bilateral, relentlessly progressive and affects the younger people.

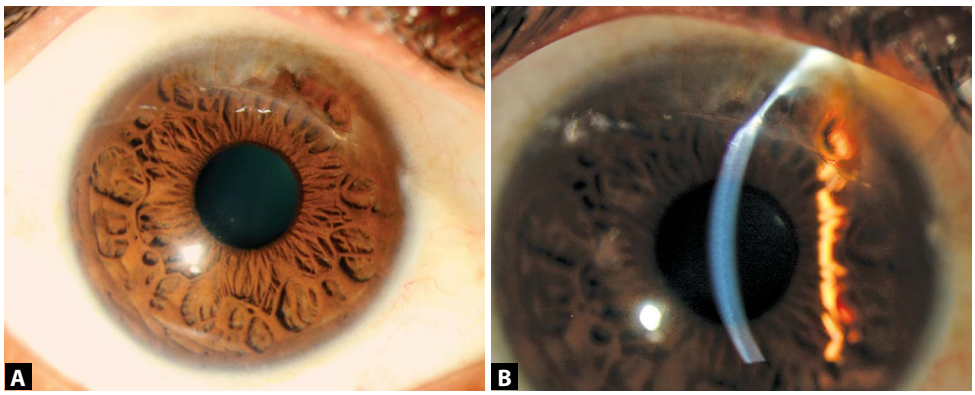
Symptoms

- Severe pain
- Photophobia and lacrimation
- Decreased vision due to irregular astigmatism and involvement of the visual axis in late stage.

Signs

- Ulcer usually starts at the ***interpalpebral area*** as patches of gray infiltrates at the margin.
- It spreads slowly undermining the epithelium and superficial stroma.
- Advancing border of the ulcer is having an overhanging edge.
- Later, it involves the entire circumference of the cornea.
- It also spreads towards center of the cornea, and also invades the sclera.
- Healing takes places from the periphery, and the healed area becomes vascularized, thinned and opaque.
- Perforation may occur with minor trauma.

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Figs 13.36A and B: Terrien's marginal degeneration. **A.** Upper limbus; **B.** Thinning

Treatment

- ***Topical corticosteroids***—at hourly intervals.
- ***Cycloplegics*** like atropine drops.
- ***Conjunctival excision (peritomy):*** A 3 mm collar of conjunctiva is excised from the limbus and parallel to the ulcer.
- ***Immunosuppressive therapy:*** Especially in bilateral cases—where the prognosis is worse.
  - Systemic corticosteroids
  - Cytotoxic drugs, like cyclophosphamide, azothioprim, or methotrexate.
- Bandage soft contact lens
- Lamellar sclerocorneal patch graft, if there is threat of perforation.

Terrien's Degeneration (Figs 13.36A and B)

It is a rare, slowly progressive, bilateral marginal degeneration of the cornea, affecting the adults and elderly people.

Clinical Features

- In has 75% of the patients are male.
- The lesion starts as fine yellow-white punctate stromal opacities at the ***upper part of the cornea***.
- It is separated from limbus by a clear zone.
- Eventually, thinning leads to the formation of a peripheral gutter.
- The sharp-edge towards the center becomes demarcated by yellow-white lipid deposits.
- Overlying epithelium is intact, and does not stain with fluorescein.
- Thinning slowly spreads circumferentially, and vascularization is prominent.
- Vision gradually falls due to increasing corneal astigmatism.
- A few patients develop recurrent episodes of pain.
- Perforation occurs in 15% of cases.

Treatment

- Contact lens may be helpful to correct astigmatism.
- Steroid drops in case of inflammation.
- Penetrating keratoplasty or deep lamellar sector keratoplasty is required in case of perforation.

PHLYCTENULAR KERATITIS (FIG. 13.37)

Although phlyctens are commonly found at the limbus, they may also occur within the

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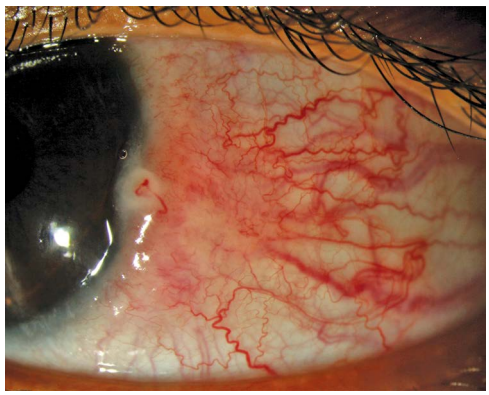


Fig. 13.37: Phlyctenular keratitis

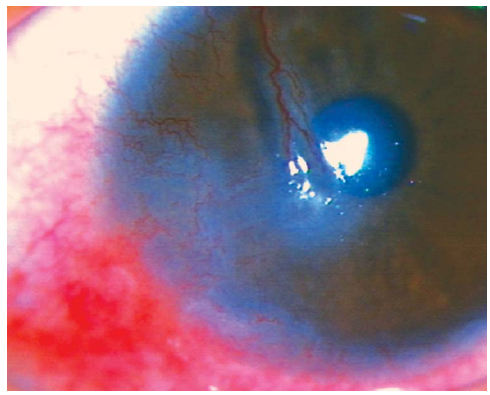


Fig. 13.38: Fascicular ulcer (note the blood vessels)

corneal margin. Like phlyctenular conjunctivitis, it predominantly affects the children.

Etiology: As already discussed in conjunctival disease.

Pathology

- A typical nodule is formed on the Bowman's membrane with adjacent epithelium, as localized lymphocytic infiltrations.
- The epithelium is readily destroyed and a phlyctenular ulcer is formed.
- The denuded surface easily becomes infected, usually by Staphylococci.
- When multiple phlyctens develop at the corneal margin, blood vessels from the limbus grow towards the nodules, thus producing phlyctenular pannus.

Clinical Features

- ***Symptoms*** include—pain, photophobia, lacrimation and blepharospasm.
- Mucopurulent discharge in presence of secondary infection.
- The corneal phlycten is a gray nodule, slightly raised above the surface, and an ulcer is yellow in color.
- It may resolve spontaneously, or may extend radially onto the cornea.
- Very rarely, it may give rise to severe ulceration and perforation.

Complications

- ***Fascicular ulcer (Fig. 13.38):*** Sometimes, phlyctenular ulcer slowly migrates from the limbus towards the center of the cornea in a serpiginous way. It carries a leash of blood vessels which lie in a shallow gutter formed by the ulcer. It is a superficial allergic type of ulcer, which never perforates. When the ulcer heals, the vessels become attenuated with formation of corneal opacity which is densest at its apex.
- ***Ring ulcer:*** It is formed by multiple phlyctens which coalesce together at the corneal periphery. It may lead to total necrosis of the cornea.
- ***Phlyctenular pannus*****:** It is usually associated with multiple phlyctens.
- ***Perforation of the cornea:*** It may occur especially when secondarily infected.

Treatment

- Topical corticosteroids, 4 times daily.
- Atropine (1%) eye drop, 2 times daily.
- Topical antibiotic, if secondarily infected, 4–6 times daily.
- If the fascicular ulcer has reached the pupillary area, it is better to allow it to progress further, as the corneal opacity left by the ulcer tract, is less dense than the site at which the ulcer stops.

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Marginal Corneal Ulcer Associated with Systemic Collagen Vascular Diseases

Marginal corneal ulceration and thinning may occur in four main diseases:

1. Rheumatoid arthritis
2. Systemic lupus erythematosus
3. Polyarteritis nodosa
4. Wegener's granulomatosis.

The peripheral corneal changes are:

- Peripheral keratitis
- ***Peripheral corneal guttering (contact lens cornea):*** Thinning or guttering occurs along the entire corneal periphery. As the central part of the cornea remains of normal thickness, the appearance resembles a hard contact lens placed on the eye—hence, the term
- Peripheral keratolysis, resulting in descemetocele formation and even perforation
- Sclerosing keratitis
- Variable degree of corneal scarring and vascularization
- Other ocular manifestations include—scleritis, choroidal vasculitis, retinal vasculitis, keratoconjunctivitis sicca, anterior ischemic optic neuropathy, etc.

Treatment

- Systemic and topical corticosteroids
- Immunosuppressive agents
- Conjunctival excision (peritomy)
- Peripheral tectonic keratoplasty.

INTERSTITIAL KERATITIS

Interstitial keratitis (IK) is an inflammation affecting chiefly the corneal stroma, without primary involvement of the epithelium or endothelium.

Causes: Three causes are congenital syphilis, tuberculosis, Cogan's syndrome.

Syphilitic Interstitial Keratitis

Pathology

The disease is fundamentally a uveitis, and the keratitis, which clinically masks the uveitis, is secondary. These reactions are largely allergic, since the ***spirochetes*** have never been detected in the cornea or uvea.

Clinical Features

It is a late manifestation of congenital syphilis developing between 5 and 15 years. It is usually bilateral, and unilateral cases occur in delayed type, after the age of thirty, particularly in acquired syphilis.

Symptoms

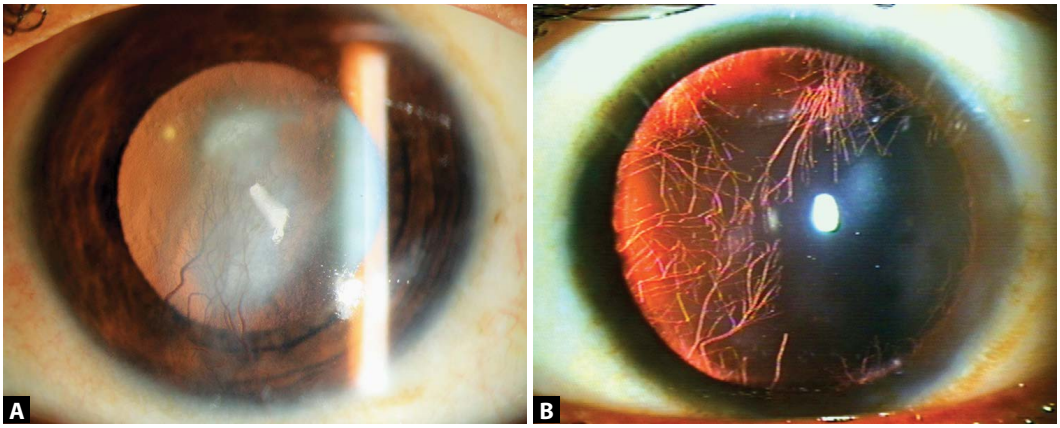
- Initially, irritation and haziness of the cornea.
- Later pain, lacrimation, photophobia and severe blepharospasm with increased haziness of the cornea.
- Profound loss of vision.

Signs

Ocular signs

- Circumcorneal congestion.
- The lesion starts as one or more hazy patches in deeper cornea from the margin towards the center (Fig. 13.39A).
- They fuse together in 2–4 weeks, and the whole cornea becomes hazy with a steamy surface, giving rise to ***ground-glass appearance***.
- Meanwhile, deep vascularization develops, and the vessels meet in the center of the cornea. Since, the vessels are covered by a layer of hazy cornea, they appear as a dull, reddish-pink patch, known as ***salmon patch of Hutchinson***.
- As a rule, there is always an associated iridocyclitis.
- After 2–4 months, the cornea begins to clear from periphery towards the center, and the vessels become nonperfused (only remain as fine lines of ghost vessels) (Fig. 13.39B). If there is re-inflammation of the cornea, the vessels may refill with blood.
- The clearing of the cornea takes many weeks or months, but little improvement

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Figs 13.39A and B: Interstitial keratitis. **A.** Lesion at the center; **B.** Ghost vessels

can be expected after eighteen months. Ultimately, majority of the patients regain some useful vision.

General signs

- ***Hutchinson's triad***
  - Interstitial keratitis (IK)
  - Permanent deafness
  - Hutchinson's teeth (notching of the two upper permanent incisors).
- Prominent frontal eminences
- Flatness of the nasal bridge
- Rhagades at the angle of the mouth
- Shotty cervical lymph nodes
- Periosteal nodules on the tibia.

Treatment

- ***Systemic penicillin:*** Its role is doubtful. It may shorten the course of the disease.
- ***Topical corticosteroids*** as drops or ointment, 4–6 times daily.
- ***Atropine (1%)*** ointment, 2 times daily.
- ***Penetrating keratoplasty***, if there is any permanent dense corneal opacity.

Tuberculous Interstitial Keratitis

This is similar to the syphilitic interstitial keratitis, but is more often unilateral and sectorial.

Treatment is with anti-tubercular drugs, topical steroids and cycloplegics.

Cogan's Syndrome

It is very rare, non-syphilitic interstitial keratitis associated with vestibulocochlear disturbances (tinnitus, vertigo and deafness). It typically affects middle-aged patients.

Treatment is with systemic steroids (to prevent permanent deafness), topical steroids and cycloplegics.

CORNEAL DEGENERATIONS AND DYSTROPHIES

Degeneration and dystrophy are not synonymous. Degeneration means, that the normal cells of a tissue undergo some pathological changes under the influences of some abnormal circumstances. Dystrophy means, the cells have some inborn defects which may cause pathological changes with passage of time (Table 13.2).

DEGENERATIONS

- Arcus senilis
- Band-shaped keratopathy
- Pellucid marginal
- Salzmann's nodular
- Arcus juvenilis
- Lipid keratopathy
- Terrien's marginal
- Limbal girdle of Vogt

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TABLE 13.2: Difference between corneal degeneration and corneal dystrophy

Corneal degeneration	Corneal dystrophy
Usually unilateral and asymmetrical	Usually bilateral and symmetrical
Located peripherally	Located centrally
Accompanied by vascularization	No vascularization
No inheritance pattern	Hereditary (usually autosomal dominant)
Onset in middle life or later	Relatively early in onset
Secondary to some compromising factors, e.g. aging, inflammation, chemicals, trauma or systemic diseases	Unrelated to any systemic or local disease, or condition

Arcus Senilis (Gerontoxon) (Fig. 13.40)

- It is bilateral lipid degeneration of the peripheral cornea, affecting the most elderly persons.
- It starts in the superior and inferior perilimbal cornea, and then progresses circumferentially to form a band.
- The band is about 1 mm wide and its central border is diffuse, whereas the peripheral edge is sharp.
- The peripheral sharp-edge is separated from the limbus by a clear zone of cornea, which may become thinned ***(senile furrow)***.
- ***Histologically:*** The lipid is first deposited anterior to the Descemet's membrane and then in the anterior stroma, just beneath the Bowman's layer.
- It does not affect the vision.

Arcus Juvenilis (Anterior Embryotoxon) (Fig. 13.41)

It is similar to arcus senilis, except it occurs under the age of 40 years. The condition is usually associated with systemic hyperlipidemias with raised serum cholesterol. So, a serum lipid profile is indicated in these patients.

Band-shaped Keratopathy (BSK) (Fig. 13.42)

- It is caused by deposition of calcium salts in the subepithelial layer of the cornea, and characterized by a horizontal band-shaped opacity.

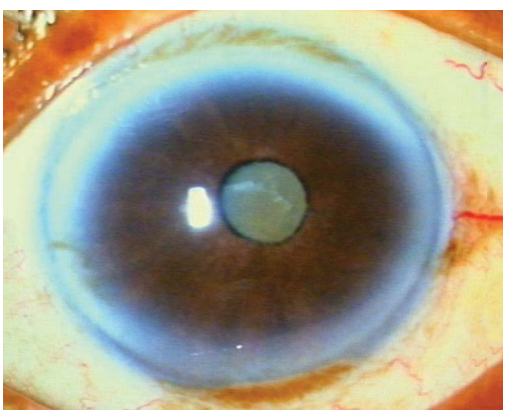


Fig. 13.40: Arcus senilis with mature cataract

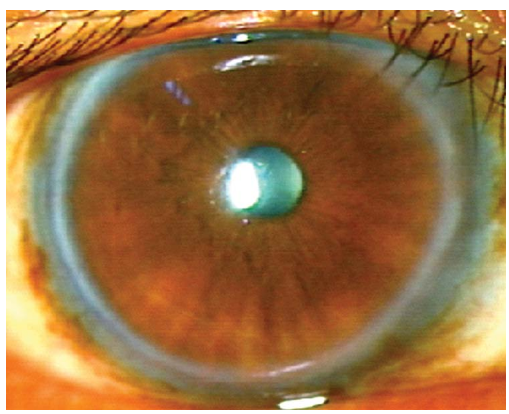


Fig. 13.41: Arcus juvenilis

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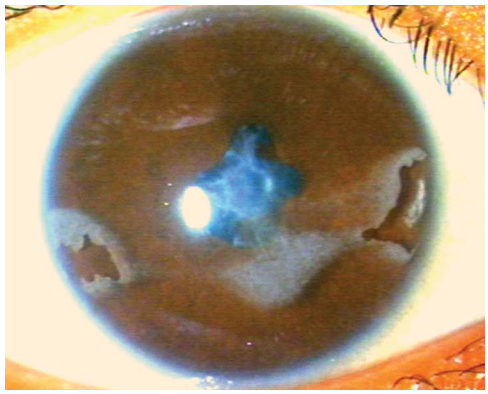


Fig. 13.42: Band-shaped keratopathy

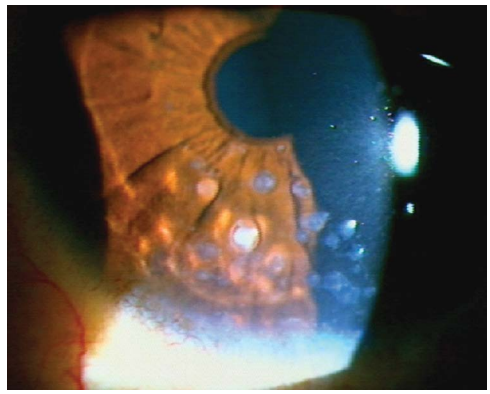


Fig. 13.43: Salzmann's nodular degeneration

- The calcific band is largely at the palpebral fissure, and separated from the limbus by a clear zone.
- The band begins in the periphery at 3 and 9 o'clock position and affects the central area later on.
- There are numerous holes, where the corneal nerves penetrate the Bowman's layer.
- As the band progresses, the epithelium becomes irregular and subsequently, its breakdown may be very painful.
- ***Pathologically:*** The calcium (mainly hydroxyapatite) is deposited in the Bowman's layer, in the epithelial basement membrane, and most superficial part of the stroma. The remaining layers of the cornea are clear.

Etiology

- Childhood chronic iridocyclitis, especially with juvenile rheumatoid arthritis (JRA)
- ***Other ocular conditions:*** Phthisis bulbi absolute glaucoma, interstitial keratitis
- Systemic hypercalcemia
- Idiopathic—in the elderly.

Treatment

- Scraping of the epithelium overlying the opacity, and application of 0.01 M solution of disodium EDTA (ethylenediaminetetraacetic acid) for 10 minutes. Then the eye is patched with antibiotics and cycloplegics till the cornea is re-epithelialized.
- Lamellar keratoplasty for optical reason.

Salzmann's Nodular Degeneration (Fig. 13.43)

- It is characterized by elevated subepithelial bluish-gray nodules (number one to nine), in either scarred cornea or at the edge of transparent cornea.
- The degeneration is unilateral in 80% cases and occurs more often in females.
- It occurs as a late sequel of corneal diseases like phlyctenulosis, trachoma, chronic keratitis, or vernal keratoconjunctivitis.
- Bowman's layer is missing over the nodules and the epithelium is irregular. The nodules represent excessive secretion of basement membrane-like material.
- Usually, there is no need of treatment. Lamellar keratoplasty may be required to improve vision when the nodules are central in location.

Pellucid Marginal Degeneration (Fig. 13.44)

- This is a rare, bilateral, slowly progressive marginal degeneration (thinning) of the cornea between 20 to 40 years of age.

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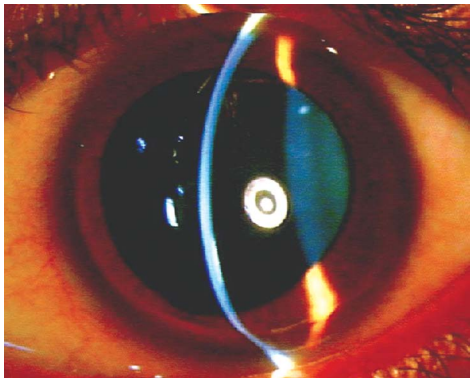


Fig. 13.44: Pellucid marginal degeneration

- The thinning involves only in the inferior cornea, and the ectasia occurs just above the thinned area, giving rise the appearance like keratoconus.
  But in keratoconus the thinning involves at the center and the maximum ectasia is within the area of thinning.
- Fleischer's ring does not occur, but this condition may be complicated by acute hydrops.
- There is no vascularization (unlike Mooren's or Terrien's degeneration) or lipid deposition (unlike Terrien's degeneration).
- ***Treatment*** is usually not required, except correction of astigmatism by rigid gas permeable contact lenses.

DYSTROPHIES

Classification

Anterior dystrophies

Epithelium and Bowman's membrane

- Cogan's microcystic
- Recurrent corneal erosion syndrome
- Reis-Bucklers (Fig. 13.45)
- Meesmann
- Map-dot-fingerprint.

Stromal dystrophies

- Granular
- Macular

Contd...

Contd...

- Lattice
- Central crystalline
- Congenital hereditary stromal.

Posterior dystrophies

- Descemet's membrane and endothelium
- Cornea guttata
- Fuchs' endothelial
- Posterior polymorphous
- Congenital hereditary endothelial.

Ectatic dystrophies

- Keratoconus
- Posterior keratoconus
- Keratoglobus.

Anterior Dystrophies

- These are probably the more common dystrophies, frequently misdiagnosed, mainly due to their variable presentation.
- Most of the patients remain asymptomatic, but the others develop recurrent corneal erosions with photophobia and lacrimation.
- All are autosomal dominant inheritance.
- Visual loss usually occurs due to irregular astigmatism.

Treatment

- Most of them do not require treatment.



Fig. 13.45: Reis-Buckler's dystrophy

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- Recurrent erosions are treated with artificial tears, antibiotic ointment, mild cycloplegic and patching of the eyes.
- Only a few cases may require lamellar keratoplasty.

Stromal Dystrophies

Granular Dystrophy (13.46A)

- Autosomal dominant inheritance.
- Usually starts around puberty and progresses very slowly.
- The lesions appear as discrete, crumb-like white granules within the anterior stroma of the central cornea.
- The peripheral cornea remains always clear.
- The lesions spread into the deeper stroma, and the stroma in between the lesions remains clear.
- The patient remains asymptomatic for many years, although a few patients complain of light scattering.
- ***Histochemically,*** the deposits are hyaline, and stain bright red with ***Masson Trichrome.***
- Visual acuity, however, remains good and penetrating keratoplasty is seldom required.

Macular (Fig. 13.46B)

- Autosomal recessive inheritance.
- The disease starts during early life with ***significant impairment of vision.***
- The lesions are focal, gray-white, poorly defined opacities in cloudy stroma.
- With time the lesions spread to the entire corneal stroma ***including the peripheral cornea.***
- ***Histochemically,*** the deposits are glycosaminoglycans (acid mucopolysaccharide) and stain with ***alcian blue.***
- Most patients require penetrating keratoplasty during early life to improve visual acuity.

Lattice (Fig. 13.46C)

- Inheritance is autosomal dominant.
- Usually presents during early life with recurrent corneal erosions.
- The lesions are branching spider-like deposits which interlace and overlap at different levels within the stroma.
- With time, a diffuse corneal haze develops, and corneal sensation is diminished.
- ***Histochemically,*** the deposits are amyloid, and stain with ***Congo red.***
- Visual acuity may be significantly impaired by the age of 30–40 years.
- Penetrating keratoplasty is then required to improve the vision. ***Recurrence of the dystrophy in the graft is common.***

Posterior Dystrophies

Cornea Guttata (Fig. 13.47)

- This is a common aging process, resulting in focal accumulation of collagen on the posterior surface of the Descemet's membrane.



Figs 13.46A to C: Stromal dystrophies. **A.** Granular; **B.** Macular; **C.** Lattice

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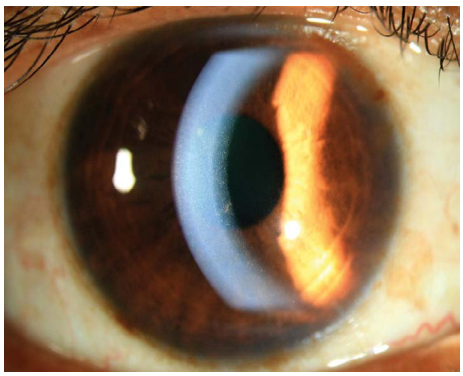


Fig. 13.47: Cornea guttata

- They appear as excrescences or warts of Descemet's membrane, formed by the stressed endothelium.
- They disrupt the normal endothelial mosaic, and appear as dark spots ***(beaten metal appearance)***.
- The corresponding peripheral lesions (Hassal-Henle bodies) are of no significance, but the central corneal lesions (guttata) may occur as a part of early stage of Fuchs' endothelial dystrophy.
- Specular microscopy often helps in arriving at a diagnosis (Figs 13.48A and B).

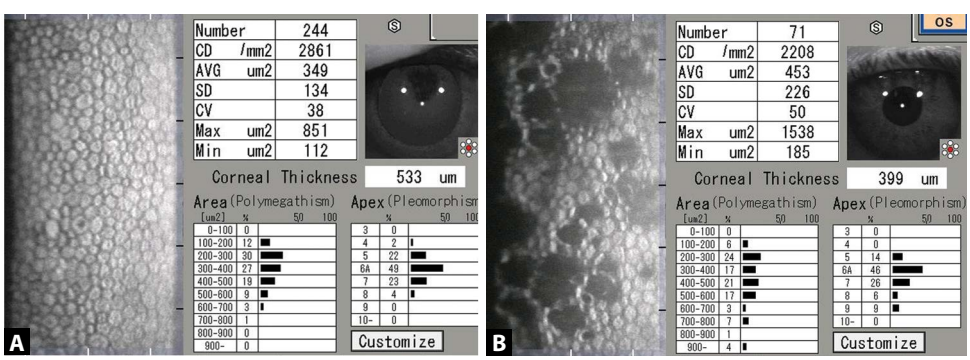
Fuchs' Endothelial Dystrophy (Figs 13.49A to C)

- Usually autosomal dominant inheritance.
- Slowly progressive, bilateral disease affects elderly people, and is more common in females.
- ***Early change—corneal guttata*** without any symptom. They gradually become more numerous and begin to spread towards the periphery.
- ***Endothelial decompensation*** soon starts causing the stroma to become edematous. This gradually increases due to malfunction of the endothelial pump.
- ***Epithelial edema*** gradually develops with increased stromal thickness. Visual acuity is grossly impaired at this stage.
- ***Bullous keratopathy (Fig. 13.48C)*** develops due to persistence of epithelial edema. When the bullae rupture, it causes severe pain, photophobia and lacrimation.

  The other causes of bullous keratopathy:
  - Postsurgical—aphakic bullous keratopathy, pseudophakic bullous keratopathy
  - Absolute glaucoma
  - Chronic iridocyclitis, disciform keratitis
  - Keratoconus (hydrops).

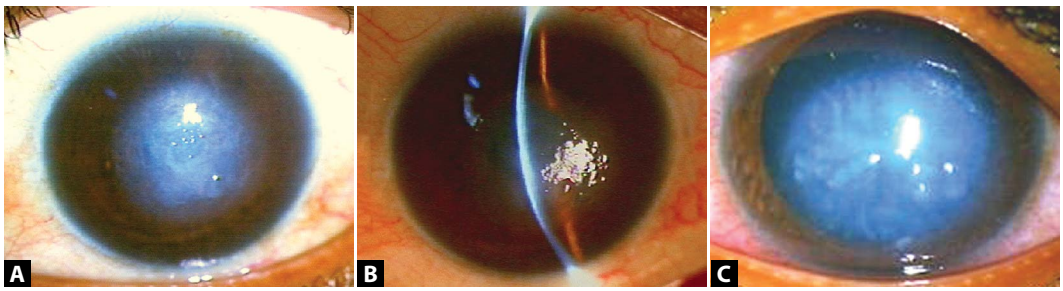
Complications

- As the disease progresses, stromal opacity develops with vascularization. Bowman's layer is replaced by degenerative pannus.
- There is increased prevalence of open-angle glaucoma in patients with Fuchs' dystrophy.



Figs 13.48A and B: Specular microscopic photography. **A.** Normal endothelial cells; **B.** Cornea guttata with low cell count

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Figs 13.49A to C: A and B. Fuchs's dystrophy with corneal edema; **C.** Bullous keratopathy

Treatment

- For early epithelial edema—5% sodium chloride drop (hypertonic saline) for 4–6 times daily and 6% sodium chloride eye ointment at night.
- ***Reduction of IOP*** by timolol maleate (0.5%) drop—2 times daily.
- ***Therapeutic (bandage) soft contact lens*** is effective to control pain by protecting the exposed corneal nerves, and also by flattening the bullae.
- ***Keratoplasty*** is the ultimate choice.

Ectatic Dystrophies

Keratoconus

It is a bilateral conical protrusion of the central part of the cornea with thinning of its central and inferior paracentral areas (Fig. 13.50).

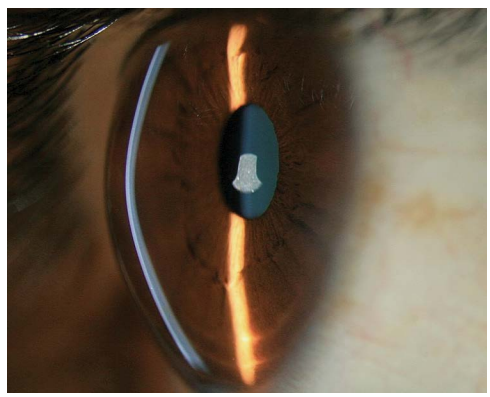


Fig. 13.50: Keratoconus—conical protrusion with thinning

Clinical Features

- It usually starts at around puberty and slowly progressive.
- Family history is usually negative.
- Although bilateral, most of the times, the involvement is largely asymmetrical.

Symptoms

- Initial symptom is impaired vision—(in one eye it is more than the fellow eye), ***due to irregular myopic astigmatism***. This visual loss can only be improved by rigid contact lenses.
- Later on, there is further impairment of vision, and contact lens can no longer correct this visual loss.

Signs

- Irregular retinoscopic reflex.
- Distortion of mires in Placido's disc or keratometer.
- Vertical folds at the level of deep stroma and Descemet's membrane (***Vogt's striae***).
- Prominent corneal nerves.
- Thinning of the central cornea with protrusion just below and nasal to the center.
- ***Munson's sign*** make it 'bulging' or 'tenting' of the lower lids when the patient looks down (Fig. 13.51).
- ***Fleischer's ring:*** Epithelial iron deposition at the base of the cone (Fig. 13.52).

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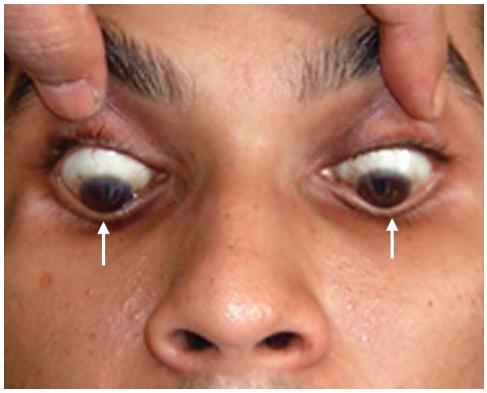


Fig. 13.51: Munson's sign (arrows)

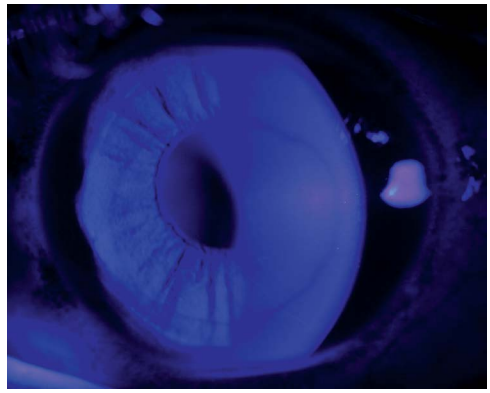


Fig. 13.52: Fleischer's ring under cobalt blue light

Associations

The systemic and ocular associations of keratoconus include—Down's syndrome, Marfan's syndrome, Turner's syndrome, atopic dermatitis, blue sclera, vernal conjunctivitis, retinitis pigmentosa and ectopia lentis.

Complications

- ***Acute hydrops (Fig. 13.53)***—due to sudden rupture of the Descemet's membrane, and acute *see* page 183 of the aqueous into the corneal stroma and epithelium. These breaks usually heal within 6–10 weeks, and the edema gradually clears. But a variable amount of apical stromal scarring is developed.
- Intolerance to contact lens after few years.

Keratoconus does not rupture spontaneously even if there is extreme thinning of the cornea.

Treatment

- ***Rigid contact lenses*** when the spectacles become ineffective. ***A piggy-back contact lens*** may be more effective.
- Five percent sodium chloride drop (hypertonic saline) and patching or soft bandage contact lens to treat acute hydrops.
- ***Penetrating keratoplasty***—indications:
  - When contact lenses can no longer correct high irregular astigmatism.
  - Intolerance to the contact lens.
  - Central apical scarring obstructing the visual axis.

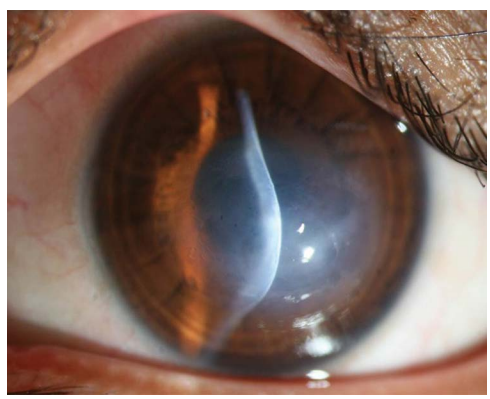


Fig. 13.53: Acute hydrops in keratoconus

The prognosis for keratoplasty is excellent (almost 95% success rate).

- Deep anterior lamellar keratoplasty (DALK) and intrastromal corneal rings (INTACS)—are also promising surgical procedures.
- ***Thermokeratoplasty:*** This is a procedure to shrink the corneal collagen by applying heat at the tip of the cone, and thereby causing flattening of the cone. This is meant for proper fitting of the contact lens. It is not very popular.

Posterior Keratoconus

It is a rare disorder, in which the posterior cornea is having excavation, and the anterior

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cornea does not protrude. The condition gives rise to mild irregular astigmatism and is non-progressive.

Keratoglobus

This is a very rare condition, characterized by thinning and protrusion of the entire cornea. Astigmatism is usually not very irregular, unless scarring has occurred following acute hydrops.

OTHER CORNEAL DISORDERS

Superficial Punctate Keratitis (Figs 13.54 to 13.56)

This is punctate epithelial keratitis, scattered all over the cornea, usually due to a viral infection.

Etiology

- ***Viral infections:*** Herpes simplex, herpes zoster, vaccinia, adenovirus (as a part of epidemic keratoconjunctivitis or pharyngoconjunctival fever), molluscum contagiosum, etc.
- Keratoconjunctivitis sicca (KCS)
- Staphylococcal exotoxin (associated with blepharitis) and Meibomianitis
- Contact lens wearer
- Foreign body in the upper palpebral conjunctiva, e.g. minute iron/dust particle, caterpillar hair, etc.
- Unknown (Thygeson's superficial punctate keratitis).

Pathology: It occurs in the deeper layers of corneal epithelium. Sometimes, it extends



Figs 13.54A and B: Superficial punctate epithelial keratitis. **A.** Schematic representation; **B.** Photograph



Figs 13.55A and B: Subepithelial punctate keratitis. **A.** Schematic representation; **B.** Photograph

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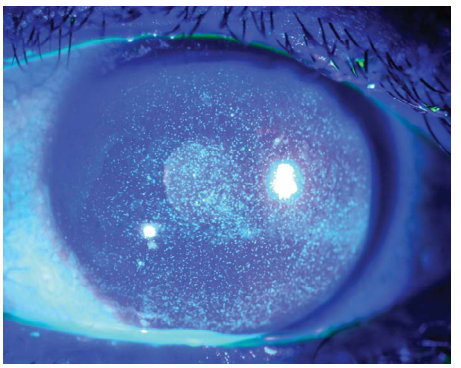


Fig. 13.56: Fluorescein staining in diffuse SPKs

into Bowman's membrane and superficial parts of the stroma (***subepithelial punctate keratitis***).
Symptoms: Irritation, photophobia and lacrimation.

Signs

- The epithelial opacities appear as raised gray dots, scattered all over the cornea.
- The lesions sometimes stain with fluorescein (Fig. 13.56), and always turn bright red with Rose-Bengal.
- Sensation may be diminished (***herpetic***).
- Ciliary congestion.
- Preauricular lymphadenopathy may be present (***adenoviral***).

Treatment

- Antibiotic ointment and cycloplegics in most cases.
- Antiviral drugs, if corneal sensation is diminished.
- Topical steroids, if associated with staphylococcal blepharitis.
- Artificial tears frequently.
- Removal of foreign body, if any.

Superior Limbic Keratoconjunctivitis (Figs 13.57A to C)

- This is a bilateral chronic inflammation of the superior tarsal and bulbar conjunctiva, with edema of the superior limbal conjunctiva.
- The disease is usually bilateral, more in females and with ***thyroid dysfunction***.
- It follows a chronic course with remissions and exacerbations, but the prognosis is ultimately good.
- ***Symptoms*** include, foreign body sensation, photophobia, pain and mucoid discharge.
- ***Signs*** include.
  - Papillary hypertrophy of the superior tarsus.
  - Hyperemia and keratinization of the superior bulbar conjunctiva.
  - Thickening of the conjunctiva at the superior limbus.
  - Superior cornea shows punctate epithelial erosions (stains with fluorescein and Rose-Bengal) and filaments.

A **B** **C**

Figs 13.57A to C: Superior limbic keratoconjunctivitis. **A.** Schematic representation; **B and C.** Photographs

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Treatment

- Artificial tears and soft bandage contact lens.
- Silver nitrate (1%) is applied to the affected conjunctival area.
- Resection of a block of superior bulbar conjunctival tissue.
- Thermocauterization of the superior bulbar conjunctiva.

Photophthalmia (Photokeratitis)

It is a condition characterised by acute multiple erosions of the corneal epithelium after exposure to ultraviolet (UV) rays.

Causes

- Exposure to bright flash of a short-circuit.
- Exposure to welding arc with naked eyes ***(welding keratitis)***.
- Exposure to UV rays, reflected from snow surfaces ***(snow blindness)***.

Clinical Features

- There is a ***latent period of 4–5 hours*** between the exposure and the onset of symptoms.
- ***Symptoms*** include, extreme burning pain, lacrimation and photophobia.
- There is swelling of the lids, conjunctival chemosis and congestion, and multiple erosions of the corneal epithelium.

Treatment

- Cold compress
- Antibiotic ointment and mild cycloplegics, and patching the eyes for 24 hours
- ***Prevention:*** Wearing protective glasses when such exposure is to be anticipated. Crooke's glass, which cuts off UV rays and infrared rays, is very useful in this situation.

Striate Keratopathy (Keratitis) (Figs 13.58A and B)

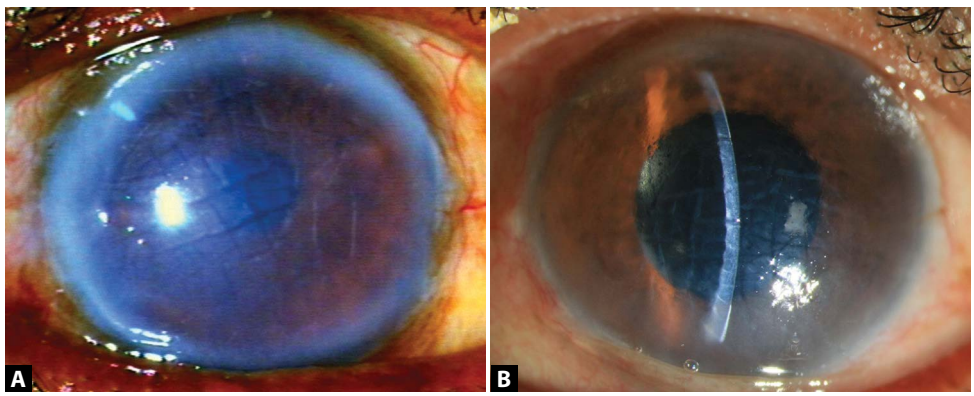
This is basically corneal edema with folds in the Descemet's membrane, which appear as delicate gray lines in the cornea.

Causes

- Following cataract operation—due to endothelial damage by instruments, by IOL, and also by tight suturing.
- Corneal ulcers or wounds—as radial striae around the lesion.
- After tight bandaging of the eye.

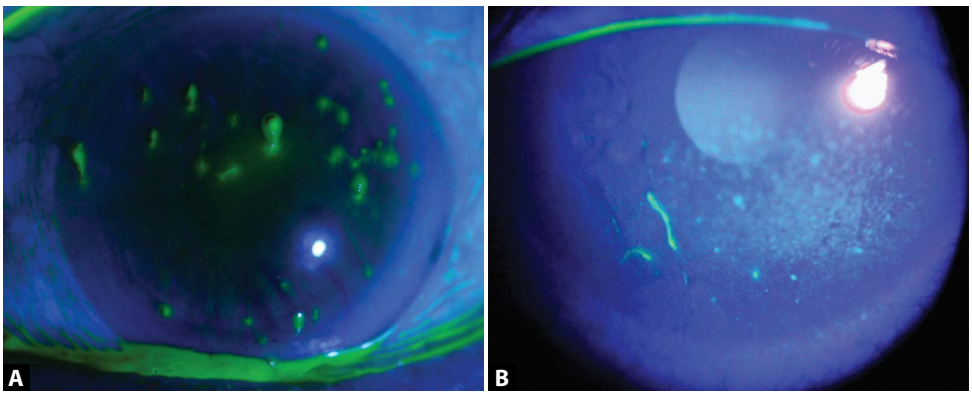
Treatment

- They disappear spontaneously as the wound heals.
- Sometimes, they persist and may cause endothelial decompensation.



Figs 13.58A and B: Mild and severe striate keratopathy after cataract surgery

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Figs 13.59A and B: **A.** Filamentary keratopathy; **B.** Superficial punctate keratitis and filaments in dry eye

Filamentary Keratopathy (Keratitis) (Figs 13.59A and B)

- This is disease with the formation of epithelial threads (filaments) on the cornea.
- The filaments (2 mm or less in length) adhere to the cornea by one end, while the other, moves about freely.

Causes

- Keratoconjunctivitis sicca
- Superior limbic keratoconjunctivitis
- Following cataract surgery
- Herpes simplex keratitis
- Recurrent erosions
- Diabetes mellitus.

Clinical Features

- Patient presents with foreign body sensation, photophobia and lacrimation.
- As the filaments are anchored to the epithelial cells, pulling on them is very painful.
- It is beautifully stained by Rose-Bengal and fluorescein.

Treatment

- Removal of the filaments, by scraping them from the underlying epithelium, and then patching
- Bandage contact lens
- Artificial tears eye drop frequently.

Keratomalacia

This is characterized by a desiccation and subsequent necrosis of the cornea due to lack of vitamin A. The characteristic feature is the absence of inflammatory reaction. For more details, *see* Chapter 26.

CONTACT LENSES (FIG. 13.60)

Contact lenses are worn directly over the cornea and beneath the eyelids.

There are three main types of contact lens:

1. ***Hard:*** It is made of PMMA (polymethylmethacrylate) or perspex.
2. ***Rigid gas permeable (RGP):*** It is made of mixture of a hard and a soft material (Fig. 13.61A).

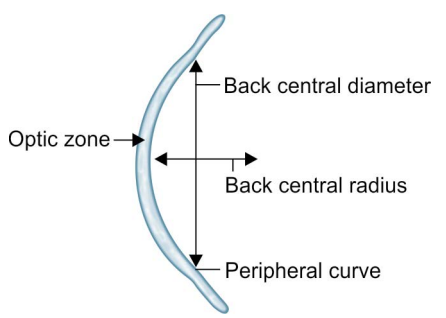


Fig. 13.60: Contact lens

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Figs 13.61A and B: A. Rigid gas permeable; **B.** Soft contact lens

3. ***Soft (hydrophilic):*** It is made of HEMA (hydroxyethylmethacrylate) (Fig. 13.61B).

Hard Lenses

These lenses cover the part of the cornea and have a diameter of 7–10.5 mm.

Advantages

- Most durable and cheap.
- Cleaning and maintenance are easy.
- It can correct the astigmatism in a greater extent.
- Less chance of corneal infection.

Disadvantages

- Only suitable for daily wear for short duration.
- Not permeable to atmospheric oxygen. Cornea suffers from quick hypoxia leading to epithelial edema ***(Sattler's veil)***.
- Most uncomfortable, and requires adaptation by progressive increase in wearing time.
- May give rise to annoying glare at night.

Rigid Gas Permeable Lenses

They also cover the part of the cornea.

Advantages

- Permeable to atmospheric oxygen in variable amount. So, the cornea does not suffer much from hypoxia.
- Suitable for daily wear, and for a longer duration than hard lens.
- Others are same as hard lens.

Disadvantages

- Less durable and costly.
- Less comfortable (though little better than hard lens) and requires slow adaptation.
- Tend to scratch and fracture more easily.

Soft (Hydrophilic) Lenses

They cover the entire cornea and extend 1–2 mm over the sclera. They have a diameter of 13–14.5 mm. The amount of oxygen passing through the lens is directly related to its water content.

They are used as ***daily-wear, extended-wear*** (more than 24 hours) and as ***therapeutic bandage lenses***.

Advantages

- It can be used both in day and at night.
- Most comfortable and does not require adaptation time.
- Lens loss is less.

Disadvantages

- More delicate, more easily damaged and have a shorter life.
- It can not correct astigmatism more than 0.5–1.0 diopter.

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Fig. 13.62: Contact lens wearing

- Cleaning and maintenance of the lenses are difficult.
- Associated with higher incidence of corneal infection.

Indications of Contact Lenses (Fig. 13.62)

Optical

About 95% of the contact lenses are worn as an alternative to glasses.

- ***Myopia*** is the most common indication.
- ***Aphakia*** especially in unilateral aphakia, and aphakia following congenital cataract operation.
- ***Astigmatism*** irregular astigmatism, as in fine nebular opacity, keratoconus, etc.
- ***Anisometropia.***
- ***High hypermetropia.***

Therapeutic

By soft bandage contact lenses and the indications are:

- Bullous keratopathy
- Recurrent corneal erosion syndrome
- Filamentary keratopathy
- Persistent epithelial healing defects
- Dry eye syndrome
- Metaherpetic keratitis
- Chemical burns—to prevent symblepharon formation
- Wound leaks, along with tissue adhesives.

Diagnostic

- Fundus contact lens
- Goldmann's three-mirror contact lens
- Radio-opaque contact lens for diagnosis of intraocular foreign body
- Goniolens
- Lenses for laser therapy.

Miscellaneous

- ***Cosmetic***—to hide an unsightly corneal scar with no visual potential.
- ***Vehicle for drug delivery***, e.g. contact lens soaked in pilocarpine.
- ***Protective***—as in aniridia or albinism with painted iris and a central clear pupillary area.
- ***Occluders***—for treatment of amblyopia in children who can not tolerate conventional occlusion.
- ***Occupational:*** Practically useful among sportsmen, sales girls, actor, models, etc.

Complications of Contact Lenses

They are more common with soft lenses, especially with the extended-wear type.

Conjunctival Complications

- ***Allergic conjunctivitis***—mainly related to allergy to the preservatives (e.g. thiomersal) present in contact lens cleaning solution.
- ***Giant papillary conjunctivitis (GPC):*** It has an immunologic origin in which contact lens deposits, especially the proteins act as allergens.

Corneal Complications

- ***Epithelial edema*** mainly due to hypoxia ***(Sattler's veil)***.
- Peripheral corneal ***vascularization***.

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- ***Sterile corneal ulceration*** which usually heals, once the contact lens wear has been discontinued.
- ***Infection***, particularly *Pseudomonas* keratitis and *Acanthamoeba* keratitis are the most serious.
- ***Warpage of the cornea***—resulting in severe and permanent astigmatism.

Problems with the Contact Lens

- ***Lens deposits***—e.g. mucoproteins, calcium, lipid or even micro-organisms can adhere to the lens surface.
- ***Lens digestion***—organisms, particularly fungi can digest lens material while growing into it.
- ***Lens spoilage***—due to ageing, loss of lens shape, discoloration, and by manual or mechanical trauma.

DISEASES OF THE SCLERA

Diseases of the sclera are relatively rare. The reasons are: (i) relative avascularity of the sclera and (ii) lack of reaction of its dense fibrous tissue to any insult.

For the same reasons, when they do occur, the diseases tend to be chronic and sluggish.

Blue Sclera (Fig. 13.63)

The sclera appears more blue than white. This is due to visibility of the underlying uveal pigment through the thinned sclera.

Causes

- Normally in babies
- ***Osteogenesis imperfecta*** (blue sclera, ***fragilitas ossium*** and deafness)
- Buphthalmos
- High myopia
- Following scleritis
- Ciliary staphyloma
- Marfan's syndrome
- Ehler-Danlos syndrome.

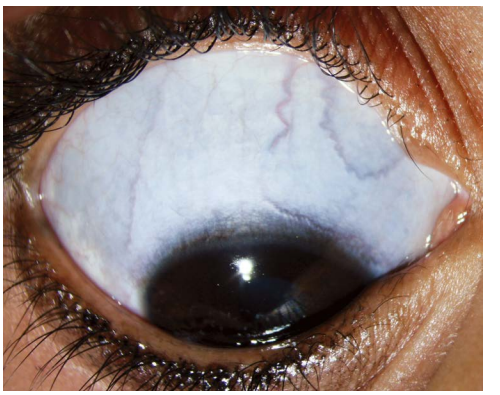


Fig. 13.63: Blue sclera

INFLAMMATION OF THE SCLERA

Classification

Episcleritis
- Simple
- Nodular

Anterior scleritis
- Diffuse
- Nodular
- Necrotizing
  - With inflammation
  - Without inflammation (scleromalacia perforans)

Posterior scleritis

Episcleritis

This is a benign inflammatory process affecting the deep subconjunctival connective tissue (episclera), and sometimes, including the superficial scleral lamella.

Etiopathology

- Young adults, more in female.
- Idiopathic (most common).
- Allergic reaction to endogenous toxins, e.g. streptococcal or tubercular.
- Infectious, e.g. herpes zoster, herpes simplex, syphilis, etc.
- Other collagen-vascular diseases.

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Pathologically, there is dense lymphocytic infiltrations in the subconjunctival and episcleral tissue.

Symptoms

- Mild pain or irritation of the eye.
- Localized redness.

Signs

Two clinical types—simple and nodular.

1. ***Simple episcleritis:*** There is sectorial redness with mild tenderness.
2. ***Nodular episcleritis:*** A small purple nodule with surrounding injection, is situated 2–3 mm away from the limbus (usually on the temporal side). The nodule is immobile and tender (Fig. 13.64).

Complications

Normally, the condition is transient with spontaneous remission within a few days. But sometimes, it gives rise to following complications:

- ***Recurrent attacks:*** When the attacks are fleeting but frequently repeated, it is called ***episcleritis periodica fugax***
- Chronic episcleritis
- Deeper extension into the sclera, leading to scleritis

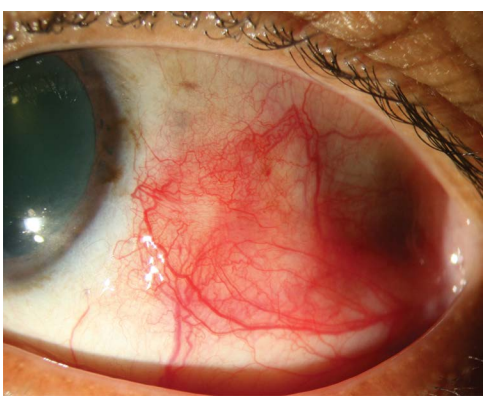


Fig. 13.64: Nodular episcleritis

- Sclerokeratitis
- Adherence of the conjunctiva with the sclera after repeated attacks.

Treatment

- Corticosteroids drops—4 times daily, and ointment at night.
- Oral anti-inflammatory agents, like ibuprofen, indomethacin or diclofenac.
- Non-steroidal anti-inflammatory drops, e.g. flurbiprofen or diclofenac drop, when steroids are contraindicated.
- Tab. salicylate or indomethacin for a prolonged period to prevent recurrences, even when no history of 'rheumatism' can be elicited.

Scleritis

Scleritis (inflammation of the sclera) is a more serious disease than episcleritis.

Etiology

- Usually bilateral and occurs most frequently in women.
- Associated with ***connective tissue disorders*** in 50% of the cases, e.g. rheumatoid arthritis, polyarteritis nodosa, systemic lupus erythematosus, Wegener's granulomatosis, relapsing polychodritis, etc.
- Herpes zoster as a local cause.
- Miscellaneous systemic conditions, like tuberculosis, sarcoidosis, syphilis, leprosy, etc.

Pathology

Deposition of immune-complex in the sclera leading to inflammation → marked infiltration of lymphocytes in the scleral lamellae with edema → breakdown of swollen lamellae with necrosis → scleral thinning → simultaneous inflammation of the uveal tract, causing uveitis.

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Differences between episcleritis and scleritis

Features	Episcleritis	Scleritis
Definition	A superficial disease of episcleral tissue, a mild condition.	A deep severe destructive disease of sclera; not a mild condition.
Symptoms	Redness is the main presentation.	Severe boring pain is the main presentation.
Signs	Less tender. Bright red in color. Only superficial edema. No KPs, no feature of uveitis.	More tender nodule. Purplish in color. Sclera appears thickened. Presence of keratic precipitates (KPs), feature of uveitis.
Drug test with 10% Phenylephrine	Quick blanching of blood vessels.	No such blanching of blood vessels.
Prognosis	Favorable, complications usually do not occur.	Poor, complications like dimness of vision, scleral thinning, staphyloma and sometimes perforation may occur.

Symptoms

- Intense deep-seated pain with radiation towards the forehead.
- Redness and lacrimation.

Signs

- ***Diffuse anterior scleritis:*** The inflammation is more widespread involving either a segment of the globe, or the entire anterior sclera with intense deep seated vascularization ***(brawny scleritis)***.
- ***Nodular anterior scleritis (Fig. 13.65)***
  - An extremely tender, firm immobile nodule separated from the overlying congested episcleral tissue. Sclera is edematous over the nodule.
  - Multiple nodules may extend around the limbus causing ***annular scleritis***.
- ***Anterior necrotizing scleritis with inflammation (Figs 13.66 A and B)***
  - Avascular patches appearing in the episcleral tissue.
  - Marked thinning of the sclera with increased visibility of underlying uvea.
  - Associated anterior uveitis.
  - Complications like, cataract, keratitis, keratolysis and secondary glaucoma may develop with severe visual loss.
  - Avascular patch may perforate acutely.

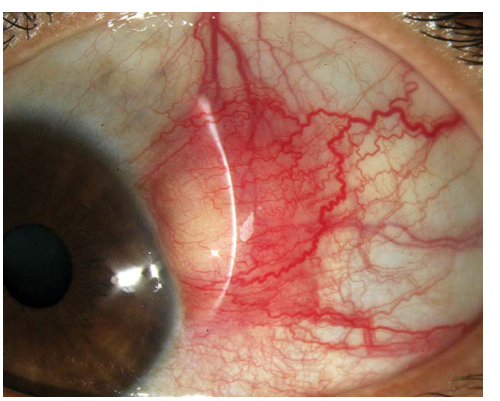
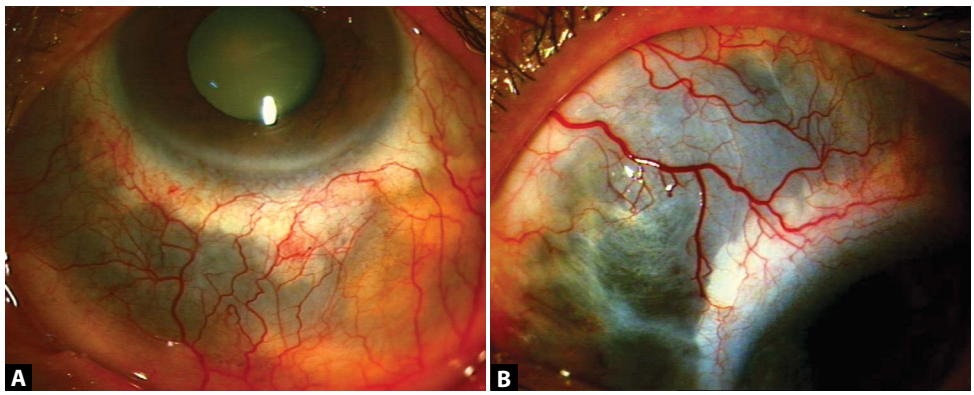


Fig. 13.65: Nodular scleritis

- ***Anterior necrotizing scleritis without inflammation* [*scleromalacia perforans (Fig. 13.67)*]**
  - Typically occurs in female patients, with long standing seropositive rheumatoid arthritis.
  - The condition is ***painless***, and starts as a necrotic patch in the normal sclera.
  - Eventually, extreme scleral thinning occurs, and the underlying uvea bulges through it.
  - Spontaneous perforation is extremely rare.
- ***Posterior scleritis***
  - It is usually not associated with specific systemic diseases.

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Figs 13.66A and B: Necrotizing scleritis with inflammation. **A.** Thinning; **B.** Bluish discoloration

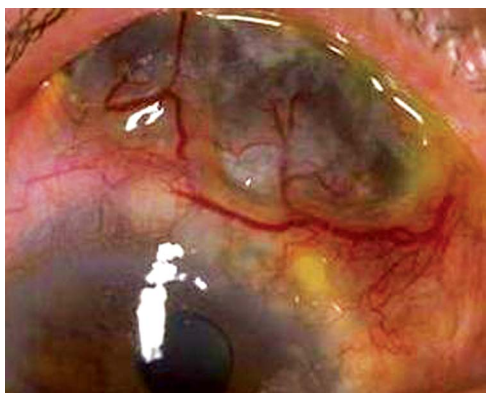


Fig. 13.67: Scleromalacia perforans

- Inward extension (towards choroid) of the inflammatory process may give rise to ***uveal effusion syndrome***—choroiditis, choroidal effusion, exudative retinal detachment, macular edema, etc.
- Outward extension onto the orbit may give rise to proptosis and extraocular muscles involvement.

Investigations

- Total hemogram, serum uric acid
- X-ray chest and sacroiliac joints
- Mantoux test
- Venereal disease research laboratory (VDRL) and fluorescent treponemal antibody absorption (FTA-ABS) for syphilis.
- Full immunological evaluation for tissue antibodies for collagen vascular diseases, e.g. rheumatiod factor, anti-nuclear factor, soluble immune-complex, lupus erythematosus (LE) cells, etc.
- B-scan ultrasonography to detect posterior scleritis.

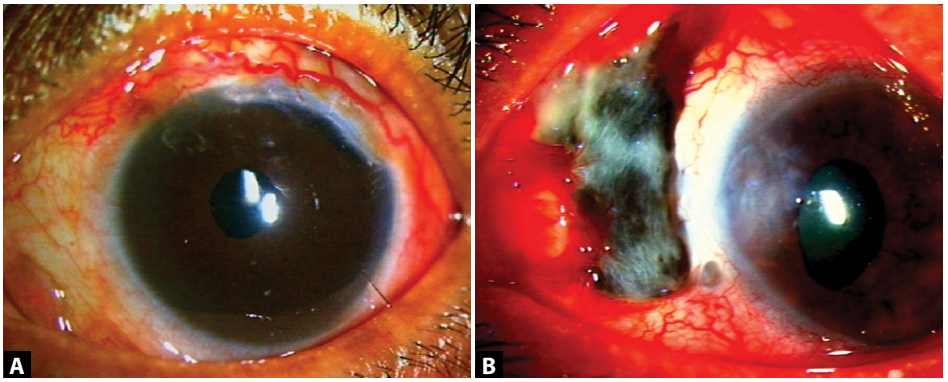
Treatment

- Tab. indomethacin—100 mg daily for 4 days, reducing to 75 mg daily until the inflammation resolves.
- Oral prednisolone—60–80 mg daily, then the dose can be tapered accordingly, as the inflammation subsides.
- Local corticosteroids are less effective, and subconjunctival injection is contraindicated for fear of perforation of the globe.
- Immunosuppressive alkylating agents, or oral azothioprim in severe and unresponsive cases.
- Atropine (1%) eye ointment—2 times daily for associated uveitis.

SURGICALLY INDUCED NECROSIS OF SCLERA (FIGS 13.68A AND B)

It is a rare postoperative immune-mediated necrosis of the sclera. It may be triggered by

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Figs 13.68A and B: Surgically induced necrosis of sclera. **A.** Post-cataract surgery; **B.** Post-pterygium surgery

excessive use of cautery, use of antimetabolites during surgery, etc.

It may be seen after cataract surgery, pterygium surgery, trabeculectomy or vitreoretinal surgery.

Treatment

- Topical and systemic steroids
- Systemic immunosuppressive therapy
- Scleral patch graft in extreme situation.

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14 CHAPTER Diseases of the Uvea

UVEAL DISEASES

The symptoms and signs vary considerably with the portion of the uvea affected.

Symptoms

- ***Pain:*** Severe, deep, boring, aching pain associated with diseases of the iris and ciliary body. Pain is absent in choroidal involvement.
- ***Redness:*** Inflammation of the iris and ciliary body causes red eye which is absent in choroidal inflammation.
- ***Visual loss:*** It is more in choroidal disease as it affects the overlying retina.
- ***Floaters:*** It is again more in choroidal disease due to exudation of inflammatory cells and protein into the vitreous.

Signs

- ***Ciliary congestion:*** It is present in inflammation of the iris and ciliary body, but absent in choroiditis.
- ***Distortion of the pupil:*** It is due to abnormalities in the iris.
- ***Accommodation:*** It is disturbed in ciliary body involvement.
- ***Release of cells and protein*** occurs in anterior chamber in iris inflammation, in vitreous in choroidal inflammation; and both in the anterior chamber and vitreous cavity in ciliary body inflammation.
- ***Keratic precipitates (KPs):*** They are seen in inflammation of the iris and ciliary body.
- ***Ophthalmoscopic*** abnormal signs are found in choroidal involvement, with associated retinal signs due to chorioretinal involvement.

CONGENITAL ANOMALIES

Anomalies of the Pupil

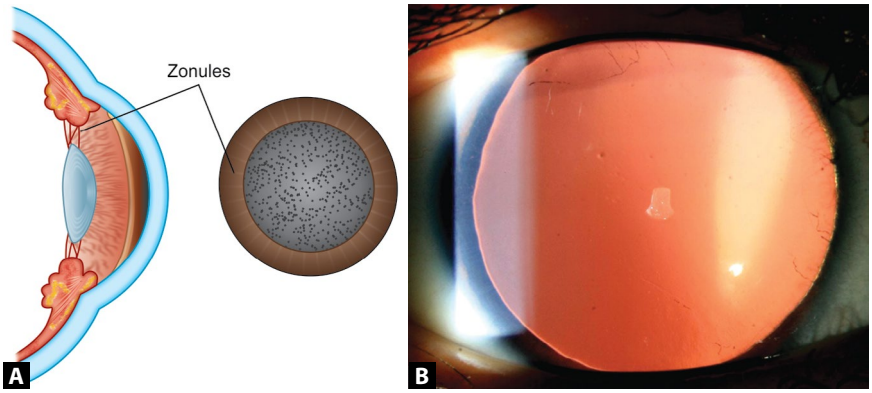
- ***Corectopia:*** Displacement of the pupil from its normal position, usually to the nasal side (normally, the pupil is situated just nasal to the center of the cornea).
- ***Polycoria:*** Multiple pupils—(a) true polycoria—multiple pupils, each having a sphincter muscle, are extremely rare, (b) pseudopolycoria—multiple pupils without sphincters, are common.
- ***Pear-shaped pupil:*** It is due to coloboma of the iris.

Aniridia

It is usually a bilateral condition in which the whole of the iris is appeared to be missing on external examination (Figs 14.1A and B).

- The rudimentary iris, concealed behind the corneoscleral limbus, is only visible by gonioscopy.
- It is due to failure of anterior growth and differentiation of the optic cup.

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Figs 14.1A and B: Aniridia. **A.** Schematic representation; **B.** Photograph

Symptoms

They are photophobia, nystagmus and low visual acuity (6/60 or less).

Signs

- Ciliary processes and suspensory ligaments of the lens are visible.
- Associated secondary glaucoma with glaucomatous optic atrophy in 25% of the cases.
- Associated foveal hypoplasia.
- Mental retardation and congenital cataract in some cases.
- Aniridia and Wilms' tumor are associated with deletion of the short arm of chromosome 11 (Miller's syndrome).

Treatment

- Provision of an artificial pupil by means of an iris-painted soft contact lens. This reduces photophobia, but does not improve vision.
- The glaucoma responds poorly to medical and surgical treatment.

Persistent Pupillary Membrane

Pupillary membrane is the anterior vascular sheath of the lens, a fetal structure, which normally disappears shortly before birth (Figs 14.2A and B).

- Persistent pupillary membrane is due to continued existence of part of this membrane.
- This appears as fine strands of membrane, running inwards from the collarette and sometimes, inserting into the anterior lens capsule or spanning the pupil.
- ***It is never at the pupillary margin*** (a point to differentiate it from the posterior synechiae which are formed at the pupillary margin).
- It usually does not interfere with vision.

Colobomas

Colobomas are developmental imperfect closure of the fetal fissure.

Consequently, they show an absence of any or all the structures of inner two layers which lie along the corresponding lower nasal sector of the eye.

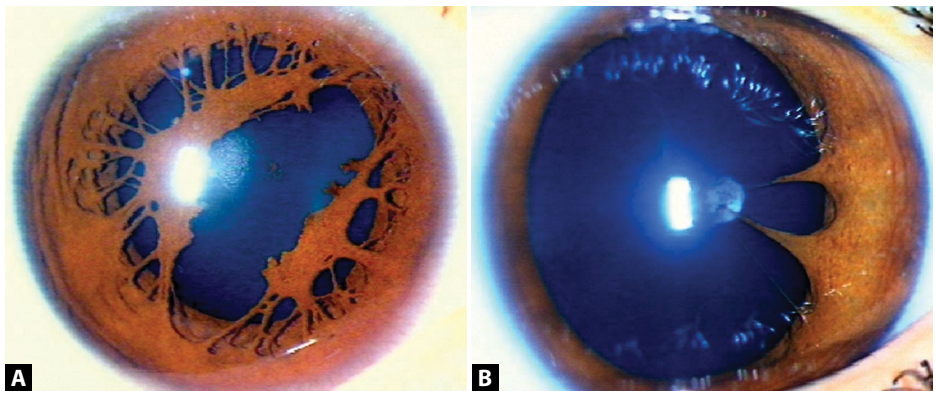
Coloboma may be ***typical*** or ***atypical***.

- ***Typical:*** It is always found along the lower nasal sector of the eye due to imperfect closure of the fetal fissure.
- ***Atypical:*** It is seen in other position and mainly confined to the iris (Figs 14.3A and B).

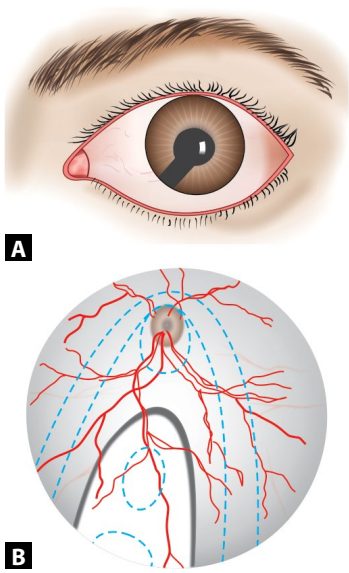
Typical Coloboma

Genesis: Overgrowth of the inner layer of the optic cup → eversion of the lips of the fetal

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Figs 14.2A and B: **A.** Persistent pupillary membrane; **B.** Persistent pupillary membrane with anterior polar cataract



Figs 14.3A and B: Coloboma of the uveal tract. **A.** Iris coloboma; **B.** Choroidal coloboma

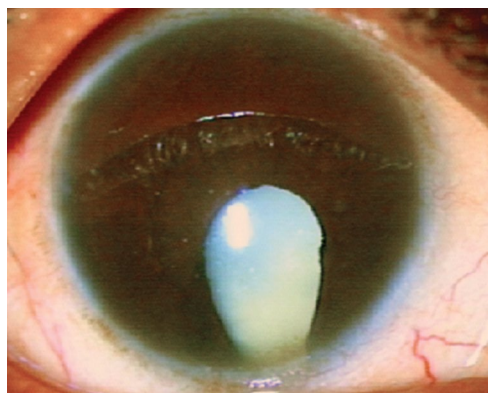


Fig. 14.4: Typical complete coloboma with cataract

fissure → obstruction of the natural union between the lips → failure of development of the retina and underlying structures.

Typical coloboma may be complete or incomplete:

- ***Complete:*** It extends from the pupil to the optic nerve, with a sector shaped gap, occupying about one-eighth of the circumference of the retina and choroid, ciliary body and iris, and causing a corresponding indentation of the lens where the zonular fibers are missing (Figs 14.3 and 14.4).
  Complete coloboma is usually bilateral, often hereditary, and may be associated with a congenital cyst or microphthalmos.
- ***Incomplete:*** When it stops short of the optic nerve, or of the iris, or else, or even partitioned by islands of normal tissue (Figs 14.5 and 14.6A).

Typical coloboma of the iris: The pupil is pear-shaped with broad base towards the pupillary margin. Isolated typical coloboma of the iris is rare. It usually extends upto the ciliary body.

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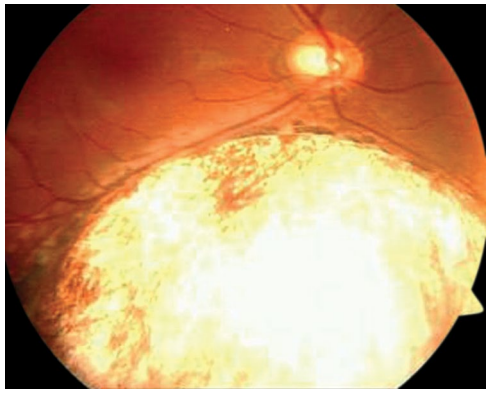


Fig. 14.5: Incomplete choroidal coloboma

Atypical coloboma of the iris: Isolated coloboma of the iris (Fig. 14.6B) is usually atypical, since the fetal fissure normally closes before the iris if formed, and it occurs at any meridian.

This is probably due to obstruction in the anterior growth of the optic cup by a large vessel of the fibrovascular sheath of the lens.

Albinism

This is a hereditary disorder in which there is absence or reduction of melanin pigmentation throughout the body.

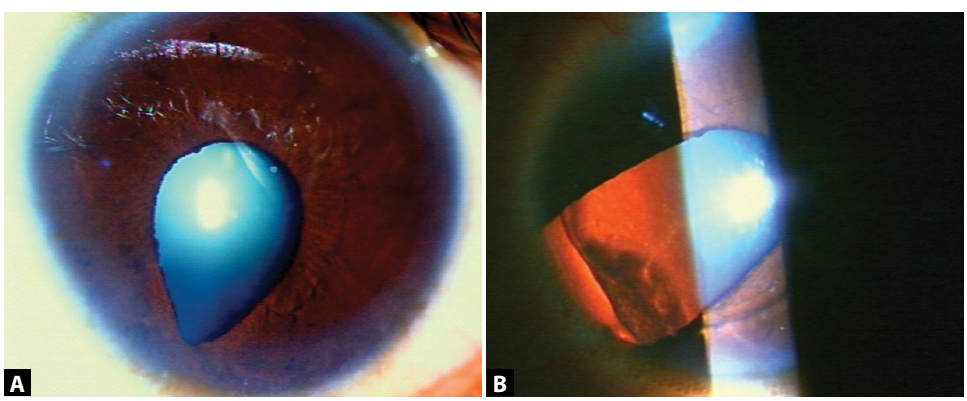
- Albinism may be divided into two main types—***oculocutaneous (Fig. 14.7)*** and ***ocular***.
- ***Clinically***, the patient suffers from dazzling. There is associated photo-phobia, nystagmus, defective vision and occasionally strabismus.
  Visual acuity is further lowered by high myopia and astigmatism.
- ***On examination***, the iris looks pink and translucent (Fig. 14.8), owing to lack of pigment.
- ***Ophthalmoscopically***, the fundus (Fig. 14.9) appears orange-pink in color. The retinal and choroidal vessels are seen with great clarity and glistening white sclera is visible in between them. Macula is also involved.
- ***Partial albinism*** is more common, where the absence of pigment is limited to the choroid and retina. Here, the iris is brown or blue colored, and macular region may also be pigmented and looks normal. Visual acuity is much better in partial albinism.

Treatment

- As photophobia is the main symptom, dark glasses may be helpful.
- Alternately, a soft contact lens with painted iris is very useful.

Heterochromia Iridum

Heterochromia iridum means the two irides show a significant difference in color (Fig. 14.10).



Figs 14.6A and B: **A.** Incomplete iris coloboma; **B.** Atypical iris coloboma

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Fig. 14.7: Oculocutaneous albinism

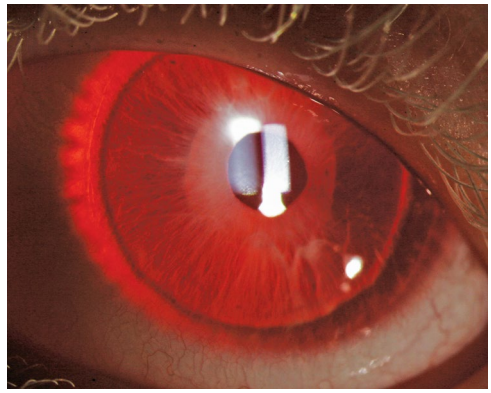


Fig. 14.8: Iris transillumination

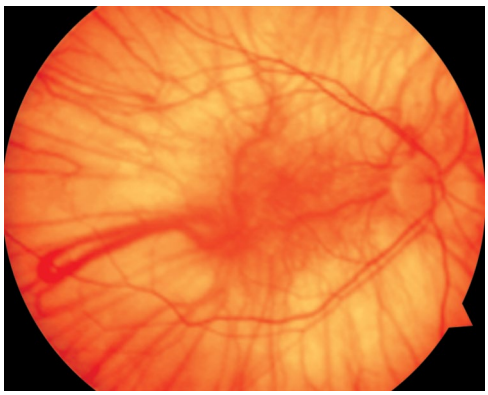


Fig. 14.9: Fundus in albinism



Fig. 14.10: Heterochromia of the iris (right—blue, left—deep brown)

Heterochromia iridis means part of the same iris, usually a sector, shows difference in color from the remainder.

The ***blue iris*** is due to absence of pigment in the iris stroma, the pigment in the retinal epithelium being seen through the translucent stroma.

Heterochromic iridum may be ***hypochromic*** or ***hyperchromic***:

- ***Hypochromic heterochromia:*** The eye with lighter-colored iris is abnormal (Fig. 14.11)
  Causes
  - Simple congenital heterochromia
  - Horner's syndrome
  - Heterochromic cyclitis of Fuchs
  - Glaucomatocyclitic crisis
  - Amelanotic tumors of the iris
  - Iris atrophy.
- ***Hyperchromic heterochromia:*** The iris on the side of the disease is darker than its fellow (Fig. 14.12).
  Causes
  - Retained intraocular iron foreign body (siderosis bulbi)
  - Malignant melanoma of the iris
  - Ocular hemosiderosis (e.g. following long standing hyphema)
  - Microcornea with heterochromia.

Heterochromic iridis—may be congenital or acquired (iris atrophy due to herpetic

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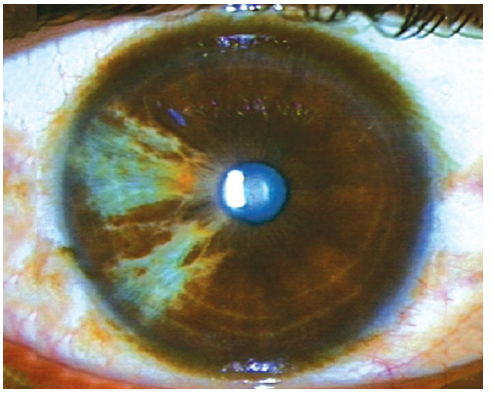


Fig. 14.11: Hypochromic heterochromia

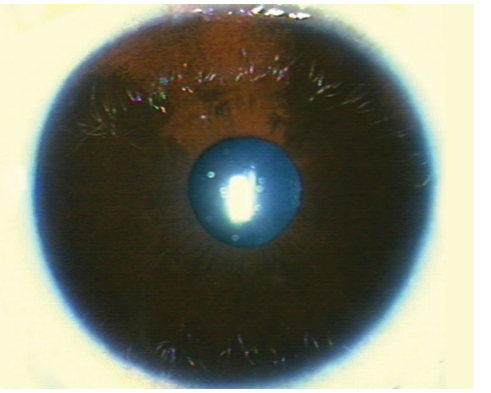


Fig. 14.12: Hyperchromic heterochromia

uveitis, trauma, acute attack of angle-closure glaucoma, after cataract surgery, etc).

UVEITIS

- ***Uveitis:*** An inflammation of the uveal tract which may also involve the adjacent structures.
- ***Endophthalmitis:*** This is a severe form of intraocular inflammation involving the ocular cavities and inner coats of the eyeball.
- ***Panophthalmitis:*** This is also a severe form of intraocular inflammation involving the ocular cavities and all three coats of the eyeball as well as Tenon's capsule.

Classifications

The useful classifications are as follows:

Standardization of Uveitis Nomenclature Classification

In an effort to better organize the classification and grading of different uveitic entities, the (Table 14.1). Standardization of uveitis nomenclature (SUN) Working Group developed a process to standardize the approach to reporting clinical data for improving the consistency.

TABLE 14.1: Anatomical classification of Standardization of Uveitis Nomenclature (SUN)

Type	Primary site of inflammation	Includes
Anterior uveitis	Anterior	• Iritis chamber • Anterior cyclitis • Iridocyclitis
Intermediate uveitis	Vitreous	Pars planitis
Posterior uveitis	Choroid or retina	• Focal, multifocal or diffuse choroiditis • Chorioretinitis • Retino-choroiditis • Retinitis • Neuroretinitis
Panuveitis	Anterior chamber, vitreous, and retina or choroid	

Onset
- Sudden
- Insidious.

Duration
- ***Limited:*** Less than 3 months duration
- ***Persistent:*** More than 3 months duration.

Course
- ***Acute:*** Sudden onset and limited duration.

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- ***Recurrent:*** Repeated episode separated by periods of inactivity without treatment more than 3 months duration.
- ***Chronic:*** Persistent uveitis with relapse in less than 3 months, after discontinuing treatment.

Activity of Uveitis Terminology

Term	Definition
Inactive	Grade 0 cells
Worsening activity	Two-step increase in level of inflammation (e.g. anterior chamber cells, vitreous haze) or increase from grade 3+ to 4+.
Improved activity	Two-step decrease in level of inflammation (e.g. anterior chamber cells, vitreous haze) or decrease to grade 0.
Remission	Inactive disease for more than 3 months after discontinuing all treatment for eye disease.

Etiological Classification

- ***Idiopathic:*** 25–30% of all cases.
- ***Exogenous:*** After external injury, either by microorganisms or by other agents from outside.
- ***Endogenous:*** Either caused by microorganisms or other agents (immune complex) from within the patient. The causes of endogenous uveitis are:
  - ***Secondary to systemic diseases***
    - Seronegative arthritis
    - Tuberculosis, syphilis and leprosy
    - Sarcoidosis
    - Ulcerative colitis.
  - ***Secondary to infections***
    - Viral, e.g. herpetic
    - Fungal, e.g. candidiasis
    - Parasitic, e.g. toxoplasmosis.
  - ***Lens-induced uveitis***
  - ***Idiopathic specific uveitis***
    - Pars planitis
    - Fuchs' heterochromic cyclitis
    - Sympathetic ophthalmitis.
  - ***Others***
    - Malignant tumors (Masquerade syndrome)
    - Associated with septic infection, e.g. sinusitis, otitis media, tonsillitis, etc.
    - Direct spread from the adjacent structures, e.g.
      - Cornea (deep keratitis)
      - Sclera (scleritis)
      - Retina (retinitis), etc.

A number of diseases associated with uveitis occur much more frequently in those persons with certain specific HLA-types, e.g:

- HLA–B 27–in acute anterior uveitis
- HLA–B 5–in Behcet's disease
- HLA–DR4–in VKH syndrome.

Pathological Classification

- Granulomatous
- Non-granulomatous

The main clinical differences between the two are summarized in Table 14.2.

TABLE 14.2: Clinical difference between granulomatous uveitis and non-granulomatous uveitis

Granulomatous uveitis	Non-granulomatous uveitis
Insidious onset and chronic course	Acute onset and short course
Relatively mild and white eye	Severe and red eye
Nodules (Keoppe's and Bussaca's) on the iris	No such nodules on the iris
Medium to large KPs (often mutton-fat type)	Fine keratic precipitates (KPs)
Mild flare	Intense flare, often with heavy fibrinous exudates
Anterior uvea and retina-choroid are equally involved	Mainly limited to the anterior uvea
Tuberculosis, sarcoidosis, leprosy, etc. are usually responsible	Mainly idiopathic and allergic in nature

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Fig. 14.13: Signs of iridocyclitis

Basic pathomechanisms in uveitis

- Vasodilatation
- Increased capillary permeability
- Migration of inflammatory cells into the ocular cavities.

ANTERIOR UVEITIS (IRIDOCYCLITIS)

Symptoms

- ***Pain:*** Acute, severe with radiation along the branches of the 5th nerve. It is typically worse at night
- ***Redness:*** It is due to vasodilatation
- ***Photophobia***
- ***Lacrimation***
- ***Dimness of vision:*** It is due to:
  - Plasmoid (turbid) aqueous
  - Vitreous exudates
  - Exudates in the pupillary area
  - Cystoid macular edema
  - Secondary glaucoma
  - Ciliary muscle spasm
  - Complicated cataract.

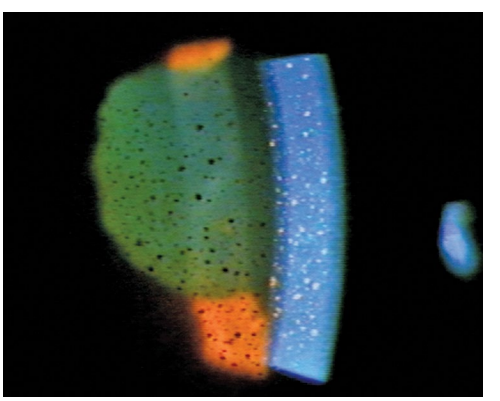


Fig. 14.14: Fine to medium keratic precipitate in acute iridocyclitis

Ocular Signs (Fig. 14.13)

Circumcorneal ciliary congestion (CCC): It is one of the hallmarks of acute anterior uveitis.

Keratic Precipitates (Fig. 14.14)

They are cellular deposits on the corneal endothelium.

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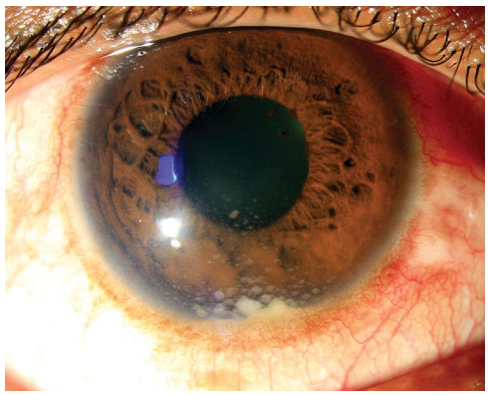


Fig. 14.15: Mutton fat keratic precipitates (within the Arlt's triangle)

- ***Distribution:*** The inflammatory cells are wandering in the aqueous by convection current, and stick to the edematous endothelial cells of the cornea. They arrange in a base-down triangular area at the lower part of the cornea ***(Arlt's triangle)*** (Fig. 14.15) due to gravitation. The smaller ones are above and the larger ones are below. However, in Fuchs' cyclitis, they are scattered throughout the endothelium.
- ***Size:*** The KPs may be small (fine), medium and large ***mutton-fat*** types.
- ***The small and medium-size KPs*** are due to deposition of lymphocytes and plasma cells, and they are seen in non-granulomatous or acute uveitis.
- ***The large KPs*** are due to deposition of macrophage and epithelioid cells, and they are seen in granulomatous uveitis (as in tuberculosis or sarcoidosis).
- ***Age:*** The KPs may be fresh or old.
- ***Fresh*** KPs are round, white, fluffy and hydrated in appearance.
- ***Old KPs*** are shrunken, faded and become pigmented with crenated edge, and halos are seen surrounding them. Old ***mutton-fat*** KPs have ***ground-glass*** appearance.

Anterior Chamber

- ***Aqueous flare:*** It is due to leakage of proteins into the aqueous through the damaged capillaries, causing a Tyndall effect. It is not necessarily a sign of active uveitis, particularly in absence of aqueous cells. The flare is graded (Table 14.3 and Fig. 14.16) (from 0 to 4+) depending upon the degree of obstruction of iris details under slit lamp.

TABLE 14.3: Grading of anterior chamber, flare

Grades	Description
0	None
1+	Faint
2+	Moderate (iris and lens details clear)
3+	Marked (iris and lens details hazy)
4+	Intense (fibrin or plastic aqueous)

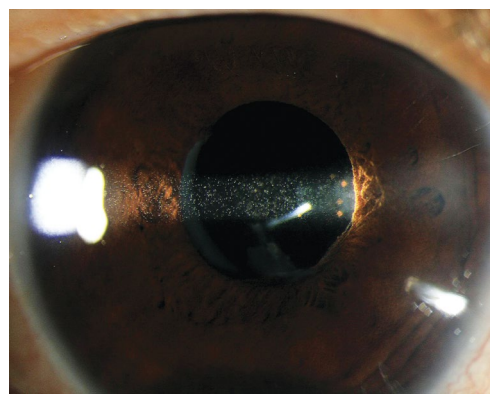


Fig. 14.16: Aqueous flare and cells

- ***Aqueous cells:*** It is due to exudation of inflammatory cells into the aqueous presence of cells in aqueous always indicates active inflammation.
  The cells should be counted and (Table 14.4) graded (from 0 to 4+) under oblique slit lamp beam (1 mm long and 1 mm wide) (Figs 14.16 and 14.17).
- ***Hypopyon:*** It is classically seen in Behcet's syndrome or infectious cases (herpetic uveitis).
- ***Hyphema:*** Herpetic uveitis and in traumatic uveitis.

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TABLE 14.4: Grading of anterior chamber cells

Grades	Cells in field*
0	<1
0.5+	1–5
1+	6–15
2+	16–25
3+	26–50
4+	50+

*Field size is 1x1 mm slit-beam

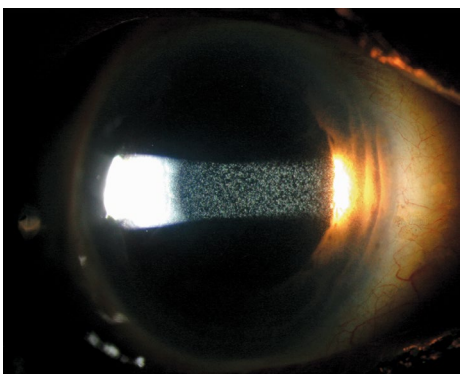


Fig. 14.17: Aqueous cells and flare-more intense

- ***Depth***
  - Deep and irregular—in posterior synechiae
  - Funnel-shaped—in iris bombe.

Iris

- ***Muddy iris or loss of iris pattern:*** This is due to accumulation of fluid and exudates over the surface of the iris.
- ***Iris nodules:*** It is seen in granulomatous uveitis.
  - ***Koeppe's nodule:*** At the pupillary border, and smaller in size.
  - ***Busacca's nodule (Fig. 14.18):*** On the surface of the iris, away from the pupil.
- ***Iris atrophy and heterochromia:*** It is seen in Fuchs' heterochromic cyclitis and herpetic uveitis.
- ***Rubeosis iridis:*** Iris neovascularization develops in chronic anterior uveitis and in Fuchs' heterochromic cyclitis.

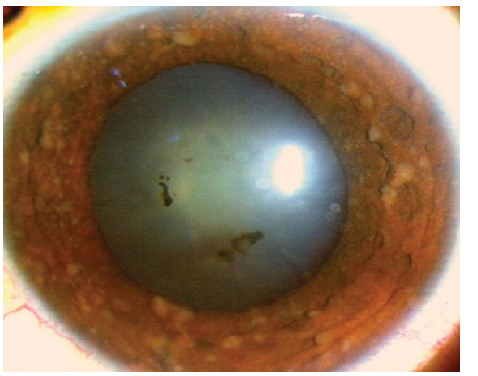


Fig. 14.18: Busacca's nodule

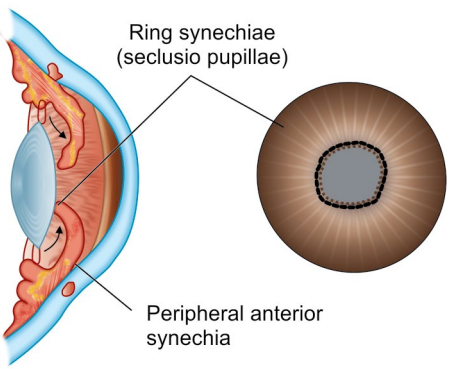


Fig. 14.19: Iris bombe

- ***Synechiae***
  - ***Posterior synechia:*** It is the adhesion between the anterior lens surface and the iris. This is due to organization of fibrinous exudate between the pupillary margin and the lens surface. This adhesion may be localized or diffuse.
    - ***Ring (annular) synechiae:*** Posterior synechiae extending for 3600 around the pupil (seclusio pupillae), prevent the passage of aqueous from the posterior to the anterior chamber. This gives rise to forward bowing of the peripheral iris causing an ***iris bombe*** (Fig. 14.19). This may lead to elevation of intraocular pressure due to secondary angle closure by the peripheral iris (Fig. 14.20).

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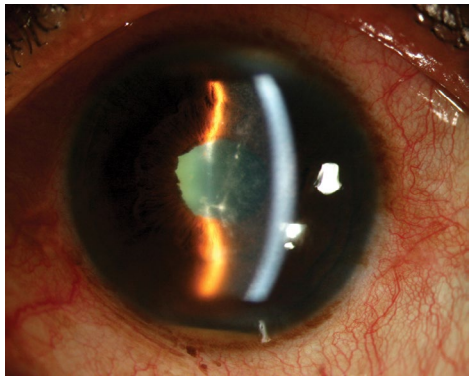


Fig. 14.20: Ring synechia with iris bombe in acute iridocyclitis with hypopyon

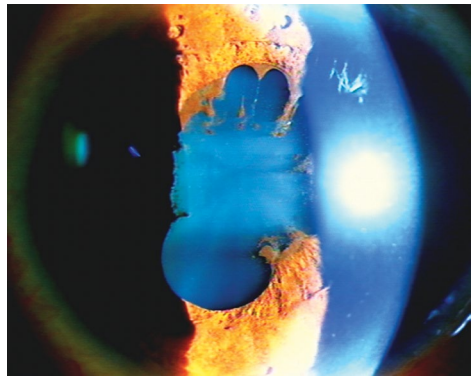


Fig. 14.21: Healed iridocyclitis—festooned pupil

  - ***Total posterior synechiae:*** Adhesion of entire iris with the anterior lens capsule.
  - ***Anterior synechiae:*** Adhesion of iris with the corneal endothelium. It is usually peripheral and secondary to iris bombe.
- Ectropion of the uveal pigment at the pupillary margin.

Pupil

- ***Sluggish or non-reacting pupil:*** This is due to edema of the iris and also due to posterior synechiae.
- ***Miotic pupil:*** It is due to (i) water logging of the iris, (ii) toxins act on powerful sphincter pupillae, and (iii) ring synechiae.
- ***Irregular or festooned pupil:*** Irregular dilatation of the pupil with mydriatic, and it is due to posterior synechiae (Fig. 14.21).
- ***Occlusio pupillae:*** Pupil is blocked or occluded by the organized fibrinous exudates.

Lens

Changes are mostly seen in chronic or healed anterior uveitis.

- ***Pigmentation*** on the anterior surface of the lens.
- ***Complicated cataract:*** Polychromatic lustre and bread-crumb appearance at the posterior cortex.
- ***Cyclitic membrane:*** It forms behind the lens, due to organization of the fibrinous exudates.

Tension

- May be normal.
- ***Decreased***—in acute iridocyclitis (due to acute ciliary shock).
- ***Increased***—in long standing anterior uveitis (due to secondary angle-closure or may be steroid-induced).

Posterior Segment

- ***Vitreous opacities:*** Due to exudation and inflammatory cells in the vitreous. It is more common in cyclitis (Table 14.5).

TABLE 14.5: Grading of vitreous opacities

Grades	Description	Clinical findings
0	Nil	None
1	Minimal	Posterior pole clearly visible
2	Mild	Posterior pole details slightly hazy
3	Moderate	Posterior pole details very hazy
4	Marked	Posterior pole details barely visible
5	Severe	Fundal details not visible

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- ***Fundus changes:*** Cystoid macular edema due to liberation of toxins. It is more with chronic anterior uveitis.

Systemic Signs

General examination is important to find out the etiological factor, e.g. examination of skin (rash, vitiligo, psoriasis), hair (poliosis, alopecia), genitourinary tract (urethritis), respiratory tract (tuberculosis), buccal mucosa (ulceration), etc.

Sequelae and Complications of Anterior Uveitis

- Posterior synechiae
- Ring synechiae → ***seclusio pupillae*** → iris bombe → peripheral anterior synechiae → secondary angle closure glaucoma
- Total posterior synechiae
- Occlusio pupillae
- Secondary glaucoma: It is due to:
  - Albuminous aqueous blocking the angle.
  - Associated trabeculitis and trabecular edema.
  - Ring synechiae → iris bombe → peripheral anterior synechiae → secondary angle-closure glaucoma.
  - ***Occlusio pupillae*** → anterior synechiae → secondary angle-closure glaucoma.
  - Steroid-induced (due to prolonged steroid therapy).
- ***Cyclitic membrane:*** It occurs more in cyclitis and forms behind the lens
- Complicated cataract
- Pseudoglioma, due to vitreous exudation
- Cystoid macular edema
- ***Phthisis (atrophic) bulbi:*** It prolonged cyclitis → ciliary body atrophy → less secretion of aqueous → atrophic bulbi
- ***Tractional retinal detachment:*** It is due to traction by the cyclitic membrane
- Band-shaped keratopathy (BSK); more common in children with juvenile (idiopathic) rheumatoid arthritis, and chronic iridocyclitis.

Investigations

- Routine hemogram
- ***Serological tests:*** Antinuclear antibody (ANA); rheumatoid factor (RF); HLA-typing; Venereal disease research laboratory (VDRL) and fluorscent treponemal antibody absorption (FTA-ABS) (for syphilis); ELISA, etc.
- ***Skin tests:*** (a) mantoux test (tuberculosis) (b) kveim test (sarcoidosis)
- ***X-ray*** (a) chest—for tuberculosis and sarcoidosis (b) joints—arthritis group
- ***Gallium scan:*** For sarcoidosis
- ***Urine culture:*** Urethritis
- ***Anterior chamber paracentesis*** for polymerase chain reaction (PCR) to diagnose organisms and cellular analysis.

Treatment

Local

- Hot compress: 3–4 times daily
- Use dark glasses
- ***Atropine (1%) eye drop or ointment–*** 3 times daily.

Atropine acts in three ways:

1. By keeping the iris and ciliary body at rest.
2. By reducing hyperemia.
3. By preventing the formation of posterior synechiae and breaking down, if any already formed.

Combined mydriatic-cycloplegics (like phenylephrine and homatropine, or phenylephrine and tropicamide) are sometimes better in place of atropine.

Atropine or any other cycloplegic should be continued for at least 10–14 days after the eyes appear to be quiet (or the aqueous cells disappear), otherwise a relapse is likely to occur.

A powerful mydriatic effect can be obtained by subconjunctival injection of 0.3 mL mydricaine (a mixture of atropine, procaine and adrenaline) often called a ***dynamite mixture*** to break the recently formed synechiae.

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- ***Corticosteroids:*** They are used as topical drops or ointment, or as subconjunctival or sub-Tenon injection.
  - Betamethasone, dexamethasone, or prednisolone drops—6 hourly to 1 hourly, depending upon the severity of the inflammation.
  - Corticosteroid ointment—at bed time.
  - Subconjunctival injection of dexamethasone (may be mixed with injection atropine)—daily (if required).
  - Sub-Tenon injection of long acting steroid (methyl prednisolone or triamcinolone)—every 2–3 weeks.

  Topical steroids are gradually tapered off with the response.
- ***Non-steroidal anti-inflammatory drops:*** Diclofenac, indomethacin or flurbiprofen, 4–6 times daily when topical steroid is contraindicated.

Systemic

- ***Systemic corticosteroids:*** If the response to topical steroid is poor, oral prednisolone—1–1.5 mg/kg body weight day, is given in a tapering dose.
- ***Anti-inflammatory (nonsteroidal) drugs:*** Like aspirin, indomethacin, diclofenac to reduce the pain and inflammation.
- Systemic antibiotic or antituberculous drugs in selective cases.
- Very rarely, ***immunosuppressive*** agents, e.g. in rheumatoid sclerouveitis.

Treatment of Complications

- ***Secondary glaucoma:*** Tab actazolamide —250 mg 3–4 times daily, and/or timolol maleate (0.5%) drops twice daily.
- ***Annular synechiae:*** Laser iridotomy.
- ***Iris bombe:*** Laser iridotomy or 4-point iridotomy ***(quadri-puncture)*** with a von Graefe's cataract knife.
- ***Cataract extracion:*** Extracapsular extraction is done under cover of systemic steroids which are also to be continued in the postoperative periods.

  Intraocular lens implantation in severe uveitic cases is still controversial.
- ***Band-shaped keratopathy:*** Epithelial scraping and application of 0.1 M solution of disodium EDTA.

INTERMEDIATE UVEITIS (PARS PLANITIS)

Etiology

The exact cause is still not clear. It is thought to be due to some immunological reactions at the pars plana and outer layers of the peripheral retina.

Clinical Features

- ***Age:*** Second to fourth decade.
- ***Sex:*** It can affect either sex.
- ***Laterality:*** It usually bilateral (80%).
- ***Incidence:*** In 5–15% of all uveitic patients.
- ***Symptoms:*** Floaters and blurring of vision.

Signs

- ***Posterior segment***
  - Inflammatory cells in the anterior vitreous.
  - Snow-ball opacities in the vitreous cavity.
  - Snow-bank exudates over the pars plana which is best seen with an indirect ophthalmoscope.
  - Absence of focal inflammatory lesions in the fundus.
- ***Anterior segment:*** It is due to spill-over anterior uveitis:
  - Moderate aqueous cells and flare.
  - Posterior synechiae.

Complications (Causes of Visual Loss)

- Complicated cataract
- Cystoid macular edema (CME)
- Vitreous hemorrhage
- Retinal detachment (tractional).

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Treatment

- Mild cases are self-limiting, and do not require any treatment except periodic observation.
- But, in moderate to severe cases (vision less than 6/12) or in the evidence of CME, the treatment is:
  - ***Sub-Tenon injection of depot steroid every*** 2–3 weeks.
  - ***Systemic corticosteroids***—oral prednisolone (60 mg/day) for two weeks—then tapered off slowly.
  - ***Cryotherapy***—if above treatment fails.
  - ***Pars plana vitrectomy***—if cryotherapy is ineffective.
  - ***Immunosuppressive agents***, very rarely.

POSTERIOR UVEITIS

Etiology

- ***Infectious:*** Toxoplasmosis, tuberculosis, syphilis, etc.
- Autoimmune
- Idiopathic.

Symptoms

- ***Floaters:*** More with peripheral lesions.
- ***Impaired vision:*** Mainly with lesions involving the fovea and papillomacular bundle.

Signs

- ***Anterior segment:*** Usually normal.
- ***Posterior segment***
  - ***Vitreous***
    - ***Opacities:*** Various types of vitreous opacities, caused by inflammatory cells and exudates are:
      - ***Fine opacities:*** They are composed of individual inflammatory cells.
      - ***Coarse opacities:*** They are result of severe tissue destruction.
      - ***Stringy opacities:*** They are caused by alterations in the vitreous gel itself.



Fig. 14.22: Disseminated choroiditis

      - ***Snow-ball opacities:*** They are the large opacities seen in pars planitis, sarcoidosis and candidiasis.

Grading of vitreous opacities (0 to 4+) is done by assessing the density of the opacity, of the fundus with direct or indirect (better) ophthalmoscope (*see* Table 14.5).

- ***Fundus:*** The lesion may be central, focal, multi-focal, geographical or diffuse.
  - ***Choroiditis (Figs 14.22 and 14.23):***
    - ***Active lesions:*** They are white, yellow or greyish patches with reasonably well demarcated margins (Fig. 14.23).
    - ***Inactive lesions:*** They are well-defined white patches of chorioretinal atrophy with pigmented margins.

    Retinal blood vessels pass over the choroiditis patch without interruption.
  - ***Retinitis:*** They appears as white, cloudy areas with indistinct margins, sometimes difficult to differentiate from healthy retina.
  - ***Vasculitis:*** Periphlebitis is more common than periarteritis. Cellular infiltrates around the blood vessels cause perivascular cuffing.

    In severe periphlebitis, there is perivascular accumulation of granulomatous materials—called ***candle-wax dripping*** (Fig. 14.24), seen in sarcoidosis.
  - ***Neovascularization:*** It may be seen in pars planitis or sarcoidosis.

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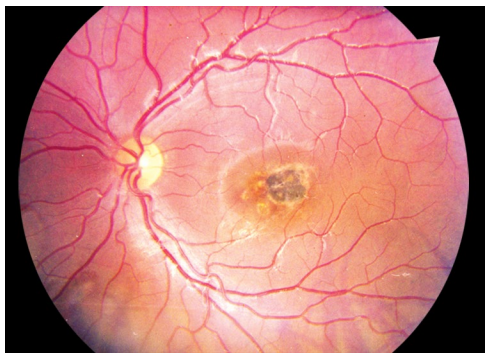


Fig. 14.23: Central choroiditis

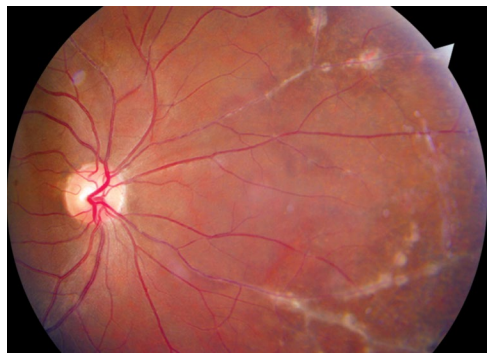


Fig. 14.24: Perivasculitis—candle-wax dripping

- ***Exudative retinal detachment:*** It is in severe choroiditis, e.g. toxoplasmosis.
- ***Optic nerve findings:*** It include optic neuritis (from contagious foci) or optic atrophy (secondary to retinal damage.)

Investigations

- Investigations for tuberculosis
- Investigations for sarcoidosis
- Venereal disease research laboratory (VDRL) and FTA-ABS for syphilis
- Toxoplasma serology (Sabin-Feldman dye test, ELISA)
- Enzyme-linked immunosorbent assay (ELISA) for toxocariasis.

Treatment

- ***For specific infective etiology***
  - Treatment for toxoplasmosis
  - Treatment for toxocariasis
  - AT drugs, etc. with systemic steroids if necessary.
- ***Idiopathic posterior uveitis***
  - ***Systemic corticosteroids:*** Oral prednisolone—1–1.5 mg/kg body weight/day, then in tapering doses.
  - Posterior sub-Tenon injections of depot steroid.
  - Rarely, immunosuppressive agents.

Panuveitis
It is the severe form of uveitis which involves the whole uveal tract.
Common causes are as follows:
- Sympathetic ophthalmitis
- Vogt-Koyanagi-Harada syndrome
- Sarcoidosis
- Behcet's disease.

SPECIFIC UVEITIC ENTITIES

Fuchs' Heterochromic Cyclitis

It is a chronic, non-granulomatous anterior uveitis with insidious onset.

It is usually unilateral and affects middle-aged adult.

Symptoms

Most of the patients are asymptomatic until the development of complicated cataract.

Signs

- Fine KPs are scattered through out the entire endothelium.
- A faint aqueous flare and cells.
- Posterior synechiae are typically absent.
- Diffuse iris stromal atrophy.
- ***Heterochromia*** affected eye is usually hypochromic.
- ***Mydriasis:*** It is due to atrophy of sphincter pupillae.

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- Fine neovascularization of the iris and at the angle (***Amsler's sign:*** Bleeding from these new fine vessels following anterior chamber paracentesis).
- Vitreous may have cells.

Complications: Two main complications—(i) cataract and (ii) glaucoma.

Treatment

- Topical corticosteroids in active stage.
- Mydriatics are unnecessary.
- Extracapsular cataract extraction (ECCE) with intraocular lens (IOL) implantation can be done safely for cataract with good prognosis.

UVEITIS IN JUVENILE RHEUMATOID ARTHRITIS

Juvenile (idiopathic) rheumatoid arthritis (JRA) is a seronegative idiopathic inflammatory arthritis developing in children below 16 years of age.

It is classified as:

- Systemic onset JRA ***(Still's disease):*** Uveitis is extremely rare.
- ***Polyarticular JRA:*** Uveitis is fairly rare.
- ***Pauciarticular JRA:*** Uveitis develops in about 20% of the cases.

Anterior uveitis in JRA is chronic (Fig. 14.25), non-granulomatous, usually bilateral and mostly affecting the girls.

Symptoms: Asymptomatic at the onset, but later, vision is impaired due to complications.

Signs

- Eye is typically white (***white iritis***) even in presence of severe inflammation.
- Small to medium-size KPs.
- Posterior synechiae are very common.
- Pigment deposition on the lens surface.

Complications

- Complicated cataract
- Secondary glaucoma

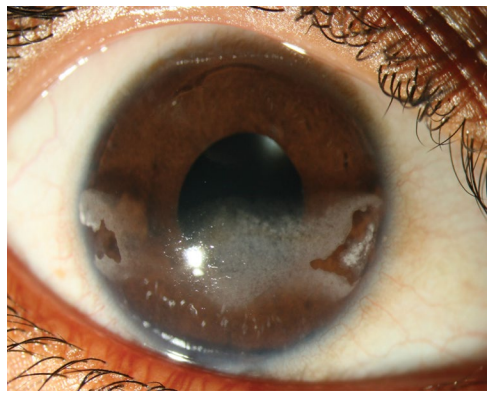


Fig. 14.25: Chronic uveitis in idiopathic rheumatoid arthritis with Barbou-Stoenner-Kelly (BSK) and complicated cataract after surgery

- Band-shaped keratopathy (common)
- Ultimately, phthisis bulbi.

Treatment

- Topical steroids and cycloplegics.
- Sub-Tenon injection of depot steroids.
- ***Oral prednisolone in selected cases:*** They are as this can affect the growth and bone formation of the young patients.
- Cataract surgery is best done by pars plana lensectomy with vitrectomy.

Reiter's Syndrome

- It consists of a triad of urethritis, conjunctivitis and seronegative arthritis.
- Uveitis is typically unilateral, acute and non-granulomatous.
- ***Treatment:*** Topical steroids and cycloplegics.

UVEITIS IN HERPES ZOSTER

- The iridocyclitis is (hyper) acute, non-granulomatous with small KPs.
- Associated severe iris ischemia causes hypopyon which may be mixed with blood.
- Sectorial iris atrophy (20%) with distortion of the pupil.
- ***Complications:*** Secondary glaucoma, complicated cataract and very rarely

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phthisis bulbi (due to ciliary body ischemia) may occur.
- ***Treatment:*** Vigorously with topical steroids, cycloplegics and if necessary with systemic steroids.

TUBERCULAR UVEITIS

It is usually a chronic, granulomatous uveitis. It may be non-granulomatous anterior or posterior uveitis (choroiditis), or vasculitis (Eales' disease) depending upon the immunological status of the patient.
- ***Anterior uveitis:*** Classically, it is a granulomatous type of uveitis with large mutton-fat KPs. It is usually bilateral, through the second eye may be affected after a few months.
- ***Posterior uveitis:*** Commonly, it occurs as a focal or multifocal choroiditis. Rarely, a large choroidal granuloma may be seen in the fundus.

Investigations

- X-ray chest, erythrocyte sedimentation rate (ESR) and Mantoux test
- ***Therapeutic (Isoniazid) test:*** A therapeutic trial of 300 mg/day isoniazid for 3 weeks is reliable. If this causes a dramatic improvement the diagnosis of tuberculosis is highly likely.

Treatment

- Topical steroids and cycloplegics.
- Full course of AT drugs.

SARCOID UVEITIS

Sarcoid is a multisystem disorder of unknown etiology, commonly affecting young adults, and usually presents with bilateral hilar lymphadenopathy and oculocutaneous lesions.

- ***Ocular findings are***—keratoconjunctivitis sicca, conjunctival granuloma and uveitis.
- ***Uveitis:*** It may be anterior, posterior or panuveitis.
  - ***Anterior uveitis***
    - ***Acute granulomatous:*** It is seen in young patients with acute sarcoidosis.
    - ***Chronic granulomatous:*** It usually bilateral and seen among old patients. Busacca's nodules are more frequently seen than Koeppe's nodules, with typical 'mutton-fat' KPs. Band-shaped keratopathy, complicated catract and glaucoma are frequent complications.
  - ***Posterior uveitis***
    - ***Snow-ball*** opacities in the vitreous.
    - Peripheral retinal periphlebitis with candle-wax drippings.
    - Retinal neovascularization at the periphery.
    - Preretinal nodules (sarcoid nodules), mostly located inferiorly and anterior to the equator (***Lander's sign***).
    - Papillitis, optic nerve granuloma, optic disc neovascularization may also be seen.

Investigations

- X-ray chest—hilar lymphadenopathy
- Serum angiotensin converting enzyme (ACE) usually elevated
- Serum calcium—raised
- Kveim test
- Gallium-67 scan of the head and neck.

Treatment

- ***Anterior uveitis:*** Topical steroids and cycloplegics.
- ***Posterior uveitis:*** Systemic steroids and posterior sub-Tenon injection of depot steroids.

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TOXOPLASMOSIS

Toxoplasmosis is an infestation by an intracellular protozoan parasite, *Toxoplasma gondii*.

The cat is the definitive host of the parasite, and human can be infested by three ways:

1. ***Ingestion of under-cooked meat*** by eating the flesh of intermediate host like pig, sheep, cattle, etc.
2. ***Ingestion of oocyte*** via feces of pet cat.
3. ***Transplacental*** from infested pregnant mother to the fetus.

Toxoplasmic uveitis may be:

- Congenital posterior toxoplasmic lesions.
- Recurrent toxoplasmic retinochoroiditis.

Acquired toxoplasmosis: It does not cause any ocular lesion, and usually remains sub-clinical. All the cases of toxoplasmic uveitis in adults are due to recurrence of an old healed congenital chorioretinitis.

Congenital Toxoplasmosis

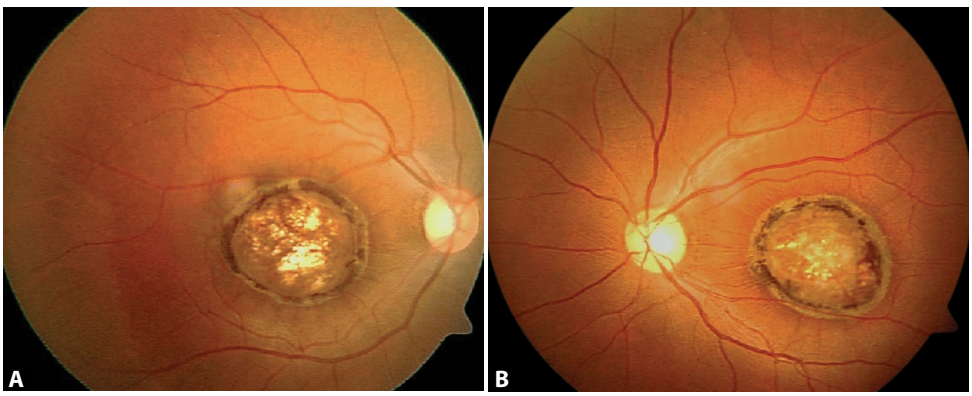
- Transplacental transmission occurs only when the mother is infested during the pregnancy. But if the mother is infested before pregnancy the fetus will not be affected.
- The 'three Cs' of congenital toxoplasmosis are ***chorioretinitis, convulsions and calcification***.
- Most cases are subclinical. In these children, bilateral (Figs 14.26A and B) healed chorioretinal scars are usually detected in later life.

Recurrent Toxoplasmosis

- The recurrence usually takes place between the age of 10–35 years.
- The primary lesion is a retinitis, and the inflammatory reaction seen in choroid, iris and retinal vessels is believed to be immunological in origin.
- ***Clinical features of recurrent lesions:***
  Symptoms: Visual loss, and due to:
  - Opacities in the media.
  - Direct involvement of the fovea.
  - Involvement of the papillomacular bundle and rarely the optic nerve head.
  - Cystoid macular edema.

Signs

- ***Severe vitritis*** resulting in inflammatory cells in the vitreous.
- ***Focal necrotizing retinitis*** is a satellite lesion adjacent to the edge of the old scar. Active retinitis is a white or yellowish-white lesion with fluffy indistinct edges at the post-equatorial fundus.
- ***Deep retinitis*** is less common.
- ***Papillitis*** sometimes, the active retinitis is located adjacent to the optic nerve



Figs 14.26A and B: Bilateral congenital toxoplasmosis

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(juxtapapillary choroiditis of Jensen), or occasionally on the optic nerve head itself.
- Occasionally, there is an associated anterior uveitis.

Complications

- Cystoid macular edema
- Sub-retinal neovascularization
- Tractional retinal detachment.

Diagnosis

- Sabin dye test
- Fluorescent antibody (FA) test
- ELISA for toxoplasma antibody.

Management

Treatment is indicated only when the retinitis is severe and threatening the macula or optic nerve head.
- ***Steroids:*** They should not be given (in any form) alone, and it should be combined with antitoxoplasmic drugs.
  - ***Topical:*** In associated anterior uveitis.
  - ***Posterior sub-Tenon injection:*** In retinochoroiditis.
  - ***Systemic (oral):*** In presence of severe vitritis or in vision-threatening lesions.
- ***Systemic anti-toxoplasmic drugs***
  - ***Sulphonamides:*** Sulphadiazine or triple-sulpha (sulphadiazine/sulphamera-zine/sulphamethazine)—2.0 gm loading dose, then 1 gm 4 times daily for 3–4 weeks.
  - ***Clindamycin:*** Orally 300 mg, 4 times daily for 3 weeks.
  - ***Pyrimethamine:*** 75–100 mg loading dose, followed by 25 mg daily for 3–4 weeks.
    If it is combined with clindamycin, only a 1 week course will then be necessary.
    Pyrimethamine is a ***folic acid antagonist***, which may cause thrombocytopenia and leucopenia. So, ***folinic acid***, 10 mg/day, orally (mixed with orange juice) is to be added with pyrimethamine.
  - ***Minocycline:*** 100 mg once or twice daily for 4 weeks.

TOXOCARIASIS

Toxocara is an intestinal round worm of cats *(Toxocara cati)* and dogs *(Toxocara canis)*. Human ocular toxocariasis is caused by accidental ingestion of food contaminated by feces of pet cat or dog.
- It usually presents between the age of 2 and 9 years with leukocoria, squinting or unilateral loss of vision.
- ***Signs include*** chronic endophthalmitis, vitreous clouding and severe cyclitic membrane formation.
- Toxacara endophthalmitis is considered in differential diagnosis of retinoblastoma where there is an associated secondary glaucoma.
- Diagnosis is confirmed by ELISA.
- ***Treatment:*** (a) Anthelminthic drugs (thiabendazole) (b) Pars plana vitrectomy in case of pseudoglioma formation.

BEHÇET'S DISEASE

It is accompanied by recurrent oral ulceration, genital ulceration, skin lesions together with neurological and articular manifestations.
- Conjunctivitis, episcleritis and keratitis are seen in a few patients.
- ***Fundus shows:*** Vitritis, retinal vasculitis, focal area of retinal necrosis, macular edema and optic neuropathy.
- Visual prognosis is poor, and is due to phthisis bulbi or secondary optic atrophy.
- ***Treatment***
  - Topical and systemic steroids
  - Oral chlorambucil for prolonged period.

VOGT-KOYANAGI-HARADA SYNDROME

- Vogt-Koyanagi-Harada is a severe panuveitis associated with cutaneous, neurological and auditory involvement.
- It is more common in Japanese patients who have an increased prevalence of HLA-DR4 and HLA-DR53 (Fig. 14.27).

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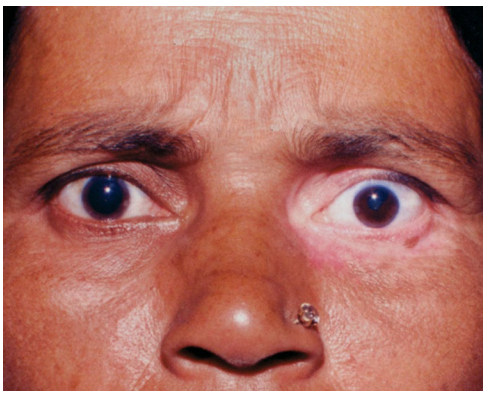


Fig. 14.27: Vogt-Koyanagi-Harada syndrome (left eye)

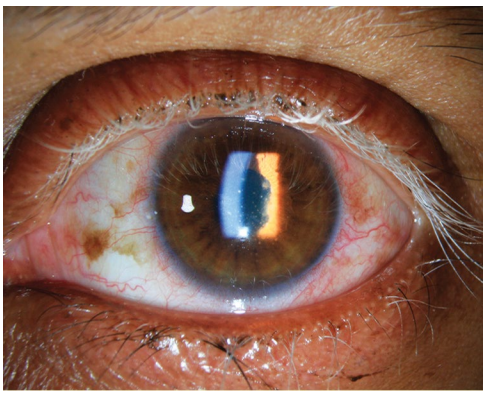


Fig. 14.28: Uveitis in Vogt-Koyanagi-Harada syndrome—note poliosis of the eyelashes

- ***Extraocular lesions***
  - ***Cutaneous***—alopecia, vitiligo and poliosis (Fig. 14.28).
  - ***Neurological***—headache, convulsion and cranial nerve palsy.
  - ***Auditory***—tinnitus, vertigo and deafness.
- ***Ocular lesions***
  - A granulomatous iridocyclitis
  - Multifocal exudative choroiditis
  - Exudative retinal detachment
  - Optic neuritis in some cases.
- ***Treatment***
  - Anterior uveitis—topical steroids and cycloplegics.
  - Exudative retinal detachment—usually subsides after systemic corticosteroid therapy.

SYMPATHETIC OPHTHALMITIS

It is a rare, ***bilateral*** granulomatous panuveitis that occurs following a penetrating injury or surgical trauma.

- The traumatized eye is called ***exciting eye***, and the fellow eye which also develops uveitis is called ***sympathizing eye***.
- Eighty percent cases of sympathetic ophthalmitis occur within 3 months, and 90% of cases occur within 1 year after trauma.

Symptoms (Insidious Onset)

Initial symptoms in the ***sympathizing eye*** are pain, photophobia, and difficulty in near vision due to loss of accommodation. In the ***exciting eye*** there is also pain, irritation and redness.

Signs

- In the ***exciting eye*** there is ciliary congestion and evidence of initial insult.
- In the ***sympathizing eye***
  - Earliest feature is ***retrolental flare***.
  - Typical granulomatous uveitis with mutton-fat KPs, iris nodules, posterior synechiae and secondary glaucoma.
- ***Posterior segment shows:*** (i) Small, deep, yellow-white spots (Dalen-Fuchs' nodules) scattered throughout the fundus, and (ii) perivasculitis and retinal edema.

Complications

Cataract, secondary glaucoma, and eventual blindness from phthisis bulbi may occur in many cases.

Etiopathogenesis

Sympathetic ophthalmitis almost always results from a penetrating injury. Wounds in the ciliary region (***dangerous zone*** of the

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eye) involving the ciliary body, and its incarceration in the scar, are considered especially dangerous. It very rarely occurs if actual suppuration has taken place in the ***exciting eye***.

It is an autoimmune reaction to antigens in uveal tissue, uveal pigments or retinal antigen (retinal-S antigen).

Pathology

Histopathology is similar in both the exciting and sympathizing eye.

- Nodular aggregation of lymphocytes and plasma cells scattered throughout the uveal tract.
- Pigment epithelium of the iris, ciliary body and choroid proliferates, and along with epithelioid cells and macrophages, they form the nodules called ***Dalen-Fuchs***' nodules.
- Retina also becomes heavily infiltrated with lymphocytes and plasma cells.

Treatment

Therapeutic

- Intensive therapy with topical, sub-Tenon injections and systemic corticosteroids. Systemic steroids are given in high doses (80–100 mg/day) and are gradually tapered, once the uveitis is controlled.
- Immunosuppressive agents in steroid-resistant cases.

Preventive

- Prompt and meticulous closure of all penetrating injury.
- If the injured eye is unsalvagable, enucleation should be done within 2 weeks following injury.
- Beneficial effect of enucleation of the exciting eye, after the development of sympathetic ophthalmitis is controversial.
- Many surgeons prefer enucleation of the exciting eye within 2 weeks of the onset of sympathetic ophthalmia, as it favorably affects the eventual prognosis of the sympathizing eye.

GLAUCOMATOCYCLITIC CRISIS (POSNER-SCHLOSSMAN SYNDROME)

Glaucomatocyclitic crisis is characterized by acute or subacute attacks of secondary open angle glaucoma associated with mild anterior uveitis.

The disease affects the young adults and usually unilateral. During an attack, the intraocular pressure is usually severely elevated (40–60 mm of Hg), and lasts for few hours to few days.

Symptoms

- Colored halos around the light
- Diminution of vision
- Mild headache, but pain is rare.

Signs

- Corneal epithelial edema
- Fine non-pigmented KPs
- Normal anterior chamber depth
- Aqueous cells and flare
- Posterior synechiae formation are rare.

Differential Diagnosis

The condition is to be differentiated from acute attack of angle-closure glaucoma, as the treatment with atropine would be disastrous in a case of angle-closure glaucoma.

A normal depth of anterior chamber and presence of few KPs establish the diagnosis of glaucomatocyclitic crisis.

Treatment

- Topical steroids and cycloplegics
- Secondary glaucoma is usually treated with tab acetazolamide, and timolol maleate drops.

CYTOMEGALOVIRUS RETINITIS (FIG. 14.29)

It is a rare chronic diffuse exudative lesion of the retina caused by cytomegalovirus (CMV) which occurs in patients with an impaired immune system, e.g:

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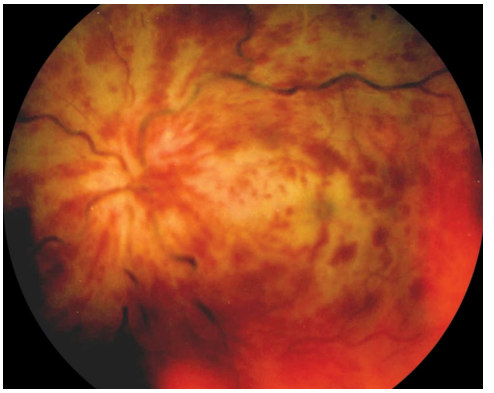


Fig. 14.29: Cytomegalovirus retinitis—central retinal venous occlusion

- AIDS
- Cytotoxic chemotherapy
- Long-term immunosuppression following renal transplantation.

Signs

- Scattered white patches with perivascular sheathing.
- Full-thickness hemorrhagic retinal necrosis with edema sometimes described as ketchup (tomato sauce) on cottage cheese appearance at the posterior pole.
- Ultimately, whole retina is involved with subsequent retinal atrophy.

Treatment

Intravenous injection of gancyclovir or foscarnet may cause regression in some cases.

ONCHOCERCIASIS (RIVER BLINDNESS)

- It is caused by a filarial worm *Onchocerca volvulus*.
- The vector is the blood-sucking black fly *Simulium*.
- Onchocerciasis is mainly seen in endemic areas of African countries.
- ***Ocular lesions*** are sclerosing keratitis, retinitis and anterior uveitis.
- Iridocyclitis and chorioretinitis are the ***cause of blindness*** due to onchocerciasis.
- ***Specific therapy*** of onchocerciasis is diethylcarbamazine (for microfilaria) and suramin (for adult worm).
- ***Ivermectin is now the drug of choice*** for the treatment.
- Onchocerciasis is now included in Vision-2020 programme (*see* Chapter 26).

PRESUMED OCULAR HISTOPLASMOSIS SYNDROME

This syndrome has been related to *Histoplasma capsulatum* infection. It is presumed because:

- Presumed ocular histoplasmosis syndrome (POHS) has never been reported in patients with active systemic histoplasmosis.
- The organism, *H. capsulatum* has not been recovered from an eye with POHS.
- Histoplasmin skin test is positive in 90% of patients with POHS.
- Increased prevalence of POHS, where histoplasmosis is endemic, e.g. Mississippi-Ohio-Missouri river valley.

Symptoms

It is asymptomatic unless it involves the macula, and the earliest symptom of macular involvement is metamorphopsia.

Signs

- Atrophic mid-peripheral spots called ***histospots***.
- Peripapillary atrophy.
- Linear chorioretinal atrophy.
- Sub-retinal choroidal neovascularization, resulting in hemorrhagic disciform lesion at the macula.
- The vitreous is never involved.

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Treatment

Argon-laser photocoagulation of sub-retinal neovascular membranes is advised to prevent the formation of disciform macular scar.

ACUTE RETINAL NECROSIS

Acute retinal necrosis (ARN) is an extremely rare devastating necrotizing retinitis, affecting the otherwise healthy individuals.

- ***Symptoms*** are periorbital pain followed by blurring of vision.
- ***Signs*** are noticed either at the posterior pole or at the periphery of the fundus:
  - Deep, multifocal, yellowish white patches with sheathing of the vessels.
  - The lesions gradually become confluent and represent a full thickness necrotizing retinitis.
  - Most of the eyes develop retinal holes, with rhegmatogenous retinal detachment.
  - It usually resolves within 4–12 weeks, leaving behind atrophic retinal areas. In some eyes, visual acuity remains fairly good despite severe necrosis.
- ***Treatment:*** Systemic antibiotics, steroids, and cytotoxic agents may be beneficial.

AIDS

Human immunodeficiency virus (HIV) is the causative organism of this invariably fatal disease.

Ocular complications occur in about 75% of AIDS patients and almost all ocular structures are affected.

The lesions are due to retinal microvasculopathy, occurrence of Kaposi's sarcoma and opportunistic super infections by *Pneumocystis carini*, *Toxoplasma*, CMV, Herpes zoster virus (HZV), *Candida*, Molluscum contagiosum, etc.

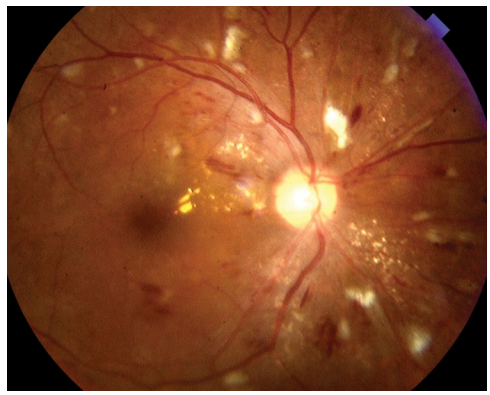


Fig. 14.30: Cotton-wool spots in retinal microvasculopathies in AIDS

- ***Retinal microvasculopathy***
  - Cotton-wool spots are the most common ocular lesion, and are seen in 50% of the patients (Fig. 14.30).
  - They are probably due to immune complex deposits in the precapillary arterioles.
  - They appear as discrete fluffy opacities, and are transient.
  - Superficial and deep hemorrhages are seen in some patients.
- ***Kaposi's sarcoma***
  - It involves the eyelids and conjunctiva.
  - It appears as a bright-red mass, most frequently in the lower fornix of the conjunctiva.
  - When it affects the lid, it can lead to entropion, trichiasis, blepharoconjunctivitis, etc.
- ***Opportunistic infections***
  - Cytomegalovirus retinitis is the most common opportunistic infection and the major cause of visual loss in AIDS patients. Its appearance is a grave prognostic sign as most of the patients will die within 6–8 weeks
  - Herpes zoster ophthalmicus
  - Candida endophthalmitis
  - Toxoplasma retinochoroiditis
  - Herpes simplex infection
  - Molluscum contagiosum of lids (Fig. 14.31).

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Fig. 14.31: Molluscum contagiosum in AIDS

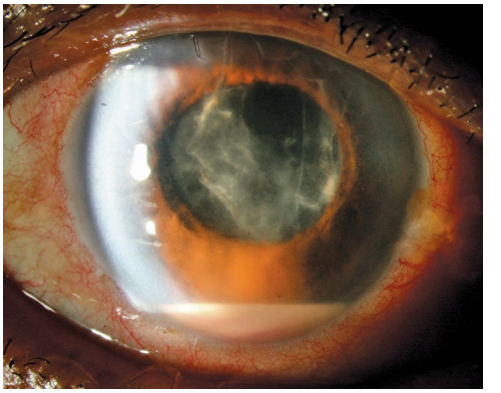


Fig. 14.32: Endophthalmitis after cataract surgery

Management

- Zidovudine (aziedothymidine) is seen to be effective in HIV-retinitis.
- In CMV retinitis—intravascular injection of ***Gancyclovir*** or ***Foscarnet*** may be effective.
- Kaposi's sarcoma is sensitive to radiotherapy.

ENDOPHTHALMITIS

It is a severe form of intraocular inflammation involving the ocular cavities and inner coats of the eyeball.

Etiology

Infective

Most of the cases are infective in nature, which may be:

- ***Exogenous***
  - Following penetrating injury.
  - Following intraocular surgery (Fig. 14.32).
  - Following wound leak or bleb infection (after glaucoma surgery).
  - Spread from the adjacent structures, e.g. perforating corneal ulcer.
- ***Endogenous:*** Septic emboli from bacterial endocarditis (Fig. 14.33), otitis media, meningitis, following urologic surgery, etc. (metastatic endophthalmitis). They are mainly seen in immunocompromized subjects.

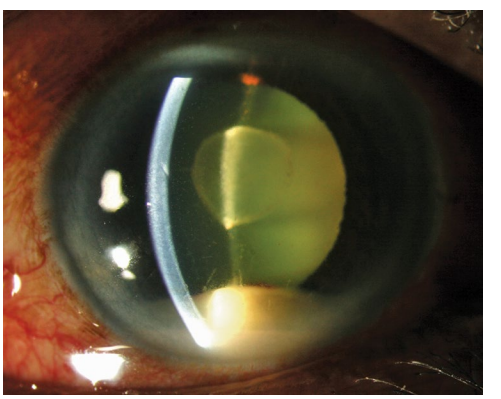


Fig. 14.33: Endogenous bacterial endopthalmitis

Common organisms include:

- ***Bacteria:*** *Staphylococci, Streptococci, Pneumococci, Pseudomonas, Bacillus subtilis, E. coli*, etc.
- ***Fungus:*** *Candida, Aspergillus, Fusarium*, etc. (Fig. 14.34).
- ***Parasitic:*** Toxocara.

Toxic (Sterile)

- Retained intraocular foreign body, e.g. pure copper.
- Following IOL implantation (due to chemicals adherent to the implant).
- Toxic reaction to the chemicals, adherent to the instruments (following chemical sterilization).
- Necrosis of intraocular tumors.

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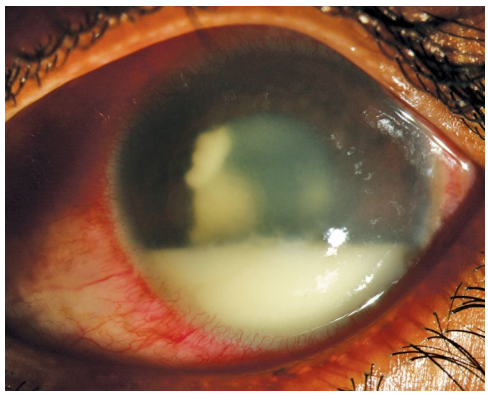


Fig. 14.34: Endogenous fungal endophthalmitis

- Phacoanaphylactic uveitis leading to endophthalmitis.

Symptoms

- Usually unilateral with subacute onset.
- Moderate pain and redness of the eye.
- Photophobia and lacrimation.
- Gross impairment of vision.

Signs

- Lid edema.
- Ciliary congestion and chemosis of the conjunctiva.
- Keratic precipitates, aqueous cells and flare.
- Vitreous is somewhat hazy which prevents clear view of the fundus.
- Later, a yellowish-white reflex appears at the pupillary area indicating massive exudation in the vitreous.
- Intraocular pressure is low and eyeball gradually shrinks to phthisis bulbi.
- Sometimes, it may progress into panophthalmitis.
- Ultimately, vision reduces to 'no PL'.

Management

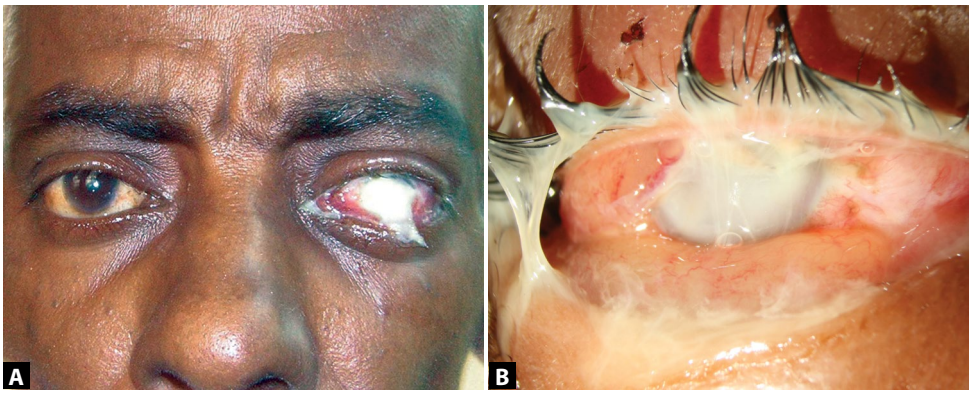
It should be very energetic and prompt.

- ***Aqueous or vitreous tap:*** A vitreous tap gives a better yield than aqueous tap.
  - Smear preparation for Gram-stain, Gimsa stain and potassium hydroxide mount.
  - ***Inoculation in culture media:*** Blood agar, chocolate agar and Sabouraud's media.
- ***Intravitreal injections:*** It is to be given within 6 hours to achieve maximum intraocular concentration of antibiotics.
  - ***Antibiotics:*** Gentamycin (400 µg), cephazoline (2.25 mg), vancomycin (1.0 mg), ceftazidime (0.2 mg), amikacin (0.2 mg). Among these ceftazidime and vancomycin are mostly preferred.
  - ***Antifungal:*** Amphotericin-B (5 µg) in case of suspected proved fungal endophthalmitis.
  - ***Steroids:*** Dexamethasone (360 µg) may be added to antibacterial agent.
- ***Antibiotic therapy:*** Systemic (intravenous or intramuscular), topical, and subconjunctival or sub-Tenon injection, are employed.
  - ***Systemic:*** (i) ***Oral:*** Ciprofloxacin—750 mg—1 g twice daily for 7 days, (ii) ***Intravenous:*** Cephazoline—1 g 4 times daily.
  - ***Topical:*** Fortified tobramycin and moxifloxacin drops every 30 minutes.
  - ***Sub-Tenon:*** Gentamycin (40 mg) and cephazoline (125 mg) daily for 5–7 days.
- ***Antifungal:*** (i) ***Systemic:*** Oral ketoconazole or fluconazole is given for 4–6 weeks. (ii) ***Topical:*** Natamycin or Amphotericin-B (0.15%) drop is given every 30 minutes.
- ***Steroids:*** They are indicated only in suspected bacterial endophthalmitis. Systemic (prednisolone—60 mg/day) and topical (dexamethasone 1 hourly).
- ***Subsequent therapy:*** It is governed by the culture results.
- ***Vitrectomy:*** Early vitrectomy is considered if the intravitreal injection fails to control endophthalmitis within 48–72 hours, and the vision is considerably reduce.

PANOPHTHALMITIS (FIGS 14.35A AND B)

This is the most severe form of inflammation of the ocular cavities which involves all three coats of the eyeball as well as the Tenon's capsule.

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Figs 14.35A and B: Panophthalmitis

Etiology

Same as endophthalmitis except the toxic causes. Panophthalmitis is mostly caused by bacterial infections.

Symptoms

- Severe pain with radiation
- Swelling of the eyelids
- Complete loss of vision
- Fever, malaise, headache, etc.

Signs

- No perception of light (no PL).
- Marked lid edema.
- Conjunctival chemosis and congestions.
- Slight proptosis, and there may be limitation of movements.
- Cornea is hazy and necrotic.
- Anterior chamber is full of pus.
- Intraocular tension is raised initially.
- Globe may rupture at the limbus or at the pre-existing wound, and pus will come out at those places.
- The signs may gradually subside, and the eye becomes a shrunken mass of fibrotic tissue (phthisis bulbi).

Complications

- Orbital and facial cellulitis
- Cavernous sinus thrombosis
- Meningitis or encephalitis.

Treatment

- Medical treatment with intensive antibiotics is very rarely successful.
- Evisceration of the eyeball is finally the treatment of choice (*see* page 481).
- Enucleation is contraindicated.

DEGENERATIONS OF UVEAL TRACT

Iridocorneal Endothelial Syndromes

This is a spectrum of diseases of unknown etiology, essentially due to abnormalities in the corneal endothelium.

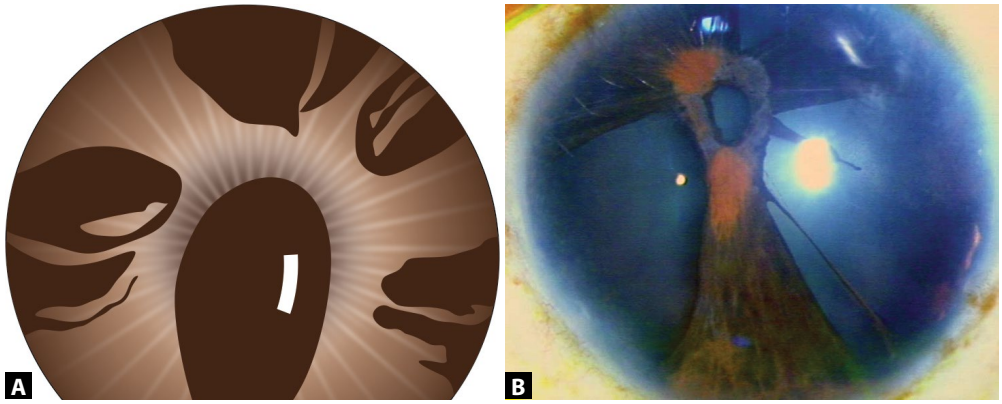
After metaplasia, the ***corneal endothelium proliferates***, and ***Descemet's membrane***-like material covers the anterior iris surface and angle of the anterior chamber, causing iris anomalies and secondary glaucoma.

These disorders are slowly progressive over a period of 10 years or more. Women are more commonly affected than men.

The spectrum includes three conditions:

1. ***Progressive essential iris atrophy (Figs 14.36A and B)***
   - It is a ***unilateral*** condition characterized by ***patchy iris atrophy*** with partial or complete hole formation.

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Figs 14.36A and B: Essential iris atrophy—polycoria; corectopia and PAS

- There is distorted and displaced pupil ***(corectopia)***.
- ***Holes in the iris*** get are gradually enlarged and coalesce together.
- ***Glaucoma*** then ensues from peripheral anterior synechiae and from damage to the trabecular meshwork.
- ***Treatment*** is directed towards secondary glaucoma, and is often, not effective either by medical or surgical means.

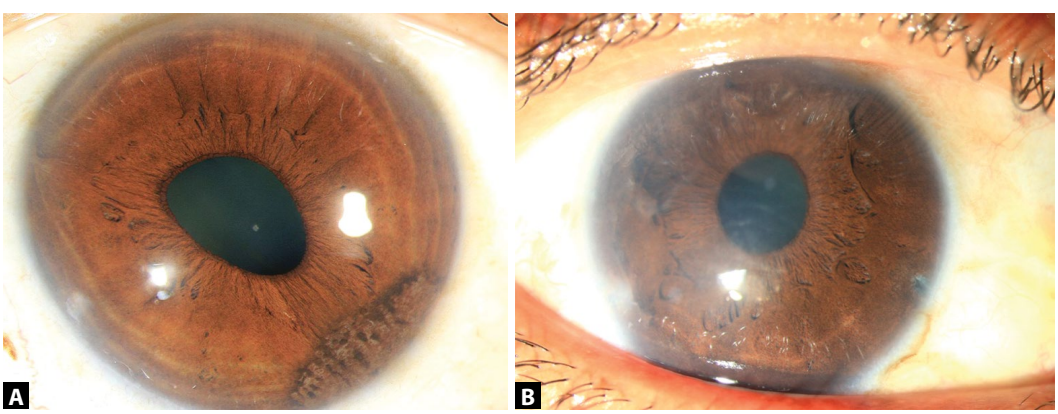
2. ***Iris nevus syndrome (Cogan-Reese)***
   - Characteristic feature is dark-brown pigmented nodules in the iris stroma (as small woolen-spherules) (Fig. 14.37A).

Iridoschisis

- It is a rare, diffuse, bilateral, senile degenerative condition of the iris. It may also be precipitated by trauma.
- Large dehiscences appear on the anterior mesodermal layer of the iris and strands of tissue may float into the anterior chamber.
- Treatment is not necessary.

   - Peripheral anterior synechiae and secondary glaucoma may also occur.
   - ***Treatment*** is directed towards the control of secondary glaucoma.

3. ***Chandler's syndrome (Fig. 14.37B)***
   - It is mainly associated with ***endothelial disturbances and corneal edema***.



Figs 14.37A and B: **A.** Iris nevus syndrome; **B.** Chandler's syndrome

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- ***Iris atrophy is minimal*** and without a tendency to hole formation.
- Peripheral anterior synechiae and ***secondary glaucoma are less common***, and the control of glaucoma is easier.
- ***Penetrating keratoplasty*** may be considered for corneal edema.

RUBEOSIS IRIDIS (FIG. 14.38)

It means neovascularization of the iris. It occurs as an irregularly distributed network of new vessels on the iris surface and the stroma.

Causes

- Diabetes mellitus
- Central retinal venous occlusion
- Heterochromic cyclitis of Fuchs
- Carotid artery occlusive diseases
- Intraocular tumors
- Anterior segment ischemia
- Following vitrectomy in aphakic eyes.

The new blood vessels and associated fibrous tissue may cover the trabecular meshwork, and cause peripheral anterior synechiae. These ultimately close the angle of the anterior chamber and cause an intractable neovascular glaucoma.

Treatment

Neovascular glaucoma is difficult to treat.

- ***Panretinal photocoagulation*** is the treatment of choice in most cases. Photoablation of the neovascularized retina is followed by involution of iris vessels.
- ***Panretinal cryocoagulation*** helps similarly, especially in presence of opacity in the media (e.g. cataract or vitreous hemorrhage).
- ***Cyclocryotherapy*** in resistant cases.

CHOROIDAL DEGENERATIONS

As the outer layers of the retina receive their nourishment from the choriocapillaries, degeneration of the choroid is often associated with atrophy of the retina, and migration of pigments in the superficial retinal layers. Secondary degenerations occur following inflammatory lesions, myopia, or in late stages of glaucoma.

Primary Choroidal Sclerosis (Fig. 14.39)

It is a degenerative vascular sclerosis of the choroid associated with secondary degeneration of the retina. It occurs as an isolated lesion, and is not associated with generalized vascular sclerosis.

Pathologically, the sclerosis is limited to the choriocapillaries and precapillary arterioles, and the veins are being less affected.

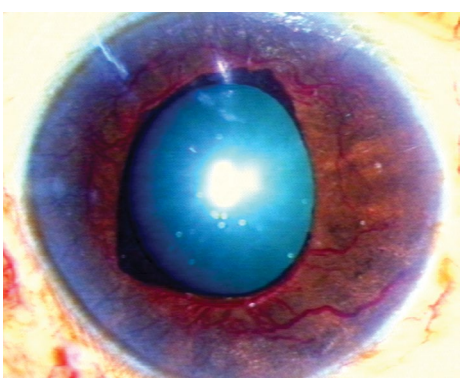


Fig. 14.38: Rubeosis iridis with ectropion uveae

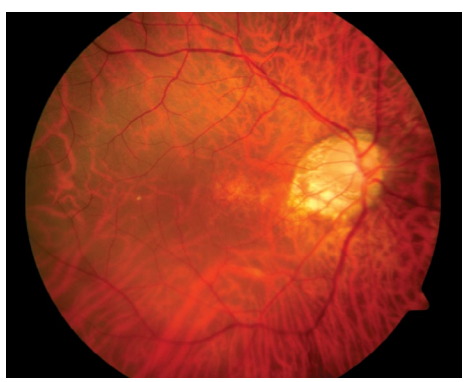


Fig. 14.39: Choroidal sclerosis

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Ophthalmoscopy shows diffuse atrophy of the retinal pigment epithelial (RPE) and choriocapillaries, and a tessellated appearance of the fundus. The larger sclerosed choroidal vessels are more visible as white lines against the irregular pigmented areas.

It occurs in two forms:

1. ***Diffuse or generalized sclerosis:*** It affects the whole fundus. It may cause bilateral progressive diminution of vision, particularly at night.
2. ***Localized sclerosis:*** It affects the central area or circumpapillary region, and is also known as central areolar choroidal dystrophy. Central vision is gradually reduced with development of central scotoma.

Gyrate Atrophy of Choroid (Fig. 14.40)

This is an inherited (autosomal recessive), inborn error of amino acid metabolism due to defective activity of ornithine ketoacid amino transferase enzyme resulting in hyperornithinaemia.

- The ***symptoms*** include progressive dimness of vision with night blindness in young adults.
- ***Ophthalmoscopy*** shows, scalloped to circular patches of chorioretinal atrophy in the far and midretinal periphery. The patches increase in size and coalesce together, and finally, whole fundus is affected, with preservation only of the macula.
- ***Perimetry*** shows, constriction of the peripheral visual field corresponding to the extent of fundus lesion.
- ***Treatment:*** A diet low in proteins and arginine (precursor of ornithine) may be beneficial.

Massive doses of pyridoxine (vitamin B_6) normalize the hyperornithinaemia, and may have beneficial effect on the ocular lesion.

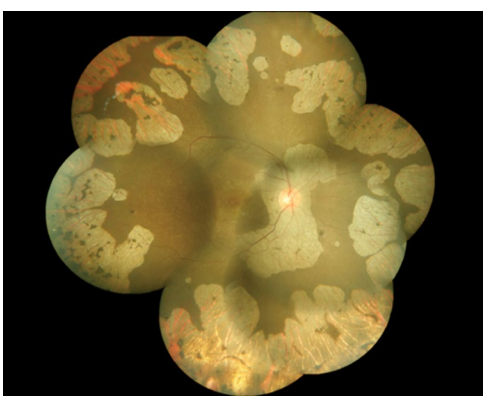


Fig. 14.40: Gyrate atrophy of the choroid

Choroideremia

It is an inherited (X-linked recessive) choroidal atrophy with secondary atrophy of the retina, resulting in night blindness.

- The disease characteristically affects only males and starts within 5–10 years of age with defective night vision.
- ***Ophthalmoscopy*** shows, diffuse mottled depigmentation of the RPE. Eventually, large patches of chorioretinal atrophy develop in the equatorial region. The atrophy then spreads centrally and peripherally, with annular scotoma and progressive constriction of the visual field. Central vision is last to be affected.

The retinal vessels are characteristically normal.

- Female carriers often clinically show mild fundus changes (patchy atrophy and mottling of the RPE at the equator), though they are asymptomatic.
- No treatment is available.

Angioid Streaks (Fig. 14.41)

Angioid streaks are irregular and jagged network of red to brown lines, mainly seen in the central fundus.

- They are produced by crack-like dehiscences in the collagenous and elastic portion of the Bruch's membrane.
- The streaks or lines typically radiate outwards in a tapering fashion from the peripapillary area.

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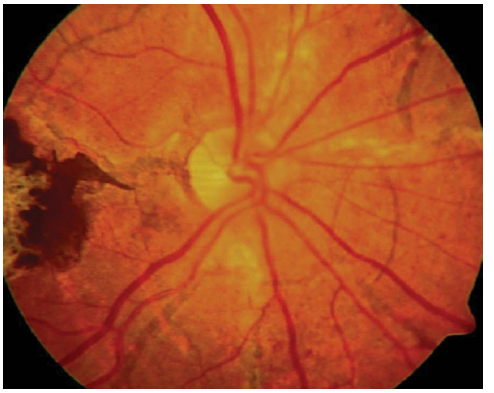


Fig. 14.41: Angioid streak

- The lesions are approximately the width of a retinal vessel, which may thus resemble (angioid). But the streaks are darker, have an irregular contour with serrated edges, and tend to terminate abruptly.
- ***Systemic associations***
  - Pseudoxanthoma elasticum (Gronblad-Strandberg syndrome)—most common.
  - Paget's disease
  - Ehlers-Danlos syndrome
  - Sickle-cell disease
  - Rarely, acromegaly, hypercalcemia and lead poisoning.
- Ocular changes are normally asymptomatic. The causes of visual impairment may be due to:
  - Involvement of the fovea by a streak,
  - Choroidal neovascularization, or
  - Choroidal rupture.

Acute Posterior Multifocal Placoid Pigment Epitheliopathy

- It is a rare, bilateral, idiopathic disease affecting the young adults.
- The ***primary site of lesion*** is at the level of RPE, although it has also been suggested as a ***vasculopathy*** of the choriocapillaries.
- Initially, the ***patient presents*** with a subacute loss of central vision, and within a few days the fellow eye is usually affected.
- ***Ophthalmoscopy*** shows, the typical deep, cream-colored placoid lesions, involving the posterior pole within the equatorial region.
- ***Fundus fluorescein angiography*** shows patchy irregular choroidal filling with late staining.
- Spontaneous resolution with good visual recovery is usual, within a few weeks.
- There is no ***effective treatment***.

Serpiginous (Geographical) Choroidopathy

It is a rare, idiopathic, recurrent disease of the RPE and choriocapillaries affecting the middle-aged patient.

- Initially, the patient remains asymptomatic.
- The chorioretinal lesion usually starts around the optic disc, and spreads outwards in all directions.
- ***Acute lesions*** are cream-colored opacities with hazy border without any inflammatory cells in the vitreous.
- With healing, they become inactive, leaving scalloped 'punched out' areas of chorioretinal atrophy.
- Fresh acute lesions usually arise as extensions from old scars, and successive attacks result in serpiginous extension of the destruction process peripherally from the peripapillary area.
- Involvement of the fovea causes a permanent loss of central vision.
- Fluorescein angiography confirms the diagnosis.
- There is no ***effective treatment***.

CHOROIDAL DETACHMENT (FIG. 14.42)

It means separation of the choroid from the sclera which occurs frequently.

Causes

- ***Following intraocular operations:*** Sudden lowering of intraocular pressure → vasodila-

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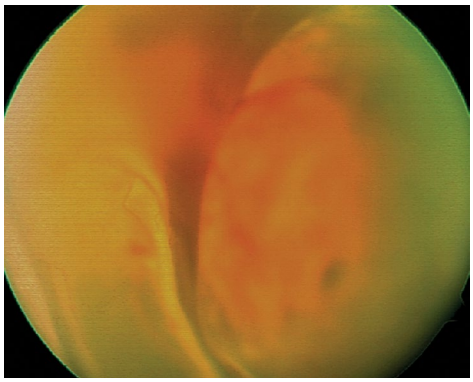


Fig. 14.42: Choroidal detachment with retinal detachment (Kissing choroidals)

tation of the choroidal vessels → exudation onto the outer lamella of the choroid → detachment
- Following intraocular hemorrhage
- Choroidal tumors
- Exudative choroiditis.

Photopsia and floaters (as seen in retinal detachment) are absent.

Signs

- Shallow anterior chamber.
- Intraocular pressure is very low as a result of hyposecretion of aqueous due to a concomitant ciliary body detachment.
- It may be visible as dark-brown mass by oblique illumination after mydriasis.
- ***Ophthalmoscopy*** shows smooth, elevated, dark-brown bullous lesions, which are more prominent on the temporal and nasal sides.

The elevations do not extend to the posterior pole because they are limited by the adhesions, where the vortex veins enter their scleral tunnels.

Treatment

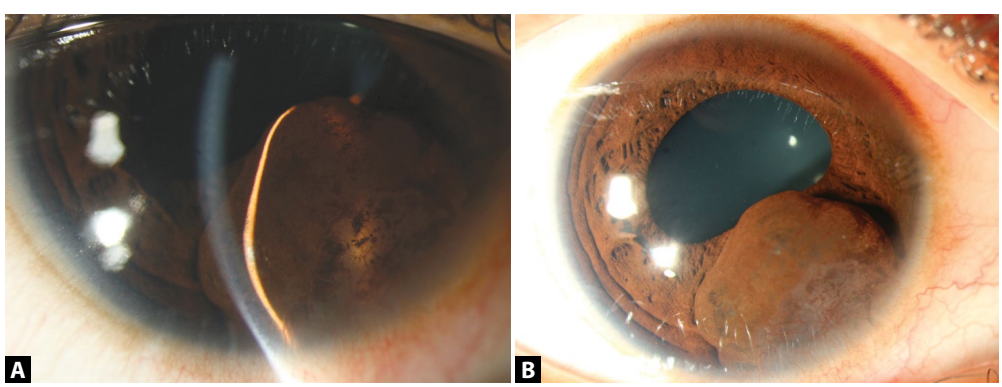
The prognosis is usually good. The choroid is re-attached, and anterior chamber is reformed, without any treatment. However, in some persistent cases, it requires treatment.
- Tab acetazolamide, 250 mg 4 times daily.
- To look for 'wound leak' and if it is there—treatment is done by regular patching, or conjunctival flap or re-suturing.
- In recalcitrant cases, drainage of the subchoroidal fluid through a diathermy puncture at the most prominent site of detachment has been suggested.

IRIS CYSTS

Iris cysts are not very uncommon, and they may be ***primary*** or ***secondary***.

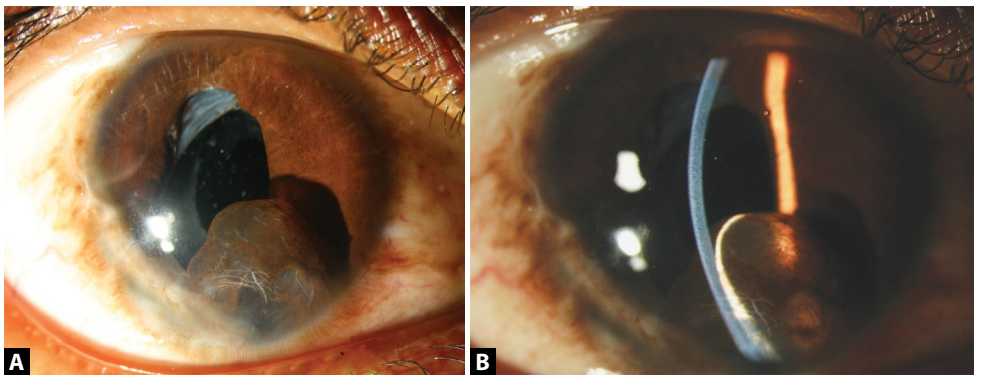
Primary Iris Cysts (Figs 14.43A and B)

- They usually arise from the iris pigment epithelium and very rarely, from the iris stroma.



Figs 14.43A and B: A. Primary Iris Cysts; **B.** Same in slit section

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Figs 14.44A and B: Secondary iris cyst. **A.** Post cataract surgery; **B.** Translucent in slit section

- Those arising from the pigment epithelial layer, are stationary and asymptomatic.
- They are globular, dark-brown structures which transilluminate.
- Treatment is required in selected cases if it touches the cornea.

Secondary Iris Cysts (Figs 14.44A and B)

- ***Implantation cysts:*** They develop from implantation of epithelial cells on to the iris following intraocular surgery or penetrating injury.
  - The cysts lie on the anterior surface of the iris, and grayish-white in color.
  - They frequently enlarge and lead to complications, like anterior uveitis and glaucoma.
  - ***Surgical excision*** is the treatment of choice.
- ***Drug-induced iris cysts:*** They occur following prolonged use of long acting miotics, such as phospholine iodide.
  - ***Treatment:*** Withdrawal of the drug and simultaneous use of phenylephrine (2.5%) with long-acting miotic, prevents formation of the iris cysts.

TUMORS OF THE UVEAL TRACT

See Chapter 20.

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15 CHAPTER Diseases of the Lens

Being avascular and composed entirely of epithelium surrounded by a capsule, the lens is incapable of becoming inflamed. Its cells, apart from those at the equator and the anterior epithelium, are incapable of proliferation. Its major function is to transmit and refract light rays. Its only way of responding to insults is to become opaque.

DISEASES OF THE LENS

Symptoms

- ***Decrease of vision for far and near:*** It is in cortical cataract.
- ***Dimness of vision for distance with good near vision without glasses:*** It is in nuclear sclerosis.
- ***Monocular diplopia or polyopia, and colored halos:*** It is in early immature cortical cataract.
- ***Acute pain, redness and watering in an eye with cataract:*** It is in lens-induced secondary glaucoma or uveitis.
- ***White pupillary reflex (leukocoria):*** As noticed by the parents or relatives.

Signs

- ***Color*** of the crystalline lens.
- ***Presence of iris shadow:*** It is indicates immature cataract.
- ***Presence of Purkinje's 3rd and 4th images:*** It is indicates normal transparent lens as they are formed by the ***anterior convex*** and ***posterior concave*** surfaces of the lens respectively.
- ***Fundal glow on distant direct ophthalmoscopy shows:*** The cataracts as dark opacities against the red background.
- ***Slit-lamp examination*** for precise location and details of the opacity.
- ***Absence of the lens:*** It is indicated by deep anterior chamber and tremulousness of the iris, due to loss of support of the iris, and by the absence of 3rd and/or 4th Purkinje's images.

CONGENITAL ANOMALIES OF THE LENS

Anomalies of the Lens Shape

Coloboma (Fig. 15.1)

There is a defect usually in the lower margin, and it may be associated with similar defects involving the iris, ciliary body and choroid. The notching or indentation in the lens is simply due to a localized defect in the zonule, allowing the lens equator to retract. Giant retinal tear may occur in these eyes. Restoration of defective zonule is not possible (Fig. 15.1).

Anterior Lenticonus

This is an anterior conical projection of the center of the lens. It interferes with vision as the central part of the lens becomes highly myopic. Ophthalmoscopically, a dark disc-like ***(oil***

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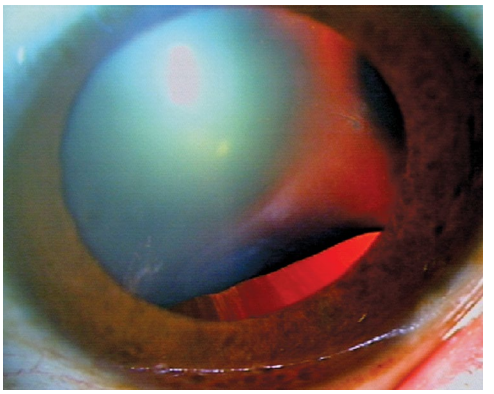


Fig. 15.1: Coloboma of the lens

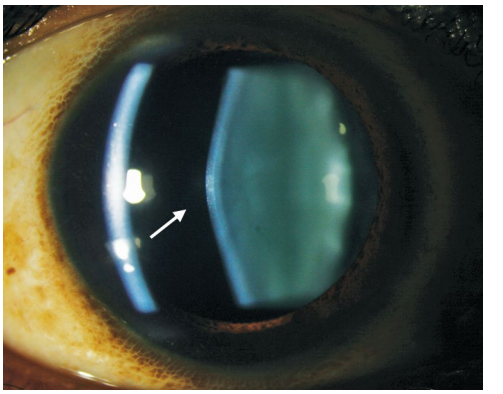


Fig. 15.2: Anterior lenticonus (arrow)

globule) reflex is visible in the center of the red-reflex, which distorts the fundus view (Fig. 15.2).

It occurs in some patients with ***Alport's syndrome*** with familial hemorrhagic nephritis and sensorineural deafness.

Posterior Lenticonus (Fig. 15.3)

It is more common than anterior lenticonus with a posterior conical or globular (lentiglobus) bulge in the axial zone of the lens.

The posterior bulge increases progressively with age, and the lens cortex in the cone may become opaque and obscure the lenticonus. It also gives ***oil globule*** appearance in ophthalmoscopy.

Spherophakia (Microspherophakia) (Fig. 15.4)

- In spherophakia, the lens is small and spherical (increased anterior and posterior curvatures of the lens).
- The zonular fibers are easily visible with pupillary dilatation. Subluxation of the lens is common, because the stretched zonules become weak and breakable.
- Angle-closure glaucoma occurs when the small, round lens blocks the flow of aqueous through the pupil. As this glaucoma is aggravated by miotics and relieved by mydriatics, it is called ***inverse glaucoma***.

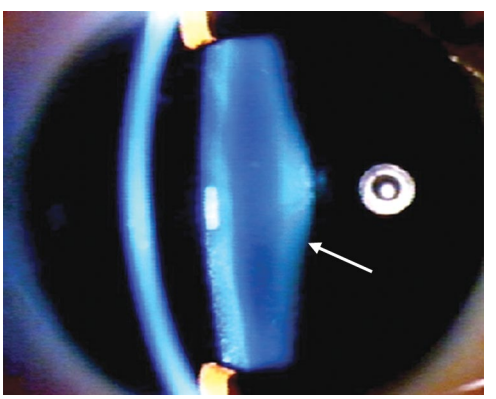


Fig. 15.3: Posterior lenticonus (arrow)

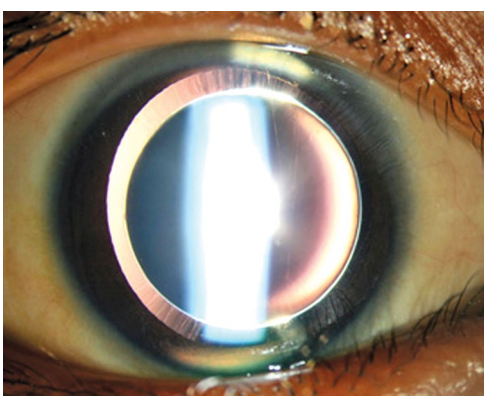


Fig. 15.4: Spherophakia

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- The causes of spherophakia are:
  - Familial microspherophakia
  - Weill-Marchesani syndrome (WMS)
  - Marfan's syndrome
  - Hyperlysinemia.

ECTOPIA LENTIS

Ectopia lentis is a congenital, bilateral, subluxation or dislocation of the lens.

- In ***subluxation*** (due to partial zonular defect), the lens is displaced sideways, but remains behind the pupil.
- But in ***dislocation*** (when the lens loses all zonular supports), the lens is moved either forwards into the anterior chamber, or backwards into the vitreous.

The symptoms are mainly optical.

- In ***subluxation***, the lens becomes more spherical producing myopic astigmatism. The division of 'phakic' and 'aphakic' zone of the pupil is usually not evident, unless the pupil is dilated.
- In ***dislocation***, the eye becomes markedly hypermetropic with loss of accommodation. Migration of the dislocated lens into the anterior chamber or vitreous causes secondary glaucoma.

  A subluxated lens usually, but not inevitably becomes dislocated with increasing age.

Signs

- An irregular, deep anterior chamber with tremulousness of the iris.
- Diagnosis should be confirmed after full dilatation of the pupil. The edge of the displaced lens appears in the pupillary area as a dark crescent in oblique illumination ***(subluxation)***.

Causes

They are mainly hereditary in nature.

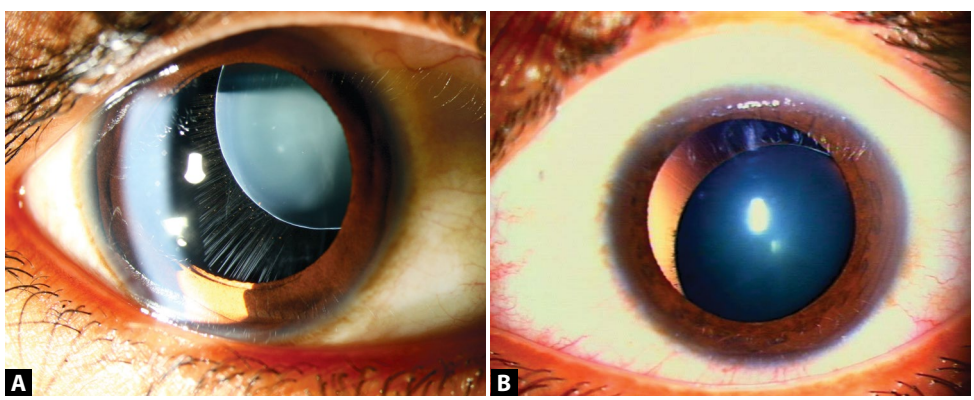
- Marfan's syndrome
- Homocystinuria
- Weill-Marchesani syndrome
- Ehlers-Danlos syndrome
- Hyperlysinemia
- Familial ectopia lentis
- Sulphite oxidase deficiency.

Marfan's Syndrome

Autosomal dominant, a multisystem mesodermal dysplasia.

Ocular features

- The lens is typically displaced upwards and inwards (Fig. 15.5A).
- Hypoplasia of the iris causes poor dilatation of the pupil.



Figs 15.5A and B: **A.** Marfan's syndrome—lens displacement upwards and inwards; **B.** Homocystinurea—lens displacement downwards and outwards

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- Axial myopia
- Secondary glaucoma
- Retinal detachment due to high incidence of peripheral retinal degeneration.

Systemic features

- Arachnodactyly (spider fingers)
- Long extremities (Fig. 15.6)
- Hyperextensibility of the joints
- High-arched palate
- Dissecting aortic aneurysm
- High incidence of hernias.



Fig. 15.6: Marfan's syndrome—long extremities

Homocystinuria

- Autosomal recessive; an inborn error of metabolism.
- Inability of convert methionine to cystine.
- Deficiency of enzyme cystathione synthatase.

Ocular features

- The lens is subluxated downwards and outwards (Fig. 15.5B).
- Secondary glaucoma, due to pupillary block.

Systemic features

- Fair complexion with malar flush
- Mental retardation
- Poor motor control
- Increased thromboembolic manifestation due to an excessive platelet adhesiveness.
- General anesthesia is hazardous
- Diagnosis is confirmed by urine test with sodium nitroprusside.

Weill-Marchesani Syndrome

- Autosomal recessive; a mesodermal dysplasia
- Short stature, stubby fingers
- Microspherophakia, which may be subluxated
- Secondary pupillary block glaucoma
- Retinal detachment.

Familial Ectopia Lentis

- Autosomal recessive
- Unassociated with any systemic defect
- May be associated with ectopic pupil—***ectopia lentis et pupillae***.

Treatment

- Spectacles or contact lenses are used to correct the optical defects, preferably through the phakic part.
- Lens extraction is difficult, complications are common and visual results are often poor.
- Pars plana lensectomy with vitrectomy is better than other surgical means.

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LENS-INDUCED OCULAR DISEASES

In addition to the glaucoma resulting from displacement of the lens, the other lens-induced ocular problems are:

- ***Phacolytic glaucoma (Fig. 15.7):*** In some eyes with hypermature cataract when the capsule leaks, large phagocytes filled with lens material obstruct the trabecular meshwork, causing a secondary open-angle glaucoma.
  Treatment: First, urgent medical control of glaucoma followed by lens extraction with intraocular lens (IOL) implantation at early as possible.

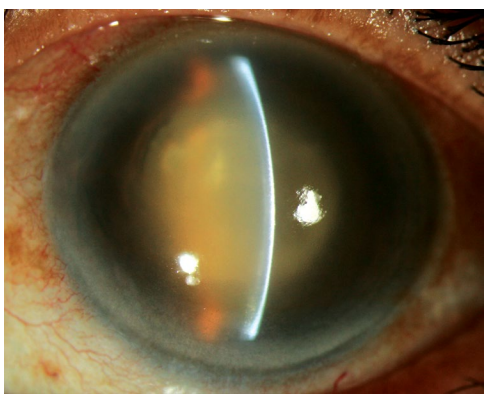
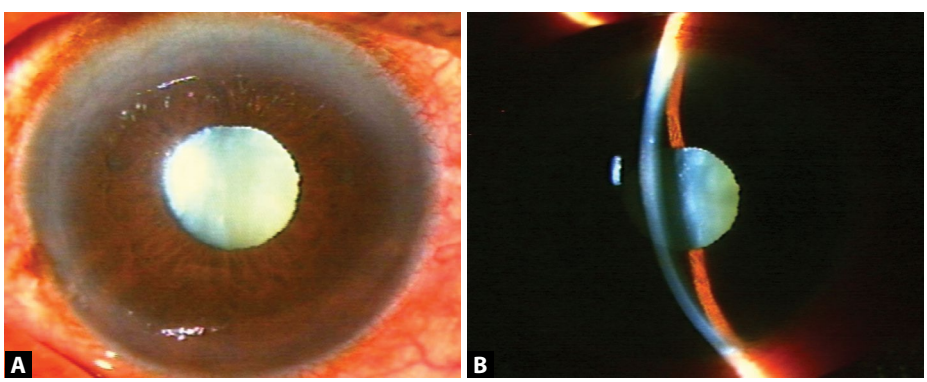


Fig. 15.7: Phacolytic glaucoma

- ***Phacogenic (phacomorphic) glaucoma (Fig. 15.8A):*** A rapid swelling of the lens (as in senile intumescent cataract, or following trauma to the lens capsule) may cause a secondary angle-closure glaucoma (Fig. 15.8B), if the anterior chamber is already shallow.
  Treatment is almost same as the phacolytic glaucoma.
- ***Phacotoxic uveitis:*** Accidental trauma to the lens capsule liberates lenticular protein within the eye. Although, lens proteins are relatively poor antigens, a granulomatous uveitis may develop and sometimes with secondary glaucoma.
  Treatment consists of administration of corticosteroids and lens extraction.
- ***Phacoanaphylactic uveitis:*** Rarely, after extracapsular cataract extraction, the immune system appear to be sensitized to lens protein.
  - Extracapsular extraction with retention of cortical material in the second eye, then may sometimes cause an endophthalmitis.
  - Dislocation of lens nucleus or nuclear fragments in the vitreous cavity during an extracapsular extraction or phacoemulsification, also causes severe uveitis.
  - The lens material must be removed from the eye by means of vitrectomy and lensectomy.



Figs 15.8A and B: **A.** Phacomorphic glaucoma; **B.** Secondary angle-closure

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CATARACT

Literally, cataract means ***waterfall***. Any opacity of the lens or its capsule causing visual impairment is called cataract.

Care must be taken in using the term 'cataract' clinically, since, it often excites anxiety which may be entirely unjustified. It is to be remembered that many congenital opacities are stationary, and even in senile cataract the opacities may remain localized for many years without causing serious visual impairment.

So, it is often wise to tell such patient that he has lental opacity and to suggest that the development of cataract may be long delayed and will be dealt with according to the need.

Classification of Cataract

No classification of cataract is entirely satisfactory. It may be classified as follows:

- Congenital or developmental cataract
- Acquired cataract
  - Senile
  - Traumatic
    - Mechanical
    - Irradiation
    - Electric shock
  - Complicated (due to some other ocular disease)
    - Anterior uveitis
    - High myopia
    - Retinal detachment
    - Retinitis
    - Glaukomflecken
  - Secondary (due to some systemic disease)
    - Diabetes mellitus
    - Hypocalcemia
    - Myotonic dystrophy
    - Atopic dermatitis
  - Toxic (due to drugs)
    - Corticosteroids
    - Chlorpromazine
    - Miotics (long-acting)
    - Busulfan
    - Amiodarone
    - Gold
  - Syndromes associate with cataract
    - Down's
    - Lowe's
    - Treacher Collins
    - Wilson's disease
    - Fabry's disease
- After cataract posterior capsule opacification.

CONGENITAL OR DEVELOPMENTAL CATARACT

It means cataract that presents at birth, or develops soon after the birth.

The lens is formed in layers, the central or fetal nucleus being the earliest formation, around which the concentric zones are subsequently laid down and the process is continuing until the late age of life.

Congenital or developmental cataract (Fig. 15.9) has therefore, a tendency to affect a particular zone, which was being formed when this process is disturbed. The lens fibers laid down previously and subsequently are often normally formed, and remain clear.

As times goes on, such an opacity is usually deeply buried into the substance of the lens by subsequent formation of normal fibers.

Developmental cataract has a tendency to follow the architectural pattern of the



Fig. 15.9: Congenital cataract—bilateral

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lens, and from its location, an estimate can be made about the stage of development at which the opacity occurred.

In strict sense, congenital and developmental cataract are not synonymous. In widest sense, most forms of cataract (even the senile cataract) may be considered as developmental, for the human lens grows until late in life, until the age of 80–90 years. The criteria of congenital cataract are:

- Any lens opacity present at birth.
- Such opacities must be situated within the fetal nucleus, or at the most within the innermost part of the infantile nucleus.
- Diameter of the opacity (partial cataract) must be less than 5.75 mm (i.e. the frontal diameter of newborn lens). If the size of the opacity is more than 5.75 mm, the cataract must be postnatal.

Causes

- Mostly unknown
- Hereditary
- Chromosomal abnormalities, e.g. Down's syndrome
- Maternal malnutrition (vitamin D deficiency and hypocalcemia as in zonular cataract)
- Maternal infections, as in rubella
- Fetal hypoxia owing to placental hemorrhages
- Drug intake during the pregnancy, e.g. corticosteroids.

Morphologically, several types of congenital cataract (lens opacities) are recognized with the slit-lamp biomicroscope.

Morphological Types (Fig. 15.10)

- Punctate (blue-dot) cataract
- Anterior polar cataract—pyramidal reduplicated
- Posterior polar cataract
- Nuclear cataract
- Coronary cataract
- Coralliform/floriform cataract
- Christmas tree cataract (in myotonic dystrophy)
- Zonular (lamellar) cataract
- Total cataract.

Punctate (Blue-dot) Cataract (Fig. 15.11)

Perhaps, this is the most common type of congenital cataract.

- It appears as multiple, tiny blue spots, scattered all over the lens, especially in the cortex by oblique illumination with the slit-lamp.
- The bluish color is due to the effects of dispersion of light in the same way that the sky appears blue.
- The visual acuity is not affected.

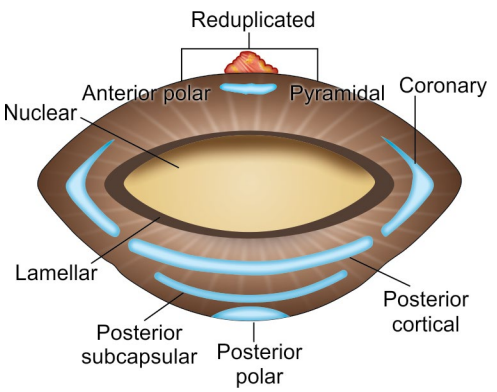


Fig. 15.10: Morphological types of cataract

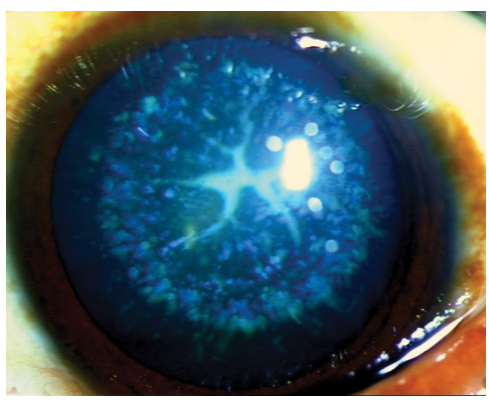


Fig. 15.11: Blue-dot and sutural cataract

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Anterior Polar Cataract (Fig. 15.12)

- It is sharply demarcated opacity at the anterior lens capsule, and usually the size of a pin's head.
- It may project forwards into the anterior chamber like a pyramid (***pyramidal cataract***); or subsequently, a subcapsular cataract may develop, just behind it with a clear zone in between (***reduplicated cataract***).
- This is due to delayed formation of the anterior chamber during development of the lens.
- These opacities are stationary and rarely interfere with vision.

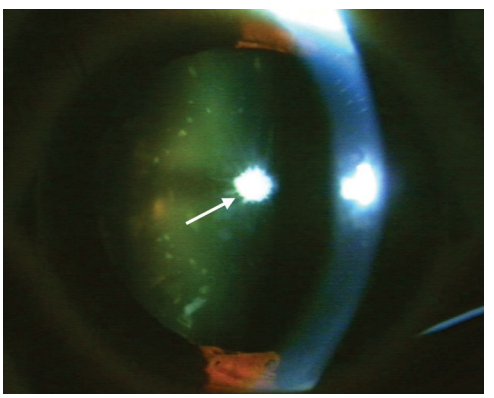


Fig. 15.12: Anterior polar cataract (arrow)

Posterior Polar Cataract (Figs 15.13A and B)

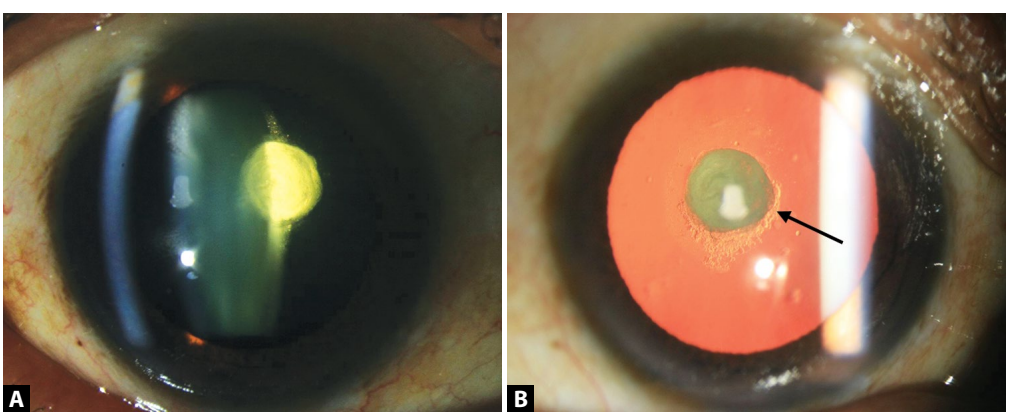
- This is due to residue of the attachment of hyaloid artery on the posterior lens capsule.
- This is usually dot-like (***Mitendorf's dot)*** and insignificant.
- However, in few cases, the lens may be invaded by fibrous tissue and a ***total cataract*** is formed.

Coronary Cataract (Fig. 15.14)

- This is the type of developmental cataract occurring ***at puberty.***
- It is situated in the deep layer of the cortex and the most superficial layer of the nucleus.
- It appears as a ***corona*** or club-shaped opacities, near the periphery of the lens, while the axial region and the extreme periphery remain clear.
- As they are hidden by the iris, dilatation of the pupil is essential to recognize these opacities.
- It does not interfere with vision.

Zonular (Lamellar) Cataract (Fig. 15.15)

- It is probably the most common type of developmental cataract presenting with visual impairment.



Figs 15.13A and B: Posterior polar cataract. **A.** Slit section; **B.** Retroillumination (arrow)

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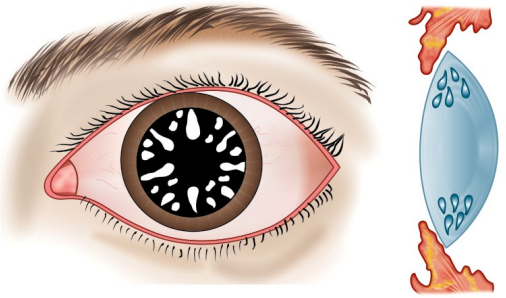
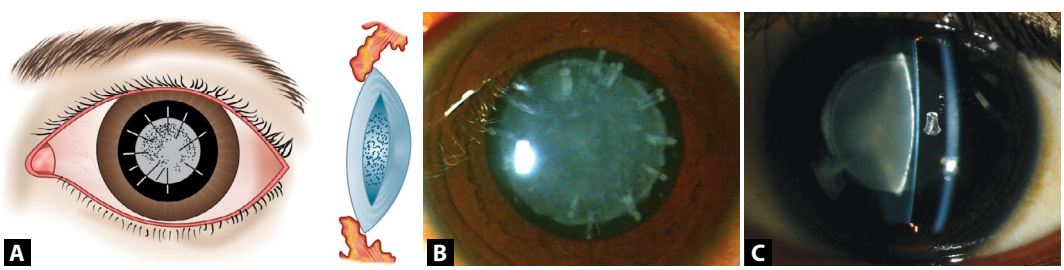


Fig. 15.14: Coronary cataract

- It may be congenital or occurs within the first year after birth.
- It is usually bilateral and often hereditary ***(autosomal dominant***) without any other ocular anomaly.
- It is associated with ***hypovitaminosis-D*** (may be with evidences of ricket), or ***hypocalcemia*** (may be with tetany or defective tooth enamel) and ***maternal malnutrition***.
- A unilateral cataract may follow blunt trauma of the eye.
- ***Characteristics***
  - It consists of concentric, sharply demarcated zones (lamellae) of opacities surrounding a core that is clear and enveloped by the clear cortex externally (Figs 15.15A to C).
  - There may be linear opacities, like spokes of a wheel (called ***riders***) that extends outwards towards the equator, and when present, it is ***pathognomonic***.
- Child with zonular cataract may present with photophobia due to light scattering.
- Surgery is considered when the visual acuity is less than 6/18.

Rubella Cataract (Fig. 15.16)

- Rubella infection during pregnancy may cause widespread ocular and systemic defects.
- Incidence of cataract is more, if the infection is contracted in the second month of pregnancy; and sometimes in the third month.
- The cataract is originally nuclear and progresses to become total (pearly-white in color) (Fig. 15.16).
- It is associated with microphthalmos, nystagmus, strabismus, glaucoma, iris hypoplasia and change to ***salt-and-pepper retinopathy.***
- The systemic associations are congenital heart disease—patent ductus arteriosus (PDA) microencephaly, mental retardation, deafness and dental anomalies.
- ***Pathologically,*** the nucleus is found to be necrotic and it may harbor the virus for upto 2 years following birth.
- Removal of such cataract frequently provokes an intense uveitis or endophthalmitis (probably by liberation of retained virus) with disappointing visual result.
- However, in view of total bilateral cataract, complete aspiration, should usually be attempted with a complete iridectomy (as



Figs 15.15A to C: Zonular cataract with riders. **A.** Schematic representation; **B.** Photograph—note the spoke-like riders; **C.** Zonular cataract in slit section

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Fig. 15.16: Congenital rubella cataract

the pupils dilate poorly) and high dose of corticosteroids.
- All the congenital anomalies associated with maternal rubella in the first trimester can be avoided by vaccination of the mother.
- The rubella-vaccine is itself toxic to the fetus, and it must therefore be administered at least 3 months before the pregnancy.

Management of Pediatric Cataract

Prognostic Factors

The prognosis of pediatric cataracts depends on few factors:
- Unilateral or bilateral
- Density of cataract during presentation
- Degree of visual impairment
- Associated ocular defects
- Associated systemic defects
- Probable cause of the cataract
- Commitment of the parents.

Investigations

- Ocular examination, especially
  - Assessment of the visual function by:
    - Fixation reflex
    - Optokinetic nystagmus (OKN)
    - Catford drum test
    - Visual evoke response (VER)
    - Forced preferential looking (FPL)
  - Look for associated nystagmus and strabismus
  - Density of the cataract
  - Fundus examination with dilated pupils.
- Thorough systemic examination including the study of the family.

Laboratory Investigations

- ***TORCH test:*** Patient and maternal serum are studied for antibodies against toxoplasma (TO), rubella (R), cytomegalovirus (C) and herpes virus (H).
- Test for galactosemia (reducing substances in urine).
- Test for Lowe's syndrome (urine chromatography for amino acids).
- Test for hypoglycemia (blood glucose).
- Tests for hypocalcemia (serum calcium, phosphate and X-ray skull).

Indications of Surgery

- About 50% of the opacities do not progress or interfere with vision and can safely be ignored.
- If the opacity is partial with useful vision, surgery should better not to be considered. The vision of 6/12 or 6/18 with accommodation is more valuable than a vision of 6/9 without accommodation, after operation.
- All dense cataracts (unilateral or bilateral) and partial cataracts with vision less than 6/18, are to be operated as early as possible.

Other modes of treatment
- ***Mydriatics:*** For axial cataract, and with convex glasses to maintain near vision.
- ***Optical iridectomy*** for axial cataract and if the opacity is stationary. It is obsolete now-a-days.

Timing of Surgery

- The critical period for development of fixation reflex is between 6 weeks and 6 months of age. So, the best time for surgery is before this period to prevent stimulus-deprivation amblyopia.

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- In dense monocular cataract, the surgery should be done as early as possible, may be on the next day after birth.

Surgical Techniques

- ***Simple discission or needling*** operation is becoming obsolete now-a-days, because of more complications (after cataract occurs in almost all cases → needs repeated surgeries → retinal detachment in future).
- ***Pars plana lensectomy with vitrectomy*** perhaps, is the most suitable surgery for congenital cataracts.
- ***Planned extracapsular cataract extraction (ECCE)*** as in adult ECCE; a posterior chamber IOL may be considered with ECCE if the child is older than 3 years of age.

The incidence of posterior capsule opacity (PCO) after ECCE with posterior chamber intraocular lens (PCIOL) is very high (may be upto 100%) in young children. A primary posterior curvilinear capsulorhexis (PCCC) with or without vitrectomy is helpful to prevent PCO in this group. It is based on the theory that removing the scaffold for the migration of lens epithelial cells will provide a permanently clear optical zone.

Visual Rehabilitation

- ***Conventional aphakic glasses:*** It is only useful for bilateral aphakia, but technical difficulties in wearing heavy spectacles (+15D or more) in young children should be kept in mind.
- ***Extended-wear soft contact lens:*** It is especially for uniocular aphakia. Extreme co-operation and commitment of the parents are very important.
- ***Posterior chamber IOL implantation:*** It should certainly, not to be considered before the age of 3 years because at that age the growth of the eye has leveled off.
- ***Amblyopia therapy:*** It should be started as soon as possible in the form of occlusion or ***atropine penalization*** in the other eye. This is a must for unilateral cataract.



Fig. 15.17: Bilateral senile mature cataract

SENILE CATARACT

The aging lens tends to become opaque after the age of 50 years and by the age of 70 over 90% of the population show some evidence of cataract.

It occurs equally in men and women, and is usually bilateral, but often develops earlier in one eye than the other (Fig. 15.17).

There is a considerable genetic influence in its occurrence, and in hereditary cases, it may appear at an earlier age (even before the age of 50) in successive generations (***presenile cataract***).

Senile cataract may be broadly divided into two types (though they may occur concurrently).

1. ***Cortical or soft cataract (75–80%):*** The classical features of hydration followed by coagulation of lens proteins, appear primarily in the cortex.
2. ***Nuclear or hard cataract (20–25%):*** The essential feature is a slow progressive sclerosis in the lens nucleus.

Cortical (Soft) Cataract (Fig. 15.18)

- ***Cuneiform:*** It starts as wedge-shaped spokes of opacity at the periphery and gradually encroaches towards center.
- ***Cupuliform cataract:*** It is also known as posterior cortical cataract and starts as a saucer-shaped opacity in the posterior cortex just beneath the capsule.

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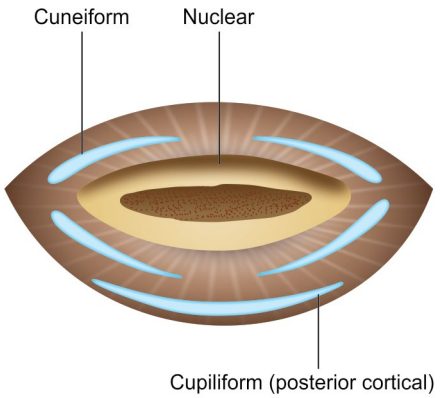


Fig. 15.18: Types of senile cataract

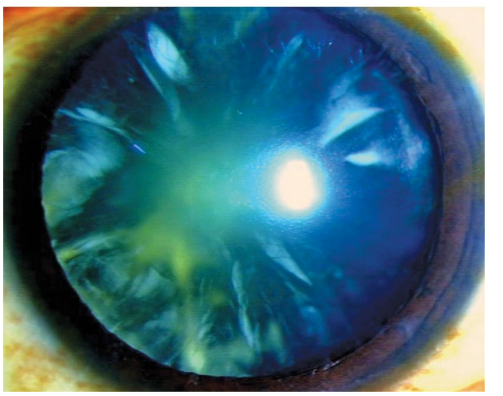


Fig. 15.19: Cuneiform cortical cataract

Cuneiform Cataract (Fig. 15.19)

Symptoms

- Painless, progressive loss of vision
- ***Uniocular diplopia or polyopia:*** It is due to irregular refractions by different sectors of the lens producing multiple images.
- ***Colored halos:*** It is due to accumulation of fluid droplets in between the lens fibers.
- ***Black spots in front of the eyes:*** The spots are fixed.
- ***Glare***, particularly at night, during facing head-lights.
- Progressive decrease in peripheral visual field.
- ***White opacity inside the black of the eye*** (as noticed by the friends or relatives).

Stages and Signs

- ***Stage of lamellar separation:*** A demarcation of the cortical fibers is due to their separation by fluid, and can only be seen with the slit-lamp.
- ***Stage of incipient cataract***
  - Cuneiform opacities with clear area in between them, appear in the periphery of the lens.
  - The bases of the wedge-shaped opacities are peripheral, and they are most common in ***lower nasal quadrant***.
  - They appear as gray opacities with oblique illumination, but appear black against the red fundal glow with the ophthalmoscope or retinoscopic mirror.
  - They gradually encroach to the axial area and vision becomes impaired.
- ***Immature stage (Fig. 15.20A):*** As time goes on, opacification becomes more diffuse, and the deeper layers of the cortex become cloudy.
  - ***Vision:*** It may be reduced to finger counting.
  - ***Lens:*** Grayish or grayish-white.
  - ***Iris shadow:*** Present.

Painless Progressive Loss of Vision

The common causes of painless progressive loss of vision are:

- Senile cataract
- Open angle glaucoma
- Presbyopia
- Degenerative myopia
- Retinitis pigmentosa
- Diabetic retinopathy
- Age-related macular degeneration (ARMD)
- Papilledema [due to intracranial space-occupying lesions (SOL)].

Differential Diagnosis

- ***Senile cataract:*** Aged patient; grayish-white or white pupillary reflex; iris shadow—present in immature type; normal IOP; and poor or absent fundal glow.

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- ***Open angle glaucoma:*** Aged patient; high IOP; typical glaucomatous field defects and cupping of the optic disc.
- ***Presbyopia:*** Aging process—gradual dimness of vision in near only; no lental opacity; normal IOP; no cupping of the disc and near vision is regained by simple 'plus-power' glasses.
- ***Degenerative myopia:*** Younger subject; increasing high 'minus-power' glasses; normal IOP; typical myopic degenerative changes in the fundus.
- ***Retinitis pigmentosa:*** Middle-aged patient; positive family history; night blindness; tubular field defect; normal IOP; typical fundus changes—bone-corpuscle pigments, arteriolar attenuation and waxy pallor of the disc.
- ***Diabetic retinopathy:*** Young adult or aged; history of long standing diabetes, often uncontrolled; with or without cataract; typical fundus picture—dot and blot hemorrhage, hard exudates, macular edema, neovascularization, vitreous hemorrhage, tractional retinal detachment (RD); fundus fluorescein angiography (FFA) is diagnostic.
- ***Age-related macular degeneration:*** Aged patients; progressive central loss of vision; anterior segment and IOP are essentially normal; retinal pigment epithelium (RPE) atrophy; detachment of RPE with subretinal neovascular membranes (SRNVMs) formation; FFA is diagnostic.
- ***Papilloedema:*** Any age; bilateral onset; associated headache and vomiting; normal reacting pupil; normal IOP, bilateral optic disc swelling and may be with superficial hemorrhage, soft exudates and macular star; CT scan or MRI may detect intracranial SOL.

Iris shadow is a concave shadow of the pupillary margin of the iris, cast upon the grayish lens, when light is thrown upon the eye from the same side.

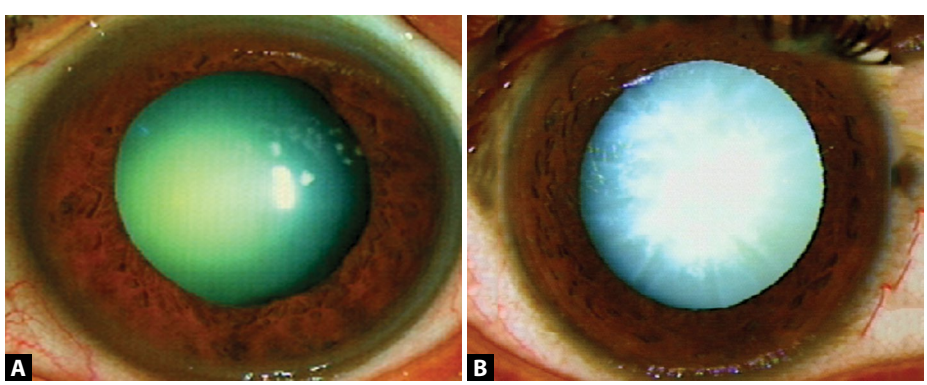
It implies that some clear cortical fibers are still present beneath the lens capsule, i.e. between the pupillary margin of the iris and the actual opacity. Therefore, it is present in immature cataract.

- ***Fundal glow:*** It is faint, as the pupillary area is becoming more occupied by the dark shadow.
- ***Purkinje's image:*** It is the 4th image may be present (but distorted), or absent in advance cases.

Intumescent Cataract

It is seen in some cases of immature cataract, in which the lens becomes swollen due to progressive hydration of the cortical layers. The anterior chamber is shallow, and there may be chance of secondary angle-closure glaucoma.

- ***Mature stage (Fig. 15.20B):*** The lens is completely opaque.
  - ***Vision***: The hand movement to perception of light (PL).
  - ***Lens:*** White or pearly-white in color.
  - ***Iris shadow:*** Absent—as the cortex is completely opaque, the pupillary margin lies almost in contact with the opacity, separated only by the capsule, the iris then casts no shadow.



Figs 15.20A and B: A. Immature cataract—iris shadow present; **B.** Mature cataract—iris shadow absent

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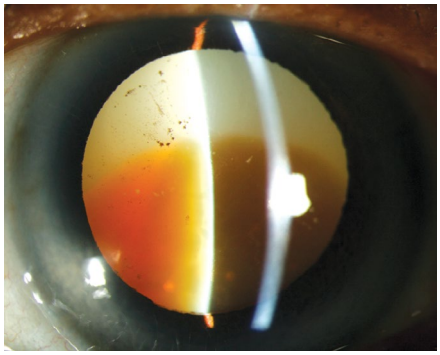


Fig. 15.21: Hypermature cataract—morgagnian

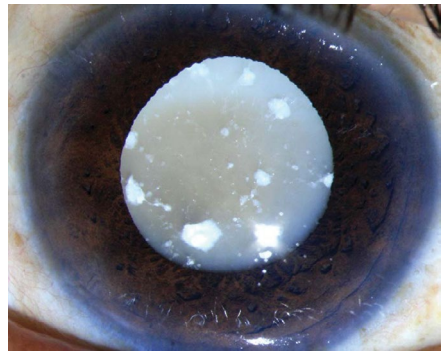


Fig. 15.22: Hypermature sclerotic cataract

- ***Fundal glow:*** Absent
- ***Purkinje's images:*** Fourth image is absent.

- ***Hypermature stage:*** If the process is allowed to go on without any intervention, the cortex becomes disintegrated and then liquefied, or transformed into a pultaceous mass. It is of two types:
  1. ***Morgagnian cataract (Fig. 15.21):*** Here, the cortex liquefies and the nucleus may sink at the bottom within the lens capsule.
     - The cortex appears milky-white and the nucleus appears as a brown mass, limited above by a semicircular line, which may change its position.
     - Anterior chamber may be shallow and may give rise to secondary angle-closure glaucoma.
  2. ***Sclerotic cataract (Fig. 15.22):*** The lens becomes more and more inspissated, and shrunken in appearance, due to loss of fluid from the lens.
     - The lens is more flat and yellowish-white in appearance.
     - The anterior capsule becomes thickened due to proliferation of the anterior cuboidal cells, and there may be calcium or cholesterol crystals on it.
  - Owing to shrunken lens, the iris becomes tremulous with deep anterior chamber, and finally the lens may be subluxated due to degeneration of the zonules.

A long-standing hypermature cataract may give rise to the following complications:

- Subluxation or dislocation of the lens
- Lens-induced glaucoma
- Lens-induced uveitis
- Ultimately, there will be absolute glaucoma with 'no PL'.

Cupuliform Cataract (Posterior Cortical Cataract) (Fig. 15.23A)

The opacity starts in the axial region of the posterior cortex, and slowly progresses to involve the entire posterior cortex. Other types of cataract may supervene on it.

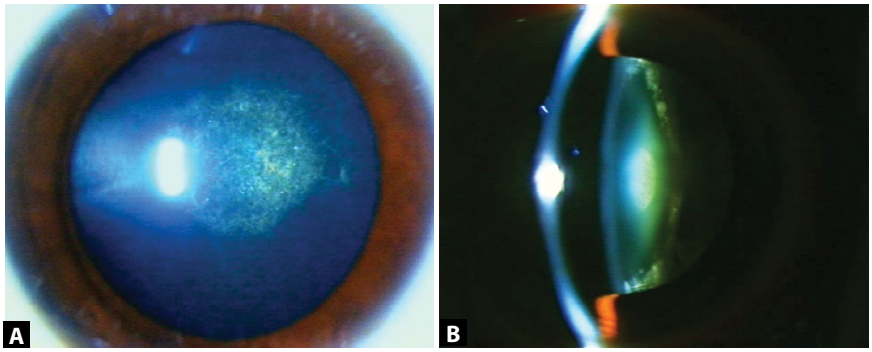
Symptoms

- Marked diminution of vision, as the opacity is near the nodal point of the eye.
- Vision is poor in bright light, especially during the day time (as the pupil becomes constricted in bright light and the opacity is axial). Patients with posterior cortical cataract always see better in darkness (dawn or dusk).

Signs

- It is difficult to see with a torch light in undilated pupil.
- The exact position of the opacity is best judged by a slit-lamp and with ***dilated pupil***. It appears as yellowish-white layer in the posterior cortex.

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Figs 15.23A and B: A. Cupuliform cortical cataract; **B.** Cupuliform and nuclear cataract

- In distant direct ophthalmoscopy (with a retinoscope in the dark room)—it appears as a central dark spot against red fundal glow.
- Forth Purkinje's image is blurred.

Nuclear (Hard) Cataract (Fig. 15.23B)

Nuclear cataract tends to occur earlier than the cortical variety, often soon after 40 years of age. At first, the nucleus appears more refractile and as time progresses, it becomes diffusely cloudy and often yellowish in color.

The cloudiness gradually spreads towards the cortex and the lens becomes tinted dark-brown called brown cataract ***(cataracta brunescens)***. Ultimately, it becomes black, and called black cataract ***(cataracta nigra)*** (Figs 15.24A to D). The discoloration of the lens in nuclear cataract, is due to deposition of melanin and other pigments derived from photo oxidation of aromatic amino acids present in the lens.

At maturity, the sclerotic process may extend almost to the capsule, so that, the entire lens functions as a nucleus, but hypermaturity does not occur.

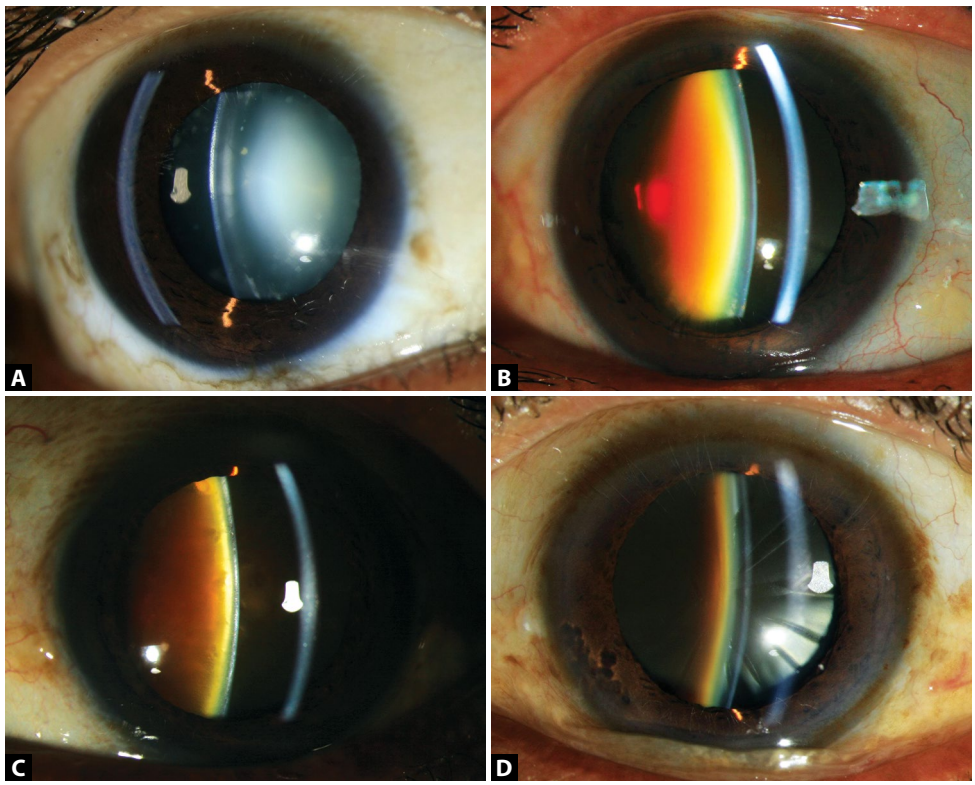
Symptoms

- ***Gradual impairment of vision for distance:*** It is initially due to a progressive

Difference between immature and mature cataract

Features	Immature cataract	Mature cataract
Symptoms	• Partial loss of vision • Diplopia/polyopia • Rainbow halos	• Total loss of vision • White opacity noticed by the patients or relatives
Signs		
• Visual acuity	• Vision reduced to varying degree	• Vision reduced to hand movement or perception of light (PL)
• Color of the lens	• Gray/grayish-white	• White/pearly-white
• Iris shadow	• Present	• Absent
• Purkinje's images	• Third image is seen, fourth image may be distorted	• Only third image is seen, fouth image is absent
• Fundal glow	• Present	• Absent
• Retinoscopy	• Possible	• Not possible
• Spectacles correction	• May improve vision	• Does not improve vision

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Figs 15.24A to D: Nuclear cataract. **A.** Grade NS II; **B.** Grade NS III: Amber color; **C.** Grade NS IV: Brown cataract; **D.** Grade NS IV+: Black cataract

myopia owing to increased refractive index of the nucleus ***(index myopia)***. This myopia neutralizes the plus power of presbyopia. Hence, a patient who was using presbyopic glasses for reading, is able to read better without any glasses when nuclear sclerosis sets in. This is felt as an improvement of vision by the patient, and is termed as ***second sight***.

- ***Change in color values:*** Particularly, with the blue end of the spectrum (as the lens is yellow-brown in color).
- Ultimately, very slowly there will be considerable loss of vision, due to increasing cloudiness of the nucleus.

Signs

- ***Vision:*** It may be reduced to hand movements in dense opacity.
- ***Color of the lens:*** It is yellow, brown or black (better appreciated in dilated pupil).
- ***Iris shadow:*** Usually present.
- ***Slit-lamp grading*** of the hardness of the nucleus can be judged by the pigmentation of the nucleus. This is essential for phacoemulsification.

Grading of hardness	Appearance of nucleus
Grade 1+ (soft) ↓	Gray or greenish-yellow
Grade 2+ ↓	Yellow nucleus
Grade 3+ ↓	Amber
Grade 4+ (rock hard)	Brown to black

- ***Ophthalmoscopy:*** Initially, little change of the fundal glow may be seen with ophthalmoscope, except that the fundus details are hazy. Later, the fundal glow may be entirely blackened.

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Fig. 15.25: Cataract classification system—lens opacities classification system (LOCS) III

- ***Retinoscopy:*** May reveal false lenticonus where the central part of the pupil is myopic and the peripheral part is hypermetropic.

Lens Opacities Classification System III (Fig. 15.25)
The most popular grading system is lens opacities classification system III (LOCS III). LOCS III uses a reference set of standard photographs that defines the extent of opacities in cortical and subcapsular zones and the color/grade of the nuclear opacity.

By using LOCS III, the cataract can be classified as: Nuclear color/opalescence = NC/NO: 1-6, cortical opacities = C: 1–5 and posterior subcapsular opacities = P: 1–5.

MANAGEMENT OF CATARACT

Medical Treatment

No treatment can restore the cataractous lens to its original transparent state. Similarly, there is no known method of preventing its progression. The only way is to disperse the lens opacities is to remove the lens.

The medical treatment of cataract is, thus purely symptomatic.

- Frequent and accurate refraction to maintain vision at the best possible level.
- Using optimum illumination and magnifying glasses.
- Weak mydriatics, e.g. phenylephrine (2.5%) for axial opacities to provide visual improvement.

Surgical Treatment

Indications of Surgery

- ***For optical reason:*** This is by far the most common indication and varies from patient-to-patient. The opacity has progressed to such an extent that the patient can no longer be able to carry out his day-to-day activity efficiently, with best visual correction.
- ***For medical reasons:*** When the presence of a cataract is adversely affecting the health of the eye, as in:
  - Phacolytic glaucoma.
  - Secondary angle-closure glaucoma due to intumescent cataract.
  - ***Vitreoretinal diseases:*** Examples are vitreous hemorrhage, diabetic retinopathy or retinal detachment, treatment

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Difference between cortical and nuclear cataract

Cortical cataract	Nuclear cataract
• Usually starts at late 50s • Starts with uniocular diplopia/polyopia or colored halos	• Tends to occur earlier than cortical • No such symptoms
• Give rise to index hypermetropia able spectacles	• Gives rise to index myopia. This myopia neurtalizes the plus power of presbyopia and the patient is to see small prints without
• Appears grayish, white to milky—white with the maturity of the cataract	• Appears yellow, brown or black with progression of cataract
• Progress is gradual and ultimately progress into hypermature stage with liquefaction of the cortex	• Progress is very slow and ultimately the entire lens functions as a nucleus, but hypermaturity does not occur
• More chance of lens-induced glaucoma	• Little chance of lens-induced glaucoma

of which is being hampered by the presence of lens opacities.

- ***For cosmetic reasons:*** Rarely, this is an indication in order to obtain a black pupil.

Investigations Prior to Surgery

The main aims of the investigations are:

- To detect underlying local or systemic pathology, which can affect the management adversely.
- To prevent intraoperative or postoperative complications.
- To calculate the power of IOL.
- To predict the visual outcome after surgery.

History

- Any trauma or inflammation of the eye.
- Any posterior segment disease, like macular lesions, venous thrombosis, vitreous hemorrhage, etc. in the past.
- Nature of glasses using in the past.
- Diabetes, hypertension, cardiac problems, bronchial asthma, dental and aural problems, urinary problems, etc.
- Drug allergy.

Local Investigations

Ocular Examination

- Vision and refraction of the eye to be operated.
- ***Perception of light (PL):*** If the eye has 'no PL'—a favorable prognosis for visual improvement is nil.
- ***Projection of rays (PR):*** It indicates the gross function of the peripheral retina. It tells nothing about the condition of the macula.
- ***Importance***
  - An 'accurate PR' excludes the possibility of a large retinal detachment, or an absolute field defect.
  - 'Inaccurate PR' may be seen in retinal detachment, big patch of chorioretinal atrophy, or in advanced stage of glaucoma.
  - 'Inaccurate PR' does not necessarily indicate an inoperable situation, as the opaque media can diffuse light so much that all directional quality may be lost.
- ***Pupil:*** To examine the light reactions and to estimate the ability of the pupil to dilate before surgery.
- ***Slit-lamp examination***
  - To evaluate the type and extent of cataract.
  - To evaluate the health of the cornea, e.g. presence of Fuchs' endothelial dystrophy precludes IOL implantation.
  - To find out the evidences of old iridocyclitis, e.g. old keratic precipitates, posterior synechiae or pigments over anterior lens surface.
  - To find out any pathology of anterior segment of the other eye.

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- ***Intraocular pressure:*** It is preferably by applanation tonometer.
- ***Patency of the lacrimal passage:*** It is to find out any pathology of the lacrimal drainage system and treat accordingly before cataract operation.
- ***Fundoscopy***
  - ***Eye to be operated*** in case of immature cataracts.
  - ***Fellow eye*** for bilateral lesions like diabetic retinopathy, age-related macular degeneration, etc.

Conjunctival Swab for Culture and Sensitivity Tests

- Operation should not be performed in presence of any pathogenic organism. The value of conjunctival swab culture is doubtful, since the conjunctival sac can harbor organisms during the period, between the reporting time and day of operation.
- Most of the surgeons do not advise conjunctival swab culture, and as a routine, advise to use antibiotic drops and ointment into the conjunctival sac for a few days prior to operation.

Calculation of Intraocular Lens Power

There are three ways to decide the power of IOL to be implanted:

1. ***Using standard power (+19D):*** This is generally no longer acceptable.
2. ***Based on basic refraction:*** It means the refractive error prior to the onset of cataract. The formula for posterior chamber IOL is:

   P = 19D + (R × 1.25)
   (P = implant power and R = basic refractive error)

   Accordingly, a patient with –2.00D myopia will need an implant power of:

   P = +19D + (–2.00 × 1.25) = +16.50 D

   It is useful where ultrasound biometry facility is not available.
3. ***Using SRK regression formula:*** It requires:
   - ***Keratometry*** is to determine the corneal power.
   - ***USG-A scan*** is to determine the axial length of the eyeball.

   'SRK' stands for ***'Sanders, Retzlaff*** and ***Kraff'*** and the ***formula is***:

   P = A – 2.5 × L – 0.9 × K

   Where, **P** = implant power, to achieve emmetropia, **L** = axial length in mm and **K** = corneal power in diopter. **A** = specific constant, which varies with the implant type, design and the manufacturer, e.g. typical A constant for PCIOL = 116.5 or 118.0 or 118.7 and for ACIOL = 115.0, etc.

Formulas for intraocular lens power calculation:

- ***First generation:*** SRK formula (regression)
- ***Second generation:*** SRK II formula (regression)
- ***Third generation:*** SRK/T; Hoffer Q; Holladay 1 formulas (theoretical)
- ***Fourth generation:*** Holladay 2; Haigis formula (regression and theoretical).

Most common regression formulas are the SRK and SRK II. They are popular because of simplicity. SRK formula is best to use when the axial length (AL) is between 22 and 24.5 mm.

Subsequently, SRK II formula is attempted to optimize axial length for short and long eyes. In this formula, a correction factor is added—to increase the lens power in short eyes and decrease it in long eyes.

P = A1 – 0.9K –2.5L. Where A1 = (A – 0.5 greater than 24.5; A1 = A for axial length (AL) between 22 and 24.5; A1 = (A+1) for AL between 21 and 22; A1 = (A + 2) for AL between 20 and 21; and A1 =(A + 3) for AL less than 20.

Even more customized formulas are required today to calculate anterior chamber depth (ACD) based on AL and corneal curvature. ***SRK/T (T for theoretical)*** is one such formula, representing a combination of linear regression method with a theoretical eye model. The SRK/T

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and other third generation formulas work best for near-schematic eye measurements; specifically, the SRK/T is best for eyes longer than 26.00 mm. SRK/T calculation, however, does not account for effective lens position (ELP).

Modern theoretical formulas are based on schematic optics and the eye is considered as dual lens system (i.e. IOL and cornea). The predicted distance between them is called ELP, which is used to calculate IOL power. ELP correlates well with IOL placement inside the eye, whether it is in anterior chamber, in-the-sulcus or in-the-bag. It also varies with IOL configuration and the location of its optical center.

In the ***original theoretical formula*** the ELP was considered a constant value of 4 mm for every lens in every patient. Better results are obtained by relating the expected ELP to the axial length, anterior chamber depth, corneal curvature and IOL itself.

The ***modern IOL power formulas are***—SRK-T, Holladay 1, Holladay 2, Hoffer-Q and Haigis.

When the AL is between 22.5 and 26.0 mm, and K value is between 41D and 46D—any modern formula can be used. Outside this range—Holladay 2 and Haigis formula is better.

These formulas are programed into the IOL Master, Lenstar and most modern ultrasonic biometry machine, thus eliminating any need for regression formulas.

There are special cases, like post refractive [radial keratotomy (RK), laser-assisted *in situ* keratomileusis (LASIK) or photorefractive keratectomy (PRK)] cases, post vitreoretinal surgery cases, especially the silicone-filled eyes—where IOL power calculation for each eye is differently calculated.

AL (axial length): Distance between the center of the anterior corneal surface and the fovea. It is usually measured by ultrasonography (USG) A-scan or optical coherence biometry. The axial length (AL) is the most important factor in IOL power calculation. 1 mm error in AL measurement results

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in a refractive error of approximately 3.0 diopter error of IOL power.

Two types of A-scan ultrasound biometry are currently in use—contact applanation biometry and immersion A-scan biometry. Immersion A-scan gives accurate measurement. A mean shortening of 0.25–0.33 mm may be possible with applanation AL measurements, which translates into an error of approximately 1D IOL power.

Macular Function Tests

These tests are important to predict the visual potential of the eyes with opaque media such as cataract, total leukoma or vitreous hemorrhage.

- ***Two-point discrimination test:*** This is performed in a dark room. The patient is asked to look through an opaque disc perforated with two pin-holes close together, behind which a light is held. If he can appreciate the presence of two lights—macular function is probably good.
- ***Maddox rod test:*** The patient is asked to look at a distant light through a Maddox rod. If the red-line of light is continuous and unbroken, macular function is probably good.
- ***Color vision test:*** If the patient can identify red, green or blue light in presence of an opaque medium—his macular function is good.
- ***Pupillary light reflex:*** This should be brisk even in presence of a mature cataract. This indicates good optic nerve function.
- ***Purkinje's entoptic view of retina:*** Patient can see his whole retinal vessels, after pressing the lower lid with an illuminated bulb. An intelligent patient may be able to detect a patch of choroiditis or macular degeneration.
- ***Blue-field entoptoscopy:*** It allows the observation of one's own leukocytes (flying corpuscles) flowing in macular retinal capillaries.
- ***Illuminated Amsler's grid test:*** This is useful in assessing macular function in eyes with visual acuity of 6/60 or less.

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- ***Modified photostress test:*** This is with the help of an indirect ophthalmoscope with at least 6 volts of illumination. A prolonged recovery time indicates a maculopathy.
- ***Laser interferometry and potential acuity meter (PAM):*** Both the methods are used to estimate the potential visual acuity in terms of Snellen's equivalent. They are helpful in immature cataracts.
- ***Foveal electroretinogram (ERG).***
- ***Visual evoked response (VER):*** It comments on integrity of the macula and optic nerve.
- ***USG B-scan***: It gives the valuable anatomical informations concerning the vitreous and entire retina.

Specular Microscopy

It is necessary to count, and to study the morphology of the corneal endothelial cells. It is especially important in cataract with suspected endothelial dystrophy. Normal cell count in elderly subject is 2000–2500 cells/sq mm. Special precautions are to be taken before an IOL implantation, if the cell count is less than 1500 cells/sq mm.

Systemic Investigations

- ***Blood pressure:*** Any systemic hypertension should be controlled prior to surgery to avoid complications like retrobulbar hemorrhage, hyphema, expulsive hemorrhage, etc. Pre-existing hypertensive retinopathy may be responsible for poor visual prognosis after surgery.
- ***Blood sugar postprandial:*** Presence of diabetes mellitus increases the risk of infection, delayed wound healing and intraocular hemorrhage. Pre-existing diabetic retinopathy is also responsible for poor vision after surgery.
- Dental and ENT check up for septic foci.
- Urine for routine and microscopic examination particularly for albumin, sugar and pus cells.
- X-ray chest, electrocardiogram (ECG) and cardiological consultation for selective patients with heart disease.
- Pre-operative anesthetic check up for complicated patients and for children, if general anesthesia (GA) is needed.

Surgical Techniques in Senile Cataract

An increasing variety of cataract extraction methods are being employed depending on the:

- Needs of the patient,
- Availability of the changing technology, and
- Training of the surgeon.

Extracapsular cataract extraction (ECCE)

- Conventional ECCE
- Manual small incision cataract surgery (MSICS)
- Phacoemulsification
- Femto laser cataract surgery.

Intraocular lens implantation with any of the above techniques.

Intracapsular cataract extraction (ICCE): It is very rarely done (in grossly subluxated or dislocated lens), resulting aphakia (Figs 15.26A and B).

Lensectomy with vitrectomy: It is in children or in grossly subluxated/dislocated lens.

Intraocular lens implantation may or may not be possible in ICCE or lensectomy cases.

Glaucoma surgery: It is combined with any types of ECCE with IOL implantation.

Vitreoretinal surgery: It is combined with any types of ECCE with IOL implantation.

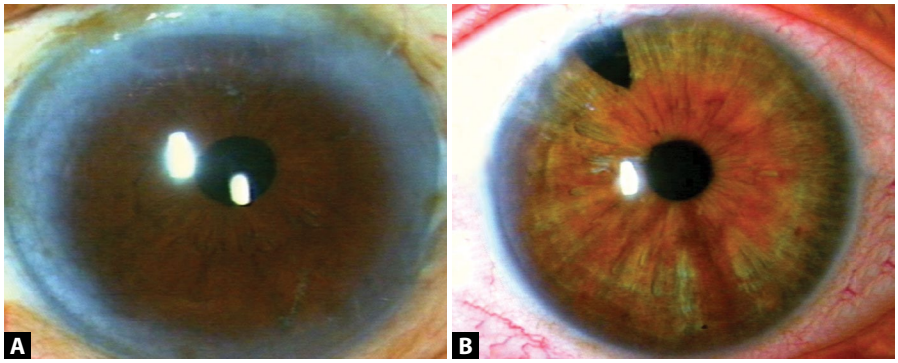
Keratoplasty: It is combined with lens extraction with or without IOL implantation. Keratoplasty with ECCE with IOL implantation is conventionally called ***triple procedure***.

Each of these techniques have an endless number of variations.

Extracapsular Cataract Extraction

It involves excision of central part of the anterior capsule (anterior capsulotomy), followed by expression of the nucleus and cortical cleaning. The posterior capsule, equatorial region and peripheral part of the anterior capsule are being left intact.

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Figs 15.26A and B: A. Pseudophakia; **B.** Aphakia

Merits

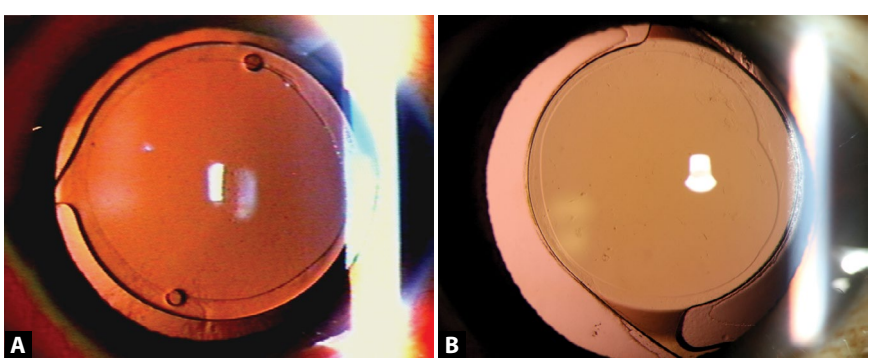
- Chances of vitreous loss is very minimal.
- The occurrence of vitreous-related anterior segment complication is negligible.
- Less chance of cystoid macular edema (CME) due to intact posterior capsule.
- Less chance of retinal detachment.
- A posterior chamber IOL in-the-bag is usually implanted along with ECCE, which is an ideal IOL (Figs 15.27A and B).
- An intact posterior capsule guards against infection (like endophthalmitis) for a prolonged period.

Demerits

- A difficult microsurgical technique, costly and takes time to master.
- Iridocyclitis and glaucoma due to lens particles are common.
- Opacification of the posterior capsule (after cataract) occurs in a significant number of cases, requiring (yttrium aluminium garnet) YAG-laser capsulotomy or needling.
- Extracapsular cataract extraction can not be done in dislocation and is difficult in subluxation of the lens.

Manual Small Incision Cataract Surgery

Manual small incision cataract surgery (SICS) (or more commonly called SICS) is a low-cost, small-incision form of ECCE that is popular in the developing countries. Compared



Figs 15.27A and B: Pseudophakia: **A.** Rigid PMMA PCIOL in-the-bag; **B.** Single piece foldable PCIOL in-the-bag

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to conventional ECCE, manual SICS has the advantages of stitchless self-sealing wound with much faster rehabilitation.

In presence poor-resource settings, manual SICS also has several other advantages over phacoemulsification; like—shorter operative time, no need for high technology and also lower cost. Recent studies have shown that, outcomes and complication rates between patients undergoing phaco and manual SICS with PCIOL implantation are comparable.

There are several techniques and modifications of performing manual SICS with comparable visual and surgical outcomes.

Phacoemulsification

It is basically an ECCE with the help of a highly sophisticated instrument called ***phacoemulsifier***. Here the lens nucleus and cortical matter are emulsified by ultrasonic vibration (using a hollow 1 mm titanium needle vibrating 40,000 times/sec), and then removed by simultaneous controlled irrigation and aspiration. Finally, the whole posterior capsule and part of the anterior capsule are left intact as a ***capsular bag***.

It is a safe method of performing ECCE with in-the-bag PCIOL implantation in almost all types of cataracts.

Merits

- Small incision cataract surgery—most of the times suture is not required called ***sutureless cataract surgery***
- More rapid wound healing
- Shorter convalescence
- Early stabilization of refraction with minimum or no astigmatism.

Demerits

- The equipment is expensive.
- Most difficult technique.
- High incidence of complications by the beginners, like iris damage, corneal decompensation, posterior capsular rent or nucleus drop into the vitreous.
- It is difficult to perform in white mature cataract and grade 4+ nuclear cataract.

Operative details (*see* Chapter 25).

Rehabilitation

A successfully performed cataract extraction is only the first step in the visual rehabilitation of the patient. The optical correction following cataract extraction is dealt elsewhere (*see* Aphakia, Chapter 7).

Intracapsular Cataract Extraction

In this technique the whole crystalline lens including the capsule is removed by various means, leaving a clear pupillary area.

Merits

- Relatively simple, quick, cheap and suitable for camp surgery.
- No chance of after cataract, since there is no posterior capsule and no need for treatment of after cataract.
- No possibility of developing uveitis and secondary glaucoma due to lens particle.

Demerits

- It can not be performed safely on patients under 35 years of age.
- Posterior chamber IOL implantation is impossible.
- The incidence of vitreous-related anterior chamber problem is higher, e.g. pupillary block, delayed wound healing, endothelial decompensation, glaucoma, or lately, infection by vitreous-wick syndrome.
- The incidence of post-operative CME is higher.
- The incidence of post-operative (aphakic) retinal detachment is higher.
- Corneal astigmatism is more as the limbal section is larger.

SPECIFIC CATARACT ENTITIES

Complicated Cataract

- Complicated cataract results from a disturbance of the lens-metabolism due to inflammatory or degenerative diseases of

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the eye, such as iridocyclitis, choroiditis, high myopia, retinitis pigmentosa, retinal detachment, etc.

- The opacity usually commences in the posterior cortex in the axial region ***(posterior cortical cataract)***. The opacity appears grayish-white with irregular border extending towards the equator and the nucleus in oblique illumination (***bread-crumbs appearance)***.
- With slit-lamp examination, the opacity shows a characteristic rainbow display of colors, the ***polychromatic lustre*** (Fig. 15.28).
- The opacity may remain stationary for many years, or it may gradually spread peripherally and towards the nucleus until the entire lens is involved.
- Vision is much impaired even in early stage, owing to the position of the opacity near the nodal point of the eye.

Treatment

- ***For inflammatory causes:*** ECCE with or without PCIOL under local and systemic steroids.
- ***For degenerative causes:*** Extracapsular extraction with PCIOL implantation is considered.

Operative and visual prognosis in complicated cataract are usually poor.

Diabetic Cataract

Diabetes mellitus is associated with two types of cataract.

1. ***Early onset senile cataract***
2. ***True diabetic cataract:*** This occurs in poorly controlled juvenile diabetic patients with gross disturbances in water balance.

   The opacities are bilateral (Fig. 15.29) and cortical, predominantly involve the anterior and posterior subcapsular region.
   - They consist of minute white dots of varying size, like snowflakes (Fig. 15.30) and are usually called ***snow-storm cataract***. A true diabetic cataract may progress to complete maturity in less than 72 hours.
   - ***Mechanism:*** Excess glucose in the lens → metabolizes to sorbitol (glucose alcohol) → increase osmolality of the lens → imbibition of water into the lens → formation of vacuoles in the cortex → opacification.
   - During early stage (during water vacuolation) the cataract is reversible, but once the lens proteins undergo coagulation, the opacities become irreversible.
   - ***Surgery*** is usually uncomplicated in diabetic cataract, but associated retinopathy often reduces the visual outcome.

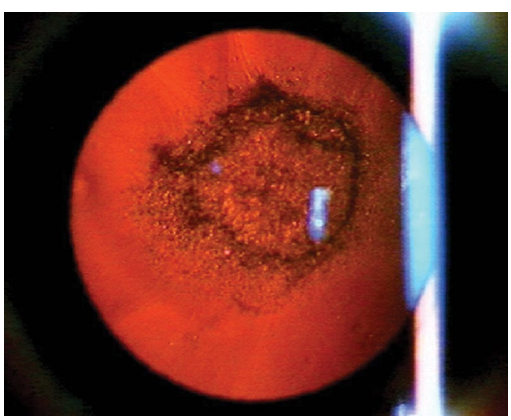


Fig. 15.28: Polychromatic lustre in complicated cataract



Fig. 15.29: Bilateral cataract in juvenile diabetes

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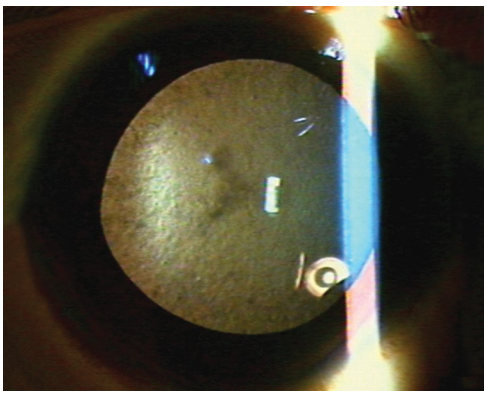


Fig. 15.30: True diabetic cataract—snowflakes

Galactose Cataract

Galactose is the metabolic product of lactose which is the main carbohydrate of milk.

Galactose cataract is associated with two kinds of ***recessively inherited*** galactosaemic enzyme deficiencies:

1. ***Classical galactosemia:*** It is caused by the absence of enzyme galactose-1-phosphate-uridyl-transferase (GPUT).
   - ***Ocular features:*** About 75% of the infants with classical galactosemia develop bilateral oil-droplet subcapsular lental opacities which may progress to maturity within a few months unless treated promptly.
   - ***Systemic features:*** It include diarrhea, vomiting, hepatosplenomegaly, nutritional failure and mental retardation.
2. ***Galactokinase (GK) deficiency:*** It is a milder form and only associated with infantile cataract in an apparently ***healthy child***.
   - ***Mechanism:*** Metabolic pathway from galactose to glucose derivatives is blocked by deficiency of GPUT and GK enzymes → thus blocking the normal metabolic pathway of lens metabolism—instead, shunting of metabolism by aldose reductase to produce dulcitol, a sugar-alcohol similar to sorbitol → increased osmotic pressure causes swelling of the lens → disruption of lens fibers with vacuolation → opacification of the lens.
   - Reducing sugar in urine and red blood cell enzyme assay confirm the diagnosis.
   - ***Treatment*** includes removing all lactose and galactose from the diet. This will prevent the development of cataract and even in early stage, it may reverse the lental changes.

Traumatic Cataract

It may be either due to a concussion or penetrating injury (*see* Chapter 24).

Heat Cataract

It is produced by prolonged exposure to infrared rays, and occurs in industry. It is seen among glass-blowers in glass factories ***(glass-blower's cataract)*** and also among ***ironworkers,*** especially tin-plate mill-men and chain-makers.

- The cataract appears as a small disc-like opacity in the posterior cortex which is sharply demarcated.
- There is an associated ***exfoliation*** (true exfoliation) of the lens capsule, in which the lamella of the capsule may be curled up in the pupillary area as large sheets.
- ***Mechanism:*** The heat (infrared) rays act not directly on the lens, but are absorbed by the pigment of the iris and ciliary body, and thus influences the lens fibers indirectly.

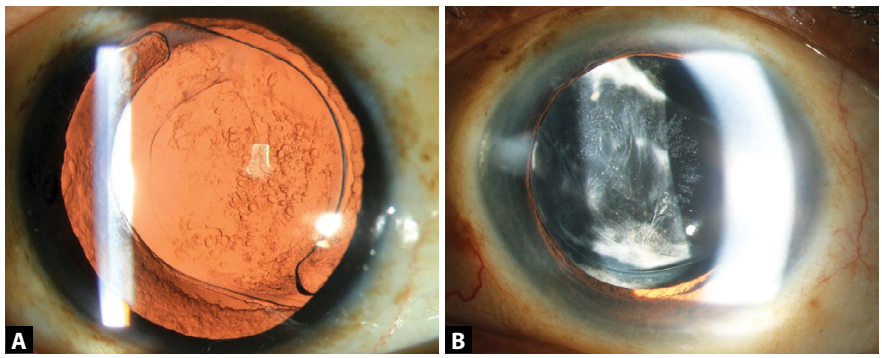
Posterior Capsular Opacification After-cataract

It is a membranous, white opacity formed by the remnants of anterior and posterior capsules of the lens, following extracapsular cataract extraction and discission of the congenital or traumatic cataract.

Incidence

In adults, it is about 5–35%, within one to five years after the surgery, and more common in children (may be upto 100%).

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Figs 15.31A and B: Posterior capsular opacification. **A.** Elschnig's pearl type; **B.** Fibrous type

Mechanisms

- When the cataract is not mature, some soft, clear cortex, sticks to the capsule which may be difficult to remove during operation.
- This gets partially absorbed by the action of aqueous, but, often gets entrapped by adhesion of the remnants of anterior capsule with the posterior capsule.
- In such cases, the cuboidal cells which line the anterior capsule, continue to fulfil their function of formation new lens fibers. These lens fibers under the abnormal condition are abortive and opaque.
- Along with it, organized inflammatory exudates (from iridocyclitis), or fibrin (from hyphaema)—may also contribute to the formation of after-cataract.

The after-cataract may be:

- Capsular,
- Capsulolenticular, and
- Pigmentary or hemorrhagic.

Again it may be ***Elschnig's pearl type*** (Fig. 15.31A) or ***fibrous type*** (Fig. 15.31B). Elschnig's type causes more visual disability than fibrous type.

- ***Elschnig's pearl:*** The subcapsular cubical cells proliferate and instead of forming lens fibers, they sometimes, develop into large balloon-like cells which fill the pupillary aperture. This balloon lens-cell looks like pearl, and is known as Elschnig's pearl.

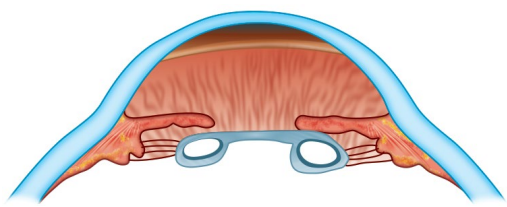


Fig. 15.32: Soemerring's ring in after-cataract

- ***Soemerring's ring:*** It is ring behind the iris, formed by the lens fibers enclosed between the two layers of lens capsule and found in some cases of after-cataract. It may dislocate into the anterior chamber and causes secondary glaucoma (Fig. 15.32).

Treatment

- ***YAG-laser capsulotomy (Fig. 15.33):*** This modern technique is especially useful in after-cataract following ECCE with PCIOL implantation.
  YAG-laser acts by photodisruption of the tissue. It is safe, non-invasive (no chance of infection) and a quick OPD procedure.
- ***Discission or needling:*** When the membrane is thin, then with the help of a Bowman's needle or Zeigler's knife, a gap is made in the central pupillary area.
  Repeated needling disturbs the anterior face of the vitreous and subsequently, vitreous gets organized at places which may ultimately lead to retinal detachment in future.

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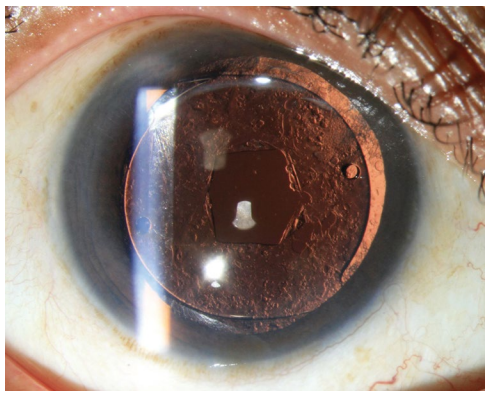


Fig. 15.33: Nd-YAG-laser capsulotomy in posterior capsular opacification

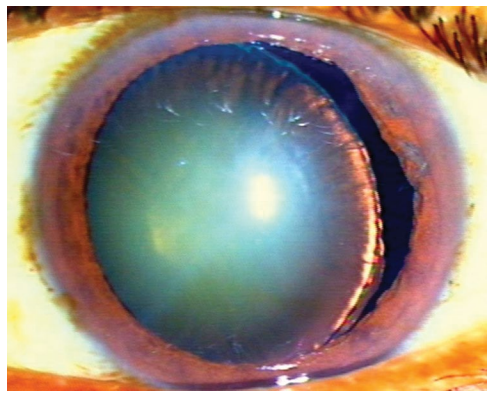


Fig. 15.34: Subluxation of the lens—after blunt trauma

- ***Capsulectomy, membranectomy or capsulo-iridectomy:*** It is done by using fine scissors, when the after-cataract is dense and adherent to the iris.

Prevention of PCO formation

- Continuous curvilinear capsulotomy (CCC).
- Good cortical cleaning including the peripheral and equatorial parts.
- Polishing of the posterior capsule.
- Polishing of the under-surface of the remaining anterior capsular ring.
- Insertion of PCIOL in-the-bag.
- Use biconvex PCIOL or planoconvex with convexity towards the posterior capsule.
- In case of capsulorhexis, the margin of the capsular opening must overlap 0.5–1 mm within the margin of the optic of IOL.
  That means, if the IOL-optic is 6.0 mm, the rhexis diameter should bae 5.0–5.5 mm.
- Hydrophobic acrylic IOL has least incidence of PCO.
- Lastly, square-edge optic designed IOL.

SUBLUXATION OF THE LENS (FIGS 15.34 AND 15.35)

This is a condition of the lens in which a portion of the supporting zonule is absent and the lens lacks support in that quadrant.

In subluxation, the lens usually remains in the pupillary area, though there may be tilting or displacement in any meridian.

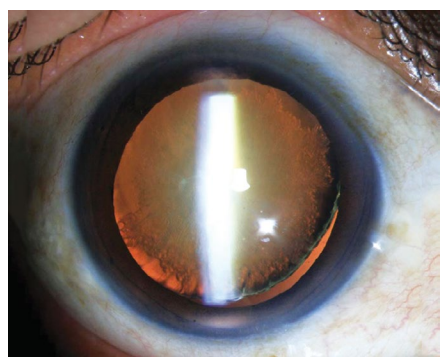


Fig. 15.35: Subluxation of the lens—high myopia

Etiology

- ***Congenital ectopia lentis*** (discussed)
- ***Acquired***
  - ***Spontaneous***: It is due to:
    - Excessive stretching of the zonules, as in buphthalmos, high myopia or perforation of corneal ulcer.
    - ***Degeneration of the zonules***, as in hypermature cataract and patient with latent syphilis.
  - ***Traumatic:*** It is due to tear of the zonules following blunt or penetrating injury.

Symptoms

- Monocular diplopia
- Dimness of vision due to myopic astigmatism.

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Signs

- Unequal depth of the anterior chamber.
- Tremulousness of the iris ***(iridodonesis)*** and the lens ***(phacodonesis)***.
- Diagnosis should be confirmed after full dilatation of the pupil. The edge of the lens is visible as a dark crescentic line in oblique illumination.
- Indirect ophthalmoscopy may show, two images of the disc due to phakic and aphakic zones in the pupil.
- Secondary glaucoma may be present in some cases.

Treatment

- If the lens remains clear and there is no irritative symptom—glasses are prescribed against phakic part to correct lenticular myopic astigmatism.
- Alternately, if the aphakic zone in the pupil is more—aphakic correction may be considered.
- In presence of cataract or irritative symptoms are:
  - ***For small zonular tear:*** ECCE with PCIOL may be considered. A capsular tension ring (CTR) is helpful in some cases of subluxation. Here, a fine PMMA (polymethylmethacrylate) made ring is placed within the capsule bag to give support to zonule.
  - ***If the zonular tear is more:*** Intracapsular cataract extraction with vectis is the treatment of choice. This is then followed by anterior vitrectomy with or without ACIOL implantation.
  - Sometimes, a scleral fixation PCIOL may be considered, when the other eye is normal or pseudophakic.

DISLOCATION OF THE LENS (FIGS 15.36A AND B)

This is the condition, in which the crystalline lens is completely unsupported by the zonular fibers, so that the lens is completely displaced from the pupillary area.

Etiology

- Same as subluxation of the lens.
- Deliberate dislocation of the cataractous lens in the vitreous cavity (by forcible backward pushing)—an ancient (and unwise) surgical procedure, called couching or reclination. It is still sometimes practised in rural India by the quacks.

Symptoms

- Marked dimness of vision for distance and near, due to high hypermetropia and loss of accommodation.
- Slight improvement of vision in dislocated hypermature cataract.

A B

Figs 15.36A and B: Dislocation of the lens. **A.** Anterior; **B.** Posterior

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- Migration of the dislocated lens into the anterior chamber or vitreous, gives rise the symptoms of uveitis and glaucoma.

Signs

- ***Signs of aphakia:*** Like, deep anterior chamber, jet black pupil, iridodonesis and absence of third and fourth Purkinje images but without an iridectomy, or a limbal scar mark.
- When the lens is dislocated into the anterior chamber and clear—it appears as an ***oil-globule*** due to total internal reflection.
- In posterior dislocation—after dilation of the pupil, the lens can be seen as translucent or opaque mass, lying at the bottom of the vitreous cavity.
- In traumatic cases, associated ocular pathology may also be seen.

Complications

It is more with dislocation of lens than with subluxation:

- Secondary glaucoma
- Severe iridocyclitis
- Low-grade cyclitis due to irritation of the ciliary body region by the lens
- Vitreoretinal degeneration.

Treatment

- ***Anterior dislocation (Fig. 15.37):*** Immediate intracapsular extraction of the lens and vitrectomy with or without implantation of ACIOL.
- ***Posterior dislocation (Fig. 15.38)***
  - No inflammatory signs—dislocated lens may be kept as such, and only aphakic glasses are prescribed.

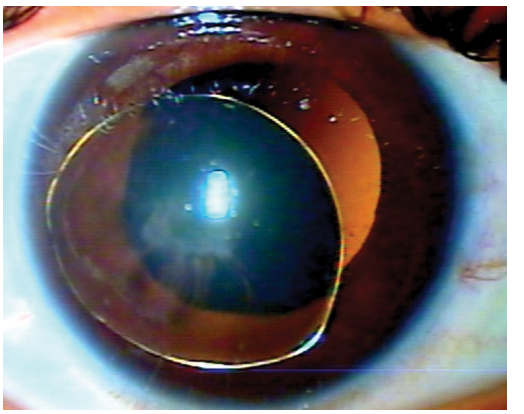


Fig. 15.37: Anterior dislocation of the lens

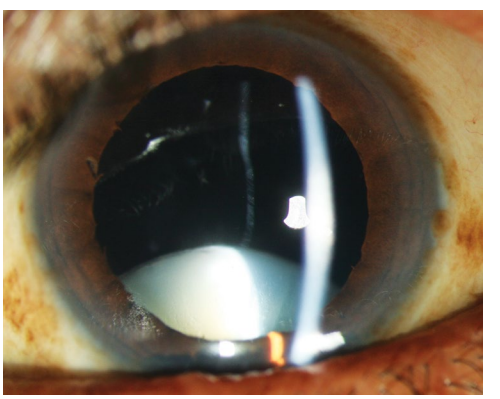


Fig. 15.38: Posterior dislocation of the lens with intact vitreous face

  - In presence of complication—the lens has to be extracted (lensectomy) along with vitrectomy and then ACIOL may be implanted.

A scleral fixation (SF) PCIOL can be implanted by the experts in this situation. The haptic of the IOL is fixed with sclera with prolene sutures or tissue-glue (called, glued IOL).

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16 CHAPTER

Glaucoma

INTRAOCULAR PRESSURE

It is the internal pressure exerted by the intraocular fluids on the coats of the eyeball. The normal intraocular pressure (IOP) is between 10 and 20 mm Hg (in contrast, the normal tissue pressure is about 2 mm Hg and intracranial pressure is 7 mm Hg).

Two main factors, concerned with the maintenance of IOP are the rate of secretion of aqueous humor and its rate of outflow from the eye. Most (75%) of the resistance to outflow is in the trabecular meshwork immediately adjacent to the canal of Schlemm (Fig. 16.1).

The ease with which the fluid exits, is indicated as the ***coefficient of the facility of outflow*** ('C'), and in the normal eyes is more than 0.2 µl/min/mm Hg of pressure within the eye. It is measured clinically by tonography.

Under normal conditions, the rate of aqueous production is relatively constant, so that, in clinical practice, the main determinants of IOP are the ***resistance of exit channels,*** and the ***outflow pressure*** (IOP minus episcleral venous pressure).

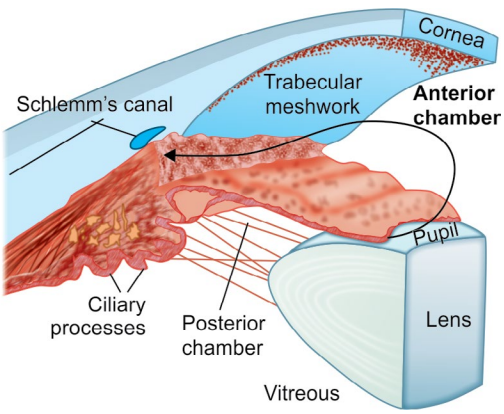


Fig. 16.1: Aqueous outflow channels

Factors Modifying Intraocular Pressure

Physiological Variations

The IOP normally fluctuates 2–5 mm Hg throughout the day.

- ***With respiration and heart beat:*** The pressure fluctuates upto 5 mm Hg with each breath and 1–2 mm Hg with each heart beat.
- ***With time of the day:*** A characteristic diurnal variation is seen, with a minimum around 6.00 PM, and a peak on waking in the morning. In normal eyes, this difference seldom exceeds 3–4 mm, but in glaucoma it is often much higher.
- ***With the venous pressure:*** The Valsalva maneuvre causes a rise in episcleral venous pressure, reduces aqueous outflow, and temporarily elevates the IOP.
- ***With the arterial pressure:*** Transient rise in IOP with the rise of systemic blood pressure.
- ***With the osmotic pressure of blood:*** When plasma osmolarity rises (as with intravenous mannitol, or oral glycerol), the IOP falls, whereas, reduced osmolarity (as with water drinking provocative test) raises IOP.

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Local Mechanical Factors

- ***Dilatation of the pupil:*** The IOP may rise if the anterior chamber is shallow, owing to a relative obstruction of the drainage angle by the iris.
- ***Changes in the solid content of the eye:*** A rise in IOP may result from rapid increase in size of the lens (intumescent cataract) or from the rapid growth of an intraocular neoplasm.
- ***Pressure from outside:*** IOP rises whenever the lids are closed, and forcible blepharospasm may cause rise of 5 mm or more; while contraction of rectus muscles may equally provoke a significant increase.

Pharmacological Factors

The ciliary muscle is inserted into the trabeculum, so the contraction of the ciliary muscle makes the trabecular meshwork more porous → increases the facility of outflow → reduces IOP.

- ***Outflow facility:*** Pilocarpine acts directly on the ciliary muscle to contract, and the coincident miosis is also important in relieving the pupillary block and obstruction of the drainage angle in acute glaucoma.
- ***Outflow facility*** is also increased by adrenaline and other α-blockers, and decreased by topical steroids.
- ***Reduction of the aqueous production:*** Adrenaline and carbonic anhydrase inhibitors are most important.
- ***Atropine*** produces a slight fall in IOP, possibly by facilitating the drainage of aqueous via uveoscleral route.

Measurement of Intraocular Pressure

- ***Manometry:*** It is measured by inserting a cannula, directly into the anterior chamber which is connected with a manometer. This is the most accurate method, but impossible in clinical practice, and only useful in experimental animals.



Fig. 16.2: Digital tonometry (finger tension)

- ***Digital tonometry (finger tension):*** Intraocular tension is roughly assessed by digital palpation, fluctuating the downturned eye between the two index fingers through the upper lid (above the tarsal plate) (Fig. 16.2).
  This is a routine clinical method and yields a fairly accurate assessment to experienced fingers. It is sometimes recorded as '+', '++', '+++', if increasingly high, or '-', '--', '---', if increasingly soft. In very high IOP—this fluctuation is absent (***stony hard***) and in very low IOP ***(phthisis bulbi)***—it is ***soft like a water bag.***
- ***Instrumental tonometry:*** Intraocular tension is measured by two types of tonometer:

1. ***Contact tonometer (Fig. 16.3)***
   i. ***Indentation tonometer:***
      – Schiotz (manual type)
      – Mackay-Marg (electronic type)
   ii. ***Applanation tonometer (AT) :***
      – Goldmann AT (slit-lamp mounted)
      – Parkins' AT (hand-held)
2. ***Noncontact tonometer (NCT)***
   - ***Schiotz (indentation) tonometry (Fig. 16.4A):*** This is based on the principle, that a plunger will indent a soft eye more than a hard eye. It is constructed

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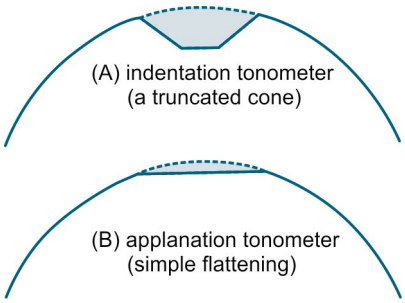


Fig. 16.3: Corneal deformation in contact tonometry

in such a way that, when placed on the eye, the plunger together with a present weight, indents the cornea.

The amount of indentation is measured on a scale, and then the corresponding intraocular tension (Fig. 16.4B) (in mm Hg) can be calculated from an accompanying table. The normal IOP in Schiotz tonometer is 14–20 mm Hg.

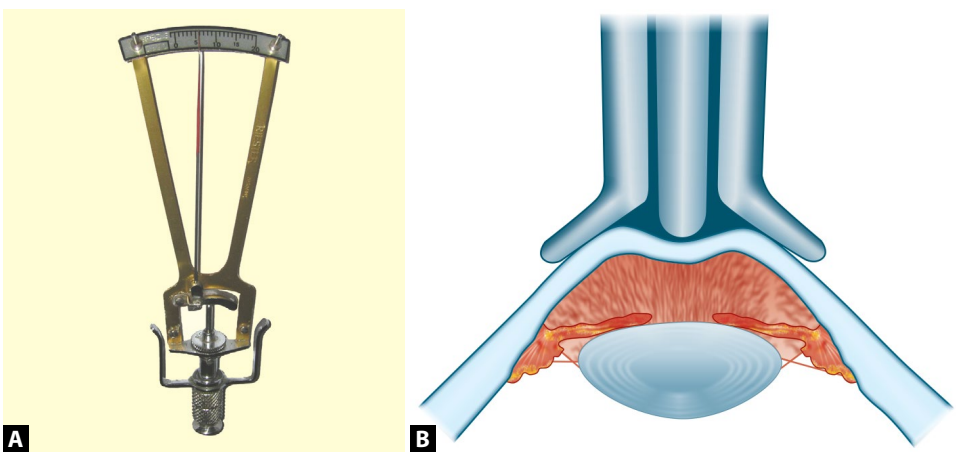
- ***Advantages:*** It is cheap, easy to use, convenient to carry and does not require a slit-lamp for measurement.
- ***Disadvantage:*** Schiotz tonometer (Fig. 16.5) is not so accurate, since the intraocular tension recorded, is influenced by ocular (scleral) rigidity.

Scleral rigidity means resistance to distensibility of the outer coats of the eyeball, especially the sclera. Increased elasticity of the outer coats of the eye, is called ***low scleral rigidity***. It may occur in high myopia, thyroid exophthalmos, miotic therapy and post-operative eyes. In these cases, Schiotz tonometer may record a falsely low reading of IOP when compared with an applanation tonometer.

Similarly, if the eye has an abnormally ***high scleral rigidity*** (as in hypermetropic eyes, microphthalmos) a falsely high level of IOP may be recorded.

Abnormalities of scleral rigidity can be detected with a Schiotz tonometer by using two different weights (e.g. 5.5 Gram and 10 Gram) If the two measurements are identical, then the scleral rigidity is normal. If there is a discrepancy, then the scleral rigidity is abnormal and the value can be determined by consulting ***Friedenwald's nomogram***. The average normal coefficient of scleral rigidity is 0.0215.

- ***Applanation tonometer:*** This is based on the principle of ***Imbert-Fick's Law***. It states that for an ideal, thin-walled sphere, the pressure inside the sphere (P) equals to the force necessary to flatten its surface f. divided by the area of flattening (A), i.e. P = F/A.

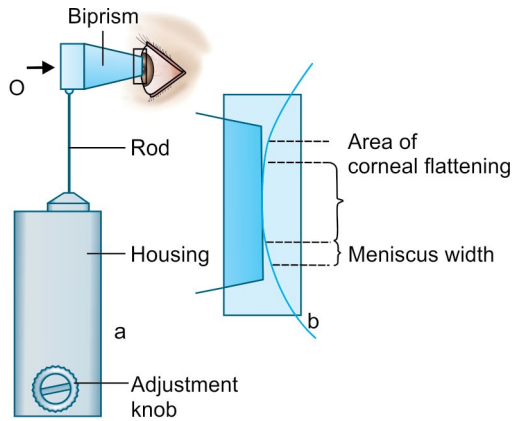


Figs 16.4A and B: **A.** Schiotz tonometer; **B.** Indentation of the anesthetized cornea by the plunger of the tonometer in order to measure intraocular tension

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Fig. 16.5: Schiotz tonometry



(O = Observer's view)

An applanation tonometer measures the IOP by flattening (rather than indent) the cornea over a specific area (applanation of circle with a diameter at 3.06 mm). This is more accurate since the pressure values recorded are not influenced by scleral rigidity.

- ***Goldmann applanation tonometer (Fig. 16.6)*** is mounted on a slit-lamp and a special plastic prism rests on the cornea, the pressure being adjusted until it achieves a standard disc of contact as outlined by a pre-instilled drop of fluorescein (Fig. 16.7).
- ***Parkins (hand-held) applanation tonometer (Fig. 16.8)*** is similar except, that it does not require a slit-lamp, and it can be used even in supine position.

The normal IOP in applanation tonometry (ATn) is 15.5 ± 2.5 mm of Hg. The normal IOP ranges from 10 to 21 mm of Hg. If a value is more than 21 mm of Hg, it is considered high IOP.

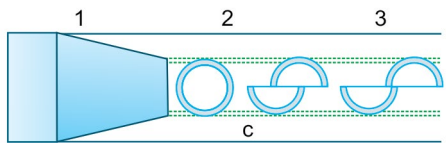


Fig. 16.6: Goldmann-type applanation tonometry. a—basic features of tonometer, shown in contact with patient's cornea, b shows tear film meniscus created by contact of biprism and cornea, c—view through biprism (1) reveals circular meniscus (2) which is converted into semicircles (3) by prisms



Fig. 16.7: Goldmann's applanation tonometry

CORNEAL THICKNESS AND GLAUCOMA

Central corneal thickness (CCT) variations affect the apparent IOP as measured by applanation tonometer. Thinner corneas tend to give a reading of lower IOP whereas thicker corneas tend to overestimate the IOP value falsely. A CCT lower than 555 microns is an

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Fig. 16.8: Parkins applanation tonometry



Fig. 16.9: Noncontact tonometry

independent risk factor for the development of primary open angle glaucoma. When originally devised, it was believed that significant variations in corneal thickness were uncommon and a CCT of 520 μ was assumed in calibration of the Goldmann applanation tonometer.

In each case of suspected glaucoma, especially for the open angle type, it is necessary to measure the CCT by pachymetry. Again, the ultrasonic pachymeter is currently the gold standard.

Different nomograms (Ehler's, Dresdner's, or Doudhty's) are used to correct IOP with CCT variations. But, no nomograms have been identified to this day to be full proof. One may use 15 μ corneal thickness as being equivalent to 1 mm of Hg as a rough average to arrive at the corrected IOP reading.

Other Types of Tonometer

- ***Pneumotonometer:*** It is an electronic tonometer. It is useful for patient's with corneal scarring or altered corneal shape.
- ***Air-puff noncontact tonometry:*** No local pediatric is required. No chance of cross infection. Ideal for use in mass glaucoma screening program. Its accuracy is also satisfactory (Fig. 16.9).

Definitions of Glaucoma

- ***Glaucoma*** is a symptomatic condition where the functional integrity of the eye is disturbed, resulting in characteristic irreversible loss of visual field due to persistent raised IOP.

> ***Glaucoma*** is a multifactorial optic neuropathy in which there is a characteristic loss of retinal ganglion cells and atrophy of the optic nerve. Though controversial, this definition of American Academy of Ophthalmology excludes IOP as a criterion and says this is a risk factor.

- ***Ocular hypertension:*** The patient is having bilateral, high intraocular tension (i.e. above 21 mm Hg) without any field defect, or cupping of the disc. These patients should be kept under observation as some of them may develop open-angle glaucoma in future.
- ***Low-tension glaucoma (LTG):*** This is also called ***normal-tension glaucoma (NTG)***, in which a few patients develop characteristic field defects and cupping of the optic disc, in spite of normal IOP.

This is a variety of open-angle glaucoma, and may be associated with some cardiovascular abnormality which is responsible for reduced optic nerve head perfusion.

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Classification of Glaucoma

Congenital or developmental

- ***Primary:*** Due to primary developmental anomaly at the angle.
- ***Secondary:*** Associated with other ocular or systemic disorders.

Acquired

- ***Primary***
  - Angle-closure glaucoma (PACG)
  - Open-angle glaucoma (POAG)
- ***Secondary:*** Associated with some ocular or systemic diseases.

CONGENITAL OR INFANTILE GLAUCOMA (BUPHTHALMOS)

It is a congenital or infantile glaucoma due to aqueous outflow obstruction, as a result of failure of the development of tissue at the angle of the anterior chamber.

ETIOPATHOGENESIS

- ***Primary congenital glaucoma*** (Fig. 16.10) is inherited as autosomal recessive trait.
- Boys are more affected than girls.
- It is usually bilateral.
- Forty percent cases are true congenital, and in 50% of cases, the disease manifest within one year of birth ***(infantile glaucoma) (Figs 16.11 and 16.12).***



Fig. 16.10: Primary congenital glaucoma—bilateral



Fig. 16.11: Infantile glaucoma—buphthalmos—right eye



Fig. 16.12: Infantile glaucoma—buphthalmos (both eyes)

- ***Pathogenesis:*** (a) Presence of a cellular membrane ***(called Barkan's membrane),*** obstructs the aqueous outflow. (b) Abnormal cleavage of the anterior chamber angle is responsible.
- ***Associated factors:*** A developmental anomaly, elsewhere in the eye is responsible for obstruction of the aqueous outflow (secondary congenital glaucoma), e.g.
  - Aniridia,
  - Neurofibromatosis,
  - Sturge-Weber syndrome,
  - Rubella syndrome,
  - Mesodermal dysgenesis (Rieger's anomaly and Peter's anomaly), etc.

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SYMPTOMS

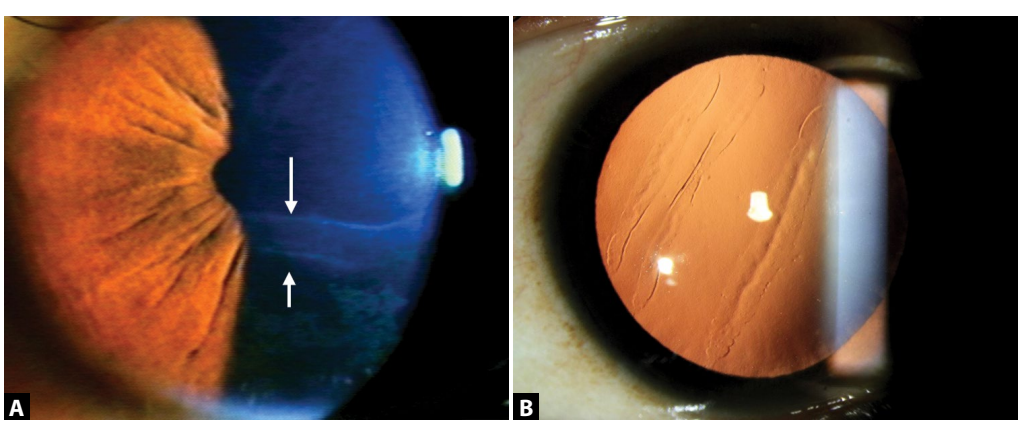
- Marked photophobia
- Blepharospasm
- Watering.

SIGNS

- ***Eyeball:*** It becomes enlarged, if the IOP becomes elevated prior to age of three years. This enlarged eyeball due to glaucoma, is known as buphthalmos (means ox-eye).
- ***Sclera:*** Bluish discoloration.
- ***Cornea:*** It is enlarged, globular and steamy. Horizontal curvilinear lines are seen on the back of the cornea, known as ***Haab's striae*** (Fig. 16.13A), and they represent healed breaks of Descemet's membrane (Fig. 16.13B). Corneal sensation is diminished.
- ***Anterior chamber:*** Deep.
- ***Iris:*** Tremulousness of the iris with patches of atrophy.
- ***Lens:*** Flattened, and displaced backwards. There may be subluxation.
- ***Fundus:*** Shows cupping of the disc, but it may regress when the IOP is normalized quickly.
- ***Intraocular tension:*** High, but it is usually neither marked, nor acute.
- ***Refractive error:*** The patient becomes myopic. But the amount of myopia is less than anticipated from the increased axial length, owing to flattening and backward displacement of the crystalline lens.

MANAGEMENT

- ***Evaluation under anesthesia:*** Examination should be performed under general anesthesia. It includes:
  - ***Measurement of corneal diameter:*** In infant, the mean horizontal corneal diameter is 10.0 mm. If a corneal diameter is more than 12 mm within first year, it is always pathological.
  - ***Intraocular tension:*** It is preferably measured with Parkin's (hand-held) applanation tonometer, as the scleral rigidity is very low in children. The normal IOP in an infant under ***Halothane*** anesthesia is approximately 9–10 mm Hg, and a pressure of 20 mm Hg or more should arouse suspicion.
  - ***Gonioscopy:*** To study the angle with the help of goniolens.



Figs 16.13A and B: **A.** Haab striae—congenital glaucoma (horizontal curvilinear line) (arrows); **B.** Descemet tear—birth trauma (vertical curvilinear line)

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- ***Treatment***
  - ***Medical:*** It is of no value. Miotics or β-blocker is given only for temporary period prior to surgery.
  - ***Surgical:*** Treatment of congenital glaucoma is essentially surgical.
    - ***Goniotomy:*** An arcuate incision is made with a special knife, halfway between the iris and the Schwalbe's line.
    - ***Goniopuncture:*** A puncture is made through the whole thickness of the trabecular region into the subconjunctival space.
    - ***Trabeculotomy:*** A fine metal probe is passed into the Schlemm's canal, and is then swept into the anterior chamber, thus exposing the Schlemm's canal directly to the aqueous humor.
    - ***Combined trabeculectomy and trabeculotomy:*** It may give the best surgical result amongst all types of surgery.
- ***Rehabilitation:*** It is extremely important to detect and treat any refractive error and amblyopia. Corneal opacity may be treated by penetrating keratoplasty.

ACQUIRED GLAUCOMA

PRIMARY ANGLE-CLOSURE GLAUCOMA

Primary angle-closure glaucoma (Fig. 16.14) (PACG) is an acute, subacute or chronic glaucoma, due to obstruction of the aqueous outflow, solely caused by closure of the angle by the peripheral iris.

Etiology

- ***Age:*** 4th to 5th decade
- ***Sex:*** Female: male = 4:1
- ***Personality:*** Emotional, nervous people with unstable vasomotor system.
- ***Type of the eye affected:***

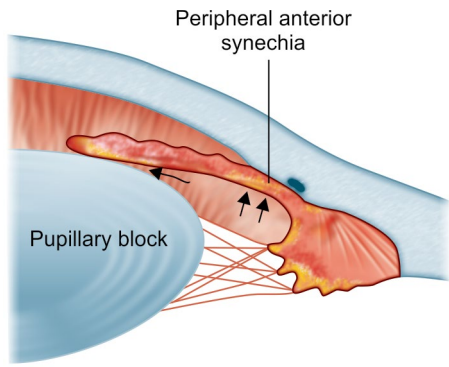
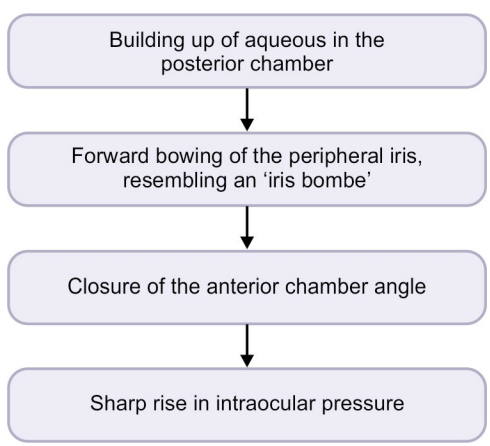


Fig. 16.14: Angle-closure glaucoma

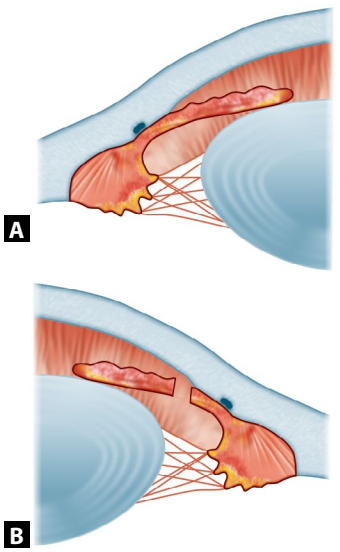
  - Small hypermetropic eye
  - Shallow anterior chamber
  - Narrow anterior chamber angle, due to:
    - Smaller cornea,
    - Bigger size of the lens, and
    - Bigger size of the ciliary body.
- ***Laterality:*** Initially unilateral, but frequently becomes bilateral.

Mechanisms of Angle-closure (Figs 16.15A and B)

- ***Pupillary block mechanism:*** The initiating event is thought to be a ***functional pupillary block*** (between the pupillary portion of the iris and the anterior lens surface) in mid-dilated position.



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Figs 16.15A and B: Mechanisms of angle-closure. **A.** Pupillary block mechanism; **B.** Plateau iris mechanism

In the second mechanism **(B)**, note the deep central anterior chamber, the flat iris plane, patent iridectomy, and infolding of the peripheral iris at the angle

- ***Plateau iris mechanism:*** It is due to an abnormal anatomical configuration of the anterior chamber angle. The angle is closed by infolding of the iris into the angle in association with pupillary dilatation, but without a significant pupillary block component. Peripheral iridectomy does not help to control IOP in this situation. Pilocarpine is the only drug which controls this situation. Otherwise, surgery is required.

Stages

It is divided into ***five stages***—(1) Prodromal stage, (2) Stage of constant instability, (3) Acute congestive attack, (4) Chronic congestive stage and (5) Stage of absolute glaucoma.

Prodromal Stage

Symptoms

- Colored halos around the light
- Blurring of vision
- Occasional headache.

Signs: Eye remains white and without congestion, even-though, there may be transient sudden rise in IOP upto 40 or 60 mm Hg.

Stage of Constant Instability

This second phase is reached, when intermittency of prodromal attacks is replaced by regularity.

The IOP may rise in the late afternoon and evening, but reduces spontaneously during sleep (as the pupil becomes constricted).

Acute Congestive Attack

An acute congestive attack occurs sooner or later, and is always associated with closure of the angle, and a severe elevation of IOP.

Pathology

The crisis in acute attack is due to acute ischemia, perhaps, associated with the liberation of prostaglandin-like substances. If the attack lasts for several hours or days, irreversible damage may occur to the ocular tissues.

The effects on different ocular structures during and following an acute attack are:

- ***Accumulation of the fluid occurs*** in all the layers of cornea. This is due to imbibition of fluid in the cornea caused by dysfunction of 'endothelial pump' owing to sharp rise in IOP.
- ***Circumcorneal congestion and chemosis*** of the conjunctiva, are due to stasis with increased permeability of the capillaries.
- ***Peripheral anterior synechiae*** occur as a result of prolonged or repeated acute congestive attacks. Initially, there is iridocorneal contact and later on, the contact becomes irido-trabecular.
- ***Pupil is mid-dilated*** and oval. Iris shows atrophic changes adjacent to the sphincter muscle. This is due to ischaemia of the sphincter pupillae.
- ***Glaucom-fleckens*** (Fig. 16.16) are small grayish-white anterior subcapsular opacities occur in the pupillary zone. They are diagnostic of previous attack of angle-closure glaucoma, and due to atrophy of the newly formed lens fibers.

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- ***Optic nerve head*** is usually edematous and hyperemic, and is due to anterior ischemic optic neuropathy. Cupping of the optic disc is usually evident after 2 weeks, if the disease passes into chronic congestive phase.

Clinical Features of an Acute Attack

Symptoms

- Acute, intense, unbearable pain, radiating along the distribution of 5th cranial nerve.
- Severe headache often with nausea and vomiting, often mistaken for an acute abdomen.
- Marked dimness of vision, is mainly due to ischemic optic neuropathy, and partly due to corneal edema.
- Redness, lacrimation and photophobia.

Signs

- ***Eyeball:*** Tenderness present.
- ***Eyelids:*** Marked edema with narrowing of the palpebral aperture.
- ***Conjunctiva:*** Both ciliary and conjuntival congestion with chemosis.
- ***Cornea:*** Steamy and insensitive.
- ***Anterior chamber:*** Very shallow, cells and flare may be present [but no keratic precipitates (KPs)].
- ***Iris:*** Pattern is lost and discolored.

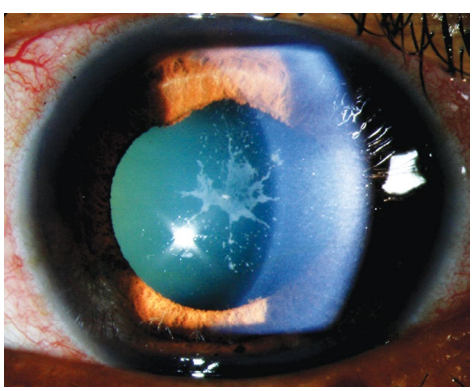


Fig. 16.16: Glaucom-fleckens (spilled-milk appearance)

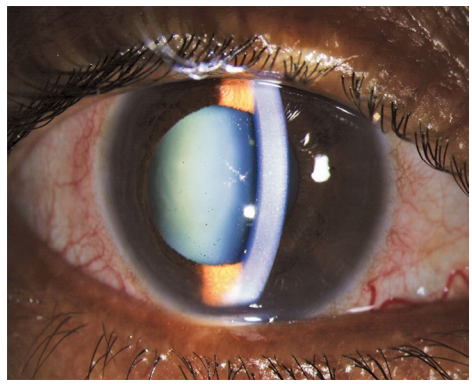


Fig. 16.17: Acute attack of primary angle-closure glaucoma—fixed mid-dilated, vertically oval pupil

- ***Pupil:*** Mid-dilated and vertically oval (Fig. 16.17). Reaction to light and accommodation are absent.
- ***IOP:*** Markedly elevated.
- ***Visual acuity:*** It may be reduced to perception of light (PL) and projection of rays (PR).
- ***Fundus:*** It can not be visualized due to hazy cornea.
- But after instillation of glycerine drop (it clears the cornea temporarily), the fundus may be visible with hyperemic optic disc with small hemorrhages, and with spontaneous pulsation of the central retinal artery. There is no cupping.
- Similarly, after instillation of glycerine drop, gonioscopy shows marked narrowing of the angle, with or without peripheral anterior synechiae.
- The fellow eye, usually has a shallow anterior chamber, and a narrow angle.

Termination of an acute attack

- Spontaneous improvement with lowering of IOP.
- It may pass into chronic congestive stage.
- Recurrence of attacks which may lead to
  - Marked impairment of vision due to acute-ischemic optic neuropathy, followed by cupping.
  - Irregular constriction of the visual field.

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  - Permanent peripheral anterior synechiae when three-fourth part of the iris is attached to the cornea, a 'false angle' is formed. Ultimately, all lead to the chronic congestive stage.
- Rarely, it passes into the absolute glaucoma stage, with total loss of vision (no PL).

Chronic Congestive Stage

This is sometimes called creeping angle closure, as the angle becomes slowly and progressively closed.

Clinical Features

- Visual acuity is always impaired.
- Congested and irritable eye.
- IOP remains permanently elevated.
- Cupping of the disc appears.
- Peripheral anterior synechiae develop, mostly in the upper part of the angle, but gradually spread around the whole circumference.
- Typical glaucomatous field defects become evident. The chronic congestive phase, if untreated, gradually passes into the final stage of absolute glaucoma.

Stage of Absolute Glaucoma

- Painful blind eye with no PL.
- Reddish-blue zone surrounding the limbus, due to dilated anterior ciliary vein.
- ***Cornea:*** Cloudy and insensitive; there may be associated bullous keratopathy or filamentary keratopathy.
- ***Anterior chamber:*** Very shallow.
- ***Iris***: Patches of atrophy, ectropion of the uveal pigments.
- ***Pupil:*** Dilated and grayish in appearance. There is no light reaction.
- ***Optic disc:*** Large and deep cupping with atrophic changes.
- ***Tension:*** Extremely high (stony hard).

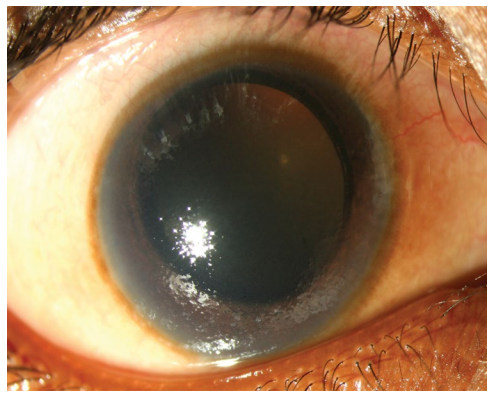


Fig. 16.18: Absolute glaucoma

Absolute glaucoma (Fig. 16.18): It is the end stage of any glaucoma, whether congenital or acquired, primary or secondary; and characterized by extremely high IOP (stony hard) with no PL.

Ultimately, the sequelae may be:

- ***Staphyloma formation***, due to continued high IOP → thinning of the sclera.
- ***Increased danger of rupture***, from slight injury.
- ***Atrophic bulbi:*** Continued pressure on the ciliary body → atrophy of the ciliary body → decreased aqueous production → eyeball shrinks → atrophic bulbi.

Diagnosis

In the acute and chronic congestive stages, the nature of the condition is usually obvious. But the diagnosis of angle-closure glaucoma, is really difficult in the prodromal stage, and is of immense importance.

Diagnostic criteria in early stages are:

- The history of seeing ***colored halos,***
- The presence of a ***narrow angle*** of the anterior chamber, and
- The inducement of a rise of tension ***(positive provocative tests)***.

Colored Halos

The colored halos are due to accumulation of fluid in the corneal epithelium and alterations in the refractive condition of the corneal lamellae. They are seen around the lighted

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bulb. The colors are distributed as in the spectrum of rainbow, with red being outside and violet innermost.

Similar colored halos may be seen in early immature cataract. These two can be differentiated by ***Fincham's stenopaeic slit test,*** where a stenopaeic slit is passed before the eye, across the line of vision. As it passes, a glaucomatous halo remains intact, but diminished in intensity, whereas a lenticular halo is broken up in segments which revolve as the slit is moved.

If the patient gives a vague history, the halo can be demonstrated to him, by his looking through a thin layer of lycopodium powder enclosed between two glass plates made up as a trial lens.

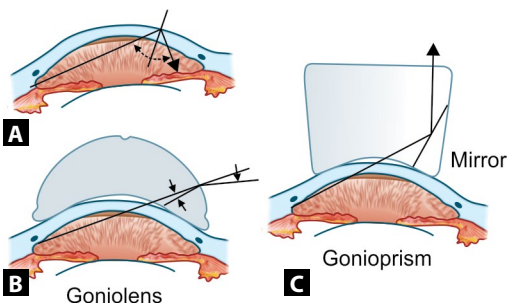
Gonioscopy

Before gonioscopy, narrow angle may be suspected in presence of shallow anterior chamber by routine torch light oblique illumination (Figs 16.19A and B).

The purpose of gonioscopy (Fig. 16.20) is to identify abnormal angle structures, and to estimate the width of the chamber angle.

Optics

Normally, the angle can not be visualized directly through an intact cornea, because light rays emitted from angle structures undergo ***total internal reflection*** (Figs 16.20A to C). A gonioscope eliminates total internal reflection



Figs 16.20A to C: Optical principle of gonioscopy. **A.** As the light from anterior chamber angle exceeds the critical angle at the 'cornea-air interface', it is reflected back in the eye; **B and C.** Contact lenses eliminate total internal reflection and allow light to enter the lens and then be refracted (goniolens) or reflected (gonioprism) beyond the 'lens-air interface'

A

B

Figs 16.19A and B: Oblique illumination by torch light—a screening measure to estimate the anterior chamber depth. **A.** With a deep chamber, nearly the entire iris is illuminated; **B.** When the iris is bowed forward, only the proximal portion is illuminated, but a shadow is seen in the distal half

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Fig. 16.21: Grading of the anterior chamber angle

by replacing the cornea-air interface by a new lens-air interface that has a greater refractive index than that of the cornea and tears.

Types of Gonioscopy

- ***Direct gonioscopy with goniolenses:*** They provide a direct view of the angle. They are used both as diagnostic and operative purposes, e.g. Koeppe, Barkan, Thorpe goniolens, etc.
- ***Indirect gonioscopy with gonioprisms:*** They provide a mirror image of the opposite angle, and can only be used under a slit-lamp, e.g. Goldmann single-mirror or three-mirror gonioscope, Zeiss four-mirror gonioscope, etc.
- ***Normal angle structures (Fig. 16.21)***
  From anterior to posterior
  - ***Schwalbe's line***, is an opaque line which represents the peripheral termination of the Descemet's membrane.
  - ***Trabecular meshwork*** (degree of pigmentation varies), with occasional visibility of the Schlemm's canal.
  - ***Scleral spur***, is the prominent white line which represents the most anterior projection of the sclera.

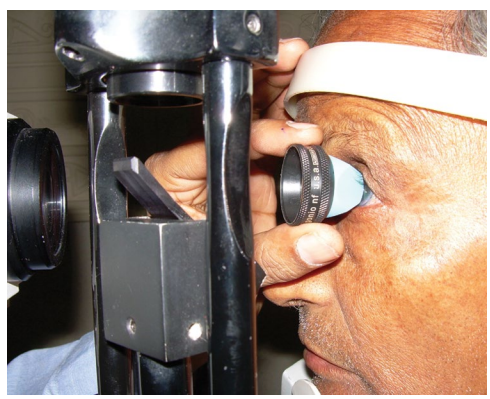


Fig. 16.22: Gonioscopy

  - ***Ciliary band***, a gray or dull-brown band of ciliary body, at the insertion of iris root.

When a gonioscope is not available, ***slit-lamp technique of van Herick*** may be used with a fair accuracy. Here, the depth of the peripheral anterior chamber (PAC) is estimated by comparing it to the adjacent corneal thickness (CT) 1 mm inside the limbus.

The presence of a narrow-angle of the anterior chamber, as evident from gonioscopy (Figs 16.22 and 16.23), is invaluable in the diagnosis of the disease.

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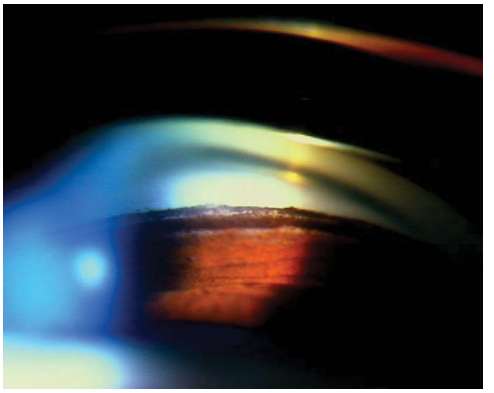


Fig. 16.23: Normal gonioscopic appearance of the angle of the anterior chamber

Van Herick slit-lamp grading of the angle (Table 16.1, Figs 16.24 and 16.25)
Grade 4 : PAC >1 CT-wide open angle
Grade 3 : PAC = ¼ - ½ CT-mild narrow angle
Grade 2 : PAC = ¼ CT-moderately narrow angle
Grade 1 : PAC <¼ CT-extremely narrow angle

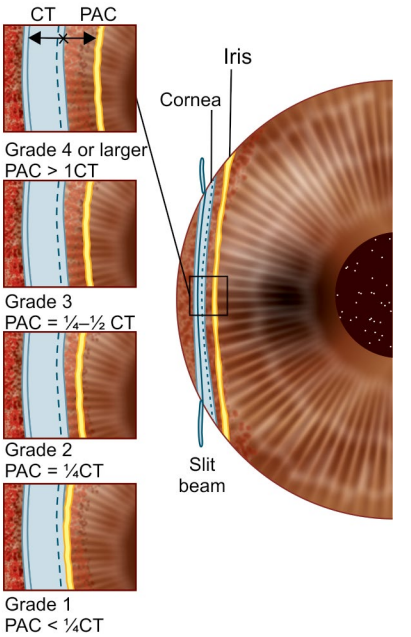


Fig. 16.24: Van Herick slit-lamp grading for estimating the depth of the peripheral anterior chamber (PAC) by comparing it to the peripheral corneal thickness (CT)

Provocative Tests

These are a variety of tests for ACG suspects, and have been designed to precipitate an attack of angle-closure glaucoma in the ophthalmologist's office (where it can be treated promptly).

Physiological provocative tests: Before the test is started, the IOP is recorded.

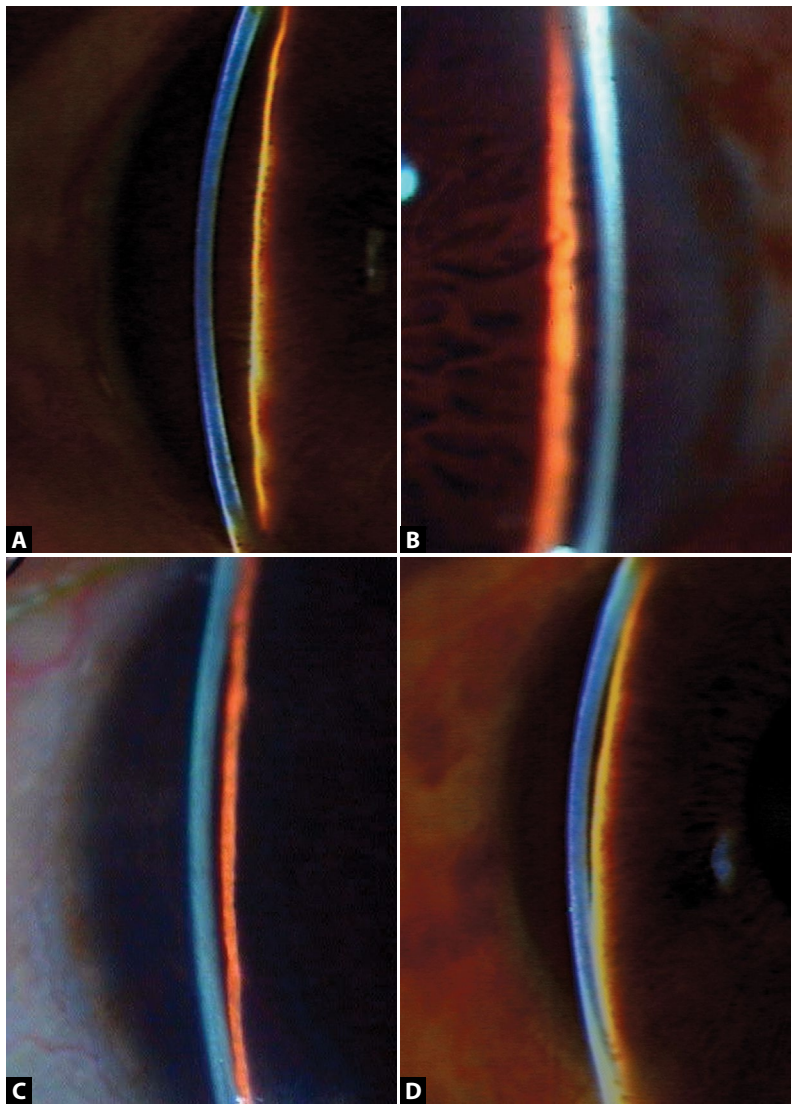
- ***Dark room test:*** The patient is left in a dark room for 1 hour. He must remain awake so that pupils remain dilated.
- ***Prone test:*** The patient lies in prone position for 1 hour.
- ***Prone-dark room test:*** The patient lies prone in a dark room (awake) for 1 hour. This appears to be the most popular and best physiologic provocative test.

After one hour, the IOP is measured, and gonioscopy is performed. A pressure rise of 8 mm Hg or more, in presence of a closed angle, is taken as a positive response.

TABLE 16.1: Grading of the angle of the anterior chamber (Schaffer's grading)

Grade	Angle	Gonioscopic view	Clinical interpretation
IV	Wide open (35–450)	All structures visible	Closure improbable
III	Open (20–350)	Scleral spur visible	Closure improbable
II	Moderately narrow (10–200)	Trabecular meshwork visible	Closure possible
I	Extremely narrow (<100)	Schwalbe's line visible	Closure imminent
0	Completely closed (00)	Nothing visible	Closure present

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Figs 16.25A to D: Van Herick slit-lamp grading of the angle (see also schematic representation in Fig. 16.24): **A.** Grade 4; **B.** Grade 3; **C.** Grade 2; **D.** Grade 1

- ***Mydriatic (pharmacological) provocative test:*** A short-acting topical mydriatic (e.g. 0.5% tropicamide) is instilled, and a pressure rise of 8 mm Hg or more, is considered to be positive.
- ***Mapstone's (pilocarpine-phenylephrine) test:*** 2% pilocarpine and 10% phenylephrine are instilled simultaneously every minute for three applications, to achieve a mid-dilated pupil. A pressure rise greater than 8 mm Hg, is considered as a positive response.
- ***If the provocative test is negative***, this does not necessarily mean that the angle is incapable of closure in near future. The patient should be warned of possible symptoms of angle-closure, and followed up periodically.

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- ***A positive provocative test*** means that the angle is capable of spontaneous closure, but it does not mean that closure is imminent or inevitable. So, the possible complications of iridectomy or Laser iridotomy have to be weighed against the risk of frank ACG.

Management

In early stages (intermittent attacks)

- ***Miotic therapy:*** Instillation of pilocarpine 2% every 5 minutes, is usually effective in pulling the iris from the angle and aborting the attack. The fellow eye should be treated with 2% pilocarpine, 3 times daily.
- ***Yttrium aluminium garnet laser peripheral iridotomy*** (YAG PI) or surgical peripheral iridectomy (surgical PI) should then be performed in both eyes.

In acute congestive attack: Although, the treatment of acute PACG is essentially surgical, the initial treatment is medical.

- ***Medical therapy***
  - ***Tabacetazolamide (250 mg):*** 2 tabs stat, and then followed by 1 tab 4 times daily, with potassium supplement.
  - ***Corneal indentation***: Simple repeated indentations of the central part of the cornea with a squint hook or a sterile swab-stick may be effective in opening the angle. By this maneuvre, aqueous will exit from the anterior chamber and the IOP will drop. This is only effective in absence of significant synechial closure of the angle.
  - ***Hyperosmotic agents:*** They act by drawing water out of the eye and reduce the IOP.
    - ***Intravascular injection of mannitol (20%):*** The dose is 1-2 gm/kg body weight, i.e. 300-500 ml is given intravenously over a period of 30–45 minutes.
    - ***Oral glycerol (50% solution):*** 30 mL (1 oz) of pure glycerol with equal amount of fruit (lemon) juice stat, and then 3 times daily. Cannot be used in diabetics.
    - ***Isosorbide:*** It is used orally, and it does not cause nausea. It can be used safely in diabetic patients.
  - ***Pilocarpine*** (2% or 4%) eye drop is instilled every 5 minutes till the pupil gets constricted, then 3–4 times daily. The ***fellow eye*** should be treated simultaneously with pilocarpine 2%, 3 times daily.
  - ***Strong analgesics*** (even injection pethidine) and antiemetic may be needed to reduce pain and vomiting.
  - ***Steroid-antibiotic drops*** are instilled frequently to reduce congestion.

  Once the IOP has been reduced medically and eye becomes quiet, the further management is:
  - ***Continue medical treatment*** with pilocarpine 2% drop 3–4 times daily. This is only indicated in old patient with poor general health, and where a laser is not available.
  - ***Surgery:*** (i) Laser iridotomy or surgical peripheral iridectomy and if necessary, (ii) Filtration surgery.
- ***Surgical therapy:*** A careful gonioscopy (indentation gonioscopy with Zeiss four-mirror gonioscope is much better) is necessary in deciding the percentage of angle closure by peripheral anterior synechiae, before considering the type of surgery.
  - If the angle-closure (by synechiae) is less than 50%, then laser iridotomy or a surgical peripheral iridectomy should suffice.
  - If the angle-closure is more than 50%, then a filtration operation (e.g. trabeculectomy) is indicated.

Laser iridotomy: In general, Nd:YAG or argon laser iridotomy (Fig. 16.26) has replaced surgical peripheral iridectomy in most cases.

Advantages

- A non-invasive procedure without any chance of infection.
- A painless, OPD procedure.
- It is cheap for the patient.

Disadvantages

- Not widely available, as the instrument is costly.
- Difficult to perform in presence of corneal edema and flat anterior chamber.
- May cause endothelial burns and localized lental opacity.
- Iridotomy hole may be blocked by scar tissue, specially in black individuals.

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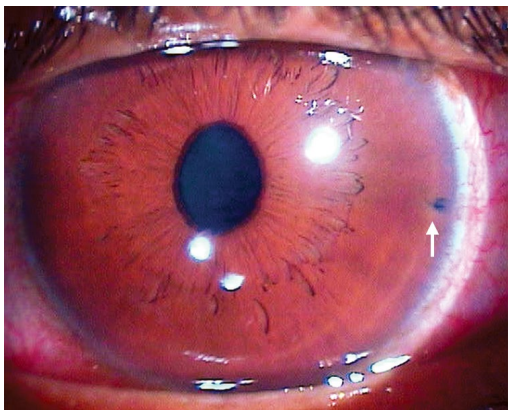


Fig. 16.26: Nd: YAG peripheral iridectomy opening (arrow)

- ***Treatment of the fellow eye:*** The 'fellow' or second eye is also to be reated by Laser iridotomy or a surgical peripheral iridectomy, as soon as possible.
  - ***In chronic congestive stage***
    - Laser iridotomy may be tried initially. If this is unsuccessful, a filtration operation (e.g. trabeculectomy) should be performed.
    - Miotics are not useful at this stage.
  - ***In absolute glaucoma stage***
    - ***Cyclocryotherapy:*** This is to reduce the aqueous secretion and thereby the IOP will reduce.
    - ***Retrobulbar injection of 70% alcohol:*** This is to destroy the ciliary ganglion.
    - If the pain is still unbearable, the eye may be enucleated.

PRIMARY OPEN-ANGLE GLAUCOMA

Primary open-angle glaucoma (POAG) is a chronic, slowly progressive, bilateral raised IOP with an open angle and associated with glaucomatous cupping and visual field loss. It was previously referred to as ***chronic simple glaucoma***.

Etiology

- ***Age:*** Common in 5th and 6th decade.
- ***Sex:*** Equal in both sexes.
- ***Inheritance:*** Probably in a multifactorial manner. In 5–20% cases, POAG runs in family.
- ***Ocular association:*** Examples are high myopia, Fuchs' endothelial dystrophy, retinal venous occlusion, retinal detachment, retinitis pigmentosa, etc.
- ***Systemic association:*** Examples are diabetes mellitus, thyroid disorders, cardiovascular abnormalities, etc.
- ***Corticosteroid responsiveness:*** Individual with POAG or a family history of the disease, are more likely to respond to chronic steroid therapy with a significant rise in IOP.

Pathogenesis

The elevation of the IOP in POAG is due to obstruction of the aqueous outflow. However, the precise mechanism of outflow obstruction is not known.

- ***Electron microscopic picture shows***
  - ***Changes in the trabecular meshwork***
    - Faulty collagen of the trabecular tissues.
    - Proliferation of endothelial lining with thickening of basement membrane.
    - Narrowing of intertrabecular spaces, amorphous material in the juxtacanalicular tissue.
  - ***Collapse of the canal of Schlemm.***
  - ***Sclerosis*** of the intrascleral channels.

  All lead to increased resistance to aqueous outflow.
- ***Immunologically,*** there is increased γ-globulin and plasma cells in the trabecular meshwork. In some patients, positive antinuclear antibody reactions have been reported. These findings support an immunogenic mechanism in POAG.

Symptoms

Because of its insidious onset and silent nature, POAG is usually asymptomatic until it has caused a significant loss of visual field. However, the patient may present with:

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- Painless, progressive loss of vision.
- Mild headache or eyeache.
- Increasing difficulty in near works, and frequent change of presbyopic glasses. This is due to accommodative failure owing to pressure upon the ciliary muscle and its nerve supply.
- A defect in the visual field (by an intelligent patient).

Signs

- Visual acuity may remain good till the late stage.
- Cornea is usually clear.
- Anterior chamber depth is normal.
- Pupillary reaction remains normal until the late stage.
- Increased IOP with a large diurnal variation.
- Cupping of the optic disc.

The diagnosis of POAG, therefore, depends on classical triad of:

- Raised IOP,
- Cupping of the optic disc, and
- Classical visual field defects.

Raised Intraocular Pressure

Patients with POAG frequently show a wider variation in IOP than normal individuals. A single pressure reading of 21 mm Hg or less does not exclude the diagnosis. So, it is necessary to measure the IOP at different times of the day.

Initially, there is a rhythmic swing in IOP, which is an exaggeration of normal diurnal variation. In most patients, the IOP falls during evening (contrary to what happens in angle-closure glaucoma).

- ***Morning rise of tension***—20% of cases.
- ***Afternoon rise of tension***—25% of cases.
- ***Biphasic rise of tension***—55% of cases.

The difference between the 'peak pressure' and the 'base pressure' gradually diminishes, and a permanent elevation is then occurred above 21 mm Hg.

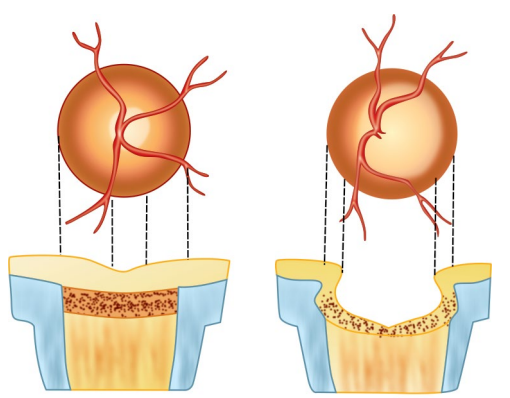
A variation of IOP over 5 mm Hg (Schiotz), should always excite suspicion of glaucoma, even, although the whole reading lies under the limit of 21 mm Hg.

Cupping of the Optic Disc (Figs 16.27A and B)

POAG is usually suspected by finding an abnormal optic disc on routine fundus examination. Documentation of progression or arrest of cupping, together with perimetry, plays a vital role in assessing the efficacy of treatment.

Ophthalmoscopic appearance of normal disc

- Optic nerve head is vertically oval.
- The central portion of the disc contains a depression, the cup and the area of pallor.
- The tissue between the cup-margin and the disc margin is called as ***neural rim***.
- Retinal vessels ride up the nasal wall of the cup often with kinking at the cup margin.
- Most normal eyes have a horizontal ***cup-disc ratio (C:D)*** of 0.3 or less (cup-disc ratio is determined by a ratio between the width of the scleral canal and the volume of glial supporting tissues).
- Large physiological cups (e.g. in myopia or normal subjects) are shallower and do not progress.
- Normal optic nerve heads are characterized by symmetry of the cups and an evenness of the neural rim.



Figs 16.27A and B: Cupping of the optic disc in compare to the normal optic disc

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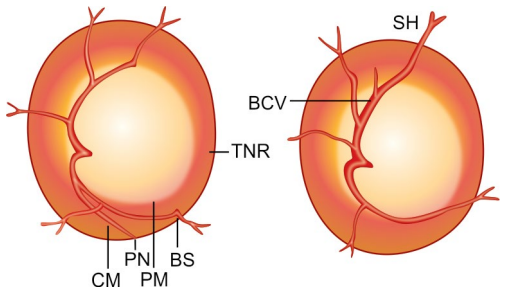


Fig. 16.28: Early disc changes in glaucoma
BS—Bayonetting sign; **CM**—Cup margin; **PM**—Pallor margin; **BCV**—Baring of circumlinear vessels; **SH**—Splinter hemorrhage; **TNR**—Temporal neural rim; **PN**—Polar notching

- ***Glaucomatous disc changes:*** The disc changes of glaucoma are typically progressive and asymmetric, and present in characteristic clinical patterns.
  - ***Early glaucomatous changes (Fig. 16.28):***
    - Cupping usually starts as ***focal enlargement*** at the inferotemporal quadrant, but it may enlarge in concentric circles.
    - ***Bayonetting sign:*** It is the double angulation of the blood vessels, as they pass sharply backwards, and then turn along the steep wall of the excavation before angling again onto the floor of the cup.
    - Area of cupping may progress ahead of the area of pallor.
    - ***Thinning of the neural rim:*** It is mostly on the temporal side. It is actually alterations in the neural rim of an eye with glaucoma that leads to changes in the cup as well as to loss of visual field.
    - ***Nasal shifting*** (Figs 16.29A to H) of retinal blood vessels.
    - ***Splinter hemorrhages*** at the disc margin.
    - ***Baring of circumlinear vessels*** may be seen at the disc margin.
    - ***Progressive enlargement and deepening of the cup*** with loss of neural tissue.
  - ***Advanced glaucomatous changes***
    - ***Total cupping (Fig. 16.30):*** It appears as a white disc with loss of all neural rim, and bending of all retinal vessels at the margin of the disc. This is also called ***bean-pot cupping***, as seen in cross-section (Figs 16.31A and B).
    - ***Total pallor of the disc,*** as there is glaucomatous optic atrophy.
    - Openings of the lamina cribrosa are visible upto the margin of the disc—***laminar dot sign***.
- ***Pathogenesis of cupping and optic atrophy:*** The present evidence suggests that obstruction of the axoplasmic flow with ultimate loss of axons, is involved in the pathogenesis. ***Two factors*** are responsible for this obstruction:
  1. ***Mechanical factor:*** The elevated IOP forces lamina cribrosa backward, and causes death of the neurones by direct compression.
  2. ***Vascular factor:*** Ischemia plays a major role in the obstruction of axoplasmic flow in response of elevated. This leads to atrophy of axons without corresponding increase of supporting glial tissue. Lamina cribrosa becomes weaker and bulges backwards.

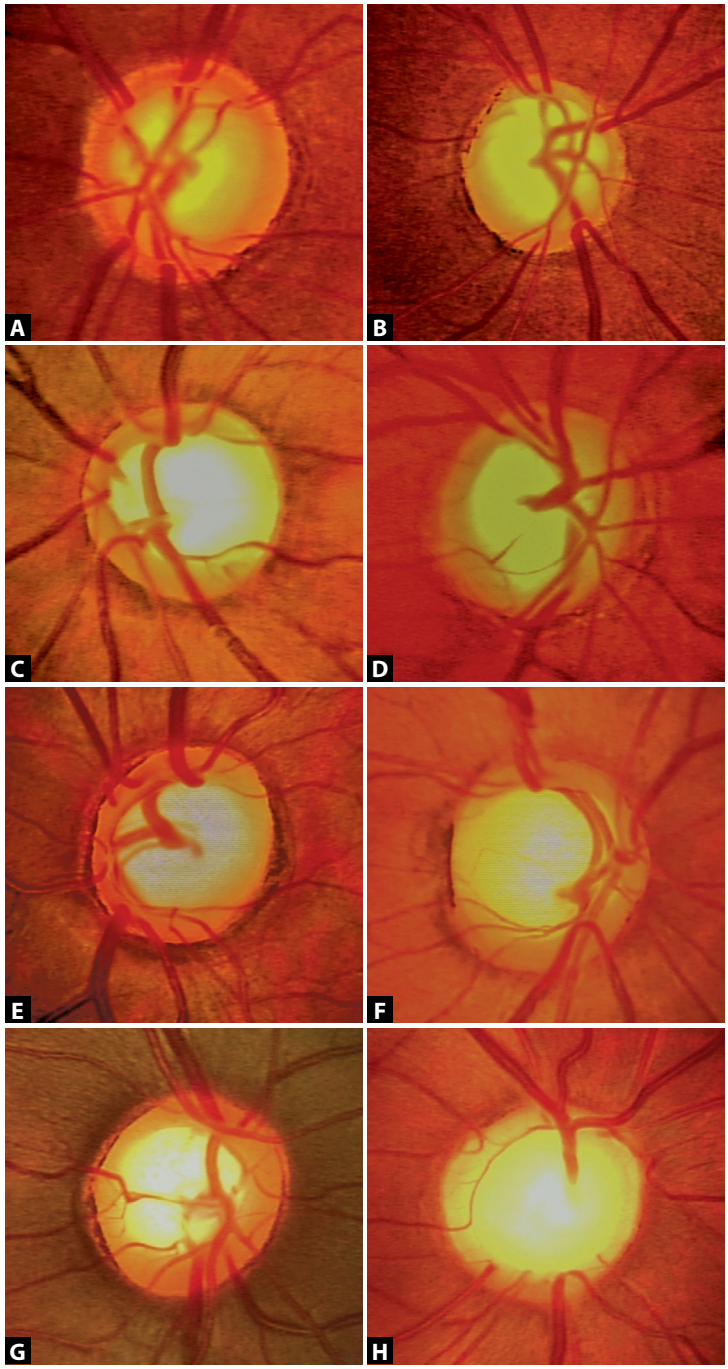
Nerve fiber bundle defects (NFBD)
In some eyes, in early stages, the nerve fiber bundle defects are detected, even prior to the development of cupping and detectable loss of visual field.

The normal nerve fiber bundles appear as fine parallel striations that cross over the larger blood vessels and enter the disc. Visualization of this striation is best by as ophthalmoscope with red-free light. The changes are:

- ***In early glaucomatous damage:*** Some of the striations will be replaced by slit-like defects or grooves, mostly one disc-diameter above and below the disc.
- ***With progression:*** The defects are wedge-shaped and mostly in the lower peripapillary region, and baring of the blood vessels.
- ***In late stage:*** There is total atrophy of the nerve fiber layer and complete baring of the large retinal vessels.

These signs may have prognostic value, since it has been shown to correlate highly with visual field changes, and may precede visual field loss.

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Figs 16.29A to H: Cupping of the disc. **A** and **B.** Normal and asymmetry of the cup size; **C.** Nasal shifting of blood vessels; **D.** Inferior notching; **E.** Bayonetting sign; **F.** Baring of the blood vessels; **G.** Over-passing phenomena, **H.** Thinning of neuroretinal rim

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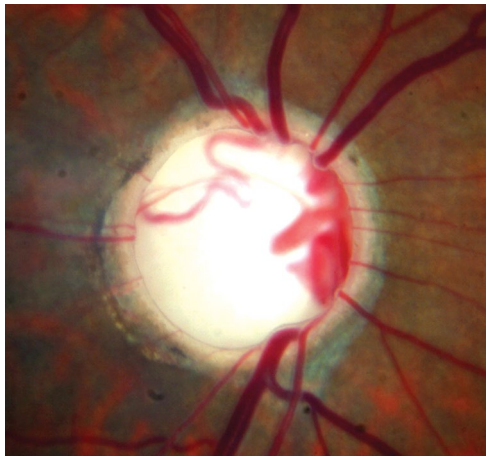
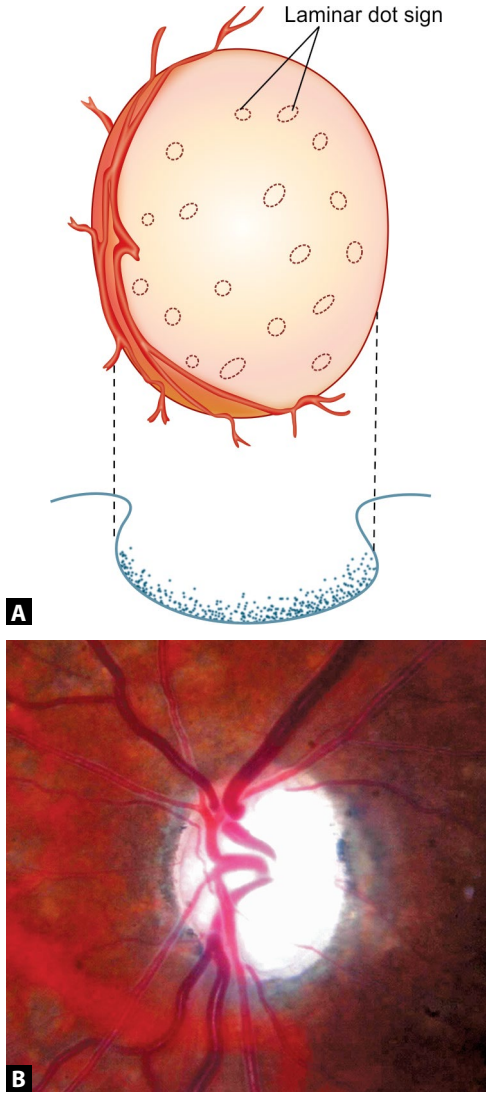


Fig. 16.30: Total cupping of the optic disc



Figs 16.31A and B: A. Bean-pot cupping in cross-section; **B.** Bean-pot cupping of the optic disc

Classical Visual Field Defects

Normal Visual Field

The normal visual field is described by Traquair as ***island of vision surrounded by a sea of blindness***.

- ***Boundary:*** The peripheral limits of the visual field, which normally measures from the fixation points are approximately—60° above and nasally, 70–75° below, and 100–110° temporally.
- ***Point of fixation:*** The area of maximum visual acuity in the normal field, and it corresponds to the foveola of the retina.
- ***Blind spot***: This is an area of absolute scotoma (***non-seeing area***) within the boundaries of normal visual field, and it corresponds to the region of optic nerve head. It is located approximately 15° temporal to the fixation point.

Testing the Visual Field

There are several techniques of testing the field of vision.

- ***Confrontation technique:*** It is a rough, but very useful clinical test which should be applied in every case, if there is any suspicion of a defect in visual field. It is useful in advanced glaucoma (not in early cases) and in neuro-ophthalmological lesions. If any defect is indicated by this method, it must be accurately mapped out and recorded with the perimeter.
- ***Kinetic techniques:*** This involves moving the test object from a non-seeing area, and recording the point at which it is first seen, in

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relation to fixation. ***Two basic types*** of screen are used for kinetic visual field testing.

- ***Perimeters***: The screen may be either an arc or a bowl of a radius of 330 mm.
  - ***Lister's perimeter:*** A rotable arc, capable of being revolved round a pivot, and along which a test object can be moved.
  - ***Goldmann's perimeter:*** A hemispherical bowl, over which a target (a spot of light of adjustable size and illumination) can be directed.

    The Goldmann's perimeter is more standardized, and is preferable for glaucoma examinations, as with this, both the central and peripheral visual fields can be recorded.

    The targets of perimeter consist of circular white disc (or a spot of light) of diameters ranging from 1–10 mm. The ***isopters*** represent the limits of the field of vision with each target, and are accordingly labelled 1/330 (1 mm target at 330 mm distance the radius of perimeter), 2/330, 10/330, etc.

    Colored discs or colored spots of light are exceptionally used for estimation of a differential field loss to color. In normal conditions, ***the blue field is largest***, slightly smaller than the white; then follow the yellow, red and green. The field for blue and yellow is roughly 10° less in each direction than that for white, that for red and green another 10° less.
- ***Tangent screen (Bjerrum screen):*** It is a black felt or flannel, on which central 30° of the visual field can be studied. Estimation of defects of the central field (scotometry or campimetry) is performed with this screen.

  The screen is normally 1 m square or 2 m square, with the patient is seated at 1 m or 2 m from the screen. With the patient fixing on a white button at the center of the screen, the examiner moves a target from the periphery towards fixation until the patient indicates recognition of the target. After plotting the blind spot, the procedure is repeated at various directions around the fixation point until the isopter has been mapped (and labelled as 1/1000, 2/1000, 3/1000, etc.). The blind spot normally covers an area, vertically oval, between 12°–18° temporal to the fixation point, and its edges are sharp and abrupt with any target (small or large).

  Lister perimetry and Bjerrum screen scotometry have the advantages of low cost and simplicity of operation. They can elicit 80% of field defect, especially in advanced glaucoma. However, their values are limited in glaucoma investigation, as they can not detect early glaucomatous field defect, and are non-reproducible (which is essential for glaucoma management).
- ***Static techniques:*** In static perimetry, the visual field can be plotted by using a stationary light target of variable brightness against a background whose luminance may be similarly adjusted (either in photopic or scotopic condition).

  In general, the static perimetry is superior to kinetic methods for glaucoma patients, as it is more accurate and reproducible.

Automated Perimeters

Examples for automated printery are Fieldmaster, Ocuplot, Autofield, Humphrey or Octopus (Fig. 16.32) they are currently being developed which utilize computers to program visual field sequences. They provide exact repeatable tests through a selection of visual field testing procedure. Each of them has an electronic fixation control and an automatic recording of missed points. They are more sensitive than manual perimetry, and always reproducible.

The primary value of automated perimetry at present is to screen the glaucoma patients to detect early field loss, and also for manage-

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Fig. 16.32: Automated perimetry - Humphrey analyzer

ment. It is to be remembered that field loss can not be detected before 35% neuronal loss due to glaucoma.

Anatomical Basis of Field Defects (Figs 16.33A and B)

Retinal nerve fibers are distributed in a characteristic pattern. Most fibers reach the nerve head by a direct route, except those are temporal to the macula, which arch above and below the fovea. These ***arcuate nerve fibers*** do not cross the horizontal line temporal to the fovea. Nasal nerve fibers follow a direct path to the optic nerve head.

The arcuate nerve fibers, related to superior and inferior temporal portion of the optic nerve head, are ***most sensitive*** to glaucomatous damage.

Macular fibers follow as straight course into the optic nerve head, and get affected last with retention of central vision until the advanced stage of the disease.

The Glaucomatous Field Defects

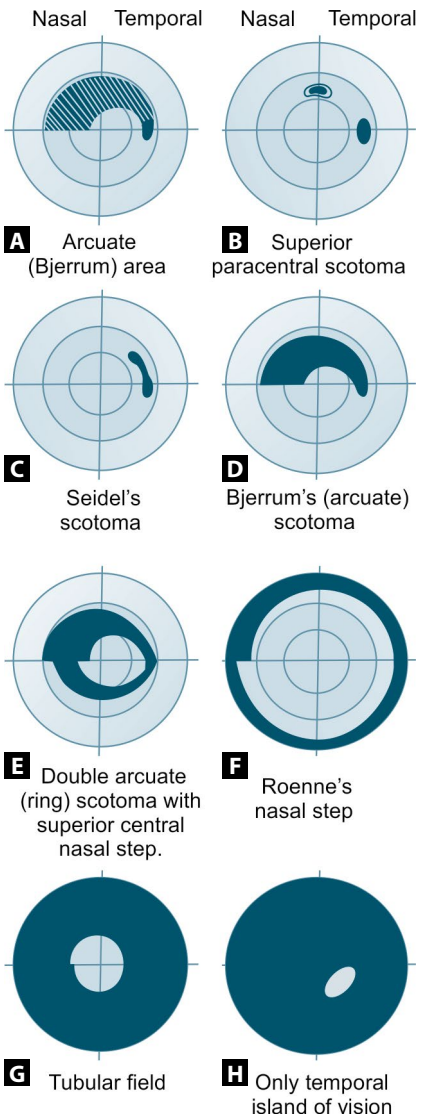
Isopter contraction: Peripheral isopter contraction may be significantly smaller prior to any field loss.

- ***Baring of the blind spot:*** It (exclusion of the blind spot) is also considered to be an early field defect in glaucoma.
- ***Angioscotomata:*** They are long, branching scotomata, above or below the blind spot, which are presumed to be resulted from shadows created by large retinal vessels, and are felt to be an early change.
- ***Isolated paracentral scotomata:*** One or more isolated paracentral scotomata develop in the Bjerrum or arcuate area (Figs 16.34A to H).

A **B**

Figs 16.33A and B: A. The course of the nerve fibers in the retina-showing the fibers involved in field defects A and B, in Figure 16.33B; **B.** Arcuate scotomata in the field of vision corresponding with lesions A and B in Figure 16.33A

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Figs 16.34A to H: Glaucomatous field defects

Arcuate or bjerrum area: An arcuate area above and below the fixation from the blind spot to the median raphe, and corresponds to arcuate nerve fibers.

The nasal extreme may come within 1° of fixation, along the median raphe and extend nasally for 10°–20°.

Defects in superior arcuate area usually occur before those in the inferior area, because of the greater tendency for early glaucomatous cupping in the inferior temporal pole of the optic nerve head.

- ***Seidel's scotoma:*** A sickle-shaped defect arises from the blind spot and tapers to a point in a curved course with concavity towards the fixation point.
- ***Bjerrum's or arcuate scotoma:*** A relatively larger area of defect in the form of arching scotoma, which eventually fills the entire arcuate area, from blind spot to the median raphe. With further progression, a double arcuate (ring or annular) scotomata will develop.

Other causes of ring scotoma: High myopia, aphakic spectacle correction, retinitis pigmentosa and panretinal photocoagulation.

- ***Roenne's nasal step:*** The arcuate defects may not proceed at the same rate in the upper and lower portion of the eye. Consequently, a step-like defect is frequently created where the arcuate defects meet at the median raphe. This is called ***Roenne's nasal step*** and it is mostly superior nasal step, as superior field is involved somewhat more frequently.
- ***Generalized constriction of the peripheral field*** along with double arcuate scotoma leads to tubular field of vision ***(tubular vision)*** in which only central vision remains clear.

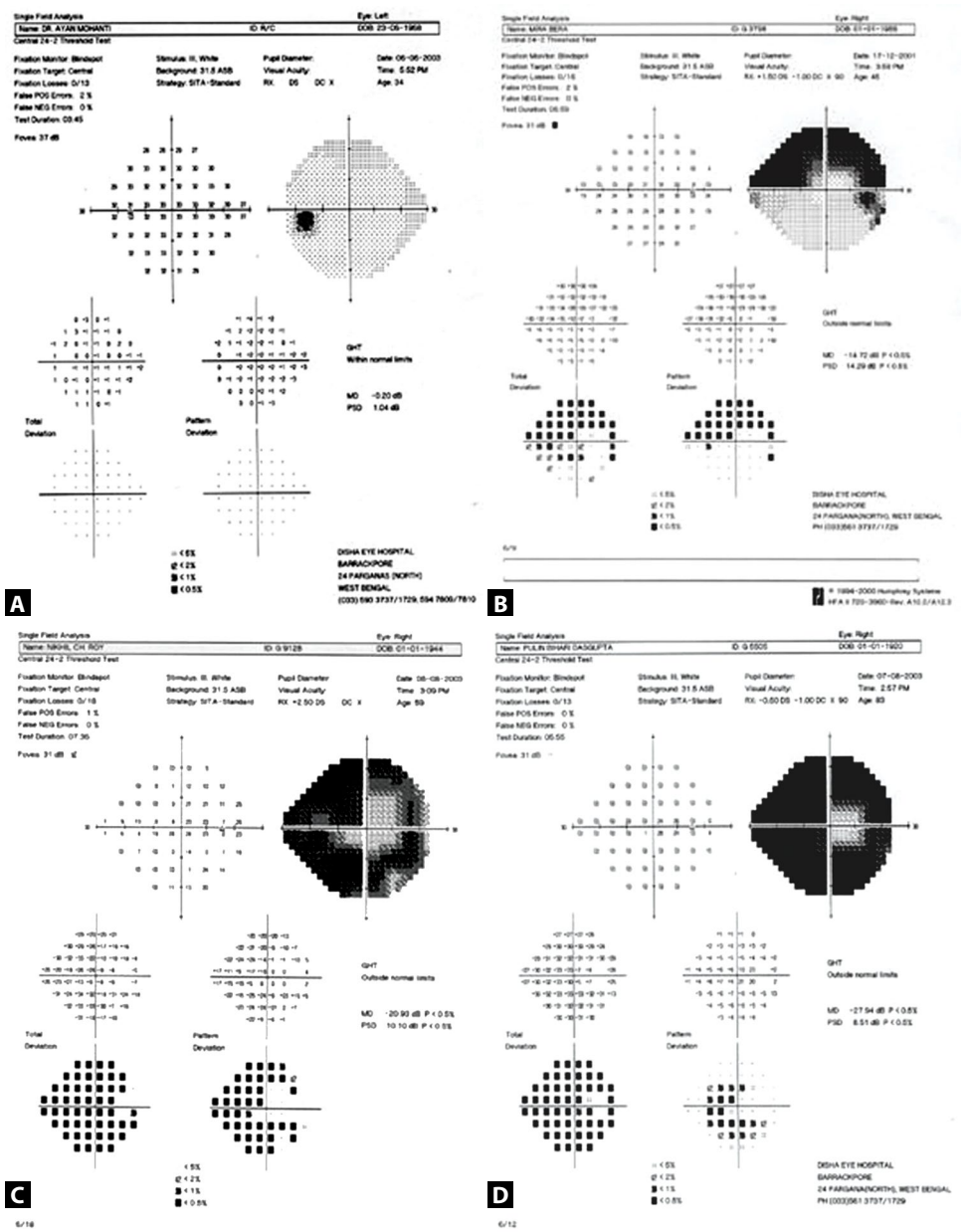
Causes of tubular vision other than glaucoma: Retinitis pigmentosa, high myopia, central retinal artery occlusion with sparing of cilioretinal artery.

- Lastly, only ***a paracentral temporal island of vision*** persists, central vision being abolished.
- Ultimately, all the nerve fibers are eventually destroyed with ***no perception of light.***

Investigations

- ***Diurnal variation*** of IOP.
- ***Gonioscopy:*** By definition, the anterior chamber angle is open, and grossly normal in eyes with POAG.
- ***Tonography:*** To estimate the facility of aqueous outflow, which is reduced in POAG. The POC ratio (more than 100) is

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Figs 16.35A to D: Field changes in glaucoma (Humphrey field analyzer). **A.** Normal field; **B.** Upper arcuate field defect with nasal step; **C.** Double arcuate or ring scotoma with nasal step; **D.** Advanced field defect with only paracentral island of vision

thought to be more sensitive parameter in this situation.

- ***Water drinking provocative test:*** Fasting base line applanation tonometric reading is taken in the morning. The patient is instructed to drink 1 litre of water, following which applanation tonometry is performed every 15 min for 1 hour.

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Schiotz tonometry is unsatisfactory in this test, as water drinking reduces the ocular rigidity.

A rise of 8 mm Hg is said to be significant. Performing tonography after water drinking test has an additional diagnostic value. The mechanism of IOP rise is due to increased aqueous secretion due to reduced serum osmolality.

Other provocative tests, e.g. Jugular vein compression, bulbar pressure test, priscol test, caffeine test, etc. are not used now-a-days.

- ***Slit-lamp examination*** of the anterior segment is to rule out other (secondary) causes of open-angle glaucoma.
- ***Perimetry*** and ***scotometry.***
- ***To detect glaucoma in pre-perimetric stage:*** Newer devices like—nerve fiber layer analyzer, optical coherence tomography (OCT), GDx VCC, etc. are important (*see* Chapter 28).

Treatment

The aim of the treatment is to prevent the field loss which results from too high an IOP. Optic nerve heads vary in susceptibility to pressure damage, so that the danger level of IOP (target pressure) varies from patient to patient.

Treatment requires continued supervision, and it involves periodic tonometry, assessment of cupping (fundus photography is more valuable), and careful mapping of the visual fields.

Modes of Treatment

- Medical therapy
- Laser therapy
- Surgical therapy
- Combination therapy.

Medical Therapy

The treatment of POAG is in the first place, always medical, with operation as a last resort.

Topical Therapy

- ***Pilocarpine nitrate*** (1%, 2%, or 4%) eye drop 3–4 times daily. Two percent solution is commonly used.
  - ***Mechanism in lowering IOP in POAG:*** Pilocarpine causes contraction of the longitudinal muscle of ciliary body → pull on the scleral spur → opens up the trabecular meshwork or Schlemm's canal, or both → increased facility of aqueous outflow → lowering of IOP.

    Miotic effect of pilocarpine is useful in the management of angle-closure glaucoma.
  - Pilocarpine may be administered as Ocusert, gel preparation, or with soaked soft contact lens.

Side effects of pilocarpine

- ***Systemic:*** Increased perspiration, spasms of smooth muscles causing abdominal cramps or bronchospasm, increased glandular secretion, etc.
- ***Ocular:*** Ciliary muscles spasm, leading to browache and induced myopia (more in young patients); miosis may reduce the visual acuity and alter the visual fields, and retinal detachment (by exerting vitreoretinal traction).

Long-acting miotics (like ecothiophate, demecarium bromide, eserine, etc.) are not popularly used now-a-days.

- β-***blockers:*** Timolol maleate, β-Bxolol and Levobunolol.
  - ***Timolol maleate*** (0.25% or 0.50%) eye drops, at 12 hourly interval (twice daily).
    - ***Mechanism of action:*** It is a non-selective β-blocker. It lowers the IOP by reducing the aqueous secretion, as a result of direct action on the secretory epithelium of the ciliary body.
    - It has been seen that IOP responsiveness to timolol will decrease with continued use in some patients, and timolol alone may not sufficient to control IOP after 3 months to 1 year after starting the treatment.

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Side effects of timolol

- ***Systemic:*** Bradycardia, hypotension and arrhythmias due to β_1 blocking effect; bronchospasm (due to β_2 blocking effect); mental depression, sexual impotence, etc. It should not be used in heart block and asthmatic patients.
- ***Ocular:*** Superficial punctate keratitis, corneal anesthesia (due to membrane stabilizing effect) and reduced tear production.

  - ***Betaxolol (0.5%)*** eye drops twice daily. It is cardioselective β_1 blocker and is almost as effective as timolol.
    It has little effects on cardiopulmonary system, and may be used safely in bronchial asthma.
  - ***Levobunolol*** (0.5%) eye drops twice daily. It is as effective as timolol with lesser ocular side effects.

Advantages of β-blockers

- It is equally effective as other antiglaucoma drugs.
- Convenient dose (twice daily).
- Does not alter the pupillary size or accommodation status.
- It can be safely used in hypertensive.
- Assessment of visual field is better as the pupillary size remains unaltered.
- ***Epinephrine*** (0.5%, 1% or 2%) eye drops twice daily, and is useful in young patients.
  - ***Mechanisms of action***
    - It reduces aqueous production due to vasoconstriction (α-adrenergic effect).
    - It increases the aqueous outflow by stimulation of β-receptors in the outflow system (β-adrenergic effect).

- ***Side effects of epinephrine***
  - ***Systemic:*** Increased BP, tachycardia, arrhythmias, tremor, etc. It should not be used in hypertensives.
  - ***Ocular:*** Irritation, reactive hyperemia, conjunctival pigmentation and 'black cornea' (due to deposition of melanin), mydriasis, epinephrine maculopathy (a form of cystoid macular edema seen in aphakic individuals),

Contd…

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discoloration of soft lenses, and may reduce the optic nerve head perfusion.

Dipivefrin or dipivalyl epinephrine (0.1%), a prodrug which is converted into epinephrine, after absorption into the eye. It penetrates the cornea better (17 times), and has fewer systemic side effects and does not discolor soft lens.

- ***Topical carbonic anhydrase inhibitor:*** Dorzolamide (2%) and brinzolamide (1%) ophthalmic solution, 3 times daily. They lower IOP by reducing aqueous production. Most significant side effect is on corneal endothelium.
- ***Brimonidine tartarate (0.15%) eye drop:*** It is a relatively selective α-2 adrenergic agonist. It has a peak ocular hypotensive effect occurring at two hours after instillation.
  - Recommended dose is ***three times daily***.
  - Brimonidine has a ***dual mechanism of action***—(i) by reducing aqueous production and (ii) increasing uveoscleral aqueous outflow.
  - It has minimal or no effect on cardiovascular or pulmonary functions.
  - But, it ***may cause fatigue and/or drowsiness*** in some patients.
- ***Bimatoprost (0.03%) eye drop:*** It is a prostamide, a synthetic prostaglandin analogue with ocular hypotensive effect.
  - Recommended dose is one drop daily in the evening or night time.
  - It decreases IOP by increasing aqueous outflow through both the trabecular meshwork and uveoscleral routes.
  - It dose not have any effect on cardiorespiratory system.
  - ***Major adverse effects are:***
    - Increased pigmentation and ***growth of eyelashes***.
    - Hyperpigmentation of the iris and periocular skin.
- ***Latanoprost (0.005%) or Travoprost (0.004%) eye drop:*** It is a prostaglandin analogue, which increases uveoscleral

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outflow by over 100%. It lowers upto IOP up to 35% from the baseline. The dose is just one drop daily, and side effects are minimal. But these are expensive drugs.

- ***Combination of topical therapy:*** Timolol and pilocarpine, or timolol and epinephrine or timolol and latanoprost are used commonly as combination drugs, when any one of the topical preparations alone fails to control IOP satisfactory.

Systemic Therapy

- ***Carbonic anhydrase inhibitors (CAIs)*** are used orally to control IOP. They are acetazolamide (250 mg), dichlorphenamide (50mg), methazolamide (50 mg) and ethoxzolamide (125 mg). Among these, acetazolamide (250 mg), 1–2 tab 4 times daily is used most popularly.
  - ***Mechanism of action:*** It lowers the IOP by a 50–60% reduction of aqueous production. It acts on secretory epithelium of the ciliary body by creating a local acid environment, as well as by developing metabolic acidosis (***diuretic effect is not a factor in the reduction of IOP)***.
    Pressure lowering effect is additive when it is used along with topical pilocarpine or β-blockers.

Side effects of carbonic anhydrase inhibitors

- ***Systemic:*** Metabolic acidosis, hypokalemia (so a potassium supplement is necessary), gastrointestinal upset, renal calculi, paresthesia (tingling sensation) of finger and toes, and blood dyscrasias.
- ***Ocular:*** transient acute myopia and rarely dry eye (due to Stevens-Johnson syndrome, an effect of sulphur-drug).

Laser Therapy

Argon laser trabeculoplasty (ALT) is indicated when POAG is not well controlled with medical therapy.

- ***Mechanism:*** Burns in the trabecular meshwork for 360° by Argon laser → shrinkage and fibrosis of collagen → opening up of intertrabecular spaces → increase in aqueous outflow. It works better in whites than among blacks.
  It is safe, non-invasive OPD procedure. The average drop in IOP is 8–10 mm Hg.

Surgical Therapy

The indications for surgical therapy are:

- POAG can not be controlled with maximal medical therapy.
- The patient can not tolerate medical treatment, due to toxicity.
- The compliance of the patient is poor, or when the follow-up is unreliable.
- Some surgeons prefer early surgical intervention.

Types of Surgery

- ***Free-filtering (full-thickness fistula) surgery,*** e.g. Scheie's thermosclerostomy, Elliot's sclerocorneal trephining operation, iridenclesis operation, etc. They are no longer practised now-a-days.
- ***Guarded-filtering (partial-thickness fistula) surgery,*** e.g. trabeculectomy, which is routinely practised now-a-days (*see* Chapter 25).
- ***Newer modifications***
  - Trabeculectomy with antimetabolites, e.g. with Mitomycin-C (MMC) or 5-fluorouracil (5-FU). Here, these drugs act as wound modulators because of their antifibroblastic activities. So the chance of fibrosis and postoperative bleb failure is less.
  - ***Glaucoma drainage devices (shunt/valve):*** Examples are Molteno, Kurpin-Denver, baerveldt implant, Ahmed glaucoma valve, or Aurolab aqueous drainage implant (AADI) are designed to divert aqueous humor from anterior chamber to an external reservoir, where a fibrous capsule forms about 4–6 weeks after surgery and regulates flow.

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This is done in recalcitrant cases, like previously failed trabeculectomy and complicated glaucoma, such as uveitic glaucoma, neovascular glaucoma, iridocorneal endothelial (ICE) syndrome, paediatric glaucoma and also in conjunction with keratoprosthesis.

The tube is usually placed in the anterior chamber (rarely through pars plana route). In valved variety, when IOP rises, the valve opens and aqueous drains into the subconjunctival space; but as soon as the IOP decreases, the valve automatically closes.

Combination Therapy

A combination of therapy may be required in some cases of POAG, e.g. trabeculectomy and medical therapy, or Argon laser trabeculoplasty and medical therapy, etc.

Treatment of Ocular Hypertension

- The patients with ocular hypertension should be regarded as ***glaucoma suspect***.
- They must be kept under observation for periodic analysis of the IOP, cup/disc ratio, and field of vision.
- But, when the IOP is more than 30 mm Hg, the patients are to be treated medically with topical timolol or epinephrine.

Treatment of Low-tension Glaucoma

- In this form of POAG, there is no firm evidence that treating the IOP, improves the visual prognosis.
- The most important aspect of the management, is the treatment of any cardiovascular abnormality to ensure maximum perfusion of the optic nerve head.
- Nevertheless, most ophthalmologists try to keep the IOP as low as possible with topical antiglaucoma medication.
- Surgery does not help much, if required, trabeculectomy with MMC is the method of choice.

SECONDARY GLAUCOMA

It means glaucoma secondary to some preexisting or simultaneous ocular pathology.

While the open-angle and angle-closure aspects are useful in classifying the primary glaucoma, but they are of less value with regard to the secondary glaucoma, since many of these may involve more than one mechanism of outflow obstruction, depending upon the stage of the disease.

Common Types

Associated with Lens Disorders

Pseudoexfoliative Glaucoma

Secondary glaucoma with pseudoexfoliation syndrome (Figs 16.36A and B).

- It is due to deposition of fibrillar basement membrane like material, and pigment granules in the trabecular meshwork.
- Characteristic white exfoliative material is seen on the anterior lens capsule in three distinct concentric zone.
- Treatment is same as POAG.

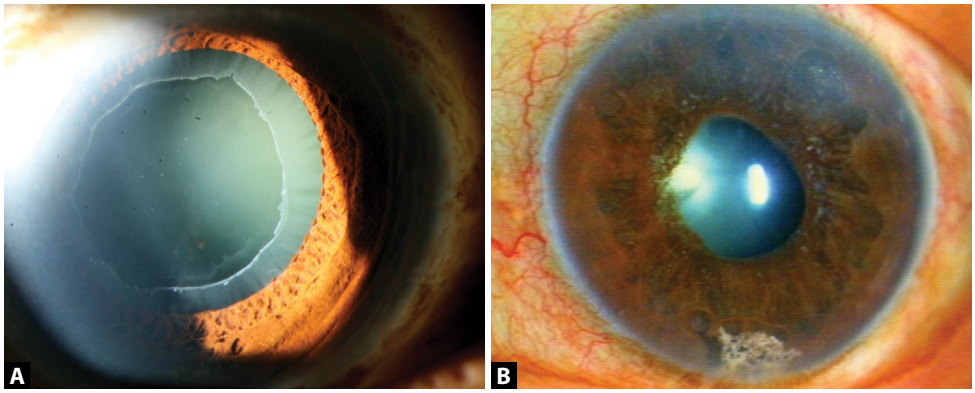
Phacomorphic Glaucoma (Figs 16.37A and B)

- A swollen intumescent cataract (immature or hypermature) may cause secondary angle-closure glaucoma.
- Initial treatment is to reduce acute rise in IOP medically, followed by extraction of cataract with or without IOL implantation.

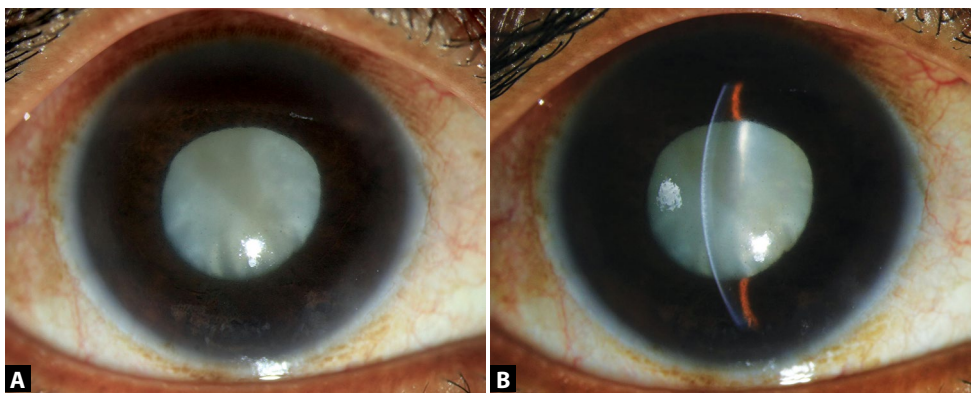
Phacolytic Glaucoma (Fig. 16.38)

- A secondary open-angle glaucoma due to microleak of lens capsule in a hypermature or rarely in a mature cataract.
- IOP-rise is due to obstruction of trabecular meshwork by macrophages which ingest the lens protein.
- Treatment is initially by medical means, followed by cataract extraction.

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Figs 16.36A and B: A. Pseudoexfoliation of lens capsule; **B.** Pseudoexfoliative glaucoma



Figs 16.37A and B: A. Phacomorphic glaucoma—with corneal edema; **B.** Phacomorphic glaucoma—note the shallow anterior chamber



Fig. 16.38: Phacolytic glaucoma—deep anterior chamber with milky cortex

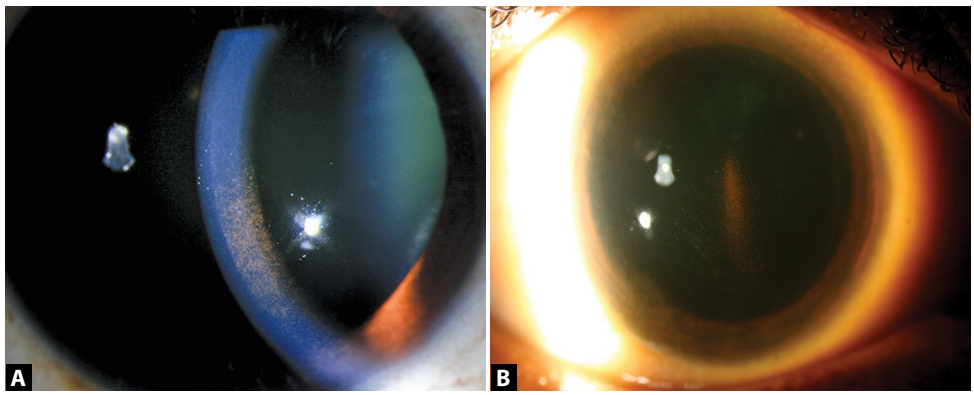
Lens Particle Glaucoma

- It occurs typically following extracapsular cataract extraction, or after penetrating injury of the lens.
- It is due to obstruction of the trabecular meshwork by the lens particles, with associated inflammation.
- Residual lens material should be removed surgically either by irrigation or by vitrectomy (if associated with rupture of the posterior capsule).

Phacoanaphylactic Glaucoma

- Always associated with a latent period, and during that period sensitization to lens protein occurs.

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Figs 16.39A and B: Pigmentary glaucoma—Krukenberg's spindle

- The typical finding is a chronic 'granulomatous' uveitis.
- Corticosteroids therapy along with antiglaucoma medication is required. When medical treatment fails, the retained lens material should be surgically removed.

Associated with Ectopia Lentis

- Secondary glaucoma may develop in congenital ectopia lentis, traumatic or spontaneous lens subluxation and dislocation.
- ***The mechanisms are*** pupillary block, phacolytic glaucoma or angle anomaly with peripheral anterior synechiae.
- Treatment by iridectomy (in pupillary block), or lens extraction (in phacolytic type and when the lens is cataractous).

Pigmentary Glaucoma (Figs 16.39A and B)

- Secondary open-angle galucoma, occurs in a young, myopic male.
- Pigment dispersion occurs throughout the anterior segment, with loss of pigment from the iris pigment epithelium.
- Deposition of pigment on the corneal endothelium in a vertical line called ***Krukenberg's spindle.***
- The glaucoma is due to pigmentary obstruction and subsequent damage to the trabecular meshwork.

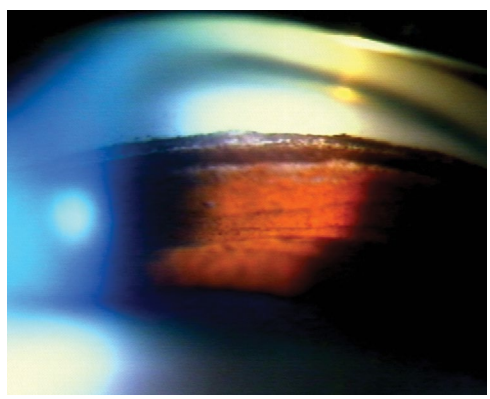


Fig. 16.40: Pigmentary glaucoma—Sampaolesi's line

- Gonioscopy shows, accumulation of pigment along the Schwalbe's line, especially inferiorly, as a dark line—***Sampaolesi's line*** (Fig. 16.40).
- Iris transillumination shows a characteristic radial spoke like pattern.
- Management is same as POAG and the prognosis is good if detected early.

Neovascular Glaucoma

- Secondary glaucoma is associated with neovascularisation of the iris (rubeosis iridis) (Fig. 16.41) and angle of the anterior chamber.
- It is classically seen in:

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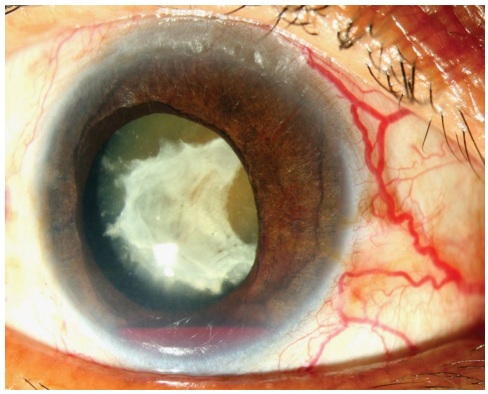


Fig. 16.41: Neovascular glaucoma—rubeosis iridis

  - Diabetic retinopathy
  - Central retinal venous occlusion (CRVO) (as the glaucoma usually develops after 3 months, it is called 90 days glaucoma)
  - Sickle cell retinopathy
  - Caroticocavernous fistula.
- Neovascular glaucoma occurs in three stages:
  1. ***Pre-glaucomatous stage*** (rubeosis iridis).
  2. ***Open-angle glaucoma stage:*** It is due to intense neovascularization at the angle.
  3. ***Angle-closure glaucoma stage:*** It is due to contracture of fibrovascular membrane at the angle leading to goniosynechiae.
- Recurrent hyphema may occur due to hemorrhage from these new vessels.
- Treatment is very difficult
  - ***Prophylactic*** panretinal photocoagulation, panretinal cryocoagulation, or goniophotocoagulation is helpful in the preglaucomatous stage.
  - ***After establishment*** of glaucoma the treatment:
    - Maximal medical therapy
    - Cyclocryotherapy
    - Filtering surgery with antimetabolites or valve implant may be helpful
    - Ultimately, a painful blind eye may require enucleation.

Inflammatory Secondary Glaucoma

The form of ocular inflammation, which most frequently produces secondary glaucoma is iridocyclitis. Other forms of ocular inflammations are keratitis or corneal ulcer, choroiditis, or scleritis.

Associated with Iridocyclitis

- It occurs in all forms of iridocyclitis.
- In acute phase, it is due to plasmoid aqueous; and also due to associated trabeculitis (trabecular edema).
- In healed phase, or in chronic iridocyclitis → it is due to posterior synechiae → iris bombe → goniosynechiae and peripheral anterior synechiae.
- Treatment is usually medical. But some times, it requires surgical intervention, like iridectomy or synechiolysis, or even filtering surgery.

Glaucomatocyclitic Crisis (Hypertensive Uveitis)

- It is known as ***Posner Schlossmann's (PS) syndrome (Fig. 16.42).***
- It is an unilateral disease, of middle-aged patients, which is characterised by recur-

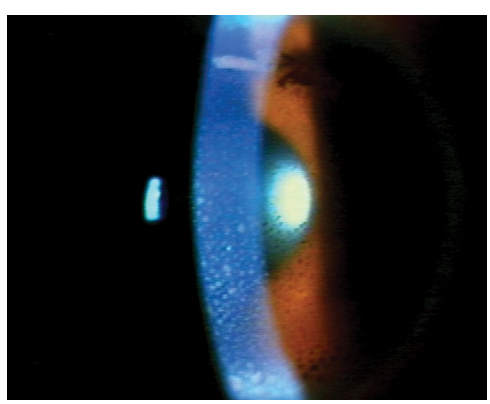


Fig. 16.42: Glaucoma with iridocyclitis—Posner Schlossmann's syndrome

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rent attacks of mild acute iridocyclitis with marked elevation of IOP.

- Patient may experience ***slight ocular discomfort with colored halos.***
- Slit-lamp examination (for cells and KPs), and gonioscopy (open-angle) confirm the diagnosis.
- ***Treatment*** is as acute iridocyclitis, in addition, topical timolol, and tab acetazolamide (***but not pilocarpine***).

Following Perforated Corneal Ulcer

- If a greater part of iris is adherent with the scar, a secondary glaucoma is developed.
- This is due to shallow anterior chamber with angle-closure in the affected quadrant.
- ***Treatment*** is synechiolysis, and if necessary, trabeculectomy operation.

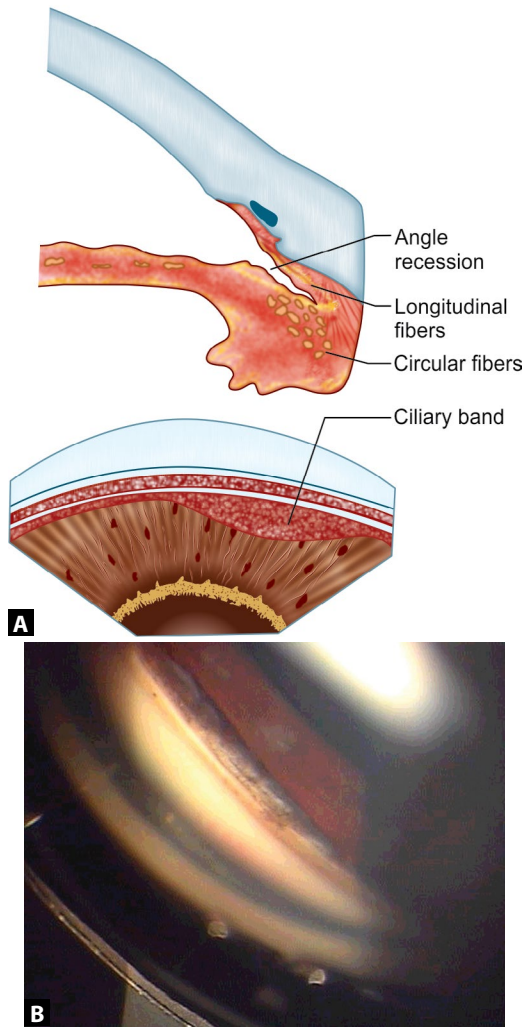
Glaucoma in Keratitis, Scleritis or Choroiditis

It is usually due to associated secondary iridocyclitis which consequently causes posterior synechiae and rise in IOP.

Traumatic Glaucoma

Blunt Injury

- A blunt injury to the eye may cause secondary glaucoma by several mechanisms. In many cases, more than one mechanism are involved. They are:
  - Traumatic iridocyclitis,
  - Hyphema,
  - Massive intraocular hemorrhage,
  - Subluxation or dislocation of the lens,
  - Trabecular damage, and
  - ***Angle-recession (Figs 16.43A and B):*** A late secondary glaucoma occurs with angle-recession. It is due to tear between longitudinal and circular muscles of the ciliary body, and later, by the formation of scar tissue (Descemet's-like membrane) at this area.



Figs 16.43A and B: **A.** Angle recession with irregular widening of the ciliary band; **B.** Angle recession in gonioscope

- ***Treatment*** is medical. Sometimes, surgical intervention is necessary in case of hyphema, trabecular damage, or in angle recession.

Penetrating injury

- The secondary rise in IOP is due to gross disruption of the ocular tissue (e.g. synechiae, hyphema, dislocation or rupture of the lens, inflammations, etc.).

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- ***Siderosis bulbi,*** results from intraocular retention of ferrous metal may also cause secondary glaucoma.

Chemical Injury

- Both alkali and acid burns produce a rapid rise in IOP, due to shrinkage of the outer collagenous layer of the eye.
- This is followed by a return of normal or subnormal pressure; then a slow and sustained rise in IOP occurs, associated with release of prostaglandins.
- ***Treatment*** is by antiglaucoma medication. Miotics are usually avoided as there is associated anterior uveitis.

Glaucoma with Intraocular Tumors

All intraocular tumor (retinoblastoma, malignant melanoma, metastatic carcinoma, dikty-oma, etc.) may cause a secondary glaucoma.
Mechanisms: (not by increased volume alone).

- Obstruction of the angle by seeding of neoplastic tissue.
- Angle-closure glaucoma due to forward displacement of lensiris diaphragm by ciliary body tumor.
- Neovascularization of the angle in eyes with retinoblastoma or melanoma.
- Direct extension of the tumor in the angle.
- ***Melanomalytic glaucoma:*** Due to blockage of trabecular meshwork by melanin containing macrophages (ingestion of necrotic tumor cells by macrophage).

The emphasis of treatment should shift from prevention of blindness to preservation of life.

Steroid-induced Glaucoma

- It is caused by topical, and sometimes, by systemic administration of corticosteroids.
- It occurs more commonly in individuals who have POAG, or a family history of POAG.
- It may occur in chronic (more common) or acute form.
- ***Mechanism:*** It is an open-angle type of secondary glaucoma, due to reduction in facility of aqueous outflow which is:
  - Due to accumulation of polymerized mucopolysaccharides in the trabecular meshwork, and
  - Due to suppressed phagocytic activity of trabecular endothelial cells.
- In general, the antiinflammatory potency of a topical steroid is proportional to its pressure-inducing effect, and βmethasone is most dangerous.
- It is most serious with sub-Tenon or intravitreal injection of Triamcinolone; and also with intravitreal injection of depot dexamethasone (Ozurdex).
- Weak steroids, like medrysone and fluorometholone have least pressure-inducing effect.
- Loteprednol (etabonate), a weak corticosteroid has the least pressure-inducing effect. Enzymatic degradation of loteprednol occurs quickly because of its ester-based formulation (Other corticosteroids have ketone-based formulations which stay prolonged within tissues with good therapeutic effect).
- ***Treatment:*** (i) IOP normalizes with steroid withdrawal and antiglaucoma medication, within 1–4 weeks. (ii) trabeculectomy may be rarely needed.
- ***Prevention*** is by use of weak or dilute steroid (1:10), or non-steroidal antiinflammatory drops in susceptible individuals.

Glaucoma Following Ocular Surgery

Secondary glaucoma may be a complication of any intraocular surgical procedure. The important ones are:

Malignant (Ciliary Block) Glaucoma (Fig. 16.44)

- It includes the following features:
  - Shallowing of both the central and peripheral anterior chamber,
  - Elevated IOP, and

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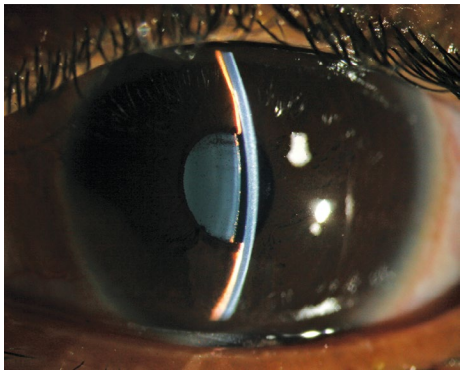


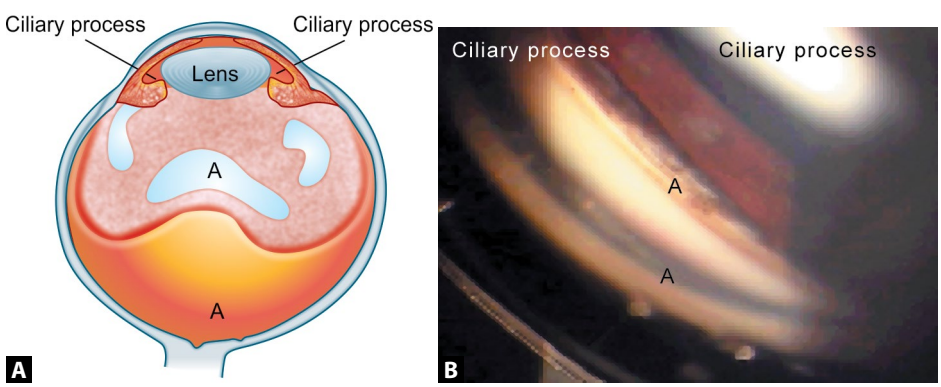
Fig. 16.44: Malignant glaucoma—very shallow (flat) AC following trabeculectomy

  - Poor response to conventional medical therapy.
- It occurs following peripheral iridectomy or filtering surgery (classical malignant glaucoma), and following aphakia or pseudophakia.
- ***Mechanisms:*** It is due to posterior pooling of the aqueous as a result of cyclorotation [ciliolenticular or ciliovitreal block (Figs 16.45A and B)] and anterior hyaloid face obstruction. The aqueous accumulates in the vitreous cavity (vitreous pockets), and further pushes lensiris diaphragm forwards.
- It must be differentiated from pupillary-block glaucoma, where there is moderate depth at the central anterior chamber with peripheral bowing of iris.
- ***Treatment***
  - Mydriatic (phenylephrine) and cycloplegic (atropine) therapy.
  - USG-localization of vitreous pockets, and then posterior sclerotomy with air injection into the anterior chamber.
  - Lens extraction in phakic cases.
  - YAG laser anterior hyaloidotomy.
  - Vitrectomy in aphakic cases.

Glaucoma in Aphakia

There are many mechanisms by which cataract surgery can lead to glaucoma.

- ***In early post-operative period***
  - By distortion of the anterior chamber angle, induced by tight suturing.
  - Delayed reformation of the anterior chamber, and subsequent development of peripheral anterior synechiae.
  - Pupillary-block by air or vitreous, leading to secondary angle-closure glaucoma.
  - Angle is blocked by vitreous or viscoelastic substance itself, leading to secondary acute open-angle glaucoma.



Figs 16.45A and B: A. Concept of ciliolenticular block as the mechanism of malignant glaucoma. Apposition of ciliary processes to the lens equator causes a posterior diversion of aqueous (A) which pools in and behind the vitreous, with a forward shift of the lens-iris diaphragm; **B.** Concept of ciliovitreal block as the mechanism of malignant glaucoma in aphakia. Apposition of ciliary processes against the anterior hyaloid leads to posterior diversion of aqueous (A) which causes a forward shift of the vitreous

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- Hyphema and iridocyclitis.
- ***Enzyme glaucoma:*** By use of α chymotrypsin for zonulysis during ICCE, (not used now-a-days).

- ***In late post-operative period***
  - Epithelial downgrowth
  - Fibrous ingrowth
  - Peripheral anterior synechiae, presumably due to a flat anterior chamber, or iridocyclitis in early post-operative period.

Treatment

- In early period with anti-glaucoma medication, cycloplegics, or laser peripheral iridotomy.
- In late stages, antiglaucoma drugs or conventional trabeculectomy does not help much. Cyclocryotherapy is the treatment of choice in most cases. One may also try for trabeculectomy with antimetabolites.

Glaucoma in Pseudophakia

The specific forms of secondary glaucoma in pseudophakic eyes are due to:

- ***Use of viscoelastic materials (like, hydroxy propyl methyl cellulose (HPMC), or Na hyaluronate):*** It causes a marked elevation of IOP during first few post-operative days.
- ***UGH (uveitis glaucoma hyphema) syndrome:*** It means uveitis, glaucoma and hyphema syndrome, and is associated with anterior chamber IOL implantation.
- ***Pupillary-block glaucoma:*** It occurs more with anterior chamber or iris-supported lens.
- ***Fibrinous iridocyclitis:*** It is particularly in children.
- ***Prolonged use of corticosteroids (steroid-induced):*** It may be an important cause.

Following Penetrating Keratoplasty, Pars Plana Vitrectomy and Retinal Detachment Surgery

- In PK the additional mechanisms are steroid-induced glaucoma, trabecular collapse due to 360° suturing, and flat anterior chamber.
- In detachment surgery, due to tight buckling, congestion and shallowing of the anterior chamber.
- In vitrectomy, the main mechanism is excessive use of intraocular gas, or silicone oil.

Due to Elevated Episcleral Venous Pressure

- The features are—(i) dilatation and tortuosity of episcleral and bulbar conjunctival vessels, (ii) moderate rise of IOP and (iii) open-angle with blood reflux into the Schlemm's canal.
- The causes are—(i) thyroid ophthalmopathy, (ii) superior vena cava syndrome, (iii) carotico-cavernous fistula, (iv) orbital varices and rarely, (v) idiopathic.
- ***Treatment:*** Anti-glaucoma medications with treatment of the cause.

Associated with Intraocular Hemorrhage

With fresh hemorrhage in the anterior chamber (traumatic hyphema)

- The pressure elevation is due to obstruction of the trabecular meshwork by fresh blood, and more with clotted blood.
- If the tension is not relieved, blood-staining of the cornea may result within a few days.
- Paracentesis, or opening of the anterior chamber to remove the clots may be necessary to prevent corneal blood-staining and permanent optic nerve damage.

With long-standing hemorrhage (in vitreous hemorrhage)

- ***Ghost cell glaucoma:*** The degenerated RBCs (less-pliable, spherical, ghost cells) from vitreous cavity enter the anterior chamber where they obstruct the outflow.
- It is a temporary process, and is usually responded by antiglaucoma medication.

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- ***Hemolytic glaucoma:*** Here, the macrophages ingest the contents of RBC in intraocular hemorrhage, and block the trabecular meshwork. It is self-limiting and should be managed medically.
- ***Hemosiderotic glaucoma:*** Iron in the hemoglobin causes siderosis (in massive intraocular hemorrhage) which produces tissue alterations in trabecular meshwork, and cause obstruction to aqueous outflow.

Epidemic Dropsy Glaucoma

- It rarely occurs in an epidemic form, due to consumption of adulterated mustard oil by ***Argemone mexicana.*** The poisonous alkaloid is called ***sanguinarin***.
- It is associated with massive generalized edema, cardiomegaly, widespread capillary dilatation, and rarely peripheral neuritis.
- ***Mechanism:*** Increased capillary permeability of the ciliary epithelium, leads to increased formation of aqueous (***hypersecretion glaucoma***).
- The IOP-rise is bilateral, acute and very high (above 50 mm Hg), and the patient presents with colored halos, eyeache and dimness of vision in both eyes.
- The eyes are non-congested, with normal anterior chamber depth and the angles are open.
- ***Treatment:*** Tab. acetazolamide and topical timolol are very effective in reducing IOP. With general treatment, the IOP usually comes down to normal.

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17 CHAPTER Diseases of the Vitreous

VITREOUS HUMOR

The vitreous humor is an inert biologic gel, which serves only optical function. It has a volume of about 4.5 mL. It comprises two-thirds of the volume of the eyeball and three-fourths of the weight. Although, its viscosity is derived from hyaluronic acid, 99% part of the vitreous humor is water. It possesses no blood vessels in postnatal life and is incapable of becoming inflamed.

Composition of Vitreous Humor

The vitreous humor (80% of the volume of the eyeball) is a delicate, optically clear gel composed of a highly-hydrated double network of protein fibrils and charged polysaccharide chains. By weight, vitreous is composed of ~99% water and 0.9% salts. Protein and polysaccharide components constitute remaining 0.1%.

The composition of vitreous humor:

- Water (~99%),
- Inorganic salts,
- Sugar,
- Ascorbic acid,
- Large molecules of hyaluronic acid,
- Peripheral cells (hyalocytes) and
- A network of collagen fibrils (type II fibers with glycosaminoglycan).

Vitreous humor has a viscosity 2–4 times that of water, giving it a gelatinous consistency and a refractive index of 1.336.

Metabolic exchange between systemic circulation and vitreous humor is very slow. That is the reason why vitreous humor is sometimes the fluid of choice for postmortem analysis of glucose levels or other substances.

Symptoms and Signs of the Diseases of the Vitreous

- ***Three main symptoms***
  1. Decreased vision due to opacities, hemorrhage or membranes.
  2. Floaters (visualization of materials floating in the vitreous).
  3. Flashes of light on ocular movement (photopsia).
- ***Examination of the vitreous***
  - Gross vitreous opacities can be seen with a direct ophthalmoscope.
  - With indirect ophthalmoscopy, opacities are visible against red background of the fundus.
  - Slit-lamp microscope with –40D or 50D contact lens equipped with inclined mirror (three-mirror gonioscope).
- ***Ultrasonography A-scan or B-scan*** to find out the exact anatomical location of the vitreous pathology in presence of hazy media.

Degenerations

Syneresis

- The vitreous humor becomes partially or completely fluid, creating an appearance

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of strands or membranes floating freely in the vitreous.
- It occurs in aging, myopia, and following injury or inflammation of the eyes.
- They are easily visible with the ophthalmoscope or slit-lamp.
- No treatment is available to restore the integrity of the vitreous.

Normal attachment of the vitreous: It is firmly attached in the following regions:
- ***At the vitreous base:*** It is a 3–4 mm wide zone at the ora serrata, where the cortical vitreous is firmly attached to the peripheral retina and pars plana.
- ***At the optic disc margin:*** It is firmly attached.
- ***Around the fovea:*** It is rather a weak attachment. In other areas, the cortical vitreous is attached loosely to the internal limiting membrane of the sensory retina.

VITREOUS DETACHMENT

Vitreous detachment occurs in three forms—(1) posterior, (2) anterior and (3) basal.

Posterior vitreous detachment (Fig. 17.1)
- It is a senile phenomenon and more common in diabetics.
- It appears spontaneously, and produces sudden onset of floaters and photopsiae.
- The photopsiae are more common on the temporal field, and are due to vitreoretinal traction.
- On examination, floaters are seen in front of an optically empty space, adjacent to the posterior retina.
- The condition is benign, unless it is associated with other pathology like retinoschisis, a retinal break or diabetic retinopathy.
- At the onset, the patient must be examined carefully, especially the peripheral retina with an indirect ophthalmoscope.

Anterior and basal vitreous detachment: They occur secondary to blunt trauma, and are accompanied by vitreous hemorrhage.

Fig. 17.1: Vitreous detachment
PVD—Posterior vitreous detachment

VITREOUS OPACITIES

Many foreign substances may be suspended in the vitreous humor—(i) exogenous material, e.g. parasites or foreign bodies, or (ii) endogenous materials, e.g. leukocytes, red blood cells (RBCs), tumor cells, pigments, cholesterol crystals or calcium salts. All opacities cause the symptoms of floaters and some of them may cause severe impairment of vision.

Muscae Volitantes

- These physiologic opacities represent the residues of primitive hyaloid vascular system.
- The patient sees them as fine dots and filaments, often drift in and out of the visual field, against a bright illumination or background (e.g. blue sky).
- The opacities may cause disturbances to sensitive individuals, especially among myopes.
- Correction of ametropia (if required), with reassurance is the only treatment.

Asteroid Hyalosis (Fig. 17.2)

- This is an involutional, mainly binocular phenomenon that affects the aged male patients.

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Fig. 17.2: Asteroid hyalosis

Fig. 17.3: Synchysis scintillans

- They appear as white, round or discoid bodies suspended throughout, or in a portion of the solid vitreous.
- The opacities exhibit slight oscillations in normal vitreous with ocular movements.
- They consist of various calcium containing lipids, and apparently result from the degeneration of vitreous fibrils.
- They do not cause symptoms, or do not require treatment (although they may obscure the view of the fundus).

Synchysis Scintillans (Fig. 17.3)

- It affects the damaged eyes which have suffered trauma or inflammation in the past.
- It is due to deposition of the cholesterol crystals in the liquid vitreous.
- The crystals appear as multicolored glittering particles which settle at the bottom of the vitreous cavity, and can be thrown up by ocular movements to form a golden shower.
- If the lens is removed intracapsular cataract extraction (ICCE), the crystals may be observed both in the anterior chamber and in the vitreous cavity.
- Associated diseased condition may be responsible for poor vision in these eyes.
- No effective treatment is known.

Fig. 17.4: Persistent hyperplastic primary vitreous—posterior type

Persistent Hyperplastic Primary Vitreous (Fig. 17.4)

- The hyaloid vascular system is atrophied by the eighth month of gestation, and sometimes, leaving only minute remnants which are visible as muscae volitantes.
- Failure of the hyaloid artery to regress, combined with the hyperplasia of the posterior portion of vascular meshwork, produces persistent hyperplastic primary vitreous (PHPV).
- It is an unilateral condition and the eye is smaller than the fellow eye due to faulty development. Bilateral cases of PHPV

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may be associated with Patau's syndrome (Trisomy 13) or Norrie's disease.
- A white pupillary reflex is noticed in the full-term infant shortly after birth.
- Associated abnormalities are microphthalmos, cataract, glaucoma, long and extended ciliary processes, and intraocular hemorrhage.
- The anterior chamber is shallow and the retrolental tissue contracts to pull the ciliary processes inwards.
- The cause of cataract is rupture of the posterior lens capsule.
- It must be distinguished from other causes of white pupillary reflex e.g. congenital cataract, retinopathy of prematurity, retinoblastoma, endophthalmitis and retinal dysplasia.
- Lens removal (preferably by pars plana lensectomy with vitrectomy) should be done as early as possible (1 day to 4 weeks of age) to preserve some vision.
- Visual prognosis is often poor due to development of amblyopia.

VITREOUS HEMORRHAGE

Vitreous hemorrhage may occur as a pre-retinal phenomenon, or an intravitreal phenomenon or a combination of both.

Etiology

- ***Retinal break:*** Due to ***trauma*** or by ***vitreous traction*** causing retinal tear, and subsequent rupture of the retinal blood vessels.
- ***Rupture of newly formed blood vessels*** originating from the optic disc and retinal capillaries. The causes are:
  - ***Eales' disease:*** It is the retinal periphlebitis (probably due to hypersensitivity of vessels to tuberculo-protein) and subsequent development of neovascularization. Rupture of these new vessels lead to recurrent vitreous hemorrhage. It occurs in young male patients.
  - ***Retinal venous obstruction:*** Usually after 3 months of occlusion, capillary microaneurysm and new vessels formation occur, and vitreous hemorrhage may arise from these delicate new vessels.
  - ***Proliferative diabetic retinopathy:*** It may be due to rupture of fine new vessel arising from the disc (NVD) or from elsewhere (NVE) (Fig. 17.5).
  - ***Sickle-cell retinopathy:*** Sea-fan neovascularization occurs, particularly at the superior temporal peripheral areas of the retina.
  - Retinopathy of prematurity (ROP).
- ***Operative:*** Following retinocryopexy, retinal detachment surgery or vitrectomy (rebleeding).
- ***Terson's syndrome:*** Rupture of a subhyaloid hemorrhage secondary to subarachnoid hemorrhage.

Fig. 17.5: Vitreous hemorrhage from neovascularization at the disc (NVD)

Symptoms

- Sudden onset of floaters to significant loss of vision.
- Blurring of vision with a red haze.

Signs

- In mild cases, there may not be any sign ophthalmoscopically.
- In moderate to severe cases, the fundus can not be visualized with an ophthalmoscope.

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Indirect ophthalmoscopy may be helpful in moderate vitreous hemorrhage.
- No fundal glow is visible by distant direct retinoscopy with a plain mirror.
- With slit-lamp, a reddish mass may be visible in the anterior vitreous.

Fate of Vitreous Hemorrhage

- The blood (small or massive) may be evenly distributed throughout the vitreous humor, localized or distributed in sheets.
- Absorption of blood usually takes place without organization and the vitreous becomes clear within 4–8 weeks. In a young person resorption may be rapid.
- Organization of hemorrhage does occur in persistent or recurrent bleeding, and is followed by formation of yellow or white debris and fibrous membrane in the vitreous (Fig. 17.6).
- Ultimately, retinitis proliferens develops, which is more common near the optic disc from which the fibrous strands stretch forwards.
- This fibrous tissue contracts, and leads to a tractional retinal detachment.

Investigations

- Blood pressure
- Routine hemogram
- Estimation of blood sugar
- Tests for tuberculosis
- ***Ultrasonography*** B-scan is helpful to establish the diagnosis, as well as to rule out any associated retinal pathology (e.g. retinal detachment)
- ***Macular function tests*** before surgery.

Treatment

Medical
- Bed rest with head elevation and eyes are padded, so as to minimize the dispersion of blood.
- Patient is periodically followed up and indirect ophthalmoscopy is done whenever possible.

Surgical: If the hemorrhage remains unabsorbed even after 6 months, a closed pars plana vitrectomy with laser photocoagulation (endolaser) should be considered.

Fellow eye: The fellow eye should be examined thoroughly and treated actively.

Prevention: Whenever a neovascularization is diagnosed, as in proliferative diabetic retinopathy, retinal venous occlusion or sickle cell retinopathy, laser photocoagulation should be done so that the new vessels are regressed.

PROLAPSE (LOSS) OF THE VITREOUS (FIG. 17.7)

- After removal of the intact lens (ICCE), or rupture of the posterior lens capsule [in extracapsular cataract extraction (ECCE)], the anterior vitreous may herniate into the anterior chamber to fill it completely.
- The consequences that result from operative loss of vitreous, are not at all related to the loss of vitreous bulk itself, but to the morphologic changes those occur in the vitreous body.

The complications of vitreous prolapse (loss) are related to one of the following mechanisms:

Fig. 17.6: Organized vitreous hemorrhage

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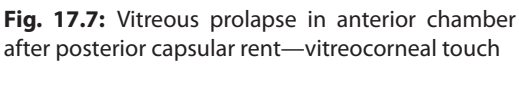


Fig. 17.7: Vitreous prolapse in anterior chamber after posterior capsular rent—vitreocorneal touch

- Direct contact of the vitreous with other structures (such as the cornea or the angle structures).
- Incarceration of the vitreous into the operative wound.
- Fibroplasia of the residual vitreous.
- Inflammation.

The list of consequences is as follows:

- Excessive degree of astigmatism
- Bullous keratopathy due to damage of the corneal endothelium
- Epithelial and fibrous ingrowth
- Secondary glaucoma
- Updrawn or hammock pupil
- Wound infection and endophthalmitis (Vitreous-Wick syndrome)
- Vitreous opacities and formation of tractional bands
- Cystoid macular edema
- Tractional retinal detachment
- Chronic ocular irritability
- In case of posterior chamber intraocular lens (PCIOL) implantation the lens can not be placed properly and the implantation is abandoned, due to fear of further problems.

Treatment

Therapeutic

- Vitreous loss at the time of surgery is treated by means of an anterior (open sky) vitrectomy to ensure that no vitreous is between the wound edges and no traction upon the vitreous base.
- A sector iridectomy is usually done, and this may be combined with inferior sphincterotomy.

Preventive

- Readjustment of the preoperative medication and choice of anesthesia.
- Surgery on a soft eye which is achieved by hyperosmotic agents (e.g. injection mannitol) and bulbar massage after giving peribulbar block.
- Satisfactory anesthesia and akinesia.
- Reduction of external pressure on the globe.
- Use Flieringa's ring to prevent scleral collapse in selected cornea grafting cases.
- Microscopic visualization and proper instrumentation are necessary in ECCE.

VITRECTOMY

Removal (excision) and replacement of the vitreous is known as ***vitrectomy***.

Indications for vitrectomy

- ***Persistent vitreous opacities***
  - Hemorrhage
  - Preretinal membranes
  - Vitreous membrane and strands.
- ***Complications of cataract extraction***
  - Vitreous touch with bullous keratopathy
  - Malignant glaucoma
  - Loss of vitreous (posterior capsular tear)
  - Incarceration of vitreous in wound with traction
  - Removal of dropped nucleus or intraocular lens from the vitreous cavity.
- ***Endophthalmitis with vitreous abscess***
- ***Trauma***
  - Removal of intraocular foreign body
  - Removal of subluxated or dislocated lens.
- ***Complicated retinal detachment***
  - Giant retinal tear
  - Retinal dialysis
  - Massive vitreous traction.
- ***Congenital cataract and ectopia lentis***
- ***Persistent hyperplastic primary vitreous.***

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Types

There are two types of vitrectomy—(1) open-sky vitrectomy and (2) pars plana (closed) vitrectomy.

Open-sky Vitrectomy

It is performed through a large corneal or limbal section. It is necessary to remove the lens first. It is especially indicated in:

- Vitreous loss occurred during cataract extraction
- Aphakic keratoplasty
- Removal of dislocated or subluxated lens
- Anterior chamber reconstruction after trauma.

Pars Plana (Closed) Vitrectomy

It is employed to restore the optical pathway of the eye. If the opacities are confined to the vitreous cavity the prognosis is good.

Vitrectomy is done through a 1–3 mm incision via the pars plana route, called pars plana vitrectomy (PPV). As the vitreous is removed from the eyeball by cutting and suction, physiologic solution is infused into the eye to balance the suction.

An operative microscope with coaxial illumination and fine sophisticated instruments are necessary for vitrectomy.

Now-a-days, 20G pars plana vitrectomy is commonly used by the vitreoretinal surgeons. It requires conjuctiva and Tenon's dissection and sclerotomy wound suturing. Modern 23G, 25G or 27G PPV is transconjunctival sutureless procedure.

Vitreous Substitutes

The vitreous replacement is necessary for:

- Restoration of intraocular pressure, and
- Repositioning the retina in different vitreoretinal surgeries.

The vitreous substitutes are as follows:

- ***Air:*** As an internal tamponade.
- ***Physiological solutions***, e.g. Ringer's lactate and balanced salt solution (BSS).
- ***Gases:*** They are better than air and give longer period of internal tamponade, e.g. sulphur hexafluoride (SF_6) and octafluoropropane (C_3F_8). They are used as a 40% mixture with air.
- ***Silicone oil***
- ***Sodium hyaluronate***
- ***Donor vitreous:*** As it is completely inert and acceptable to the recipient eye.
- ***Perfluorocarbon liquid (PFCL):*** It is heavy and mainly used to assist removal of dislocated lens nucleus or IOL from the vitreous cavity.

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18 CHAPTER Diseases of the Retina

SYMPTOMS AND SIGNS

- Diminution of vision without pain.
- ***Night blindness:*** In diseases those predominantly affect the function of rods.
- ***Peripheral constriction of the visual field*** or ***scotoma*** may be present corresponding with the areas, especially affected.
- ***Metamorphopsia (distorted images) micropsia*** (***small images), or macropsia (large images):*** It may be present in macular lesions.
- ***Photopsia*** (sparks or lightning flashes) occurs due to traction upon the retina.
- ***Frequently***, the patient is unaware of symptoms, and the abnormality is only detected during routine ophthalmoscopic examination.

OPHTHALMOSCOPIC FINDINGS

- ***Disturbances of the blood vessels:*** Examples are vascular pulsation, venous dilatation, arteriovenous ratio and arteriovenous crossing changes, any abnormal vascular reflex, neovascularization (NVD, and/or elsewhere—NVE), of disc hemorrhages (superficial or deep), and microaneurysms.
- ***Opacities in the sensory retina***: Examples are soft exudates (cotton-wool patches), hard exudates, hemorrhages, edema residues and glial tissue proliferation.
- ***Disturbances in the attachment of the sensory retina:*** Examples are rhegmatogenous retinal detachment (with a retinal break) and non-rhegmatogenous retinal detachment (without a retinal break).
- ***Disturbances in the retinal pigment epithelium (RPE):*** Examples are decreased or increased pigmentation (dark areas).
- ***Abnormalities of Bruch's membrane:*** Examples are breaks in Bruch's membrane with replacement by fibrous tissue, as in angioid streak and drusen. Both of these conditions favour the formation of subretinal neovascular membrane.
- ***Peripheral retinal lesions***, are best seen with an indirect ophthalmoscope, or Goldmann's three mirror gonioscope by a slit-lamp.
- Appearance of the normal fundus as seen with the direct ophthalmoscope (Fig. 18.1).

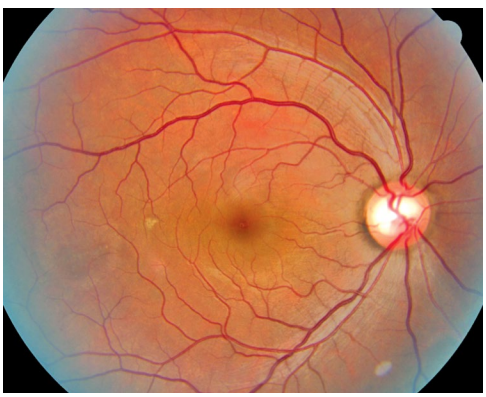


Fig. 18.1: Normal fundus

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EXAMINATION OF THE FUNDUS

- ***Examination with a retinoscopic plane mirror from 1 meter distance:*** To know the fundal glow and refractive status of the patient's eye.
- ***Examination by an ophthalmoscope or a plane mirror at 22 cm distance (distant direct ophthalmoscopy):*** Opacities in the anterior segment can be readily visualized.
- ***Direct ophthalmoscopy:*** The details of central retina are examined under higher magnification (15 times). Direct and erect image as uniocular.
- ***Indirect ophthalmoscopy:*** With this upto the peripheral portion of retina is visualized. Magnification is 3–5 times. But the image is real and inverted. Depth of lesion is better judged as it is binocular.
- ***Slit-lamp*** and special lenses:
  - Hruby lens
  - Fundus contact lens
  - Three-mirror contact lens
  - +90 D lens.

Fundus examination includes:

- ***Fundal glow:*** Good, poor or absent.
- ***Optic disc:*** Margin, color, size, shape, cup-disc ratio, neural rim, venous pulsation, any abnormal vessels or lesions.
- ***Retinal blood vessels:*** Vascular reflexes, A-V ratio, A-V crossing, any abnormal or new vessels.
- ***Macula and foveal reflex:*** Any abnormality, i.e. cyst, hole, hemorrhages, edema, scar, etc.
- ***General fundus:*** Any abnormality, e.g. exudates, hemorrhage, pigmentation, scar, new vessels, detached area, coloboma, folds, etc.
- ***Peripheral fundus:*** It is (best seen by an indirect ophthalmoscope) for retinal break (hole/tear) degenerated area, pigmentation, exudation, etc.
- ***Choroidal blood vessels:*** Normally not visible, but in case of sclerosis they are visible as ribbons.

METHODS OF OPHTHALMOSCOPY

There are two forms of ophthalmoscopy—(1) ***Direct*** and (2) ***indirect***.

Direct Ophthalmoscopy

The hand-held direct ophthalmoscope is designed to provide a direct magnified (×15) view of the fundus. The source of illumination is projected by means of a mirror or prism coinciding with the observer's line of vision through the aperture.

- The ophthalmoscope is held close to the observer's eye and approximately 15 cm from the patient's eye.
- It is held in the right hand to examine patient's right eye and in the left hand to examine patient's left eye.
- The observer uses his/her right eye to examine patient's right eye and left eye for patient's left eye.
- The patient should fix on a distant target with the eye as steady as possible.
- The observer have to adjust ophthalmoscope power setting to accommodate for the patient's refractive error and/or his/her own.
- If both patient and observer are emmetropic, the lens is set at zero. A red reflex will be seen and is considered normal.
- Moving the ophthalmoscope as close to the patient's eye as possible and using the plus or minus lens, the observer will able to see central retina in details upto the equator.
- Lens setting at more plus power will focus the ophthalmoscope in the vitreous or more anterior.

Indirect Ophthalmoscopy

This technique is generally used by retina specialist and involves the use of a head mounted, prism directed light source coupled

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with use of a high condensing lens (+14, +20 or +30D) to see the retinal image. A +20D condensing lens is more commonly used.

The retinal image formed by indirect ophthalmoscopy is inverted, real and capable of being seen at the focal plane of the lens.

The image is brilliantly illuminated, binocular stereoscopic one that covers approximately 10 times of the area compare to direct ophthalmoscope. The image is, however, smaller (×3 times). It is essential to see retinal periphery to locate different types of lesions. Because of its stronger illumination, it allows light to pass through moderate opacities in the media which are obstructive to direct ophthalmoscope.

CONGENITAL AND DEVELOPMENTAL ABNORMALITIES

Myelinated Optic Nerve Fibers (Fig. 18.2)

- The myelin sheaths of the optic nerve fibers cease normally at the lamina cribrosa.
- Sometimes, this myelination extends a short distance over the retinal surface adjacent to the disc.
- ***Ophthalmoscopically***, they appear as white patches with radial striations at peripheral edges. Usually the patches are continuous with the disc, but occasionally they are isolated. The retinal vessels are covered at places by the opaque fibers.
- When present, the blind spot is often enlarged, or a scotoma corresponds with the position of the patch.
- They must be differentiated from soft exudates, as seen in hypertensive retinopathy, and from papilledema.
- The myelin sheaths disappear in optic atrophy (due to any cause) and no trace of abnormality remains.
- No treatment is required.

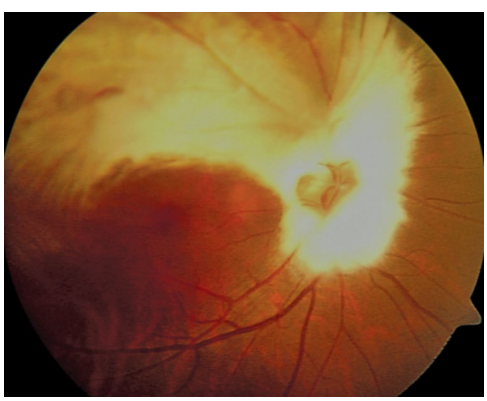


Fig. 18.2: Myelinated optic nerve

Phakomatosis

This is a group of conditions (hamartomas) in which there are congenital, disseminated, usually benign tumors of the blood vessels or neural tissues. They are often ocular, cutaneous, and intracranial in location.

The variety of conditions are:

- Neurofibromatosis ***(von Recklinghausen)***
- Tuberous sclerosis ***(Bourneville)***
- Angiomatosis retinae with cerebellar hemangioblastoma ***(von Hippel-Lindau)***
- Encephalotrigeminal angiomatosis ***(Sturge-Weber)***
- Ataxia telengiectasia ***(Louis-Bar)***
- Encephalo-retino-facial angiomatosis ***(Wyburn Mason)***.

Neurofibromatosis (von Recklinghausen's Disease)

- It is the most common type, with typical subcutaneous nodules and ***cafe-au-lait spots***.
  Ocular manifestations: Plexiform tumors of lids with ptosis, thickened corneal nerves, Leish's spot (Fig. 18.3) on the iris pulsating proptosis (due to transmitted cerebral pulsations through the defects in the orbital walls), glioma of the optic nerve, and congenital glaucoma.
- Operative measures are seldom satisfactory.

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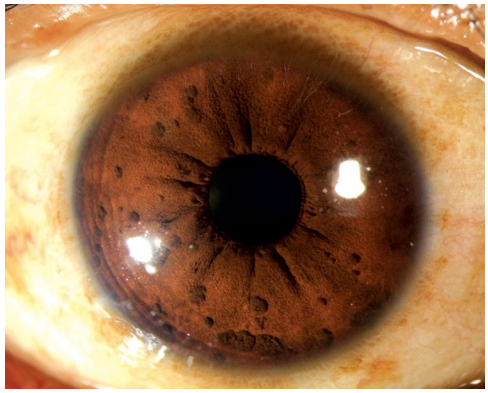


Fig. 18.3: Leish's nodules in neurofibromatosis

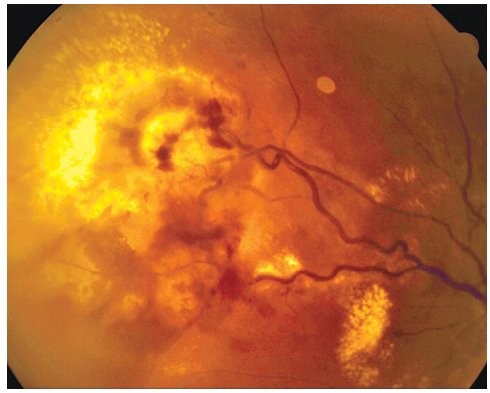


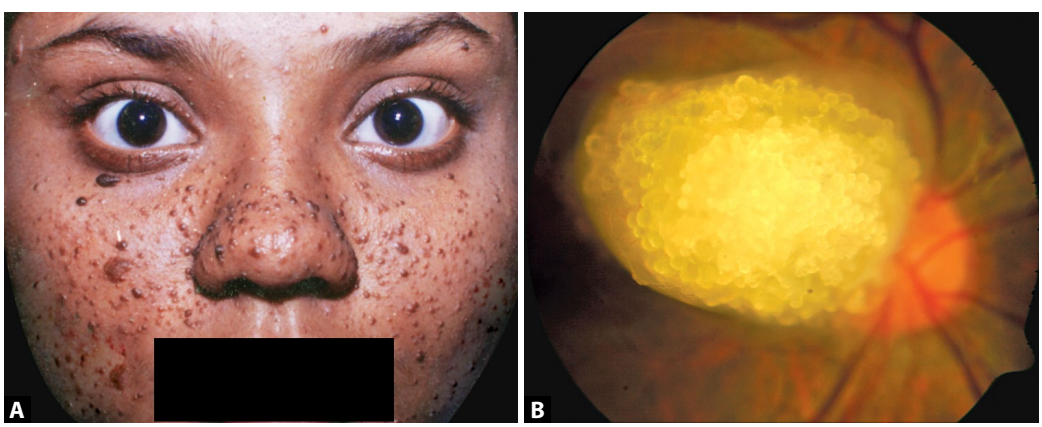
Fig. 18.5: Angiomatosis retinae

Tuberous Sclerosis (Figs 18.4A and B) (Bourneville's Disease)

- The diagnostic triad are epilepsy, mental retardation and adenoma sebaceum (angiofibroma). It is also called *epiloia*-***epi*** (epilepsy)-***loi*** (low IQ)-***a*** (adenoma sebaceum).
- ***Ocular lesion:*** Multiple nodular tumors are seen springing from the optic disc, like white mulberries (astrocytoma).
- Glial hamartomas of the cerebrum result in seizures, and acoustic nerve is most frequently involved.

Angiomatosis Retinae (Fig. 18.5) (von Hippel-Lindau's Disease)

- It is classically seen in the retina and in the cerebellum.
- ***Ophthalmoscopically:*** It is a reddish, slightly elevated tumor is seen in the retina, which is nourished by dilated large retinal artery and vein. The retina becomes spattered with hemorrhages and exudates, ultimately leading to vitreous hemorrhage, retinal detachment and secondary glaucoma.
- When localized, the secondary complications can be prevented by photocoagulation.



Figs 18.4A and B: Tuberous sclerosis. **A.** Adenoma sebaceum; **B.** Astrocytoma of the optic disc

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Fig. 18.6: Sturge-Weber's syndrome

Encephalotrigeminal Angiomatosis (Sturge-Weber's Syndrome) (Fig. 18.6)

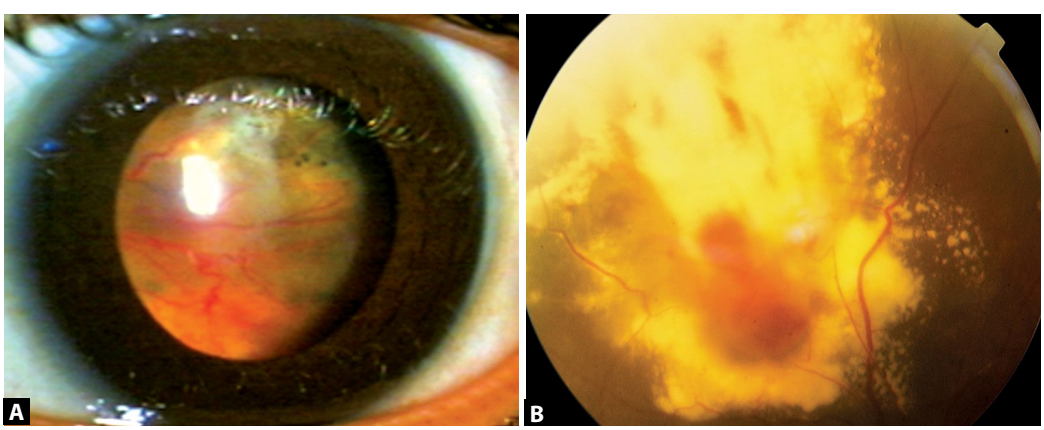
- There is ***nevus flammeus*** (Port-Wine stain) along the distribution of the trigeminal nerve of the affected side.
- There is an associated ***choroidal hemangioma*** which may result in a congenital or childhood glaucoma.
- Focal seizures and mental retardation are also common.
- A characteristic X-ray finding is cortical calcifications which appear as double densities (***train-tract*** or ***rail-road*** sign).

Coats' Disease (Figs 18.7A and B)

- This is a chronic, progressive, vascular abnormality in which telangiectatic retinal vessels leak fluid, which results in an exudative bullous retinal detachment.
- It usually affects boys between 18 months and 18 years of age.
- It is usually unilateral, but both eyes may also be affected.
- The main symptoms are decreased visual acuity and a ***white pupillary reflex***.
- ***Ophthalmoscopically,*** yellowish-white exudative patches are seen behind the retinal blood vessels. The blood vessels may have a tortuous course with aneurysms, fusiform dilatations, and loop formations. Hemorrhage in the vitreous may occur subsequently. Eventually, there may be detachment of retina with iritis, cataract and glaucoma.
- Treatment is frequently ineffective in established cases, but early photocoagulation or cryotherapy is helpful.

RETINOPATHY OF PREMATURITY (FIG. 18.8)

- It is a bilateral abnormality with retinal neovascularization.



Figs 18.7A and B: Coats' disease. **A.** White pupillary reflex; **B.** Fundus picture

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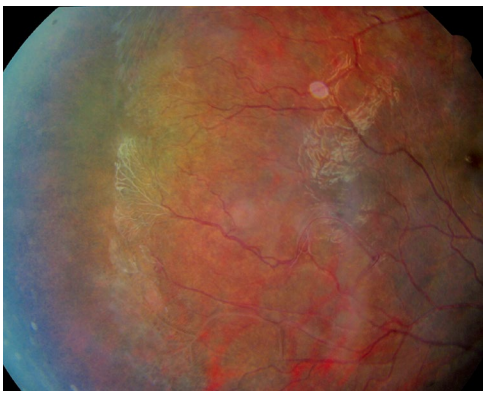


Fig. 18.8: Retinopathy of prematurity

- It occurs some weeks after birth, in premature infants (less than 1500 gm birth weight), who have been given high concentration of oxygen during the first 10 days of life.
- The earliest signs are dilatation of the retinal veins, and the appearance of hazy white patches in the peripheral retina.
- This is due to neovascularization in the retina itself which hubs into the vitreous.
- This is followed by the development of fibrous tissue which eventually proliferates to form a continuous mass behind the lens, appearing as a type of pseudoretinoblastoma (pseudoglioma).
- In many cases the retina gets detached, and of course, the vision is lost.

Pathogenesis

- Retinal vascularization is only completed by the end of full-term gestation, and the retina anterior to the equator (especially that of the upper temporal quadrant) being the last to develop its vessels.
- In prematures, these immature vessels may be occluded by high oxygen concentration. On return to normal atmospheric air, there is intense new vessels formation at the border of these ischemic areas.
- Milder forms of disorder result in tractional bands in the upper temporal quadrant, producing an ectopic macula or dragged optic disc.

Contd…

Contd…

ROP stages: To describe the fundus findings at the junction between the vascularized and avascular retina.

- Stage 1 = a faint demarcation line
- Stage 2 = an elevated ridge
- Stage 3 = extraretinal fibrovascular tissue
- Stage 4 = subtotal retinal detachment
- Stage 5 = total retinal detachment
- In addition, ***plus disease*** may be present at any stage. It describes a significant level of vascular dilation and tortuosity observed at the posterior retinal vessels.

Treatment

- In 80% of infants, the retinopathy of prematurity will regress spontaneously.
- Treatment is required, only when the definite progressive lesion are noted in the vitreous:
  - Photocoagulation or cryotherapy to ablate the avascular immature retina.
  - Scleral buckling for retinal detachment.
  - ***Vitamin-E*** (100 mg/kg/day) may protect the susceptible infants.

Prevention

- Monitor arterial (umbilical) PaO_2 levels during treatment of asphyxia neonatorum, and it should be preferably between 50–100 mm Hg.
- Premature infants (less than 32 weeks) or weighing less than 1500 gm, who have received oxygen therapy, should be screened for retinopathy of prematurity (ROP).
- Once the signs of retinopathy develop, the child should be reexamined every 3 to 4 months upto the age of 4 years, to learn the progress of the disease.

RETINAL VASCULAR DISORDERS

Retinal Artery Occlusion

The occlusion may affect the central retinal artery occlusion (CRAO) (Fig. 18.9) itself,

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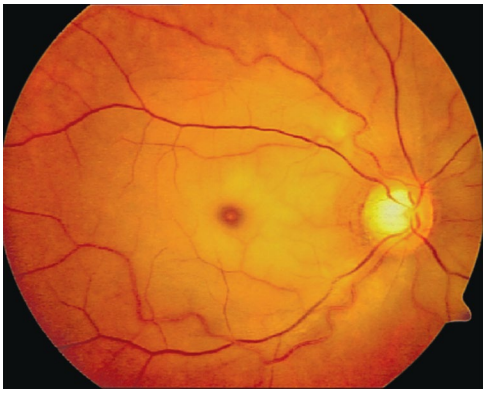


Fig. 18.9: Central retinal artery occlusion with cherry-red spot

when the entire retina is involved, or a peripheral branch (arteriole), when the effect is localized.

Etiology

- Emboli from the carotid artery, e.g. atherosclerotic plaques in older patient.
- Emboli from valvular heart disease in young patient.
- Thrombus from arteriolosclerosis.
- Hypertension
- Arteritis.

Symptoms

- Painless, unilateral sudden loss of vision.
- ***Amaurosis fugax*** (transient sudden loss of vision lasting for few minutes, due to occlusion by minute emboli) may be a premonitory symptom.

Signs

Ophthalmoscopically, the fundus shows a typical picture:

- Larger arteries are thread-like, and the smaller arterioles are invisible.
- Veins are almost normal.
- Within a few hours, the retina loses its transparency, and becoming opaque and milky-white, especially around the posterior pole.
- A day or two later, a ***cherry-red spot*** appears at the fovea, since the vascular choroid is visible underneath. It is beautifully seen against strong contrast of the cloudy-white background.

A ***cherry-red spot*** is also seen in:

- Blunt injury (Berlin's edema of the macula),
- Tay-Sach's disease,
- Niemann-pick disease,
- Sandhoff's disease, and
- Quinine amblyopia.

- When the obstruction is incomplete, the blood flow is partially restored. In that case, gentle pressure upon the globe may break up the column of venous blood into red-beads which move in a jerky fashion—the ***cattle-track*** appearance.
- After a week or so, the retina resumes its normal ophthalmoscopic appearance, but the optic nerve becomes atrophic and appears white.
- The vision is no perception of light (PL), and there is no direct pupillary reaction.
- ***In obstruction of a branch of the retinal artery,*** the occlusion is usually at a bifurcation, and invariably due to embolism. An atheromatous embolus may be visible as a pale refractile body within the artery (***Hollenhorst plaque***), and the distal area of the retina that the vessel supplies, becomes edematous. A sectorial scotoma corresponding to the affected area is detected in fieldcharting.

Diagnosis

It depends on the history, since the patient is very rarely seen during an acute attack. All the patient of ***amaurosis fugax*** should be thoroughly investigated for ***cardiovascular problems***.

Treatment

It is only effective, if given within the first few hours of such an occlusion.

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- Immediate intermittent digital massage to the globe.
- Intravenous acetazolamide, if available.
- Inhalation of 5% CO_2 and 95% O_2 mixture for 10 minutes may be helpful.
- Paracentesis of the anterior chamber.
- Retrobulbar injection of acetylcholine may produce vasodilatation.
- Anticoagulants, as intravenous heparin.

Prognosis

- As the patient usually presents after few hours, the most frequent result of an occlusion of central retinal artery, however, is blindness.
- No return of macular vision is anticipated if the obstruction has lasted over six hours.
- Where a cilioretinal artery is present (about 25% of cases), blood from the choroid will reach the posterior pole, preserving a useful island of vision which often includes the macular vision.

RETINAL VEIN OCCLUSION

Retinal vein occlusion may involve the central retinal vein which causes immediate severe loss of vision, or a branch retinal vein which results in partial loss of vision, depending on the affected region drained by the vein.

Predisposing Factors

- ***Age:*** Sixth and seventh decades.
- ***Systemic hypertension:*** The vein is compressed by a thickened artery where the two shares a common adventitia (at the arteriovenous crossing).
- ***Raised intraocular pressure:*** Patients with primary open-angle glaucoma (POAG) or ocular hypertension are at increased risk of central retinal vein occlusion (CRVO), but no correlation with branch retinal vein occlusion (BRVO).
- ***Diabetes:*** Increased risk of CRVO/BRVO.
- ***Hyperviscosity syndrome:*** As in chronic leukaemia and polycythaemia vera (hypercellularity of blood): In macroglobulinaemia and cryoglobulinemia (change in plasmaproteins) or obstructive pulmonary diseases (COPD).
- ***Periphlebitis*** as in sarcoidosis and Behcet's disease.

Three basic mechanisms involved both in CRVO and BRVO are:
1. External compression on the vein,
2. Venous stasis, and
3. Degenerative diseases of the venous endothelium.

CENTRAL RETINAL VENOUS OCCLUSION (FIG. 18.10)

Types

Their are two main types of CRVO:
1. ***Non-ischemic CRVO (Fig. 18.11):*** It is also referred as venous stasis retinopathy or partial CRVO. This accounts for 75% of cases.
2. ***Ischemic CRVO (Fig. 18.12):*** It is also called complete CRVO, This accounts for the remaining 25% of cases.



Fig. 18.10: Central retinal artery occlusion—macular sparing due to patent cilioretinal artery

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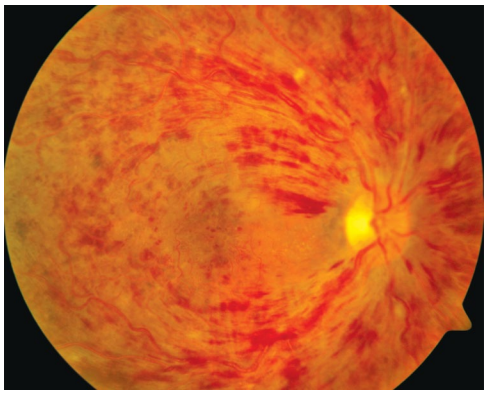


Fig. 18.11: Non-ischemic central retinal venous occlusion

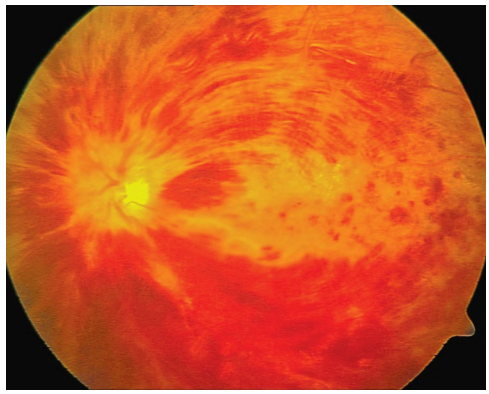


Fig. 18.12: Ischemic central retinal venous occlusion

Non-ischemic Central Retinal Artery Occlusion

- ***Clinical features***
  - Visual acuity is slightly or moderately reduced.
  - Relative afferent pupillary defect (Marcus-Gunn pupil) is usually mild, indicating mild retinal ischemia.
- ***Ophthalmoscopically***
  - Mild tortuosity and dilatation of all branches of the central retinal vein.
  - ***Dot and blot; and flame:*** It shaped hemorrhages are seen throughout all four quadrants of the retina.
  - Cotton-wool exudates are usually absent.
  - Mild to moderate swelling of the optic disc.
  - Macular edema may or may not be present.
- ***Fluorescein angiography:*** It shows, venous stasis but with good retinal capillary perfusion.
- ***Prognosis:*** In 50% of cases, the retinopathy resolves, and visual acuity returns to normal or near normal. The permanent impairment of vision is due to chronic macular edema.
- ***Treatment:*** No specific treatment is required except to treat the predisposing factor, if any.

Ischemic Central Retinal Artery Occlusion

- ***Clinical features***
  - Marked reduction in visual acuity.
  - Relative afferent pupillary defect marked.
- ***Ophthalmoscopically***
  - Marked tortuosity and engorgement of the retinal veins.
  - Massive superficial and deep hemorrhages throughout the fundus.
  - Cotton-wool exudates are common.
  - Optic disc is swollen and hyperemic.
  - Macular edema and hemorrhages.

This appearance is sometimes called ***blood and thunder*** fundus.

- ***Fluorescein angiography*** in early stage is not useful because of hemorrhages, but in less severe cases or in late stage, there are extensive areas of capillary non-perfusion.
- ***Course and prognosis:*** About 50% of eyes develop rubeosis iridis and neovascular glaucoma within 3 months of the initial occlusion ***(90-days glaucoma)***. A small percentage of eyes develop pre-retinal or vitreous hemorrhage, secondary to NVD and/or NVE.
- ***Treatment***
  - Patient should be followed-up closely for rubeosis iridis, as prompt treatment in early cases, by panretinal photocoa-

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gulation (PRP) (or cryoapplication if the media is hazy), prevents the subsequent development of neovascular glaucoma.
  - The management of established neovascular glaucoma is discussed in Chapter 16.

BRANCH RETINAL VENOUS OCCLUSION

It (Fig. 18.13) may occur near the optic disc and involves a major quadrant of the retina, or it may occur at a peripheral crossing with an artery. Blockage of the superior temporal vein frequently involves the macula.

- ***Symptoms:*** These depend on the location of the occlusion. Visual loss is marked, if the superior temporal branch is occluded. ***Ophthalmoscopically*** the affected part of the retina, drained by the obstructed vein shows:
  - Dilated and tortuous veins
  - Flame-shaped, and dot and blot hemorrhage in the affected quadrant.
  - Retinal edema and cotton-wool spots
- ***Course and prognosis:*** Following a major branch retinal vein occlusion, visual acuity is initially reduced due to macular hemorrhage and edema.
  - Within 6 months, 50% of eyes develop efficient collaterals and visual acuity returns to 6/12, or better.



Fig. 18.13: Branch retinal venous occlusion

  - However, complications may arise from vascular leakage and capillary non-perfusion.
  - Two most important vision-threatening complications are:
    1. Macular edema and
    2. Vitreous hemorrhage due to neovascularization.
  - Secondary neovascular glaucoma occurs very rarely.
- ***Treatment***
  - Fundus fluorescein angiography to know the macular non-perfusion, or macular edema with good perfusion, and also to identify the ***collateral vessels*** (because they must not be treated by laser).
  - For macular edema, grid photocoagulation with argon laser or double-frequency yttrium aluminium garnet (YAG) (532) laser may be helpful.
  - For neovascularization following BRVO, the eye should be treated by scatter argon laser photocoagulation, in order to prevent vitreous hemorrhage.

CENTRAL SEROUS RETINOPATHY

Central serous retinopathy (CSR) (Figs 18.14A and B) is characterized by sudden edema of the macular area, which typically affects the young adult males due to unknown cause. It is basically a localized retinal detachment, and caused by exudation from the parafoveal or choroidal capillaries.

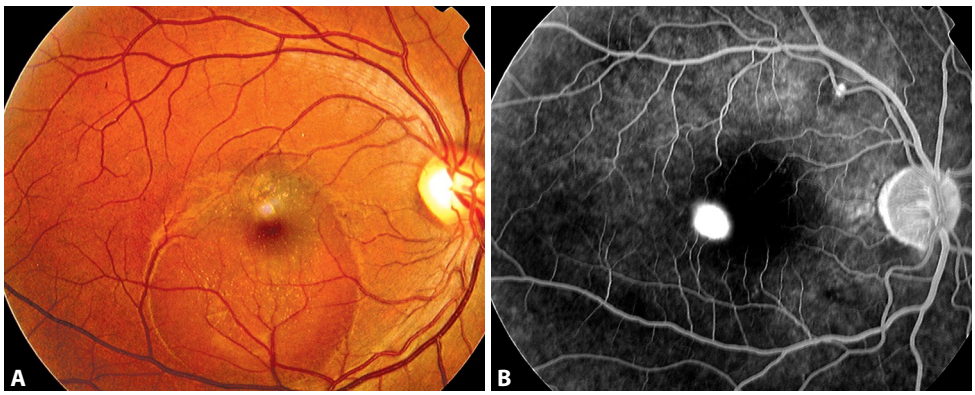
Symptoms

- Sudden onset of blurring of vision in one eye.
- Black fixed spot (a positive scotoma) at the center of the visual field.
- Metamorphopsia.

Signs

- Visual acuity reduced to 6/9 to 6/18, and often correctable to 6/6 with weak plus lens. This is due to elevation of neurosensory retina which gives rise to an acquired hypermetropia.

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Figs 18.14A and B: A. Central serous retinopathy; **B.** Ink-blot pattern on fundus fluorescein angiography

- ***Ophthalmoscopically,*** macula appears as an oval or circular dark swelling, larger than the size of the optic disc, which is surrounded by a glistening ***ring-reflex***.

FUNDUS FLUORESCEIN ANGIOGRAPHY

Fundus fluorescein angiography (FFA) is helpful for a definitive diagnosis of CSR. Two patterns are seen on FFA:

1. ***Ink-blot pattern:*** Small hyperfluorescent spot, gradually increases in size.
2. ***Smoke-stack pattern (Figs 18.15A to C):*** A small hyperfluorescent spot, ascends vertically like a smoke-stack, and gradually spreads laterally taking on a mushroom or umbrella configuration.

Prognosis

- Eighty to ninety percent cases of CSR undergo spontaneous resolution, and the visual acuity returns to normal or near normal, within 4–12 weeks.
- Mild metamorphopsia or micropsia may persist for a longer period.
- Fourty percent of patients develop recurrent attacks.

Treatment

- In majority of the cases, no treatment is required except reassurance.
- Argon laser or double-frequency YAG (532) laser photocoagulation to the site of leakage may be considered in some cases:
  - Recurrent CSR with visual loss.
  - If the vision in fellow eye is permanently reduced due to previous attacks of CSR.
  - If the duration is more than 4 months.
  - In presence of turbid subretinal fluid.



Figs 18.15A to C: Central serous retinopathy. **A to C.** Smoke-stack pattern on fundus fluorescein angiography

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CYSTOID MACULAR EDEMA (FIG. 18.16)

Cystoid macular edema (CME) is an accumulation of fluid in the outer plexiform (Henle's) layer and inner nuclear layer of the retina, in the macular region.

Etiology

Cystoid macular edema is a very common macular disorder that has many diverse causes:

- ***Irvine-Gass syndrome:*** Probably, it is the most common cause of CME, which occurs as a common complication of cataract extraction, especially when associated with vitreous prolapse. The majority of the cases are transient and have little clinical significance.
- ***Background diabetic retinopathy:*** CME is an important cause of visual impairment among diabetics.
- ***Retinal vein occlusion:*** Chronic CME may occur following CRVO or BRVO.
- Pars planitis and acute iridocyclitis.
- ***Retinitis pigmentosa:*** Typical CME may be present upto 70% of eyes with retinitis pigmentosa.
- ***Epinephrine:*** CME may develop in aphakic eyes treated with epinephrine for open-angle glaucoma (epinephrine maculopathy).

Clinical Features

- Visual acuity is often reduced to 6/60.
- ***Ophthalmoscopy*** shows an irregularity and blurring of the foveal reflex.
- The macula is wrinkled, edematous, and may show multiple small cystic changes.
- In long-standing cases, there is formation of lamellar holes at the fovea with irreversible damage to central vision (Fig. 18.17).

Fluorescein angiography demonstrates leakage of the dye from the perifoveal capillaries, and accumulation within the macular region. The accumulation of dye in the outer plexiform layer, with its radial arrangement of fibers (Henle's layer) is responsible for the typical ***flower-petal*** (Fig. 18.18) or ***spoke***-pattern of CME on FFA.

Treatment

- The edema is usually self-limited and improvement is spontaneous.
- Systemic steroids are often used in vascular or inflammatory conditions.
- Systemic steroids, sub-Tenon long-acting steroid, and non-steroidal antiinflammatory agents (systemic or local) are helpful in post-operative cases.



Fig. 18.16: Cystoid macular edema

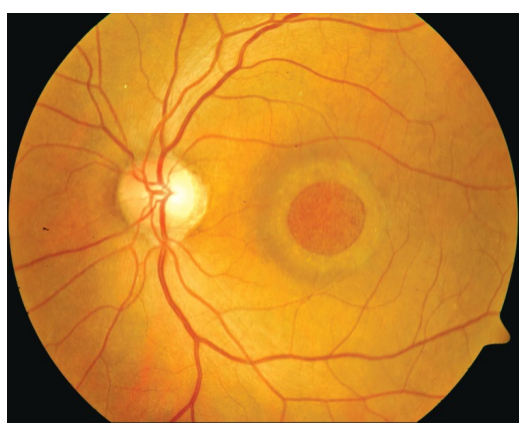


Fig. 18.17: Macular hole formation

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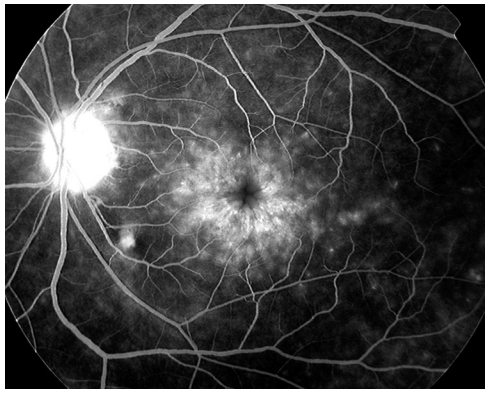


Fig. 18.18: Cystoid macular edema—flower-petal pattern on fundus fluorescein angiography

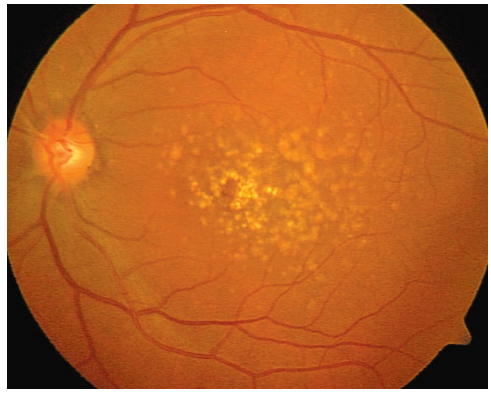


Fig. 18.19: Central hard drusen

AGE-RELATED MACULAR DEGENERATION

Age-related macular degeneration (ARMD) or senile macular degeneration (SMD) is a bilateral disease, characterized by formation of subretinal neovascular membranes (SRNVMs) at the macula. It is one of the leading causes of blindness in the Western world.

Histopathologically drusen, retinal pigment epithelial degeneration and/or atrophy, SRNVMs, retinal pigment epithelium (RPE) and sensory retinal detachment due to subretinal hemorrhage, and disciform scars at the macula, are the different manifestations of the disease in different stages.

Drusen

Drusens or colloid bodies are the most frequent clinical manifestation of aging, and are present in about 70% of people after the age of 50 years.

Clinical Features

They appear as small, discrete, yellow-white, slightly elevated spots at the posterior poles of both fundi. With advancing age they increase in size and number.

Secondary calcification in long-standing lesions gives them a glistening-white appearance.



Fig. 18.20: Central soft drusen

Classification

- ***Hard drusen (nodular) (Fig. 18.19):*** It appear as small, discrete yellowish-white nodules.
- ***Soft drusen (Fig. 18.20):*** It have indistinct edges, are larger than hard drusens, and frequently become confluent.
- ***Diffuse drusen* (confluent):** It represent a widespread abnormality of RPE.
- ***Calcified drusen:*** It have glistening-white appearance with conspicuous margin.

Histopathology

- Drusens consist of focal aggregation of hyaline material located between the basal

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lamina of the RPE and inner collagenous layer of the Bruch's membrane.
- Localized thickening and thinning of the Bruch's membrane.
- Variable degrees of RPE atrophy and depigmentation.
- Degenerative changes of the retinal receptors.

Fluorescein Angiography

- ***Retinal pigment epithelium window defects*** appear as multiple hyperfluorescent spots.
- ***Staining of the drusens*** by the dye, which may persist for a long time.

Drusen and ARMD
- The exact role of drusen in the pathogenesis of ARMD is still not clear.
- But a significant number of elderly patients with drusens may develop impairment of vision due to ARMD.
- Increased risk of subsequent visual loss in patient with confluent drusens, particularly if one eye has already developed visual loss from ARMD.

Types of Age-related Macular Edema

They are of two main types:
1. Non-exudative (dry) ARMD
2. Exudative (wet) ARMD.

Non-exudative (Dry) Age-related Macular Edema

- It is the most common type, and is due to a slow and progressive atrophy of RPE and photoreceptors.
- Progressive loss of vision for several months to years.
- ***Clinically,*** it appears as sharp circumscribed areas of RPE atrophy with varying degrees of loss of choriocapillaries. In late stage, the larger choroidal vessels become visible within the atrophic areas.

Treatment
- There are no definite medical treatments for dry AMD at this time.
- ***Lifestyle changes:*** It can slow down the process or contribute to the development and progress of AMD.
- Antioxidant medication that included vitamin C, vitamin E, beta carotene (or vitamin A), and zinc can slow the progression of the disease by 25%.
- Changing the diet which includes more fruits and vegetable, choosing healthy unsaturated fats, such as olive oil, refined grains and adding fish with high omega-3 fatty acids.
- ***Low vision rehabilitation:*** The use of magnifying devices, e.g. binoculars or telescopes can often improve vision in macular degeneration to allow for reading or watching of television.
- Use of tablet devices or large smart phone—as reading device or to watch video.
- Miniature implantable telescope may help to improve the central vision. It involves surgical removal of crystalline lens and replace it with a miniature telescope. This is followed by an extensive training program.

Exudative (Wet) Age-related Macular Edema

- It is also sometimes referred to as ***neovascular*** or wet ARMD, and is less common than non-exudative type (Fig. 18.21).
- This is a more serious form, and the patient may lose all central vision within a few weeks. ***Amslers grid test*** (Figs 18.22A to C) is convenient and accurate way to detect ARMD in early stage.
- Two important features of exudative ARMD are ***detachment*** of the RPE and ***choroidal neovascularization***.
- ***RPE detachment*** appears as sharply circumscribed dome-shaped elevation with ophthalomoscope.

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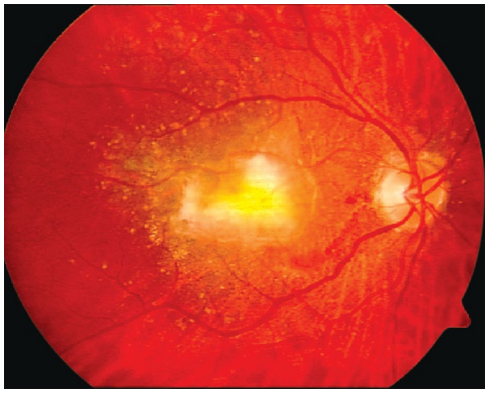


Fig. 18.21: Age-related macular degeneration—exudative type

- Subretinal neovascular membranes (SRNVMs), consisting of proliferations of fibrovascular tissue, begin to grow from the choriocapillaries → through the defects of the Bruch's membrane → into the sub-RPE space → and later into the subretinal space.
- ***Clinically,*** SRNVMs are undetectable in early stage. But later, they appear as gray-green elevated lesions in the macular region.
- ***Fluorescein angiography*** plays an important role in the detection and localization of SRNVMs in relation to foveal avascular zone (FAZ).
- ***Sequelae of exudative—ARMD***
  - Hemorrhagic detachment of the RPE and neurosensory retina
  - Vitreous hemorrhage
  - Disciform scarring of the macula
  - Exudative retinal detachment.

Treatment

Wet macular degeneration cannot be cured. If diagnosed early, treatment may help slow progress of wet macular degeneration and reduce the amount of vision lost.

There are three main treatment options for wet AMD:

1. Anti-VEGF (vascular endothelial growth factor) medication to prevent the growth of new blood vessels in the eye.
2. Photodynamic therapy (PDT) to treat foveal abnormal vessels.
3. Laser surgery to destroy abnormal blood vessels in the eye.

- ***Anti-VEGF*** intravitreal injection are considered the first-line treatment for all stages of wet macular degeneration. They include (*see* anti-VEGF in Chapter 5):
  - Bevacizumab (Avastin)
  - Ranibizumab (Lucentis)
  - Pegaptanib (Macugen)
  - Aflibercept (Eylea).
- ***Photodynamic therapy:*** Using cold laser light to activate an injected medication called, verteporfin to prevent progression



Figs 18.22A to C: Amsler grid test in ARMD, total nos. of square—20 × 20 in the chart. One square denotes 10 from the central part of fovea. Central diamond-shaped black spot represents foveola; **A.** Normal grid pattern; **B.** Distorted or missing pattern and **C.** Dark central spot

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of SRNVMs. PDT is still used to treat some cases of Wet AMD some times in conjunction with anti-VEGF injection.

- ***Photocoagulation:*** Using an Argon (blue-green) laser to destroy abnormal extrafoveal SRNVMs outside the area of FAZ. Only a small number of patients are eligible for this procedure, as most patients have growth in the area of the fovea, which cannot be treated by laser.
- Counseling of the patients reading continued therapy.

RETINITIS PIGMENTOSA (FIGS 18.23A TO C)

Retinitis pigmentosa (RP) is a group of hereditary diseases, characterized by progressive night blindness and constricted visual fields.

Typical Retinitis Pigmentosa

Typical RP is a bilateral symmetrical diffuse pigmentary retinal dystrophy, predominantly affecting the rods.

Inheritance

- ***Autosomal recessive:*** Most common.
- ***Autosomal dominant:*** Benign nature.

Pathogenesis

- Degeneration of the rods and cones.
- Migration of pigments into the retina.
- Degenerated ganglion cells and their axons are replaced by neuroglial tissue.
- Attenuation of the blood vessels.
- Atrophy of the optic disc.

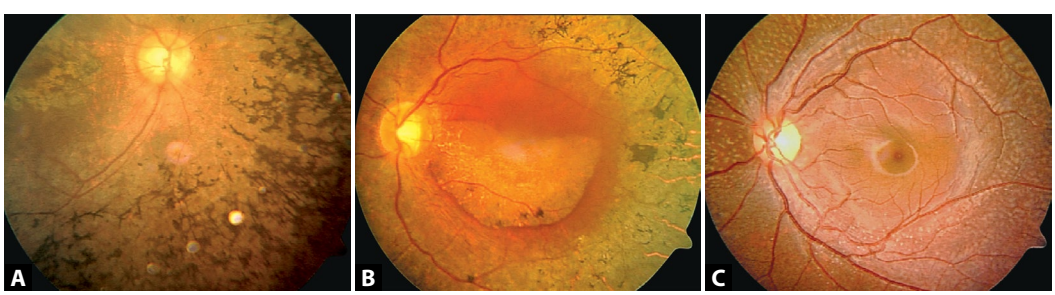
Clinical Features

- ***Symptoms:*** Include night blindness and progressive visual field defect.
- ***Signs:*** The classical triad of RP are:
  - Bony-spicule pigmentation,
  - Arteriolar attenuation, and
  - Waxy-pallor of the optic disc.
- Pigmentary changes are typically perivascular, and have a bone-spicule appearance, which are observed mostly at the equatorial region of the retina.
- In late stages of the disease, the unmasking of the larger choroidal vessels (due to pigment migration), gives the fundus a tessellated appearance.
- ***Macular lesions***
  - ***Cystoid macular edema*** may be present upto 70% of the patients with RP
  - Cellophane maculopathy
  - Atrophic maculopathy.

Investigations

They are helpful in early stages:

- ***Electroretinopathy (ERG):*** Markedly subnormal in amplitude.
- ***Electrooculography (EOG):*** Absence of light peak.



Figs 18.23A to C: A. Retinitis pigmentosa; **B.** Retinitis pigmentosa with cystoid macular edema and **C.** Retinitis punctate albicans

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- ***Visual field:*** Initially shows annular or ring-like scotoma. But ultimately, the progressive constriction of the visual field leaves only a tiny island of central vision, which may be lost eventually.

Atypical Retinitis Pigmentosa

- ***Retinitis punctata albescens:*** This is characterized by multiple scattered white dots, mostly between the posterior pole and the equator. Other findings are similar to typical RP.
- ***Retinitis pigmentosa sine pigmento:*** The typical pigmentary changes appear very late, while the presence of arteriolar attenuation and waxy-pallor of the disc with subnormal ERG, confirm the diagnosis.
- ***Sectorial retinitis pigmentosa:*** Involvement of only one quadrant (usually nasal), or one half (usually inferior) of the fundus.
- ***Pericentric retinitis pigmentosa:*** It is similar to typical RP, except that the pigmentary changes are confined to pericentral area, sparing the periphery (Fig. 18.24).

Systemic Associations of Retinitis Pigmentosa

- ***Laurence-Moon-Biedl syndrome (Bardet-Biedl) (Fig. 18.25):*** Autosomal recessive; RP, mental retardation, polydactyly, obesity and hypogonadism.

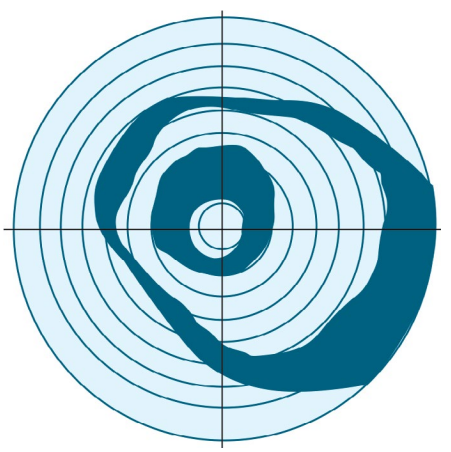


Fig. 18.24: Ring scotoma in retinitis pigmentosa



Fig. 18.25: Laurence-Moon-Biedl syndrome

- ***Bassen-Kornzweig syndrome (a beta-lipoproteinaemia):*** Autosomal recessive; RP, ataxia, acanthocytosis and fat malabsorption. Jejunal biopsy is diagnostic.
- ***Refsum's syndrome:*** Autosomal recessive; RP, peripheral neuropathy, cerebellar ataxia, elevated CSF-proteins, deafness and ichthyosis. It is due to defective metabolism of ***phytanic acid*** which accumulates in the body.
- ***Usher's syndrome:*** Autosomal recessive; RP and sensoryneural deafness.
- ***Cockayne's syndrome:*** Autosomal recessive; RP, dwarfism, 'bird-like' facies, flexion contracture of the limbs, mental retardation, ataxia and premature aging.
- ***Kearns-Sayre syndrome:*** The classical ***triad*** of RP, chronic progressive external ophthalmoplegia (ocular myopathy) and heart block; a rare mitochondrial DNA (mtDNA) deletion syndrome.
- ***Friedreich's ataxia:*** Autosomal recessive; RP, posterior column disease, cerebellar ataxia, nystagmus and subaortic stenosis.

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- ***Neuropathy, ataxia, and retinitis pigmentosa (NARP) syndrome:*** It is a rare hereditary condition. It is related to changes in mitochondrial DNA (mutations in the MT-ATP6 gene).

Treatment

- No definite treatment is available for RP patients.
- However, there are therapies available to maximize their vision with refraction and low-visual aids (LVA).
- They should be examined annually for visual field testing and ERG evaluations. Often after this, the patients may be reassured that the changes are slow. Additionally, regular examinations can ensure appropriate time to seek community and legal assistance.
- ***Medications***
  - Antioxidants and omega-3 fatty acid; or vitamin A (15,000 IU/day) may be useful in long-term to slow the progression.
  - ***Acetazolamide:*** It may be useful in macular edema in the later stages of RP.
  - Some medications have potential adverse effects in RP like, isotretinoin, vitamin E (400 IU) and sildenafil (viagra).
  - It may worsen night vision and ERG response.
- ***Cataract extraction:*** Often beneficial in RP with posterior subcapsular opacity with an improvement by 2 lines on Snellen chart.
- ***Miscellaneous:*** Dietary plan as in Refsum disease, Audiology in Usher's syndrome, etc.
- Stemcells transplantation and retinal prosthesis (photoconducting chip on retinal surface) are being used in clinical trials in patients with RP, AMD and Stargardt disease.
- ***Gene therapy:*** It is also under investigation.
- ***Counseling:*** Genetic testing and counseling to know the mode of inheritance. Counseling can also help the patients for their future plans, such as pregnancy, job choices, medical treatments and psychological boost up.

RETINAL DETACHMENT

- ***Retinal detachment (RD):*** It is the separation of the sensory retina from the retinal pigmentary epithelium (which represent the two layers of the primary optic vesicle) by subretinal fluid (SRF).
- ***Retinoschisis:*** It is the splitting of the sensory retina into two layers.
- ***Retinal break:*** It is the full-thickness defect in the sensory retina.
  - ***Hole:*** It is a small retinal break (usually circular) without any vitreous traction.
  - ***Tear:*** It is a retinal break (usually horse-shoe-shaped) with the tractional element. A tear is more dangerous than a hole.
- ***Syneresis:*** It is the contraction of vitreous gel which separates its liquid from solid components.
- ***Synchisis:*** It is the liquefaction of the vitreous gel.
- ***Choroidal detachment***: It is an effusion of fluid into the suprachoroidal space.

Classification

- Rhegmatogenous (primary) (Greek word ***rhegma*** means a break).
- Non-rhegmatogenous (secondary)
  - Tractional
  - Exudative.

Rhegmatogenous

Subretinal fluid, derived from the fluid vitreous gains access into the subretinal space through a retinal break. Rhegmatogenous RD may be traumatic or spontaneous.

Non-rhegmatogenous

- ***Tractional:*** The sensory retina is pulled away from the RPE by the vitreoretinal traction membranes. Two important causes of tractional RD are proliferative retinopathies, and penetrating ocular injury. The source of SRF is unknown.

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Figs 18.26A toD: Rhegmatogenous retinal detachment. **A.** Horse-shoe tear; **B.** Hole; **C.** Retinal dialysis and **D.** Accumulation of subretinal fluid

- ***Exudative:*** SRF, derived from the choroid gains access to the subretinal space through the damaged RPE. Exudative RD may be due to choroidal tumors and inflammation.

RHEGMATOGENOUS RETINAL DETACHMENT (FIGS 18.26 AND 18.27)

Etiology

- ***Incidence***: 1 in 10,000 population each year, and bilateral in 10% of the cases.
- ***Trauma***: Most common in young adult males.
- ***Myopia:*** Over 40% of all RDs occur in myopic subjects. This is due to higher incidence of lattice degeneration, liquefaction of the vitreous, and posterior vitreous detachment.
- ***Peripheral retinal degeneration:*** Lattice is the most important degeneration directly related to RD, and is found more commonly in myopes over—3.0D.

Peripheral Retinal Degeneration

About 60% of all retinal breaks develop in the peripheral retina that shows specific changes. These lesions may be associated with a spontaneous hole formation, or they may predispose to retinal tear formation. The important predisposing degenerations are:

- ***Lattice degeneration:*** It is present in about 8% of general population, and 40% of eyes with RD. It is characterized by white arborising lines, arranged in a lattice pattern occurring

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in the upper peripheral fundus between the equator and posterior border of the vitreous base, with long axis parallel to the ora seratta.

- ***Snail-track degeneration:*** It consists of sharply, demarcated bands of tightly packed snowflakes with a white frost-like appearance.
- ***White-with-pressure:*** It is a grayish-white appearance of the retina, induced by indenting the sclera (in case of normal fundus, scleral indentation induces a reddish elevation).
- ***White-without-pressure:*** It is an exaggeration of the above phenomenon, and is present without scleral indentation.
- ***Focal pigment clumps:*** It may be found with a retinal tear.

Other benign degeneration are:

- ***Peripheral (micro) cystoid degeneration:*** It may be a constant finding in old age with increasing severity. This is not itself causally related to RD.
- ***Paving-stone degeneration:*** It consists of yellowish-white areas of chorioretinal atrophy (present in 25% of normal eyes).
- ***Snowflakes:*** They are minute, yellowish-white dots which are scattered diffusely in the periphery.

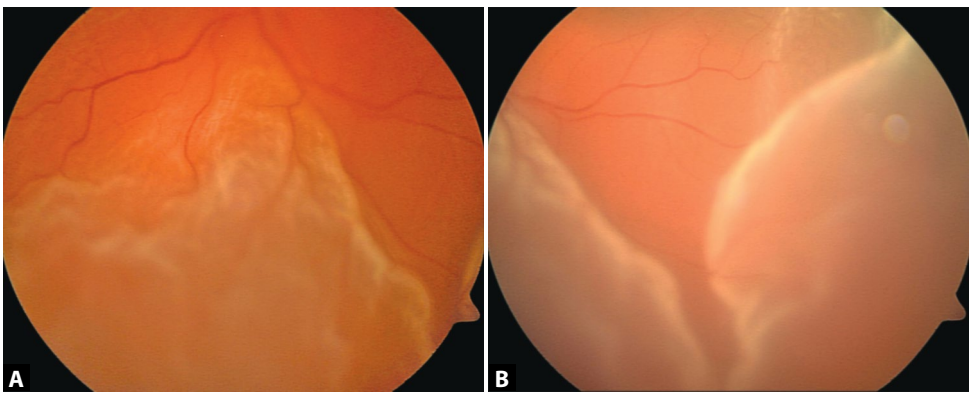
- ***Cataract surgery:*** It is more common with intracapsular cataract extraction, particularly if there had been vitreous loss. The incidence following extracapsular cataract extraction (ECCE) is less, possibly because the intact posterior capsule acts as a support to the vitreous, lessening its pull on the retina. It is also common after repeated needling in congenital cataract or after YAG-laser capsulotomy.

Pathogenesis of Rhegmatogenous

- The breaks responsible for RD, are caused by interplay between vitreoretinal traction, and an underlying weakness of the peripheral retina.
- 30% of horse-shoe tears proceed to RD within 6 weeks, and often within a few hours.
- If the retinal operculum becomes completely detached from the tear to lie separated from the retina, the risk of RD is reduced.
- Once the fluid (from liquefied vitreous) accumulates between the sensory retina and RPE, the retina becomes detached.
- As the subretinal fluid (SRF) accumulates, it tends to gravitate downwards.
- The final shape of the RD is being determined by the position of the break, and the anatomical limits of the disc and ora serrata.

Symptoms

- The classical premonitory symptoms reported in 60% patients with RD, are:
  - ***Photopsia:*** It is a flash of light and this is due to vitreoretinal traction.
  - ***Floaters***: Which may appear as large ring, small black spots or cobwebs.
- ***Visual field defect:*** It is due to the spread of SRF posterior to the equator, and is perceived by the patient as a black curtain.
- ***Loss of central vision:*** It is due to involvement of the fovea.



Figs 18.27A and B: Rhegmatogenous retinal detachment

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Signs

In fresh RD:

- Relative afferent pupillary defect (Marcus-Gunn pupil) in eyes with extensive RD.
- Intraocular pressure is low.
- Mild anterior uveitis.
- ***Posterior segment:*** Indirect ophthalmoscopy is more important.
  - Detached retina is slightly opaque, convex and corrugated in appearance (due to intraretinal edema), with loss of underlying choroidal pattern.
  - Detached retina undulates freely with ocular movements.
  - There is no shifting of fluid.
  - Breaks appear as red areas (holes or tears) of discontinuties mainly in the peripheral retina.
  - Vitreous shows ***tobacco dust*** in the anterior vitreous, with posterior vitreous detachment.

In long-standing RD:

- Retinal thinning
- Subretinal demarcation lines
- Secondary intraretinal cysts
- Multiple opaque strands of subretinal fibrosis.
- Subsequently, proliferative vitreoretinopathy (PVR) develops
- If untreated, the vast majority of RDs become total, and eventually give rise to complicated cataract, chronic uveitis, hypotony and eventually phthisis bulbi.

Investigations

- ***Indirect ophthalmoscopy*** is most important, and both eyes should be examined. Scleral indentation is necessary to enhance visualization of the extreme peripheral retina.
- Fundus drawing with appropriate color codes on a fundus chart.
- ***Slitlamp examination*** with three-mirror gonioscope:
  - To detect breaks.
  - To evaluate vitreous condition.
- ***Ultrasonography (USG):*** Both A-scan and B-scan are helpful when the media is hazy.

Treatment (Figs 18.28A to C)

The treatment of rhegmatogenous RD is essentially surgical, and should be immediate, once the diagnosis has been established.

The patient should take rest in recumbent position as much as possible.

- There are several methods of treating a detached retina, each of which depends on retinal breaks. The three general principles are:
  1. To find out the retinal breaks.
  2. To seal the breaks.
  3. To relieve present (and future) vitreoretinal traction.
- ***Sealing the retinal breaks:*** It is required in any form of rhegmatogenonus RD surgery—retinal breaks are closed by producing an area of chorioretinitis in the region of the defect, so that, the adhesions between the

A B C

Figs 18.28A to C: Treatment of rhegmatogenous retional detachment. **A.** Preoperative appearance; **B.** Encirclage (360°) and **C.** Postoperative appearance

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edge of the break and the RPE will seal the opening.

The chorioretinal reaction may be produced by cryosurgery (cryoretinopexy) at –70°C, or diathermy to the sclera, or by laser photocoagulation.

Three types of retinal detachment surgery are practiced:

1. ***Vitrectomy:*** Pars plana vitrectomy (PPV) is the most commonly performed operation treatment for retinal detachment. It works by removing the vitreous from the eye and replacing it with either a gas (SF6 or C3F8) or silicone oil. This helps in reattaching retina.

   Small dissolving sutures are used to close the wound. It is also possible to perform such surgery without the use of stitches, using 23G or 25G instruments.

   After the procedure, the patient requires positioning of the head for a while, so the bubble settles in the correct position.

   - ***Advantages*** of using gas in this surgery is that there is no myopic shift after the operation and gas is absorbed within a 2–3 weeks. There is less chance of future vitreoretinal traction. Silicone oil is more commonly used in cases associated with PVR changes. Silicon oil, if filled needs to be removed 2–6 months after surgery depending on surgeon's preference.
   - ***Disadvantage:*** Vitrectomy always leads to more rapid progression of a cataract in the operated eye. Emulsified silicon oil may sometime seep into the anterior chamber, causing keratopathy and inverse hypopyon.
2. ***Scleral buckling:*** It is a surgical procedure to create an inward indentation of the sclera and the choroid. Scleral buckling is achieved by inserting a plomb (exoplant), which is usually a solid silicone band or a silicone sponge, and sutured firmly to the sclera.
   - ***Radially-oriented*** plombage is most effective in sealing an isolated horseshoe tear, and ***circumferential*** plombage (encirclage) is indicated when multiple holes are present parallel to the equator.
   - ***Subretinal fluid drainage:*** It is customary to drain SRF by inserting a fine needle through the sclera and choroid, into the subretinal space, and allowing SRF to drain away
3. ***Pneumatic retinopexy:*** If the retinal detachment is relatively small and uncomplicated, a procedure called pneumatic retinopexy may be used. This involves injecting a small bubble of gas (SF6 or C3F8 gas) into the eye, and may be followed by laser photocoagulation or cryoretinopexy to create scar tissue around the break. The patient's head is then positioned so that the bubble rests against the retinal hole. Patients may have to keep their heads tilted for several days to keep the gas bubble in contact with the retinal hole. The bubble is slowly absorbed into the eye over the following weeks.

   This strict positioning requirement makes the treatment of the retinal holes and detachments that occurs in the lower part of the eyeball impractical.

   Pneumatic retinopexy has significantly lower success rates compared to scleral buckle surgery and vitrectomy.

   When an expansile gas is used with vitrectomy or for pneumoretinopexy, there is restriction of another surgery under GA or on flying.

   - ***Prognosis***: About 80–90% of RDs can be reattached, often with excellent visual results with immediate surgery, particularly, when macula is not involved. As, a detachment in the other eye occurs later in a high proportion of cases, a thorough retinal examination (indirect ophthalmoscopy), and prophylactic cryoretinopexy or laser photocoagulation is done, if there is any retinal break.

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A B

Figs 18.29A and B: Tractional retinal detachment (note the concave appearance of the detached retina)

TRACTIONAL RETINAL DETACHMENT (FIGS 18.29 AND 18.30)

Symptoms

- Photopsia and floaters are usually absent, because the vitreoretinal traction develops slowly.
- Slow and progressive loss of visual field.

Signs

- Retinal breaks are usually absent.
- Rhegmatogenous configuration is concave. The highest elevation of retina occurs at the site of vitreoretinal traction.
- Mobility of the detached area is severely reduced.
- Subretinal demarcation lines are absent.

EXUDATIVE RETINAL DETACHMENT (FIGS 18.31 AND 18.32)

Symptoms

- Floaters are present occasionally.
- Photopsia is absent, as there is no vitreoretinal traction.
- Sudden and rapid loss of visual field.

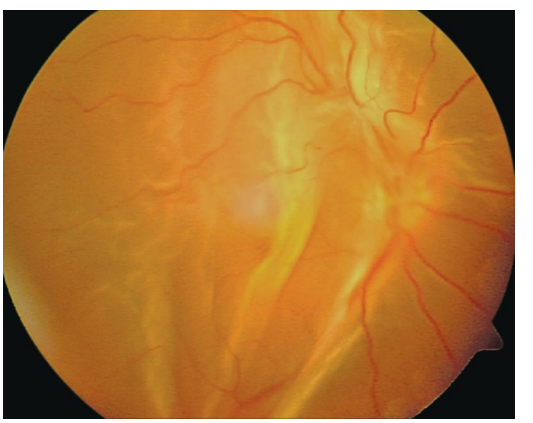


Fig. 18.30: Tractional retinal detachment

Signs

- Retinal breaks are absent.
- Rhegmatogenous configuration is convex. The detached retina is smooth, noncorrugated and bullous, and it may even touch the back of the lens.
- Shifting of fluid is ***the hallmark of exudative RD***. The SRF responds to the force of gravity, and detaches the area under which it accumulates.
- Other associated ocular features (e.g. choroidal tumors), or systemic features (e.g. rheumatoid arthritis, Harada's disease, toxaemia of pregnancy, etc.) may be present.

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A B

Figs 18.31A and B: Exudative retinal detachment (note the bullous appearance of the detached retina)

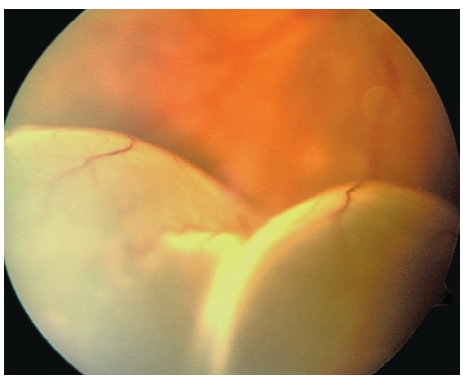


Fig. 18.32: Exudative rhegmatogenous—bullous detachment

HYPERTENSIVE RETINOPATHY

- In systemic hypertension, it is important to consider two aspects separately—the severity and the duration.
- The ***severity of hypertension*** is reflected by the degree of hypertensive vascular changes and retinopathy.
- The ***duration of hypertension*** is reflected by the degree of arteriolosclerotic changes and retinopathy.
- The ***primary response*** of the retinal arterioles to systemic hypertension is ***narrowing***, and it occurs only in young individual in its pure form.
- But, the degree of response in aged patient, also depends on the amount of pre-existing involutional sclerosis.
- So, the grading of retinal changes in hypertension has caused confusion, because of the replacement fibrosis of arterial hypertension, and that of aging are identical.

Pathogenesis

- Systemic chronic hypertension → narrowing of retinal arterioles → retinal ischemia → hypoxia → increased capillary permeability → focal retinal edema, retinal hemorrhage and hard exudates.
- Hard exudates deposit around the fovea in the Henle's layer, and lead to their radial distribution, in the form of ***macular star***.
- In severe or acute hypertension → obstruction of the precapillary arterioles → fibrinoid necrosis of the axons of nerve-fiber layer → interruption of axoplasmic flow → build-up of transported material within the nerve axons → ***cotton-wool patches***.

Classification

Keith-Wagner-Barkan

This is traditional and widely cited. It relates only systemic hypertension and arteriolar sclerosis (not for involutional sclerosis).

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- ***Grade-I:*** Mild generalized arteriolar narrowing.
- ***Grade-II:*** More narrowing of the arterioles and also focal arteriolar attenuation. Arteriovenous crossing changes (e.g. nicking) present.
- ***Grade-III:*** Grade II changes with hemorrhage, cotton-wool spot and exudates.
- ***Grade-IV:*** All the changes of grade III with papilledema including the neuroretinal edema.

Scheie's Classification

Here, the retinal changes of hypertension and arteriolar sclerosis are graded separately.

Hypertensive Features

- ***Normal:*** No ophthalmoscopic retinal changes.
- ***Grade-1:*** Narrowing of smaller retinal arterioles.
- ***Grade-2:*** Severe narrowing with localized irregular constriction of the arterioles.
- ***Grade-3:*** Narrowing and irregularities of the arterioles, with retinal hemorrhage and exudates.
- ***Grade-4:*** All changes in grade-3 along with neuro-retinal edema, and/or papilledema (Figs 18.33A and B).

Arteriolosclerotic Features

These changes develop if the hypertension is present over a period of many years, and are mainly seen in aged individual.

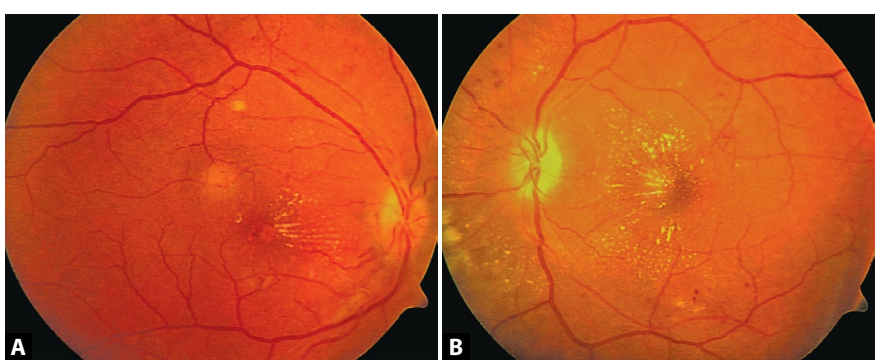
- ***Normal:*** No sclerotic changes.
- ***Grade-1:*** Widening of arteriolar light-reflex with simple venous concealment.
- ***Grade-2:*** Grade-1 changes with deflection of veins at the arteriovenous crossing (Salus' sign).
- ***Grade-3:*** Grade-2 changes with copper-wire arterioles, and marked arteriovenous crossing changes.
- ***Grade-4:*** All changes of grade-3 with silver-wire arterioles and marked arteriovenous crossing changes. It may be associated with branch vein occlusion.

Arteriovenous crossing changes (Figs 18.34A to D) may be:

- Banking of the vein where it appears dilated, distal to the crossing ***(Bonnet's sign)***.
- Tapering of the vein on either side of the crossing ***(Gunn's sign)***.
- Right-angled deflection of the vein, which gives the vein as S-shaped bend.

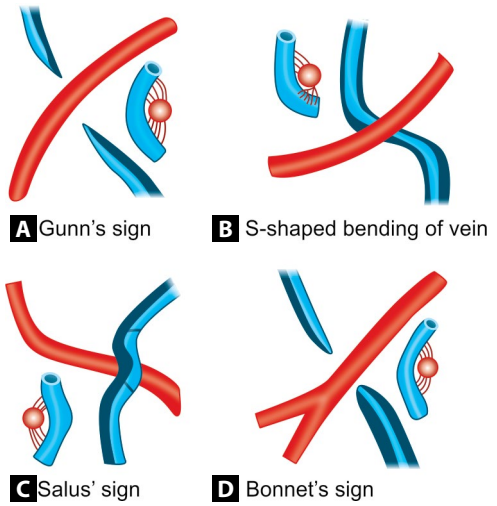
Associated choroidal changes:

- Severe hypertension may cause fibrinoid necrosis of the choroidal vessels which leads to atrophy of the RPE.
- Pigment deposition along the course of occluded choroidal vessels, is called ***Seigrist's line***.
- Irregular scattered bright-yellow spots are also seen, called ***Elschnig's spots***.
- Exudative bullous retinal detachment may occur in severe cases.



Figs 18.33A and B: Hypertensive retinopathy (Grade IV)

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Figs 18.34A to D: Arteriovenous crossing changes in hypertensive retinopathy

Renal Retinopathy

The term describes the retinal changes seen in chronic renal failure. These may mimic a hypertensive retinopathy of Grade-III or IV, but the tendency to exudate formation is marked, particularly at the macula.

The severe hypertension in eclampsia may precipitate a florid retinopathy of Grade-IV, often with exudative retinal detachment. This detachment clears rapidly with the termination of pregnancy when the hypertension is under control.

Treatment

The treatment of hypertensive retinopathies is, necessarily, control of hypertension and that of underlying disease.

DIABETIC RETINOPATHY

- The prevalence of diabetic retinopathy (DR) is about 4–28%, and about 2% of diabetic population is blind as a result of retinopathy.
- The ***best predictor*** of diabetic retinopathy is the duration of diabetes. There is no clinically apparent diabetic retinopathy for 5 years after initial diagnosis.
- The severity of the diabetic retinopathy, generally parallels the duration of the disease and the adequacy of its control, and not the severity of the diabetes.
- The incidence of diabetic retinopathy is more with insulin dependent diabetes mellitus (IDDM) than with non-insulin dependent diabetes mellitus (NIDDM), but the severity of maculopathy is more with NIDDM.
- The course and severity of DR are affected by the presence of nephropathy, systemic hypertension, pregnancy, and positive family history of DR.

Pathogenesis

- The exact pathogenesis of DR is still not clearly understood.
- Diabetic retinopathy is essentially a ***microangiopathy*** which affects the retinal precapillary arterioles, capillaries and venules.
- This microangiopathy causes microvascular occlusion and leakage.
- Microvascular occlusion → retinal ischemia → retinal hypoxia.
  Two main effects of retinal hypoxia are neovascularization and arteriovenous shunts formation.
- ***Microvascular weakness*** is due to loss of pericytes and damage to the endothelial cells → saccular pouch formation → microaneurysm, which may leak.
- ***Microvascular leakage*** → increased vascular permeability → hemorrhage and retinal edema.
- ***Chronic localized retinal edema*** leads to the deposition of hard exudates at the junction of healthy and edematous retina. The hard exudates are composed of lipoprotein and lipid-laden macrophages, and they are typically arranged in a circinate pattern.

Classification

- Background diabetic retinopathy
- Diabetic maculopathy
  - Focal

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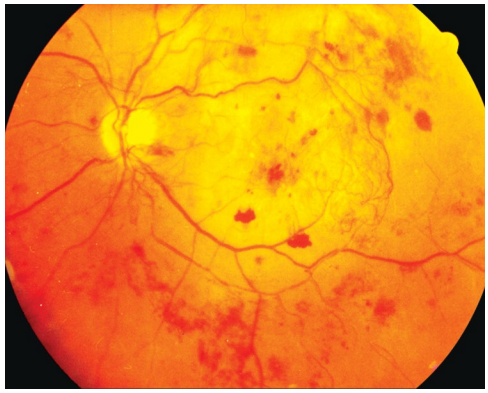


Fig. 18.35: Proliferative diabetic retinopathy neovascularization elsewhere

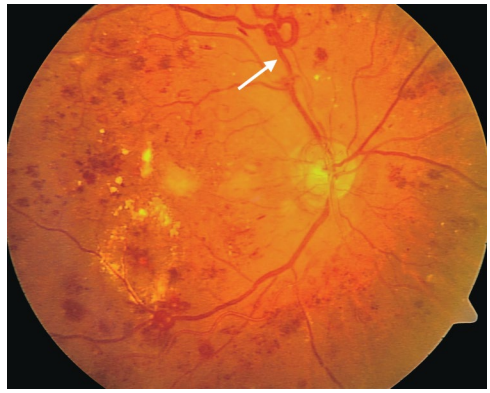


Fig. 18.36: Non-proliferative diabetic retinopathy—loop, dot and blot hemorrhage (arrow)

- Diffuse
- Ischemic

- Pre-proliferative diabetic retinopathy
- Proliferative diabetic retinopathy (Fig. 18.35)
- Advanced diabetic eye diseases (PDR)
  - Vitreous hemorrhage
  - Tractional retinal detachment
  - Preretinal membrane
  - Neovascular glaucoma.

Background diabetic retinopathy or non-proliferative diabetic retinopathy (NPDR) (Fig. 18.36).

Clinical Features

- ***Microaneurysms:*** They are the most characteristic ocular lesions in diabetics.
  They also occur in:
  - Retinal venous occlusion,
  - Coat's disease,
  - Arterial hypertension,
  - Anemia,
  - Dysproteinaemia, etc.

Microaneurysms associated with diseases other than diabetes, are located on the arterial side of the capillary, rather than on the venous side at the posterior pole.

Microaneurysms consist of minute, round or ovoid distensions ranging from 20–200 μ in size, located on the venous side of capillary network at the inner nuclear layer.

The resolving power of the direct ophthalmoscope is about 70–80 μ. Thus majority of microaneurysms are not seen with the direct ophthalmoscope. They are visible with FFA.

They may be indistinguishable from 'dot' hemorrhages.

- ***Dot and blot hemorrhages:*** They originate from the venous end of the capillaries, and are situated in the inner nuclear layer of the retina.
- ***Flame-shaped hemorrhages:*** They are located superficially in the nerve-fiber layer.
- ***Hard exudates*** have a yellow-waxy appearance with a distinct margin, and become confluent, and may form a circinate pattern around the macula. They are located between the inner plexiform and inner nuclear layer of the retina.

Diabetic Maculopathy

Diabetic maculopathy is the most common cause of visual impairment in patients with diabetic retinopathy, and is more frequent in NIDDM. Maculopathy is of three types—focal, diffuse and ischemic.

Focal

- Background diabetic retinopathy
- Macular edema

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- Hard exudates, may be ring-shaped.
- Microaneurysms [absent within foveal avascular zone (FAZ)].
- Visual acuity is severely impaired.
- It is caused by leakage from microaneurysms and dilated capillaries.
- FFA shows—focal area of leakage, but adequate macular capillary perfusion.

Diffuse

- Microaneurysms and hemorrhages
- Focal retinal thickening around the macula
- Few hard exudates
- Cystoid macular edema in long-standing cases
- Lamellar hole may result
- FFA shows diffuse leakage at the posterior pole with typical flower-petal pattern (as in CME).

Ischemic

- Clinically, it may be similar to those seen in diffuse maculopathy.
- Fluorescein angiography is necessary to differentiate it from the diffuse type. FFA shows areas of capillary non-perfusion in the macular and paramacular regions.

Pre-proliferative Diabetic Retinopathy

It develops in some eyes, those initially show only simple background retinopathy. The lesions are caused by retinal ischemia (Fig. 18.36).

- ***Cotton-wool patches (soft exudates)*** are due to capillary occlusion of the nerve fiber layers.
- ***Intraretinal microvascular abnormalities (IRMA):*** They are seen adjacent to the areas of capillary closure. IRMA may resemble focal areas of flat retinal neovascularization. The main features of IRMA are intraretinal location, absence of profuse leakage on FFA, and failure to cross over the main retinal vessels.
- Venous changes consist of dilatation, beading, looping, and sausage-like segmentation.
- Arteriolar narrowing resembling branch retinal arteriolar occlusion.
- Large dark blot hemorrhages due to hemorrhagic retinal infarcts.

Recent classification of diabetic retinopathy

- Non-proliferative diabetic retinopathy (NPDR)
- Proliferative diabetic retinopathy (PDR)

NPDR: The lesions include microaneurysms, small dot and blot hemorrhages, IRMA and cotton wool spots. NPDR is further classified as mild, moderate, severe and very severe NPDR.

- ***Mild NPDR:*** At least one microanaeurysms, and also dot and blot or flame-shaped hemorrhages in all four quadrants.
- ***Moderate NPDR:*** The findings are more severe than mild NPDR. Cotton wool spots venous beading and IRMA are present but mild.
- ***Severe NPDR:*** At least one of the following should be present: (a) Severe hemorrhages and microaneurysms in all four quadrants, (b) Severe venous beading in two quadrants, and (c) IRMA-more severe in one quadrant.
- ***Very severe NPDR:*** Two or more of above criteria, but without any proliferative changes.

Diabetic maculopathy: Clinically significant macular edema (CSME) (Fig. 18.37) is described as:

- Retinal thickening at or within 500 μm at the center of macula.
- Hard exudates at or within 500 μm of the center of macula.
- An area of retinal thickening greater than 1 dd area in size, at least a part of which is within 1 dd of the center of the macula.

PDR: Neovascularization is the hallmark of PDR.

Advanced PDR: Same as advanced diabetic eye diseases.

Proliferative Diabetic Retinopathy

It affects about 5% of the diabetic population, and more with insulin-dependent diabetes mellitus (IDDM).

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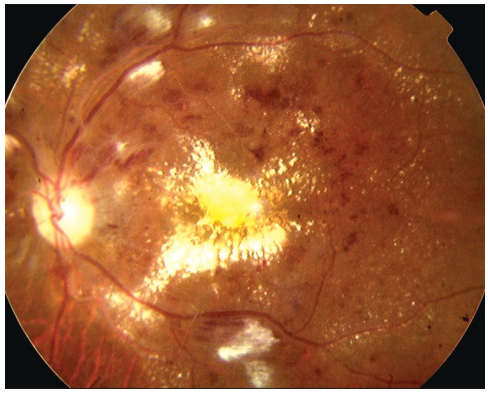


Fig. 18.37: Non-proliferative diabetic retinopathy—clinically significant macular edema

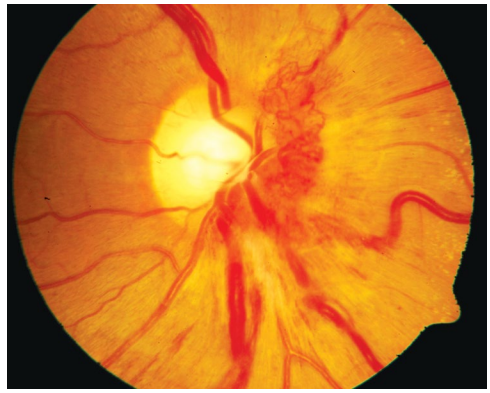


Fig. 18.38: Neovascularization of the disc

Clinical Features

- ***Neovascularization*** is the hallmark of PDR.
  It may be on the optic nerve head [NVD—***new vessels at the disc (Fig 18.38)***], or along the course of major blood vessels (NVE—new vessels elsewhere). Over one-fourth of the retina has to be ischemic (non-perfused), before NVD appear. The new vessels start as endothelial proliferations, arising from the veins. The vessels are enveloped by mesenchymal fibroblasts forming a fibrovascular epiretinal membrane. NVD are more dangerous than NVE, as they have a greater propensity to bleed.
  FFA shows hyperfluorescent areas with leakage from the new-vessels.
- ***Posterior vitreous detachment*** is extremely important in the progression of PDR.
- ***Hemorrhage*** may occur in the form of intraretinal, preretinal and vitreous hemorrhage.
  A preretinal [subhyaloid (Fig. 18.39)] hemorrhage has a crescentic or boat-shaped configuration, which demarcates the level of posterior vitreous detachment.

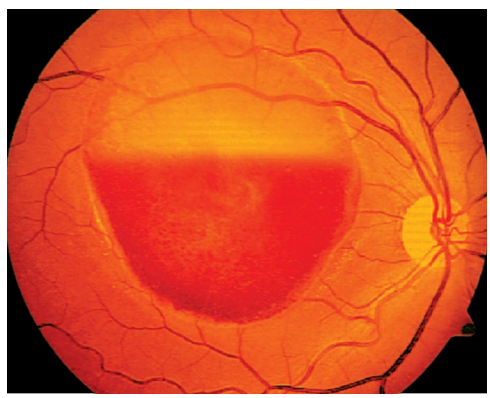


Fig. 18.39: Subhyaloid hemorrhage

Advanced Diabetic Eye Diseases

The end results of uncontrolled PDR are:

- Recurrent vitreous hemorrhages.
- Tractional retinal detachment.
- ***Burnt-out proliferative diabetic retinopathy:*** In persistent PDR, after a variable period of time, the fibrous component of the fibrovascular proliferation becomes more evident, and the vascular component decreases. Then, the blood vessels become nonperfused, and no further proliferation occurs. This is called ***burnt-out PDR***.
- ***Neovascular glaucoma:*** Rubeosis iridis is a fairly common finding in eyes with PDR, and may lead to the development of neovascular glaucoma. It is more in patients following unsuccessful pars plana vitrectomy.

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Management of Diabetic Retinopathy

General

- ***Metabolic control:*** Careful control of diabetes during first 5 years of the disease may reduce the severity, or delay the onset of DR. But, it apparently has little, or no effect on the established condition.
- ***Control of hypertension:*** Strict control of systemic hypertension is important.
- ***Anemia:*** Should be treated promptly.
- ***Aspirin and dipyridamole:*** It may have beneficial effect as they decrease platelet adhesiveness.
- ***Clofibrate:*** It is used to decrease lipid concentration.

Specific Treatment

- ***Background DR:*** No treatment is required for BDR with normal visual acuity except, periodic annual examination.
- ***Diabetic maculopathy***
  - ***Focal maculopathy:*** Focal argon-laser photocoagulation is required, around the microaneurysms or leaking vessels.
  - ***Diffuse Maculopathy:*** Macular grid (Fig. 18.40) Photocoagulation is required, but avoiding the fovea (the burns should not be closer than 500 μ from the foveola). The results are much less favorable than for focal maculopathy.
  - ***Ischemic maculopathy:*** Photocoagulation will not improve vision. Follow-up is essential, as about 30% will develop PDR within 2 years.
- ***Pre-proliferative DR:*** The patients should be watched very closely. Treatment by photocoagulation is usually unnecessary, unless FFA shows extensive areas of capillary non-perfusion. Gentle argon laser photocoagulation is done to the areas of capillary non-perfusion, or in a panretinal fashion.
- ***Proliferative DR:*** Photocoagulation is the most important modality for the treatment of PDR. Double frequency YAG (532) laser, Argon laser or Diode laser is used for photocoagulation. Xenon arc photocoagulation is no longer used. Pituitary ablation, to treat PDR, is abandoned.

  Argon-laser or double frequency YAG (532)-laser photocoagulation: The treatment of choice in PDR, is panretinal photocoagulation (PRP) (Fig. 18.41) with a slit-lamp delivery system.

  The aim of the treatment is to convert the hypoxic area into anoxic area, and thereby, to induce involution of new vessels, and to prevent recurrent vitreous hemorrhage.

  PRP is performed in two or more sessions, as, if done in one session, it causes more

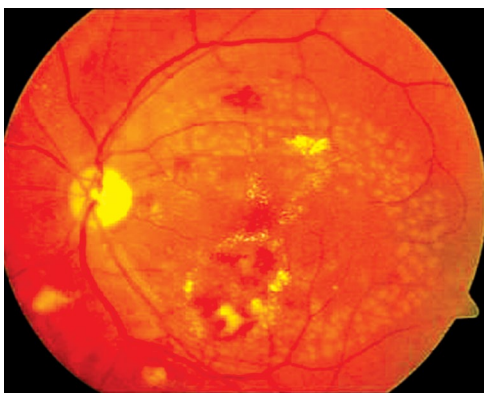


Fig. 18.40: Macular grid in diabetic maculopathy

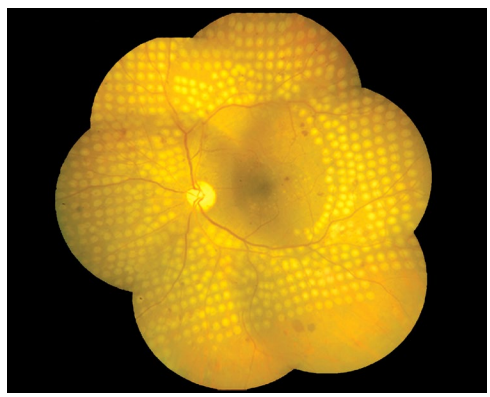


Fig. 18.41: Panretinal photocoagulation

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serious complications. The new vessels usually take 4–8 weeks time to regress after adequate PRP.

- ***Advanced diabetic eye diseases***
  - ***Vitreous hemorrhage:*** Pars plana vitrectomy with panretinal photocoagulation (endolaser).
- ***Tractional RD:*** Pars plana vitrectomy is only to be undertaken when detachment has progressed to involve the macula.
- ***Neovascular glaucoma:*** As described in Chapter 16.

OTHER OCULAR SIGNS IN DIABETES MELLITUS

Visual defects

- Transient variations in refraction:
  - Hyperglycemia → increased refractive index of the lens → myopic shift.
  - Hypoglycemia → decreased refractive power of the lens → hypermetropic shift.
- Photopsia and diplopia in cerebral hypoglycemia.
- Decreased accommodation.
- Diplopia in ophthalmoplegia caused by neuropathy.
- Decreased vision in cataract, maculopathy and vitreous hemorrhage.
- Complete blindness from tractional RD, massive vitreous proliferation and neovascular glaucoma.

Ocular movements

- Neuropathy with muscle paralysis (pupillary sparing in third nerve palsy).
- Painful ophthalmoplegia.

Lids

- Increased incidence of stye or internal hordeolum.
- Xanthelasma (due to associated hyperlipidaemia).

Conjunctiva

- Tortuous and constricted blood vessels
- Sludging of the blood.

Cornea

- Delayed epithelial healing due to abnormality in epithelial basement membrane.
- Punctate keratopathy.
- Increased incidence of infective keratitis with delayed healing.
- Wrinkling of the Descemet's membrane.
- Decreased corneal sensation (due to trigeminal neuropathy).
- Increased prevalence of dry eye.

Iris

- Hydrops of the pigment epithelium (due to transient glycogen storage) release of pigment into the anterior chamber during surgery, or simply, even after dilatation of the pupil.
- Rubeosis iridis.

Lens

- Variation in refractive power [during hyperglycemia increased glucose content (sorbitol) of the lens cortex → imbibition of water → increased thickness of the lens → increased refractive power].
- Typical snowflake (sugar) cataract in IDDM.
- Posterior cortical cataract.
- Early onset of senile cataract,

Vitreous

- Vitreous hemorrhage
- Fibrovascular proliferation in the vitreous.

Retina

- Diabetic retinopathy (as already discussed).
- Lipaemia retinalis (due to associated triglyceridaemia, in ketoacidosis).
- Central retinal venous occlusion.
- Diabetic retinopathy with superimposed changes of severe vascular hypertension —as seen in IDDM with renal failure.

Intraocular pressure

- Decreased in diabetic ketoacidosis (due to increased plasma bicarbonate level).
- Increased in neovascular glaucoma.
- Increased incidence of POAG.

RETINOBLASTOMA

Discussed in Chapter 20.

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19 CHAPTER

Diseases of the Optic Nerve

OPTIC NERVE

Optic nerve is divided into four parts:

1. ***Intraocular:*** 1 mm
2. ***Intraorbital:*** 24 mm
3. ***Intracanalicular:*** 9 mm
4. ***Intracranial:*** 16 mm

Total length: 5.0 cm

- Its diameter increases from about 1.6 mm within the eye (intraocular) to 3.5 mm in the orbit to 4.5 mm within the cranial space (intracranial).
- The non-myelinated intraocular portion contained within the scleral canal may be divided into three parts—(1) ***inner*** retinal, (2) ***middle*** choroidal and (3) ***outer*** scleral.
- The retinal portion is seen ophthalmoscopically as the disc, or optic papilla (***papilla is a misnomer*** because the disc is at the same level or slightly deeper than retinal nerve-fiber layer).
- A central depression, the ***physiological cup*** with its edges concentric to those of the disc, is lined with glial tissue, which may be sparse, so that the perforations of the lamina cribrosa are often visible.
- Human optic nerve contains between 7.7 lakh and 17 lakh nerve fibers, which are axons of the retinal ganglion cells of the retina. They synapse either in the lateral geniculate body (visual pathway), or in the pretectal region (pupillary pathway).
- Optic nerve has the structures and shares the diseases of a neurone of second order of the central nervous system (CNS) rather than a peripheral nerve.
- The nerve fibers have myelin sheaths normally proximal to the lamina cribrosa. These sheaths are separated by glial tissue, and ***neurilemma is absent***. For this reason, they do not regenerate after damage, as the tracts in the CNS.

OPTIC NERVE DISEASES

Symptoms

- ***Loss of vision:*** The main symptom.
- ***Scotoma:*** Loss of central vision.
- Disturbances in color vision.
- Pain is conspicuously absent, except it may be a prominent symptom in retrobulbar neuritis.

Signs

- Conduction defects in the optic nerve usually cause ill-sustained pupillary reaction, and ***relative afferent pupillary defect*** (RAPD) or Marcus-Gunn pupil.
- ***Ophthalmoscopy*** plays an important role in the diagnosis of optic nerve diseases. Attention is directed specially, to the margins of the disc, the surface of the disc and its color, pulsation of the central retinal vein, and the size and shape of the physiological cup.
- The variety of diseases affecting the optic nerve is large, and accurate diagnosis req-

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uires a complete history, search for systemic diseases, and awareness of variety of diseases that may cause similar findings.

DEVELOPMENTAL ABNORMALITIES

Persistent Hyaloid Artery

- The hyaloid artery nurtures the lens during the first 10 weeks of embryonic life. After the fetal fissure closes, the hyaloid artery enters the eye at the optic cup and then obliterates.
- Occasionally, a short stub of this vessel projects into the vitreous cavity from the center of the optic disc and is surrounded by a small mass of glial tissue called ***Bergmeister's papilla (Fig. 19.1)***. It is usually of no clinical significance, although it may rarely cause a recurrent vitreous hemorrhage.
- Failure of hyaloid artery to regress cause ***persistent primary vitreous*** which may proliferate.
- Minute residual fragments of the hyaloid system, may be seen as faint thread or small floaters ***muscae volitantes***.

Drusen

Two kinds of drusen (***drusen*** means stony nodule) occur in the intraocular portion of the optic nerve.

1. ***Common drusens*** are laminated, nodular, and may give the disc margins a blurred appearance (differentiated from papilledema by the absence of dilated retinal veins). Drusens often cause visual field defects but rarely affect visual acuity (Fig. 19.2).
2. The second type is ***giant drusens,*** which are astrocytic hamartomas, that occur with ***tuberous sclerosis***.

Conus

- In conus, or congenital crescent, the choroid and retinal pigment epithelium do not extend upto the optic disc.
- A large, white, semilunar area of sclera is seen adjacent to the disc, in the region of primitive retinal fissure (***inferonasal***).
- Defective vision, hypermetropic astigmatism and visual field defects are often present.
- A myopic crescent has a similar appearance, and is located at the temporal side of the disc.
- It is not present at birth and is associated with high myopia.

Fig. 19.1: Persistent hyperplastic primary vitreous—Bergmeister's papilla

Fig. 19.2: Optic disc drusen (arrows)

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Figs 19.3A and B: Optic disc coloboma—morning glory syndrome. **A.** Schematic representation; **B.** Photograph

Coloboma of the Optic Disc

- Incomplete closure of the embryonic fissure causes optic nerve defects ranging from a deep physiological cup to a pit in the optic disc, or to a deeply excavated optic nerve.
- The condition is usually unilateral, but bilateral optic disc colobomas occur as an autosomal dominant hereditary defect.
- The disc looks large and the vessels have an abnormal distribution appearing only around the edges (morning glory syndrome) (Figs 19.3A and B). The apparent disc is really the sclera and the inner surface of the sheath, of the nerve. The nerve itself being spread out as a pink horizontal linear band at the upper part. The floor of the coloboma is white and measurably depressed, often quite ectatic. The eye usually has defective vision.
- ***Pit in the optic disc*** is probably an incomplete coloboma. It is usually single, and located in the inferotemporal region surrounding the fissure, and usually round or oval. A central scotoma is often present, and in 30% of the cases, a central serous retinopathy (CSR) develops (Fig. 19.4).

Hypoplasia of the Optic Disc

- Failure of the axons of the ganglionic cells to develop or to reach the disc causes a small hypoplastic optic disc.

Fig. 19.4: Optic disc pit

- It is bilateral in 60% of cases, and often associated with decreased size of the optic foramina.
- Typical appearance consists of a small gray optic disc surrounded by yellowish peripapillary halo of hypopigmentation due to concentric choroidal and retinal pigment epithelial abnormality often called ***double-ring sign.***
- Despite the small optic disc the retinal vessels are of normal calibre.
- Sometimes, it may be associated with CNS anomalies, especially following the maternal ingestion of an anticonvulsant drug—***phenytoin sodium***.

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Fig. 19.5: Tilted optic disc

Fig. 19.6: Optic neuritis

Tilted Disc

This is due to an oblique entrance of the optic nerve into the globe and usually bilateral.

- The appearance of the disc is extremely oval, with the vertical axis directed obliquely, so that, its upper temporal portion lies anterior to the lower margin (Fig. 19.5).
- Eyes with tilted discs, frequently have inferior crescent, myopia, a moderate degree of oblique astigmatism, and hypopigmentation of the inferonasal aspect of the retina.
- Tilted disc may be associated with upper temporal field defect which may be mistaken for chiasmal compression.

OPTIC NEURITIS (FIG. 19.6)

Optic neuritis is an inflammatory or demyelinating disorder of the optic nerve (from the optic disc to the lateral geniculate body).

Classification

- Optic neuritis
- Retrobulbar neuritis—acute and chronic (toxic amblyopia)
- Neuroretinitis.

Etiology

- ***Idiopathic***
- ***Demyelination*** or ***multiple sclerosis (MS):*** Optic neuritis is the presenting feature in about 25% of MS patient, and in 70% cases, it occurs in established disease. Recurrence in the same eye or opposite eye occurs in about 25% patient. The impairment of vision is more with increased body temperature (***Uhthoff's phenomenon***).
- ***Neuromyelitis optica (of Devic):*** An acute bilateral optic neuritis in young patient with paraplegia (due to myelitis).
- ***Post-viral:*** They are the most common causes in children, and frequently bilateral. It usually follows an episode of influenza-like symptom, then followed by acute disseminated encephalomyelitis. Less frequently, it is associated with mumps, measles or chickenpox.
- ***Granulomatous inflammation:*** Like sarcoidosis, tuberculosis or syphilis, and these are rare.
- ***Infection of the adjacent structures:*** Like uveitis, retinitis, meningitis, orbital cellulitis, sinusitis (sphenoidal or ethmoidal), etc.
- ***Metabolic disturbance:*** Like diabetes, anemia, pregnancy, vitamin B deficiency, etc.

Clinical Features

Optic neuritis of the idiopathic type, or from demyelination, typically affects the patients between 20 and 40 years of age, but post-viral type typically occurs in children.

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Symptoms

- Uniocular sudden dimness of vision which may vary in severity (from a small scotoma to complete blindness).
- Visual loss progresses very rapidly, and usually maximum by the end of second week.
- A discomfort or pain behind the eyeball, especially when the eye is moved superiorly (due to involvement of the origin of superior rectus muscle).

Signs

- Visual acuity may be 6/60 or even less, although some patients have only mild loss of vision (6/12).
- Presence of local tenderness, especially near the site of attachment of superior rectus tendon.
- Pupillary reactions may be:
  - Sluggish and ill-sustained.
  - Relative afferent pupillary defect (Marcus-Gunn's pupil).
- Impaired colored vision.
- A delayed dark adaptation.
- Field of vision—central, centrocecal or paracentral scotoma.

Ophthalmoscopic Features

- ***Optic neuritis*** is most common in children and is frequently post-viral.
  Here, the intraocular portion of the optic nerve is involved and gives rise to engorgement and edema of the optic disc with obliteration of the physiological cup, small hemorrhages on the disc and inflammatory cells into the adjacent vitreous.
- ***Retrobulbar neuritis*** is the most common findings in adults. Here, the optic disc and retinal nerve-fiber layer are normal, as the site of involvement is behind the globe.
- ***Neuroretinitis*** is the least common type. It has all the signs of optic neuritis in addition to a macular star (Fig. 19.7). This form of optic neuritis is not usually associated with multiple sclerosis.

Fig. 19.7: Neuroretinitis with macular star

Differential Diagnosis

- ***Papilledema:*** Discussed in table *(see page 356)*.
- ***Pseudoneuritis (pseudopapillitis)*** is seen in:
  - High hypermetropia
  - Congenital malformed disc
  - Myelinated nerve fibers
  - Optic nerve drusen or
  - Opacity in the media (nuclear sclerosis).

In these cases, the disc appears hyperemic with blurred margin, but is not significantly elevated, there are no vascular changes and the condition is stationary.

Investigations

Usually X-ray skull, CT scan (computed tomography scan) and MRI (magnetic resonance imaging) are unnecessary in a typical case. In some cases, these tests (especially the MRI scan) are required to establish demyelinating disorders. They should also be done in order to exclude papilledema due to intracranial lesions.

Course and Prognosis

- The rate of visual recovery is slower than that of the initial visual loss and usually takes 4–6 weeks time.

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- Almost 90% of the patients recover normal or near normal visual acuity, but minor defects in color vision may persist.
- There is no correlation between the initial visual loss and the final outcome.
- In 10% of cases, the patient may develop partial or total optic atrophy (secondary or postneuritic optic atrophy).

Treatment

- Treatment must be directed to the cause.
- ***Oral prednisolone*** (1–1.5 mg/Kg/day) are given to accelerate the speed of recovery of vision. Intravenous injection of ***methylprednisolone*** (10 mg/Kg/day) for three consecutive days followed by oral prednisolone is advocated recently.
- Simultaneous posterior periocular injection of depot steroid may be tried.
- ***Injection hydroxycobalamine*** (in the form of B_1, B_6 and B_{12} injection) may be added.

TOXIC AMBLYOPIAS (TOXIC OPTIC NEUROPATHIES)

These include a number of conditions in which optic nerve fibers are damaged by exogenous toxins.

- Although, generally described as a form of chronic retrobulbar neuritis, the primary lesion is probably demyelination of the nerve-fiber layer.
- These toxic effects are of two types:
  1. In ***the majority*** initially affect the papillomacular bundle, presenting with ***central*** or ***centrocecal*** (between blind spot and fixation point) scotoma (Fig. 19.8).
  2. In ***the minority,*** there is simply a general depression of vision with contraction of the visual field.

Etiology

The common agents are tobacco, ethyl alcohol, methyl alcohol, lead, carbon disulfide, cannabis, or drug-induced e.g. quinine, ethambutol, isoniazid acid hydrazide (INH) etc.

Fig. 19.8: Centrocecal scotoma

Tobacco-alcohol Amblyopia

- A deficiency of protein and B vitamins (particularly thiamine) is an important association among heavy drinkers and pipe-smokers.
- The onset of visual impairment is gradual, progressive and bilateral.
- Visual field shows bilateral centrocecal scotomas, especially prominent with colored (red) targets.
- ***Treatment*** is by means of weekly injection of 1,000 units of hydroxycobalamine for 3 months. Patient should abstain from drinking and smoking and also be advised to take a balanced diet.

Methyl-alcohol Amblyopia (Hooch Poisoning)

- Poisoning from wood-alcohol occurs sporadically, from drinking methylated spirit (mixed with country-liquor).
- Methyl alcohol is a severe neurotoxin which is lethal to the retina. It is oxidized into formic acid and formaldehyde, in the vitreous, causing destruction of the ganglion cells resulting total optic atrophy.
- It may occur in ***acute*** or ***chronic*** form.

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  - ***In acute form:*** Vomiting, prostration, coma, and sometimes death, if more than one ounce (30 mL) has been consumed.
  - ***If the patient survives:*** Complete blindness is usually noticed within 1–2 days due to total optic atrophy.
  - ***In chronic form:*** Progressive loss of vision due to progressive optic atrophy.
- ***Treatment***
  - Rapid correction of acidosis.
  - Correction of dehydration.
  - Administration of ethyl alcohol, which competes with methyl alcohol for metabolic sites.
  - Injection of vitamin B_1, B_6 and B_{12}.
- ***Prognosis:*** The vision may improve temporarily, but usually relapses, again becoming gradually diminished to complete blindness.

Drug-induced Toxic Amblyopia

Ethambutol

- This is a standard oral chemotherapeutic agent, used in tuberculosis.
- It may produce optic neuritis, resulting in sudden impairment of vision, and color vision (especially for red-green).
- The optic disc is edematous and hyperemic with an eventual central scotoma.
- The optic neuritis is usually ***dose dependent***. It does not occur, if the dosage of not more than 15 mg/Kg/day are administered (***upper limit of safety***).
- ***Treatment:*** The neuritis is reversible when the drug is discontinued, and the majority recover their vision by 6–12 months. Injection hydroxycobalamine has an important role for the successful treatment.

Quinine

- Sudden, total loss of vision may follow even in small dosage (60 mg) in susceptible persons. 150 mg is the maximum amount of quinine sulfate which should be given within 24 hours.
- The pupil is dilated and non-reacting.
- ***Ophthalmoscopically,*** there are extreme narrowing of the arterioles, retinal edema with cherry-red spot and pallor of the optic disc.
- It may be associated with deafness and tinnitus.
- ***Treatment:*** Treated by vasodilator drugs and withdrawal of quinine.

ANTERIOR ISCHEMIC OPTIC NEUROPATHIES

Anterior ischemic optic neuropathy (AION) is a segmental or generalized infarction of the anterior part of the optic nerve caused by the occlusion of short posterior ciliary arteries. It causes a severe visual loss with an ***altitudinal visual field defect*** in the middle-aged, or elderly.

Causes

- ***Atherosclerosis:*** Non-arteritic AION
- ***Giant cell arteritis:*** Arteritic AION.
- ***Systemic collagen vascular disorders:*** Like polyarteritis nodosa or systemic lupus erythematosus.
- ***Miscellaneous:*** Examples are emboli, malignant hypertension, anemia or migraine.

Non-arteritic Anterior Ischemic Optic Neuropathies

- Elderly patient with systemic hypertension.
- Visual loss is monocular, sudden and painless, without any premonitory symptom.
- Moderate to severe loss of vision.
- Typical visual field defect is an altitudinal hemianopia, mainly involving the inferior half (Fig. 19.9).
- ***Ophthalmoscopy*** shows, sectorial (usually upper part) edema with hyperemic disc which may be surrounded by splinter hemorrhages (Fig. 19.10).
- No effective treatment is available.

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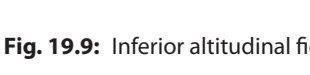


Fig. 19.9: Inferior altitudinal field defect



Fig. 19.10: Anterior ischemic optic neuropathy (arrow showing splinter hemorrhage)

Arteritic Anterior Ischemic Optic Neuropathies

- It occurs in 25% of patients with untreated giant cell arteritis, in elderly individuals.
- The visual loss is sudden, profound and usually permanent. Premonitory symptoms like periocular pain and transient blurring of vision are usually present.
- Visual acuity is reduced to hand movements or counting fingers.
- Visual field defect is profound.
- ***Ophthalmoscopy*** shows a swollen white or pale disc with splinter hemorrhages around. With time, the entire optic disc becomes pale.

Systemic associations include: Headache, scalp tenderness (with ischemic necrosis of the scalp in severe cases), jaw claudication, and pain and stiffness of the proximal muscles.

A ***high erthrocyte sedimentation rate (ESR)*** (more than 100 mm/hour), raised C-reactive protein and temporal artery biopsy confirm the diagnosis.

Treatment: Heavy doses of systemic steroids (intravenous hydrocortisone 250 mg along with prednisolone—80 mg orally), and then with gradual reduction of oral prednisolone over a period of 3 months. In many cases, treatment may be needed for 1–2 years with a small maintenance dose.

PAPILLEDEMA

Papilledema is the bilateral, noninflammatory passive swelling of the optic disc, produced by raised intracranial tension (ICT).

- Papilledema (due to raised ICT) is one of the causes of disc edema, and they are not synonymous. The other causes of disc edema are mentioned.
- Generally, unilateral causes of disc edema should be considered to be due to a local inflammation, vascular disorders, or compressive lesions in the orbit.
- Unilateral papilledema with optic atrophy on the other side, suggests a frontal lobe tumor or olfactory meningioma of the opposite side—the ***Foster-Kennedy syndrome***.
- As a rule, papilledema does not develop if the optic nerve has already become atrophic.

Etiology

- ***Raised intracranial tension*** in papilloedema may be due to:
  - ***Congenital stenosis*** of ventricular system.
  - ***Blockage of the ventricular system*** by space occupying lesions, e.g. tumors abscess, hemorrhage, etc.
  - ***Obstruction of cerebrospinal fluid (CSF) absorption by the arachnoid villi***—e.g. direct blockage of villi by blood or protein, or due to cavernous sinus thrombosis.

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- ***Hyper secretion of CSF*** by the choroidal plexus tumors.

Any intracranial tumor in any position excepting of medulla oblongata may induce papilledema. It is most evident with tumors in the posterior fossa which obstruct the aqueduct of Sylvius, and least with pituitary tumors. The site of the tumor is, thus, more important than its nature, its size and rate of growth.

- ***Idiopathic intracranial hypertension (IIH)*** or ***pseudotumor cerebri*** causes gross papilledema without any striking neurological changes. It occurs mainly following intake of certain drugs, e.g. tetracycline, nalidixic acid, high doses of vitamin A, corticosteroids, oral contraceptive, etc. Presence of normal-size ventricles (as seen in CT scan of brain) and CSF study by lumbar puncture confirm the diagnosis.
- Malignant hypertension.

Causes of disc edema
- Optic papillitis, neuroretinitis
- Ocular hypotony
- Anterior ischemic optic neuropathy
- Leber's optic neuropathy
- Optic nerve glioma or meningioma
- Central retinal venous occlusion
- Orbital cellulitis
- Metastasis on the optic nerve head
- Chronic anterior uveitis.

Causes of papilledema
- ***Congenital:*** Examples are aqueductal stenosis, craniosynostosis, etc.
- Space-occupying intracranial lesions.
- Head injury.
- ***Microbial infection:*** Examples are chronic meningitis, encephalitis, etc.
- ***Intracranial vascular lesions:*** Examples are subarachnoid hemorrhage, cavernous sinus thrombosis, etc.
- Malignant hypertension (Fig 19.11).
- ***Metabolic disorder:*** Examples are Addison's disease, diabetic ketoacidosis, hypoparathyroidism, etc.
- ***Pseudotumor cerebri***: With toxicity of drugs, like tetracycline, nalidixic acid, vitamin A, oral contraceptives and corticosteroids.
- ***Blood dyscrasias:*** Examples are lukemias, polycythemia, etc.
- ***Miscellaneous:*** Examples are sarcoidosis, serum sickness, status epilepticus, etc.

Fig. 19.11: Disc edema in malignant hypertension

Symptoms

General symptoms
- Headache (made worse by coughing or straining).
- Vomiting without nausea.
- Focal neurological deficit with changes in level of consciousness.

Ocular symptoms
- ***Visual acuity remains fairly normal*** until the late stage of the disease.
- ***Amaurosis fugax:*** It is a transient black out of vision for a few seconds may be present in some patients.
- Visual fields are usually normal, unless the lesion itself causes a hemianopia.

Signs

Pupillary reactions are normal until the secondary optic atrophy sets in.

Ophthalmoscopy: The features are different in the different stages of the disease:
- ***Early papilledema (Fig. 19.12)***
  - ***Earliest change*** is blurring of superior and inferior margins of the disc.
  - Disc hyperaemia and dilated capillaries.
  - Spontaneous venous pulsation is absent.
  - Splinter hemorrhages at or just off the disc-margin.
  - Normal optic disc-cup is preserved.
- ***Established papilledema (Fig. 19.13)***

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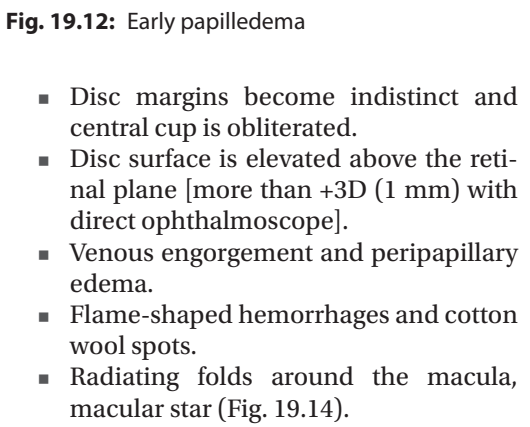


Fig. 19.12: Early papilledema

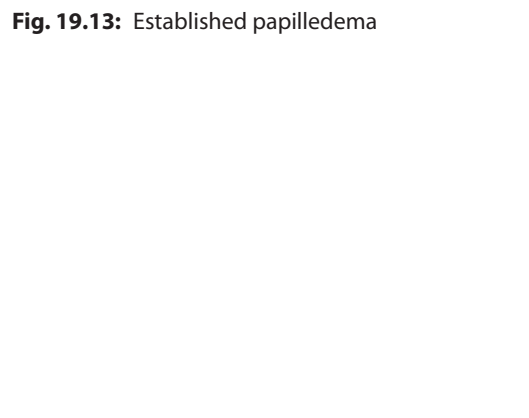


Fig. 19.13: Established papilledema

- Disc margins become indistinct and central cup is obliterated.
- Disc surface is elevated above the retinal plane [more than +3D (1 mm) with direct ophthalmoscope].
- Venous engorgement and peripapillary edema.
- Flame-shaped hemorrhages and cotton wool spots.
- Radiating folds around the macula, macular star (Fig. 19.14).

- ***Chronic papilledema (Fig. 19.15)***
  - Central cup remains obliterated.
  - The hemorrhagic and exudative components gradually resolve.
  - Optic disc appears as a champagne cork (Fig. 19.16).
- ***Atrophic papilledema***
  - Retinal vessels are attenuated with perivascular sheathing.
  - Dirty-white appearance of the optic disc due to reactive gliosis leading to secondary optic atrophy.

Fig. 19.14: Established papilledema with macular star (Note the disc elevation compared to normal disc).

Field Changes

- ***In early stage*** there is no field defect.
- ***In established stage*** there is enlargement of the blind spot.
- ***In chronic stage*** there is associated peripheral constriction of the visual field with appearance of nerve-fiber bundle defects.
- ***Finally,*** there is total loss of visual field.
- ***In addition,*** the lesion itself may cause hemianopic or quadrantic field defect.

Treatment

- Treatment must be directed to detect the cause, and then to treat it.
- Surgical decompression of the optic nerve may be indicated to preserve vision.

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Fig. 19.15: Chronic papilledema—Patton's line

Fig. 19.16: Chronic papilledema—champagne cork appearance

Difference between papilledema and optic neuritis

Features	Papilledema	Optic neuritis
History	Headache and vomiting, initially no visual symptom	Sudden loss of vision: History of fever or upper respiratory tract infection
Laterality placement	Usually bilateral	Usually unilateral
Visual acuity	Remains normal until late stage	Severely reduced (6/60 or less)
Pain or tenderness of the eyeball	Absent	May be present
Pupil	Normally reacting	Relative afferent pupillary defect (Marcus-Gunn's pupil)
Disc swelling	More than +3D (1mm) elevation in the established case	Usually + 2D to +3D elevation
Hemorrhage and exudates	More in established case	Relatively less
Visual fields	Enlargement of blind-spot and later gradual constriction Colored field not much affected	Central or centro-caecal scotoma More with colored objects
CT scan or MRI	Intracranial space occupying lesion may be detected	Demyelinating disorder may be seen
Recovery of vision	May not be complete even after treatment	Usually complete with adequate treatment

Abbreviations: **CT**—Computed tomography; **MRI**—Magnetic resonance imaging

OPTIC ATROPHY

Optic atrophy is the degeneration of optic nerve fibers with loss of their myelin sheaths, and characterized by the pallor of the optic disc.

The pallor of the disc is not due to atrophy of the nerve fibers, but to loss of vascularity owing to obliteration of the disc capillaries.

Classification

Etiological

- ***Primary optic atrophy:*** It occurs without any local disturbance, but associated with CNS disease, or without any discoverable cause. The important causes are:
  - Tabes dorsalis (classical cause)
  - Multiple sclerosis (most common)
  - Leber's optic atrophy

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Fig. 19.17: Postpapilledemic optic atrophy

Fig. 19.18: Primary optic atrophy

- Glaucomatous optic atrophy
- Tumor compression of the optic nerve.

- ***Secondary optic atrophy:*** It is preceded by swelling of the optic disc e.g.:
  - Papilledema (Fig. 19.17)
  - Optic neuritis
  - Neuroretinitis.
- ***Consecutive optic atrophy:*** Optic atrophy that follows extensive disease of the retina, causing extensive degeneration of the ganglion cells, as in:
  - Retinitis pigmentosa
  - Central retinal arterial occlusion
  - Extensive retinochoroiditis.

Anatomical

- ***Ascending optic atrophy:*** If the lesion is in the retina, an ascending optic atrophy occurs, which terminates at the lateral geniculate body, e.g. retinitis pigmentosa, central retinal arterial occlusion, etc.
- ***Descending optic atrophy:*** It follows diseases involving the optic nerve fibers anterior to the lateral geniculate body, and terminates at the optic disc, e.g. chiasmal compression, retrobulbar neuritis, etc.

Symptoms

- The chief symptom is loss of central or peripheral vision which may be sudden or gradual.
- Impairment of color vision (red-green).

Signs

- Visual acuity is impaired in proportion as the nerve fibers die.
- ***Relative afferent pupillary defect:*** In total optic atrophy the pupil is dilated and immobile.
- ***Ophthalmoscopically***
  - ***Primary optic atrophy (Fig. 19.18)***
    - Pallor of the disc, with gray or white color.
    - The margins of the disc are sharply defined, and peripapillary retina is normal looking.
    - There is slight atrophic cupping.
    - The blood vessels are only slightly constricted without any sheathing.
  - ***Secondary optic atrophy (Fig. 19.19)***
    - Pallor of the disc (dirty-gray color) with blurred margins.
    - Physiological cup is full and lamina cribrosa is obscured.
    - Narrowing of the blood vessels with sheathing.
    - Gliosis over the disc surface extending towards the peripapillary retina.
  - ***Consecutive optic atrophy (Fig. 19.20)***
    - Yellowish-waxy pallor of the disc.
    - Disc margins are less sharply defined.
    - Marked narrowing (even obliteration) of the retinal blood vessels.

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Fig. 19.19: Secondary optic atrophy—neuroretinitis

Fig. 19.20: Consecutive optic atrophy—retinitis pigmentosa

Investigations

- ***Field of vision:*** In partial optic atrophy, the central vision is depressed with concentric contraction of the visual field, according to the cause.
- Fluorescein angiography of the optic nerve head.
- Visual evoke response (VER) is useful, especially in children.
- Total neurological evaluation by a neurophysician, and investigations, like, X-ray skull, CT scan and MRI of brain and optic nerve, etc.

Treatment

- Treatment of the cause.
- As the prognosis is usually poor, community based rehabilitation is important in bilateral cases.

Temporal pallor (Figs 19.21A and B)
Most forms of partial optic atrophy involve a loss of temporal fibers including the papillomacular bundles. This results in temporal pallor, which is an important sign of an evanescent neuritis as in multiple sclerosis. But, this should be confirmed by special investigations, since, temporal side is normally relatively pale, because the retinal vessels emerge from the nasal side and the temporal side is normally less vascular.

OPTIC NERVE TUMORS

Discussed in Chapter 21, page 382.

A B

Figs 19.21A and B: Temporal pallor. **A.** Right eye; **B.** Left eye

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20 CHAPTER

Intraocular Tumors

Tumors of the uvea and retina are rare. They are of great importance, since most of them are malignant and endanger the life of the patient.

IRIS TUMORS

Iris Nevus (Fig. 20.1)

- It consists of slightly elevated, localized, discrete, small pigmented mass and is composed of proliferated melanocytes.
- The nevi are not progressive, and do not distort the pupil.
- As a rule, this is benign, but occasionally it takes on malignant proliferation.

Such a development is suggested by an increase in size, vascularization and pigmentation, which sometimes distorts and everts the pupillary margin ***(ectropion uveae)***.

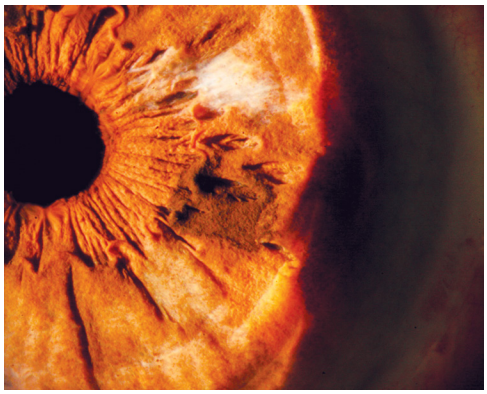


Fig. 20.1: Iris nevus

Malignant Melanoma (Figs 20.2A and B)

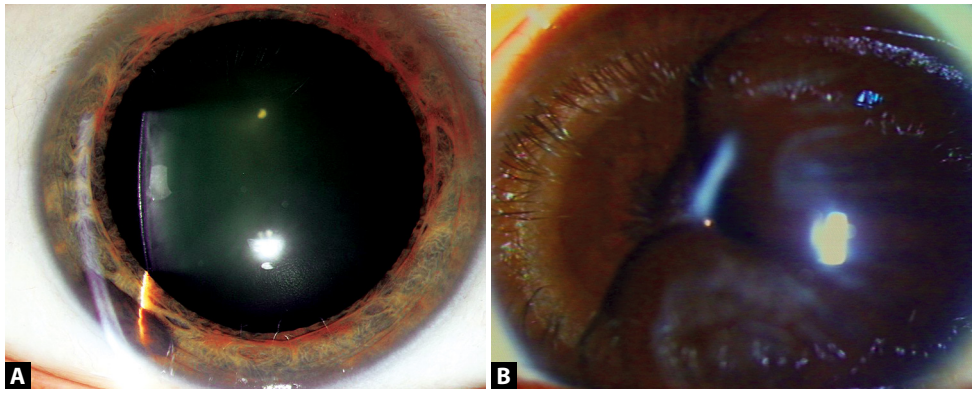
- It often arises from pre-existing nevus.
- It is usually symptomless and the tumor is noticed by the relatives or friends as a brown mass on the iris surface.
- It has blood vessels growing in it and may be associated with seeding.
- Pupil is distorted and there may be glaucoma when it extends into the anterior chamber angle.
- The growth should be watched carefully, and is best treated by broad iridectomy or iridocyclectomy if the angle is involved.

CILIARY BODY TUMORS

Medulloepithelioma (Diktyoma)

- Intraocular medulloepithelioma arises in the non-pigmented epithelium of the ciliary body, and resembles embryonic tissue of the primitive retina and ciliary body.
- It is thought to be congenital, though it occurs between 4 and 12 years of age.
- The chief symptoms are pain and poor vision.
- A white pupil (leukocoria), secondary cataract and secondary glaucoma are often present.
- Enucleation is often delayed due to failure to diagnose the condition.

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Figs 20.2A and B: **A.** Iris melanoma at anterior chamber angle; **B.** Malignant melanoma—iris

Malignant Melanoma

Ciliary body melanoma is more common than iris melanoma, but less common than the choroidal melanoma.

- It tends to involve either the choroid or the iris, or both.
- It can not usually be visible unless the pupil is widely dilated.
- Clinically, it may present in a variety of ways:
  - As subluxation of the lens with secondary glaucoma.
  - As dilated episcleral blood vessels in the same quadrant as with the tumor ***(sentinel vessels)***.
  - As retinal detachment.
  - As diffuse mass in a ring around the ciliary body (Fig. 20.3).
- Because of its location and minimal symptoms, the diagnosis may be often delayed.
- Indirect ophthalmoscopy, three-mirror gonioscopy, transillumination test and USG B-scan or ultrasonic biomicroscopy (UBM) are helpful for early diagnosis.
- Treatment is by enucleation (for large tumors), or by local resection (for small tumors).
- Since, ciliary body melanomas have a low degree of malignancy, the prognosis is usually good.

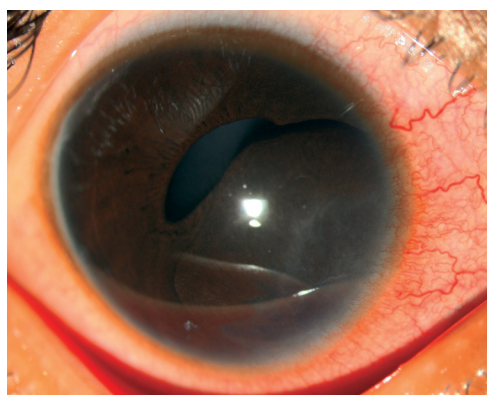


Fig. 20.3: Ciliary body melanoma

CHOROIDAL TUMORS

Benign Melanoma (Nevus) (Fig. 20.4)

- Typical benign melanomas of the choroid are flat or slightly elevated, oval or circular state-gray lesions.
- They are usually less than three disc diameters in size and occur most frequently at the posterior half of the fundus.
- They are usually present at birth and maximum growth occurs during puberty.
- Most of the benign melanomas are asymptomatic, although those near the macula may present with impairment of central vision.

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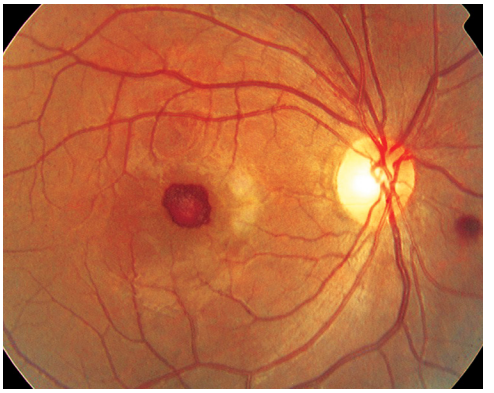


Fig. 20.4: Benign melanocytoma of the choroid

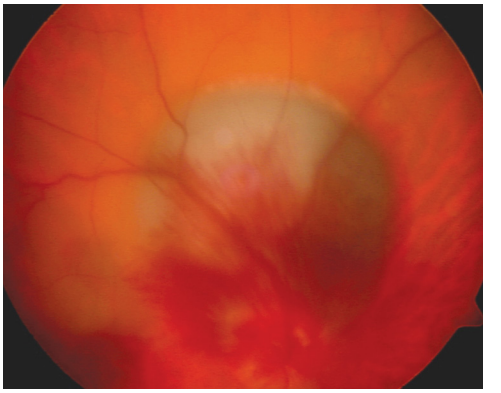


Fig. 20.5: Choroidal melanoma

- Benign melanomas may become malignant. An increase in size, elevation of the mass, irregular pigmentation over the tumor and visual field loss suggest malignancy.
- Treatment is not indicated except the patient should be followed up regularly with serial fundus photographs.

MALIGNANT MELANOMA OF THE CHOROID

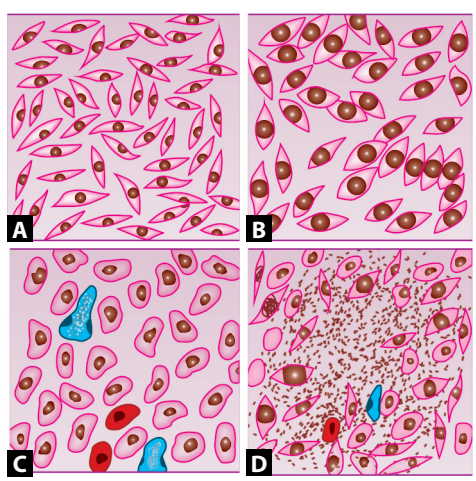
The most common primary intraocular tumor in adults is malignant melanoma of the choroid (Fig. 20.5).

- The average age of the patients with choroidal melanoma is 50 years.
- They are extremely rare among blacks, and slightly more common in males.
- Most malignant melanomas develop from pre-existing benign melanomas (nevi).
- They originate most frequently in the outer layers of the choroid, and may spread like a carpet between the sclera and the Bruch's membrane.
- Tumors may remain quiescent for many years, and then without apparent reason, suddenly begin to grow rapidly.
- Eventually, the Bruch's membrane perforates, and the growth appears as mushroom or collar-button shaped mass.

Histological Classification (Callender's)

Uveal malignant melanomas can be divided into six types (Figs 20.6A to D) according to cellular features:

1. ***Spindle-A cell type:*** Tumors composed of this cell type have the best prognosis.
2. ***Spindle-B cell type:*** It is having the second best prognosis.
3. ***Fascicular type:*** The cells in a palisading arrangement are composed of either



Figs 20.6A to D: Histological types of uveal melanoma. **A.** Spindle-A type; **B.** Spindle-B type; **C.** Epitheloid type; **D.** Mixed type

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spindle-A or spindle-B cells, and has the same prognosis as primary cell type.

4. ***Epithelioid cell type:*** This is the least common type and these cells carry the worst prognosis.
5. ***Mixed-cell type:*** This is composed of a combination of spindle and epithelioid cells with intermediate prognosis. This is the most common type of malignant melanoma.
6. ***Necrotic type:*** This is the tumor in which the specific cell type can not be recognized. The necrotic process may initiate a severe inflammatory reaction which may be mistaken clinically, as endophthalmitis or uveitis.

Clinically, malignant melanoma of the choroid is commonly divided into four stages:

1. Quiescent stage
2. Glaucomatous stage
3. Stage of extraocular extension
4. Stage of metastasis.

The chief symptoms result from the exudative retinal detachment and secondary glaucoma caused by increase in the choroidal volume. The patient may present with decreased visual acuity, or a defect in the visual field, and ocular pain accordingly.

In some of the cases, the patient is ***asymptomatic*** and the tumor is detected by a routine ophthalmoscopic examination.

Ophthalmoscopically

- The tumor is invariably unilateral and solitary.
- A typical melanoma appears as a pigmented and elevated oval mass.
- The color of the mass is frequently brown, although it may be mottled with black or dark-brown pigment or it may be amelanotic (Fig. 20.7).
- As the tumor grows a brown exudative detachment results owing to break in the Bruch's membrane.
- An accumulation of orange pigment ***(lipofuscin)*** in the retinal pigment epithelium is commonly seen over the detached area but is not diagnostic.

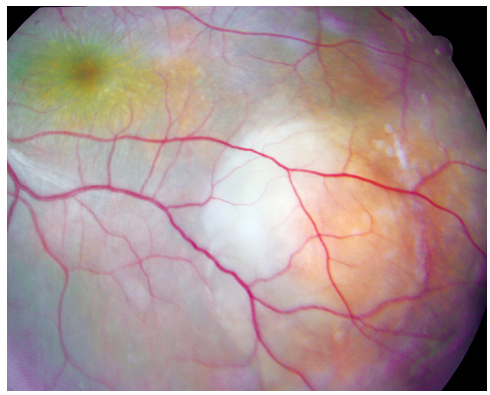


Fig. 20.7: Choroidal amelanotic melanoma

- ***Other ocular features include:*** choroidal folds, subretinal hemorrhage, vitreous hemorrhage, secondary glaucoma cataract and posterior uveitis.
- The lymph nodes are not affected, but distant metastasis in the liver and elsewhere are the common cause of death.

Investigations

- ***Medical evaluation:*** It is to exclude the possibility of a metastatic tumor of the choroid and to check for distant metastasis from the primary ocular growth. Liver function tests and X-ray chest are most valuable.
- ***Indirect ophthalmoscopy*** for better depth assessment.
- Three-mirror and contact lens examinations of the fundus.
- ***Transillumination*** test is useful in differentiating a pigmented tumor from a non-pigmented lesion.
- Fundus photography.
- ***Fluorescein angiography:*** In general, most melanomas show a mottled fluorescence during the arteriovenous phase of the angiogram with a progressive staining of the lesion and prolonged retention of the dye.
- ***Ultrasonography:*** It is specially useful in detecting the presence of a tumor in an eye with hazy media. Both A-scan and B-scan

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are helpful to detect the solid nature of the mass.
- ***P32-uptake:*** Malignant melanoma has an increased rate of phosphate uptake and retains ***P32-isotope*** longer than other non-malignant lesions.
- ***Visual field examination:*** It is of limited value.
- ***CT scan and MRI:*** They are valuable in detecting the extraocular extension.
- ***USG-guided tissue biopsy:*** It is to obtain cellular aspirates for analysis.

Differential Diagnosis

Rhegmatogenous retinal detachment (RD), metastatic tumor of the choroid, choroidal hemangioma, large choroidal nevus and choroidal detachment.

Management

The correct management at present is controversial as the traditional treatment of enucleation has been questioned by many ophthalmologists, especially for small tumors.
- ***Observation:*** It is indicated for small tumors, where the diagnosis is not certain; patients over the age of 65 years; patients with liver metastasis; and with a slow growing tumor with normal vision.
- ***Enucleation:*** It is indicated for very large melanomas, especially, if all useful vision has been irreversibly lost. The over all 5 years survival rate is 75% after enucleation for all types of melanoma.
- ***Radioactive scleral plaques (Cobalt-60 or Iodine-125):*** They are suitable for small to medium size tumors.
- ***Heavy-charged particle irradiation (protons or helium):*** It is a new approach to radiotherapy and may be better than enucleation in eyes with large tumors. It is not available widely.
- ***Photocoagulation:*** Xenon arc photocoagulation is better than Argon laser and indicated for small tumors away from the fovea.
- ***Choroidectomy (local resection):*** This difficult and complicated operation may be useful for the selected peripheral tumors.
- ***Palliative therapy:*** Chemotherapy and immunotherapy may be useful in cases with distant metastases.

METASTATIC CARCINOMA OF THE CHOROID (FIG. 20.8)

Metastatic choroidal tumors are probably the most common type of intraocular malignancy (which are often undetected).
- The most frequent primary sites are the bronchus (bronchogenic carcinoma) in males and breast (breast carcinoma) in females (Fig. 20.9).
- Other primary sites are kidney, testis and gastrointestinal tract. The prostate is an extremely rare primary site.
- Choroidal metastases have a definite predilection for the posterior pole of the fundus, with early loss of central vision.
- Typically, they appear as solitary or multiple, creamy-white, placoid or oval lesions which infiltrate laterally.
- They rarely become significantly elevated, and have ill-defined borders.
- A careful examination of the opposite eye is important as bilateral metastases are common.

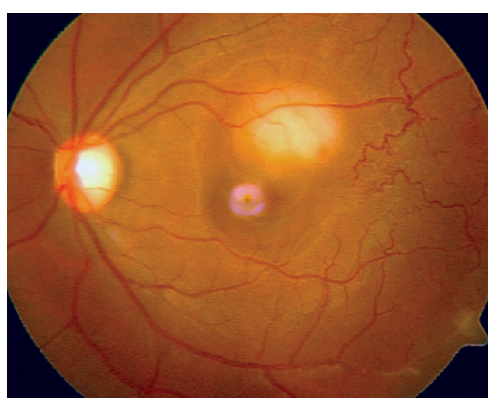


Fig. 20.8: Metastatic carcinoma of the choroid—from bronchogenic carcinoma

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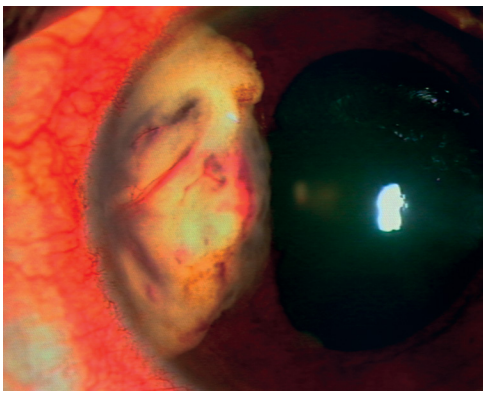


Fig. 20.9: Metastatic carcinoma of the iris from breast carcinoma

- ***Diagnosis*** depends mainly on accurate history and careful general physical examination.
- ***Treatment*** must be directed to the primary disease. Enucleation is contraindicated unless the eye is painful and blind. Palliative treatment is carried out with chemotherapy in conjunction with external-beam irradiation.

CHOROIDAL HEMANGIOMA

This is rare and is associated in about half of the patients with skin angioma (as in ***Sturge-Weber syndrome***).

- Typically, it appears as a dome-shaped or diffuse, reddish-orange lesion mostly at the posterior pole.
- Frequently, secondary changes, like exudative retinal detachment, pigment mottling and cystoid degeneration may occur.
- A useful clinical sign is the blanching of the lesion with pressure on the globe.
- P32-uptake test, ultrasonography (USG) and fluorescein angiography are helpful to differentiate it from other lesions (e.g. metastatic tumors and amelanotic melanoma).
- ***Treatment:*** Asymptomatic tumors do not require treatment. If the vision is threatened, extensive photocoagulation should be applied around the tumor.

RETINOBLASTOMA

It is the malignant intraocular tumor originating in the outer nuclear layer of the retina and strongly resembles fetal retina.

Etiology

- ***Incidence:*** 1 in 20,000 live births.
- ***Laterality:*** 25–30% of cases are bilateral.
- ***Age:*** The neoplasm is probably congenital, but the average age of diagnosis is 18 months. The vast majority of cases become clinically apparent before the age of 3 years. It is exceptional after the age of 7 years.
- ***Inheritance:*** A positive family history is present only in 6% of cases (mostly in bilateral cases). Mode of inheritance is autosomal dominant. Remaining 94% of cases are sporadic.
- ***Chromosomal abnormalities:*** In 5% of cases, it is associated with deletion of the long arm of chromosome-13 and with trisomy-21.

Other Facts

- Orbital sarcoma may occur in the irradiated field in children with enucleated retinoblastoma.
- Osteogenic sarcoma of femur and skull bones are more common in patients who have survived from bilateral retinoblastoma.
- Very rarely pinealoblastoma may be associated with some cases of bilateral retinoblastoma and then it is called ***trilateral retinoblastoma***.

Clinical Features

Clinical Presentation

- ***Leukocoria or amaurotic cat's eye reflex:*** The most common mode of presentation (60% of cases) and is due to reflection of light from the yellowish-white mass in the retrolental area (Fig. 20.10).
- ***Squinting of the eye:*** The second most common mode of presentation (20%).

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Fig. 20.10: Right sided retinoblastoma—amaurotic cat's eye reflex



Fig. 20.11: Bilateral retinoblastoma with proptosis of the left eye due to orbital involvement

- ***Secondary glaucoma*** which may be associated with buphthalmos.
- ***Proptosis*** (due to orbital involvement) (Fig. 20.11).
- ***Endophthalmitis or anterior uveitis*** may also be a presenting feature.
- ***Visual difficulties:*** Noticed by the parents.
- Nystagmus in bilateral cases if it occurs within 6 months.

Signs

- Unilateral dilated pupil
- White pupillary reflex
- Strabismus
- Intraocular tension is higher
- ***Heterochromia of the iris:*** It is due to rubeosis irides owing to massive posterior segment ischemia.

Stages of Retinoblastoma

Stage 1: The quiescent stage (6–12 months).
Stage 2: The glaucomatous stage.
Stage 3: The stage of extraocular extension.
Stage 4: The stage of metastasis.

Spread of Retinoblastoma

- ***Direct spread***
  - Into the intraocular tissues.



Fig. 20.12: Retinoblastoma—extensive direct extension into the orbit

  - Into the extraocular tissues:
    - Extension into the central nervous system (CNS) via optic nerve.
    - Extension into the orbit as a fungating mass (Fig. 20.12).
- ***Lymphatic spread:*** Along the orbital lymphatics into the preauricular and cervical lymph nodes.
- ***Spread by blood stream:*** Choroidal invasion is the main route of escape. Most common sites are the bones and liver, and lung is the least common site.

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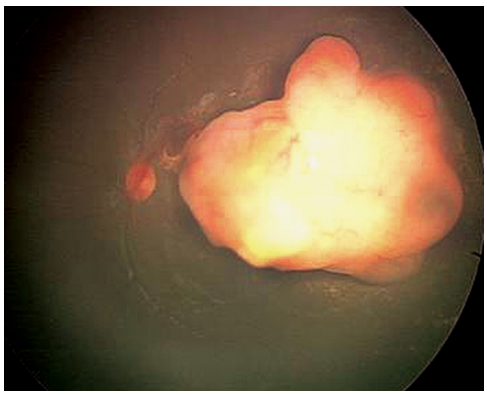


Fig. 20.13: Large endophytic retinoblastoma

Diagnosis

Indirect Ophthalmoscopy

Indirect ophthalmoscopy with scleral indentation, following full dilatation of pupil should be performed in both eyes under general anesthesia. Tumors arising anterior to the equator are missed with direct ophthalmoscope. The appearance of the tumor may be:

Endophytic type (glioma endophytum) (Fig. 20.13)

- This projects from the retina into the vitreous cavity.
- White or pearly-pink colored mass with sharply demarcated margin.
- Presence of calcium deposits in most cases gives the appearance of ***cottage-cheese***.
- Multiple seeding of the tumor cells may be seen in the vitreous cavity.

Exophytic type (glioma exophytum)

- This grows in the subretinal space and gives rise to a total exudative retinal detachment.
- The tumor itself is difficult to visualize.

Plane type (glioma planum): It is difficult to visualize the tumor.

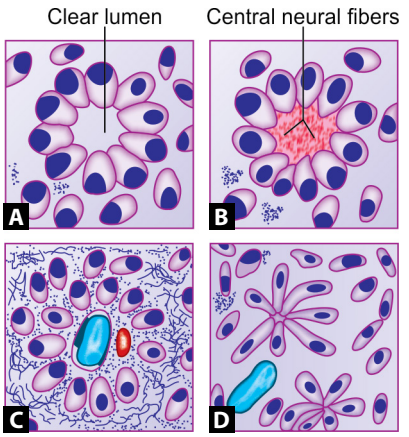
Special investigations

- ***X-ray of the orbit:*** It is for the presence of calcification and erosion of the optic foramen.
- ***Ultrasonography:*** USG B-scan shows presence of tumor with calcification.
- ***CT scan of the orbit and brain*** to demonstrate calcification and CNS spread.
- ***Aqueous humor paracentesis***: It is for cytology and lactate dehydrogenase (LDH) enzyme assay. An aqueous to serum LDH ratio of greater than 1.0 is suggestive of retinoblastoma.
- ***Carcinoembryonic antigen (CEA)*** may be found in retinoblastoma patient.
- ***Enzyme-linked immunosorbant assay (ELISA)*** test to differentiate it from Toxocara endophthalmitis.
- ***Lumbar puncture*** and bone marrow aspiration for the evidence of metastases.
- ***Fine needle aspiration cytology (FNAC)*** when other diagnostic tests are inconclusive.
- ***Fluorescein angiography:*** It is not done routinely.

Histopathology

- ***Gross examination:*** A chalky-white, friable tumor with dense foci of calcification.
- ***Microscopic:*** Retinoblastoma consists of small, round, densely packed cells with large basophilic nuclei. It may be well differentiated or poorly differentiated. Well differentiated retinoblastoma is characterized by presence of ***rosettes*** and ***flurettes*** (Figs 20.14A to D).
  - ***Flexner-Wintersteiner rosette:*** Cells arrange in a single layer around a central clear lumen. This is true rosette and highly characteristic.
  - ***Homer-Wright rosette:*** It is a radial arrangement of cells around a central triangle of neural fibers (rather than clear lumen) and is mainly found in neuroblastoma or medulloblastoma.
  - ***Pseudorosette:*** In necrotic retinoblastoma, several layers of cells may be seen around a blood vessel, within the areas of extensive necrosis with the formation of pseudorosette.

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Figs 20.14A to D: Microscopic appearance of retinoblastoma. **A.** Flexner-Wintersteiner rosette area of necrosis; **B.** Homer-Wright rosette; **C.** Pseudorosette; **D.** Flurettes

  - ***Flurette:*** It is composed of group of tumor cells and contains pear-shaped eosinophilic processes which project through a fenestrated membrane.
- ***Histology of metastatic lesions*** outside the retina shows a change of character of the cells. They resemble sarcomatous cells and rosettes are in rarity.

Treatment

- ***Enucleation:*** Excision of the eye with a long optic nerve stump is the treatment of choice for most tumors affecting the first eye. The treatment of the second eye depends on the size and location of the tumor.
- ***Exenteration:*** Exenteration of the orbit is indicated in case of orbital involvement of the tumor.
- ***Radiotherapy:*** It is done by external irradiation or by a Cobalt-60 scleral plaque. Cataract, radiation retinopathy and scleral necrosis may occur. It is indicated in small to medium size tumors and for recurrent or residual tumor of the orbit.
- ***Photocoagulation:*** Xenon arc photocoagulation is useful for certain small retinoblastomas not involving the optic nerve or the macula.
- ***Cryotherapy:*** It is useful for small peripheral tumors. The tumor should be frozen by triple-freeze and thawing technique.
- ***Chemotherapy:*** It is indicated following enucleation in advanced cases and in presence of distant metastases. Cyclophosphamide and vincristine are used every 3 weeks for 12–15 months.
- ***Combination of therapy:*** To achieve best results.

Genetic counseling: This is important in bilateral cases and if two or more cases of retinoblastoma occur in a family.

- Normal parents with one affected child in sporadic case—the chance of transfer to next sibling is 1.3–6%.
- When an affected person survives to maturity and procreates—chance of having retinoblastoma in offspring is about 25–30% in unilateral case, and the risk is more in bilateral cases.

Prognosis: The overall mortalities from retinoblastoma are between 15 and 20%. Mortality rate is higher with optic nerve involvement, large size tumor, poorly differentiated tumor and when there is choroidal invasion.

Follow-up

Follow-up is extremely important in case of treated retinoblastoma.

In general, the rule is every 4 months until 3 years of age, every 6 months until 4 years of age and yearly for next 3 years.

Examination under anesthesia (EUA) is done in each visit. The examination includes:

- Inspection and palpation of the socket after removing the prosthesis.
- Indirect ophthalmoscopy of the fellow eye.
- USG B-scan of the fellow eye to know the status of the tumor (if treated with other methods).

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DIFFERENTIAL DIAGNOSIS OF LEUKOCORIA IN CHILDREN

The common causes of leukocoria or white pupillary (Figs 20.15 and 20.16) (***amaurotic cat's eye***) reflex in children are:

- Retinoblastoma
- Pseudoretinoblastoma (pseudoglioma)
- Congenital cataract
- Retinopathy of prematurity
- Toxocara endophthalmitis
- Persistent hyperplastic primary vitreous
- Retinal dysplasia
- Coat's disease
- Choroidal coloboma.

Retinoblastoma

- Usually unilateral (Fig. 2.15)
- Usual age at diagnosis is 18 months
- No inflammatory sign in anterior segment
- Ophthalmoscopy shows, a pearly-white mass with presence of secondary calcification
- Lens is usually transparent
- Intraocular pressure is high
- The lesion is progressive
- X-ray orbit, USG and enzyme assay of the aqueous confirm the diagnosis.

Congenital cataract (Fig. 20.16)

- Unilateral or bilateral
- Opacity in the lens clearly indicates the presence of cataract
- It does not cause diagnostic problem, but a cataract does not exclude other causes of leukocoria especially persistent hyperplastic primary vitreous (PHPV).

Retinopathy of prematurity

- History of prematurity and low birth weight are present.
- History of prolonged exposure to oxygen.
- Bilateral in 100% of cases.
- First noted in neonatal period.
- Presence of tractional retinal detachment.
- Intraocular pressure is normal.

Toxocara endophthalmitis (Fig. 20.17)

- Usual presentation is between 2 and 9 years of age.
- History of contact with pet cat or dog.
- Usually unilateral.
- Signs of inflammation in anterior segment and vitreous are present.
- Intraocular pressure is low and eventually the eye may be phthisical.
- ELISA test is very diagnostic.

Persistent hyperplastic primary vitreous (Fig. 20.18)

- Developmental disorder of the vitreous.
- Usually unilateral, and first noted in the neonatal period.



Fig. 20.15: White pupillary reflex—retinoblastoma in left eye



Fig. 20.16: White pupillary reflex—congenital cataract

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Fig. 20.17: White pupillary reflex—toxocariasis



Fig. 20.18: White pupillary reflex—persistent hyperplastic primary intreous

- Associated with microphthalmos.
- Lens may be cataractous.
- Elongated ciliary processes are visible through the dilated pupil.
- Intraocular pressure may be high.
- USG B-scan confirms the diagnosis in presence of cataract.

Retinal dysplasia

- Unilateral or bilateral usually present at birth.
- Pink or white retrolental mass.
- The eye is microphthalmic with shallow anterior chamber and elongated ciliary processes.
- It is due to failure of the retina to develop normally.
- Usually associated with severe systemic abnormalities.
- USG B-scan helps to differentiate it from PHPV in unilateral cases.

Coats' disease (Fig. 20.19)

- It is almost always unilateral.
- It occurs primarily in older boys (although, it may occur at an early age).
- Large areas of retinal or subretinal exudates with cholesterol crystals.
- Dilated and tortuous retinal blood vessels at the posterior pole.
- Exudative detachment as a retrolental mass occurs over a period of years.

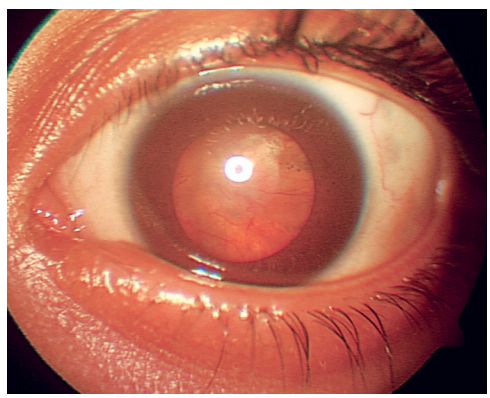


Fig. 20.19: White pupillary reflex—Coats' disease

- It is a severe form of retinal vascular telangiectasia.
- Fundus fluorescein angiography may be required to confirm the diagnosis.

Choroidal coloboma

- Leukocoria is only with a large complete choroidal coloboma.
- Usual presentation is at birth.
- Unilateral or bilateral.
- They are always located inferonasally.
- The eye is usually microphthalmic in case of large coloboma.
- Ophthalmoscopy shows the defect is at the embryonic position of choroidal fissure with shiny-white sclera.

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21 CHAPTER

Diseases of the Orbit

Symptoms of orbital diseases	
Symptoms	**Causes**
Forward displacement of the globe (proptosis) • Unilateral • Bilateral • Rapidly progressive • Slowly progressive • Periodic exacerbation and remission with upper respiratory tract infection (URTI)	• Space occupying lesion within the orbit • Thyroid ophthalmopathy • Orbital cellulitis • Rhabdomyosarcoma • Pseudotumor • Benign neoplasm • Lymphangioma
Backward displacement of the globe (enophthalmos)	• Microphthalmos • Horner's syndrome • Blow-out fracture of the orbital floor
Ocular pain	• Orbital cellulitis • Rhabdomyosarcoma • Pseudotumor
Diplopia	• Space-occupying lesion outside the muscle cone
Visual loss	• Optic nerve compression by the neoplasm • Posterior scleral indentation by the tumors
Congestion of the conjunctiva	• Inflammatory orbital diseases • Thyroid ophthalmology

Signs of orbital diseases	
Signs	**Causes**
Reduced visual acuity	• Optic nerve compression • Exposure keratopathy • Induced hypermetropia • Choroidal folds
Relative afferent pupillary defect (Marcus-Gunn)	• Optic nerve compression • Optic neuritis
Lid lag and retraction	• Thyroid ophthalmopathy
Congestion and chemosis of lids and conjunctiva	• Inflammatory diseases • Rhabdomyosarcoma • Thyroid disorder • Carotid-cavernous fistula

Contd…

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Signs of orbital diseases	
Signs	**Causes**
Increased resistance to orbital compression	• Neoplasms • Thyroid ophthalmopathy
Ocular movement abnormality	• Neurological lesions • Thyroid myopathy • Blow-out fracture • Optic nerve-sheath meningioma
Optic disc • Optic atrophy • Disc edema • Opticociliary shunt vessels	• Optic nerve compression • Raised intraorbital pressure • Optic nerve-sheath meningioma
Pulsatile proptosis	• Carotid-cavernous fistula • Orbital roof defect
Presence of bruit	• Carotid-cavernous fistula
Change of proptosis by head posture	• Capillary hemangioma • Orbital varices
Positive forced duction test (FDT)	• Blow-out fracture • Contracture of muscle

DEVELOPMENTAL ANOMALIES

The orbit and its contents may be affected by a number of developmental anomalies involving the bones of the skull or face.

They are commonly hereditary (autosomal dominant) in nature.

Frequently, there is a characteristic shape of the head or facial appearance, associated with proptosis, papilledema, optic atrophy and strabismus.

Common Abnormalities

Craniosynostosis

It follows premature closure of one or more cranial sutures. This closure causes a complete arrest of bone growth perpendicular to the closed suture and the compensatory growth of the cranium in other diameters, which causes the typical shape of the skull.

Types

- ***Scaphocephaly (boat-shaped skull):*** Premature closure of sagittal suture.
- ***Oxycephaly (tower-shaped skull):*** Premature closure of coronal suture.
- ***Trigonocephaly (egg-shaped skull):*** Premature closure of frontal suture.
- ***Brachycephaly (clover-leaf skull):*** Premature closure of all sutures.

The common clinical features are:

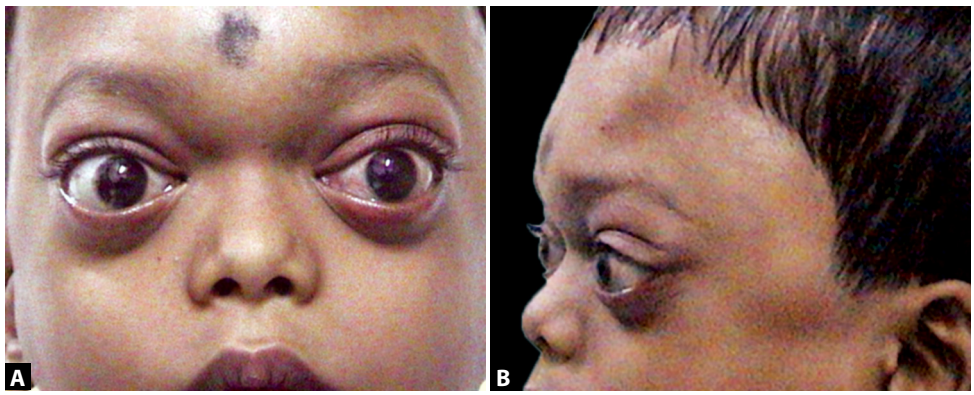
- Bilateral proptosis due to shallow orbit.
- Esotropia or exotropia.
- Papilledema, due to increased intracranial pressure.
- Optic atrophy, primarily due to traction on the optic nerve, or secondary to papilledema.

Treatment by craniotomy or orbital decompression to reduce cerebrospinal fluid (CSF) pressure and papilledema.

Craniofacial Dysostosis (Crouzon) (Figs 21.1A and B)

- Brachycephaly is combined with hypoplasia of the maxilla.
- ***Ophthalmic features***
  - Widely separated eyeballs (hypertelorism)
  - Shallow orbits with bilateral proptosis
  - Corneal problems due to exposure
  - Divergent squint
  - Optic atrophy.

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Figs 21.1A and B: Cruozon syndrome. **A.** Front view; **B.** Lateral view

- ***Non-ophthalmic features***
  - High-arched palate
  - Irregular dentition
  - Hooked (parrot-beak) nose
  - Mental retardation.

Mandibulofacial Dysostosis (Treacher Collins)

- Hypoplasia of the zygoma and mandible.
- ***Ophthalmic features***
  - Indistinct inferior orbital margin
  - Coloboma (notching) of the lower lid
  - Anti-mongoloid slanting.
- ***Non-ophthalmic features***
  - Bird-like face
  - Macrostomia with high-arched palate
  - External ear deformity.

Median Facial-Cleft Syndrome

- Hypertelorism with telecanthus
- Cleft nose, lip and palate
- V-shaped frontal hair line (widow's peak)
- Divergent squint.

Oxycephaly-Syndactyly (Apert)

- Tower skull with flat occiput
- Mental retardation
- Ventricular septal defect
- High-arched palate
- Hypertelorism, shallow orbits and proptosis
- Anti-mongoloid slanting with ptosis and exotropia
- Syndactyly of the fingers and toes.

Hypertelorism (Figs 21.2A and B)

- Increased separation of the eyes.
- Widely separated orbits, and broad nasal bridge.
- The interpupillary distance (IPD) may be 85 mm or more.
- Divergent squint, telecanthus and anti-mongoloid slanting.
- There may be optic atrophy due to associated narrowing of the optic canals.
- Hypertelorism also occurs in:
  - Median facial-cleft syndrome,
  - Apert's syndrome,
  - Crouzon's syndrome, etc.

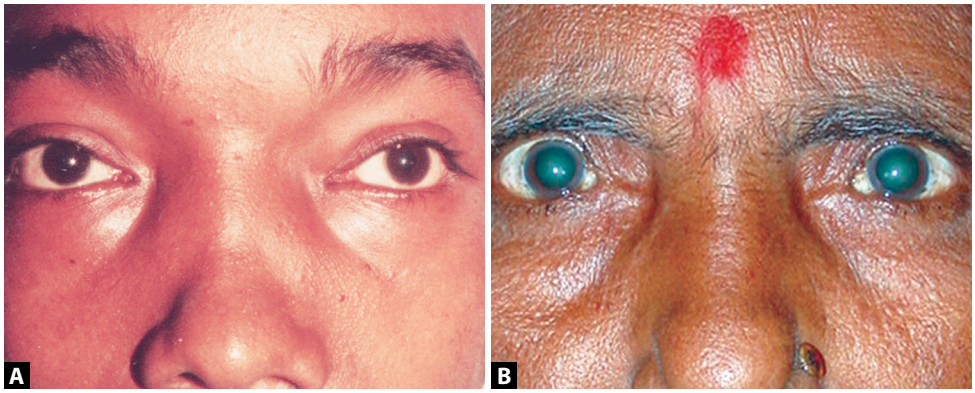
PROPTOSIS AND ITS INVESTIGATIONS

Forward protrusion of the eyeball beyond the orbital margin is called ***proptosis*** or ***exophthalmos***.

In general, proptosis and exophthalmos are loosely synonymous. But in strict sense:

- ***Exophthalmos means*** an active or dynamic protrusion of the eyeball (usually

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Figs 21.2A and B: A. Hypertelorism; **B.** Telecanthus

bilateral). Classically seen in thyroid ophthalmopathy.

- ***Proptosis means*** a passive protrusion of the eyeball, classically seen in space occupying lesion in the orbit and hence, usually unilateral. Proptosis may be:
  - Unilateral
  - Bilateral
  - Rapidly-growing (acute)
  - Slowly-growing (chronic)
  - Intermittent
  - Pulsating.

Unilateral Proptosis (Fig. 21.3)

Inflammatory

- ***Acute:*** Orbital cellulitis, panophthalmitis.
- ***Chronic:*** Periostitis, pseudotumor.

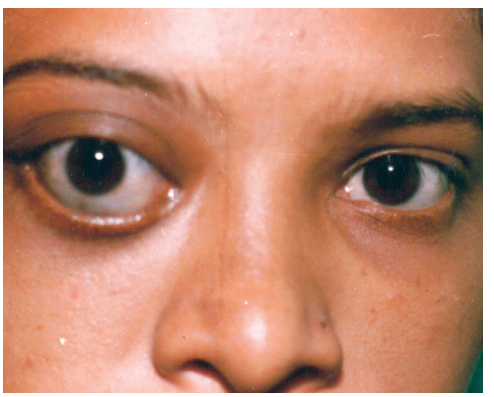


Fig. 21.3: Right-sided proptosis

Neoplastic

- ***Benign:*** Hemangioma, osteoma, fibroma, neurofibroma, dermoid, lymphangioma, glioma of the optic nerve, xanthomatosis, mixed tumor of the lacrimal gland, etc.
- ***Malignant:*** Lymphosarcoma, rhabdomyosarcoma, meningioma (optic nerve-sheath or sphenoidal-ridge); retinoblastoma and malignant melanoma extending into the orbit, maxillary antral carcinoma, metastatic deposit into the orbit (from Ca-breast, or Ca-lung), etc.

Vascular

- Retrobulbar hemorrhage (RBH)
- Orbital varices
- Aneurysm of the blood vessels.

Parasitic

- Cysticercosis
- Hydatid cyst.

Systemic diseases

- Leukemic deposits in acute myeloid and lymphoid leukemia (***chloroma***)
- Thyroid ophthalmopathy.

Bilateral Proptosis (Exophthalmos) (Fig. 21.4)

- ***Congenital:*** Craniosynostosis, uveal colobomatous cyst
- ***Endocrine:*** Thyroid ophthalmopathy
- Cavernous sinus thrombosis
- Xanthomatosis of orbit
- ***Metastatic:*** Leukemia, neuroblastoma.

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Fig. 21.4: Bilateral proptosis—Crouzon's syndrome



Fig. 21.5: Lymphangioma

Rapidly-growing (Acute) Proptosis

- ***Orbital emphysema*** due to fracture of the medial wall of the orbit
- ***Orbital hemorrhage (RBH)***
- Orbital cellulitis
- Rhabdomyosarcoma.

Slowly-growing (Chronic)

Proptosis that grows over months or years.

- Optic nerve glioma
- Optic nerve meningioma
- Orbital hemangioma
- Thyroid ophthalmopathy
- Benign neoplasm of the orbit

Intermittent Proptosis

- Orbital varices (most common)
- Lymphangioma (Fig. 21.5) (periodic enlargement with URTI)
- Recurrent orbital hemorrhage
- Recurrent orbital emphysema.

Pulsating Proptosis

- ***Transmitted vascular pulsation***
  - Caroticocavernous fistula
  - Saccular aneurysm of the ophthalmic artery
  - Highly vascular tumor in the orbit.
- ***Transmitted CSF pulsation***
  - Congenital absence of the orbital roof, as in meningocele and encephalocele.
  - Erosion of the orbital roof, as in neurofibromatosis and dermoid cyst.

Investigations

Careful history and clinical examination should precede radiological and laboratory investigations.

History

- Benign lesions tend to have a longer history than malignant lesions.
- Acute onset with pain suggests an inflammatory lesion (orbital cellulitis or pseudotumor); and also in rhabdomyosarcoma.
- In the past history, enquire about thyroid dysfunction, malignancies, orbital trauma, and sinus diseases.

Clinical Examination

- ***Measurement of the degree of protrusion*** is most readily assessed clinically by standing behind the patient and viewing the eyes from above.
  First rule out the possibilities of the pseudo proptosis and enophthalmos of the opposite eye.

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- ***Pseudoproptosis:*** The important causes are:
  - Enlargement of the same eye from high axial myopia and buphthalmos.
  - Retraction of the eyelids on the same side.
  - Shallow orbits as in craniosynostosis.
  - Enophthalmos of the opposite eye.
- ***Enophthalmos:*** About 50% of patients with enophthalmos are initially misdiagnosed as having contralateral proptosis, or ipsilateral ptosis.
  The important causes are:
  - Microphthalmos
  - Phthisis bulbi
  - Horner's syndrome
  - Absence of greater wing of the sphenoid as in neurofibromatosis
  - Blow-out fracture of the orbital floor.

- ***Measurement of proptosis (exophthalmometry):***
  - It measures the distance between corneal apex to the deepest portion of the lateral orbital margin.
  - It is measured by simple plastic ruler, and by ***Hertel's exophthalmometer*** (in both eyes simultaneously).
    - Normal value less than 20 mm (average 16 mm).
    - In ***proptosis*** a reading of 21 mm or more; or a difference of more than 2 mm between the two eyes.
- ***Exophthalmometry may be:***
  - ***Absolute:*** As compared to normal value (i.e. more than 20 mm).
  - ***Comparative:*** As compared, from time to time in the same eye.
  - ***Relative:*** As compared between the two eyes.
- ***Detailed ocular examination:*** Already discussed in table under ***signs of orbital diseases*** (*see* page 359).
- ***Palpation of the orbital rim:*** It may reveal a mass, bony erosion or bony thickening. The character of the mass (surface, consistency, thrill, tenderness, etc.) is noted carefully.
- ***Dynamic properties of proptosis:***
  - ***Try to precipitate proptosis:*** By dependent head posture, a Valsalva maneuvre or by jugular venous compression. A proptosis may be induced or exacerbated, in orbital varices or in capillary hemangioma.
  - ***Look for pulsation:*** A mild pulsation is better judged with a slit-lamp. A pulsating exophthalmos is seen in defect in the orbital roof (transmitted CSF pulsation), or in arteriovenous communication.
  - ***A bruit on auscultation:*** It may be heard in carotid-cavernous fistula or a highly vascular tumor. CSF pulsation is not associated with a bruit.
- ***Transillumination:*** It is often valuable in evaluation of anterior orbital lesions, either solid or cystic.
- ***Systemic examination:***
  - Thyroid evaluation.
  - Examination particularly for paranasal sinuses and nasopharynx.
  - To search for primary neoplasm elsewhere in the body, whenever a metastatic deposits are suspected in the orbit.
  - The important primary sites are breast, lungs, stomach, prostate, kidney and adrenals. Liver and spleen must be examined in leukemic patients.

Special Investigations

- ***Laboratory tests***
  - ***Hematologic study:*** Total leukocyte count (TLC) and differential leukocyte count (DLC) for inflammatory lesions, or blood dyscrasias, e.g. leukemia.
  - ***Thyroid function tests:*** Examples are serum T_3, T_4 and thyroid stimulating hormone (TSH) levels.
  - ***Casoni's test:*** For hydatid cyst.
  - ***Urine analysis:*** Bence-Jones protein in multiple myeloma.

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- ***Plain X-rays of the orbit***
  - ***The standard frontal anterior posterior (AP) view:*** For a composite view of the bones of the orbits, and to compare between the two orbits.
  - ***Caldwell view:*** To demonstrate the superior orbital fissure.
  - ***Water's view:*** To detect orbital floor fractures.
  - ***Rhese view:*** To visualize the optic foramina.
  - ***Lateral view:*** To study the nasopharynx.

X-ray signs in proptosis

Enlargement of the orbit

- ***Symmetrical:*** In intraconal lesions (optic nerve glioma, hemangioma, etc.)
- ***Asymmetrical:*** In extraconal lesions (rhabdomyosarcoma, dermoid cyst, etc.)

Change in bone density

- ***Increased:*** In meningioma, Paget's disease, fibrous dysplasia and osteoblastic metastasis.
- ***Decreased or destruction:*** In malignant tumors.

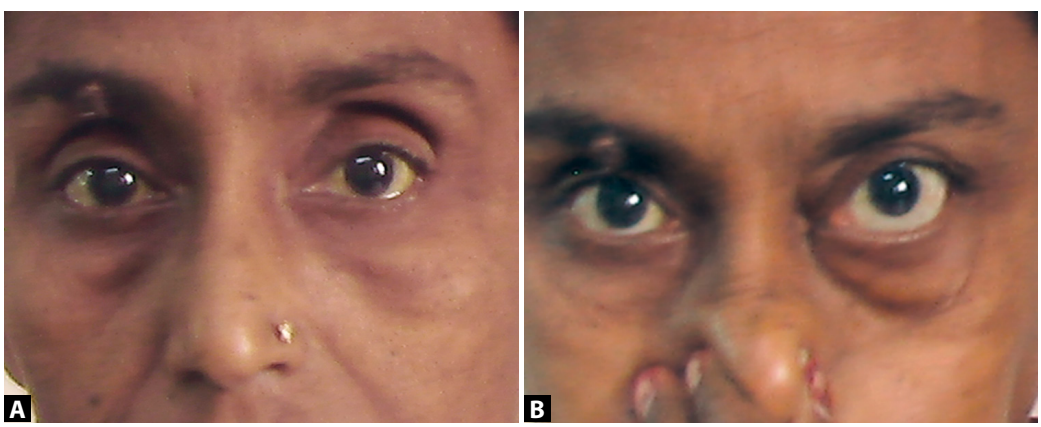
Intraorbital calcification: Seen in orbital varix (Figs 21.6A and B), optic nerve-sheath meningioma, retinoblastoma, etc.

Superior orbital fissure enlargement: Seen in infraclinoid carotid aneurysm, intracavernous aneurysm, intracranial extension of orbital tumors, etc.

Optic canal enlargement: In optic nerve glioma.

Dehiscence of orbital bones: In neurofibromatosis, mucocele, aneurysm, etc.

- ***Orbital venography:*** Presently, orbital venography is only indicated in patients who are clinically suspected to have *orbital varices.*
- ***Computed tomography (CT) scan***
  - CT scan is the most valuable non-invasive method of diagnosis for orbital lesions.
  - It is capable of visualizing the eyeball, extraocular muscles and the optic nerve.
  - It is extremely useful in determining the location and size of the orbital lesion.
- ***Ultrasonography (USG) B scan***
  - USG B scan gives an anatomical display which is easier to interpret than A scan.
  - USG is superior to CT scan in specific tissue diagnosis, and can usually differentiate a vascular lesion from a solid tumor, or an inflammatory pseudotumor from thyroid ophthalmopathy.
- ***Magnetic resonance imaging (MRI)***
  - MRI is major recent advance in imaging method, and superior to CT scan.
  - It distinguishes between the lesions based on their location, tissue structures and metabolic profile.
  - The imaging is not hampered by the bony shadow.



Figs 21.6A and B: Orbital varix with proptosis in left eye on Valsalva maneuvre

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- ***Fine needle aspiration cytology (FNAC)***
  - The aspirate is obtained under CT scan or ultrasound guidance using a 23-gauge needle.
  - It is specially valuable in suspected cases of orbital metastasis or secondary neoplasm, from the adjacent structures.
  - Diagnostic accuracy may be upto 95%.
- ***Incisional biopsy***
  - It may have a deleterious effect on the course of the tumor.
  - Incisional biopsy along with frozen section tissue study is a better alternative.
- ***Excisional biopsy***
  - It should always be preferred to incisional biopsy, for orbital tumors which are well encapsulated or circumscribed.
  - Exact diagnosis in many orbital lesions can only be made by histopathological studies of the orbital mass.

Principles of Treatment in Proptosis

- ***Medical:*** For orbital cellulitis, pseudotumor, thyroid ophthalmopathy, leukemic deposits in the orbit, etc.
- ***Radiotherapy:*** For secondary deposits and rapidly-growing malignant tumors.
- ***Surgical***
  - ***Tarsorrhaphy:*** To protect the exposed cornea.
  - ***Orbitotomy:*** For benign lesions, e.g. hemangioma, glioma, lacrimal gland tumor, dermoid cyst, etc.
    - ***Anterior orbitotomy:*** For a mass in the anterior part of the orbit.
    - ***Lateral orbitotomy (Kronlein's):*** For a mass in the posterior (retrobulbar) part or at the apex of the orbit.
    - ***Transcranial approach:*** For tumors extending into the cranial cavity.
  - ***Exenteration of the orbit:*** Total excision of the orbital contents and eyelids, followed by immediate grafting of the orbit. This is performed for secondary malignancy of the orbit from contagious ocular structures (e.g. advancing malignant melanoma of lid or tarsal conjunctiva) or radioresistance orbital malignancy.

ORBITAL INFLAMMATIONS

Orbital Cellulitis

Etiology

- ***Age:*** Usually child or young adult.
- ***Sinus infections:*** From the frontal, maxillary or ethmoidal sinusitis (sphenoidal sinusitis is very rare). This is the most common cause.
- ***Penetrating orbital injury:*** It may be with retained intraorbital foreign body.
- ***Thrombophlebitis:*** From a focus in the adjacent skin.
- ***Post-operative:*** Following enucleation of the globe.
- Dental or nasopharyngeal infection
- ***Causative organisms:*** *Streptococcus pneumonae*, *Staphylococcus aureus*, *Streptococcus pyogens* and *Haemophillus influenzae* in children under 5 years of age.

Types

The ***orbital septum***, the dense fascia (which separates the anterior structures from the orbit), divides the cellulitis into two types: (1) Preseptal and (2) Orbital.

1. ***Preseptal cellulitis (Fig. 21.7)***
   - Acute periorbital swelling and redness.
   - Increased warmth and tenderness of the eyelids.
   - Conjunctival chemosis.
   - Sometimes, fever with leukocytosis.
   - A fluctuating mass signifies abscess formation.
   - ***Treatment:*** Hot compress oral antibiotics and analgesics, and topical antibiotics.
2. ***Orbital cellulitis (Fig. 21.8A)***
   Symptoms
   - A sudden onset of unilateral severe pain and swelling of the lids.

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Fig. 21.7: Preseptal cellulitis

- Diplopia due to limitation of movements.
- Lacrimation and photophobia.
- Impairment of vision lately.
- Frequently associated with fever.

Signs

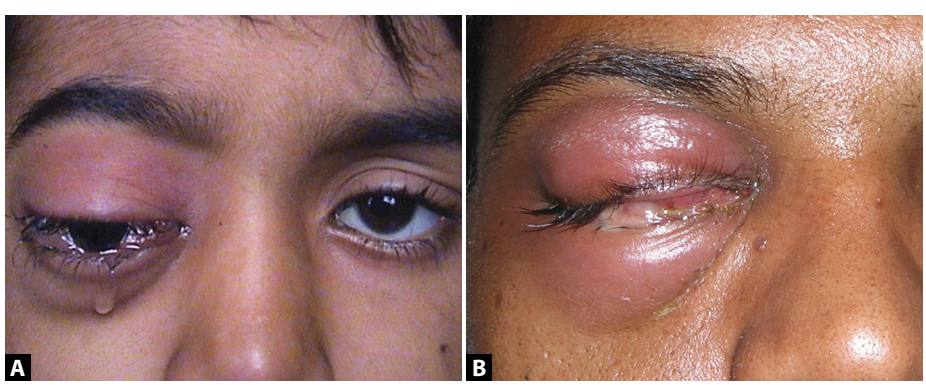
- Lid marked edema and tenderness.
- Conjunctiva chemosis and congestion.
- Proptosis commonly the eyeball is displaced laterally and downwards.
- Limitation of ocular movements.
- Ophthalmoscopy may show features of optic neuritis in severe cases.

Course and Complications

- Subsides completely with prompt treatment.
- ***Orbital abscess*** (Fig. 21.8B) formation, which may point and burst on the skin near the orbital margin.
- Meningitis, brain abscess, and cavernous sinus thrombosis, which may be fatal.
- Central retinal arterial occlusion, optic neuritis and optic atrophy may lead to blindness.

Management

- Patient should be admitted immediately.
- Antibiotic therapy is started promptly:
  - ***In children under 5 years of age:*** The antibiotics should cover *H. influenzae.*
  - Ampicillin combined with a ***penicillinase resistant antibiotic*** (e.g. oxacillin are administered parenterally.
  - ***In adult or older children:*** Intravascular high dose of ***penicillin,*** alternately with ***oxacillin*** is given every 4 hours. Alternately—***cefotaxime***, ***ciprofloxacin*** **or** ***vancomycin*** may be used in appropriate dose.
- Hot compress and analgesics to relieve pain.
- Simultaneously, the patient should be ***investigated*** for etiological factors:
  - Nasal and conjunctival swab culture.
  - X-ray of the paranasal sinuses (PNS).
  - Complete hemogram [may have normal erythrocyte sedimentation rate (ESR) and white blood cell (WBC) count].
  - Ultrasonography B scan to detect an orbital abscess.
  - Cerebrospinal fluid study and CT scan of the brain, if intracranial spread is suspected.



Figs 21.8A and B: **A.** Orbital cellulitis; **B.** Orbital abscess—severe chemosis

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- ***Surgery***
  - Drainage of the orbital abscess.
  - Incision and drainage of the lid abscess.
  - In most cases, it is necessary to drain the infected sinuses as well.

Inflammatory Orbital Disease (Pseudotumor) (Fig. 21.9)

Inflammatory orbital disease (IOD) is a term for idiopathic, non-neoplastic, nonmicrobial space-occupying periocular lesion, which may simulate an orbital neoplasm (pseudotumor).

Clinical Features

- Typically affects the middle-aged males.
- Most cases are unilateral.
- The onset is sudden with:
  - Periocular pain,
  - Edema of the lids and conjunctiva,
  - Conjunctival congestion,
  - Limitation of ocular movements, and
  - Proptosis.
- Depending on the site of involvement, a palpable mass may be present.

Investigations

- ***Utrasonography B scan:*** It shows a sonolucent area (edema) posterior to the globe with thickening of the extraocular muscles.
- ***CT scan:*** Shows muscle enlargement and scleral thickening.



Fig. 21.9: Inflammatory orbital disease (left eye)

Differential Diagnosis

- ***Thyroid ophthalmopathy:*** More gradual onset, bilateral, presence of lid signs, and altered thyroid function tests.
- ***Orbital cellulitis:*** Usually affects the children and with more severe picture.

Clinical Course

- Spontaneous remission after a few weeks.
- Intermittent course and eventual remission.
- Severe prolonged inflammation → progressive fibrosis of orbital tissues → frozen orbit with visual impairment.

Treatment

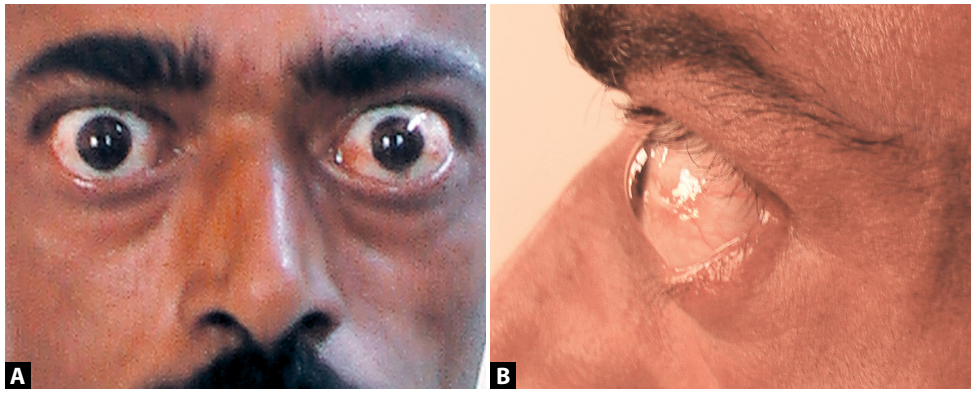
- ***Observation:*** Mild cases require no specific treatment, except close observation.
- ***Systemic corticosteroids:*** Oral prednisolone, 60–80 mg/day for 2 weeks. The dose is gradually tapered according to the response. Fifty to seventy five percent of patients show a favorable response.
- ***Radiotherapy:*** In patients who are steroid-resistant. A biopsy or fine needle aspiration cytology should be performed to rule out true neoplasm, prior to the initiation of orbital irradiation.
- ***Cytotoxic agents:*** In few cases who are resistant to both steroids and radio therapy, cyclophosphamide 200 mg/day may be the choice.

THYROID OPHTHALMOPATHY (FIGS 21.10A AND B)

The ophthalmopathy may be present in hyperthyroid, hypothyroid or euthyroid state.

- ***Grave's disease*** is the most common variety of hyperthyroid state with an autoimmune basis.

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Figs 21.10A and B: Thyroid ophthalmopathy. **A.** Front view; **B.** Lateral view

It typically affects the women between 20–45 years of age, and is characterized by goitre, infiltrative ophthalmopathy, thyroid acropathy (clubbing) and pretibial myxoedema.

- ***Ophthalmic Grave's disease (OGD)*** is a variant of Grave's disease where usual clinical and laboratory findings of hyperthyroidism are absent.

Pathogenesis

- Neither LATS (long-acting thyroid stimulator), nor EPS (exophthalmos producing substance) is responsible for ophthalmopathy.
- It has been proposed recently that circulating thyroglobulin (Tg) and anti-Tg immune-complex may bind to the eye muscle and orbital tissue to produce thyroid ophthalmopathy.

Pathology

Ophthalmopathy is due to:

- Hydrophilic mucopolysaccharide deposition, and inflammatory cells infiltration (plasma cells and lymphocytes) in the orbital tissues including the muscles.
- Proliferation of retrobulbar fat and connective tissues.

Clinical Features

They consist of one or more following signs.

- Retraction of the eyelid, due to:
  - Muller's muscle overaction due to sympathetic stimulation.
  - Overaction of levator-superior rectus muscle complex.
  - Contraction of the levator muscle due to infiltration.
- Lid lag
- Exophthalmos (unilateral or bilateral)
- Extraocular muscle involvement
- Conjunctival chemosis and hyperemia
- Optic neuropathy.

WARNER's classification of ocular changes (*Mnemonic* = "NO SPECS")

Class	Ocular changes
0.	**N**o symptom and sign
1.	**O**nly sign (lid retraction, lid lag or proptosis)
2.	**S**oft tissue changes **symptoms**—photophobia, lacrimation, foreign body sensation, **signs**—hyperemia and chemosis of conjunctive, resistance to retroplacement, palpable lacrimal gland
3.	**P**roptosis
4.	**E**xtraocular muscles involvement. (most common: inferior rectus, then—medial rectus; least common: lateral rectus)
5.	**C**orneal involvement (exposure keratopathy or dry eye)
6.	**S**ight loss due to optic neuropathy and corneal involvement

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Key lid and other signs with their eponyms

	Ocular signs	Eponyms
1.	Lid retraction	Dalrymple
2.	Upper lid-lag on downgaze	von Graefe
3.	Staring and frightened appearance	Kocher
4.	Infrequent blinking	Stellwag
5.	Tremor of the closed lid	Rosenbach
6.	Poor forehead wrinkling on upgaze	Joffroy
7.	Upper lid resistance to downward traction	Grove
8.	Lower-lid lags behind the globe on, upward gaze	Griffith
9.	Globe lags behind upper lid on upgaze	Means
10.	Increased pigmentation of the lid-skin	Jellinek
11.	Prevent eversion of upper lid	Gifford
12.	Fullness of the eyelids	Enroth
13.	Weakness of convergence	Mobius

Investigations

- ***Thyroid function tests:*** Serum T_3, T_4 and thyroid stimulating hormone (TSH); thyrotrophin releasing hormone (TRH) test; T_3 suppression test and tests for thyroid antibodies.
- ***Ultrasonography:*** Both A scan and B scan show enlargement of extraocular muscle. It can also demonstrate orbital involvement, and optic nerve involvement in some cases.
- ***CT scan:*** Enlargement of the extraocular muscles is a common finding. Optic nerve compression can be well demonstrated by CT scan.
- ***Forced duction test (FDT):*** To rule out contracture of the muscles.

Treatment

- ***Medical treatment for thyrotoxicosis***
  - ***Antithyroid drugs:*** Carbimazole (neomercazole) or propyl thiouracil, and β-blockers (propranolol).
  - Radioactive iodine therapy.
- ***Ocular treatment***
  - ***Topical therapy***
    - Decongestant eye drop and lubricants, in the form of artificial tears for symptomatic relief.
    - Guanethidine (5%) eye drop for lid retraction.
  - ***Systemic therapy***
    - ***Corticosteroids:*** They are indicated for optic neuropathy and rapidly progressive exophthalmos. A high dose of 80–100 mg/day, oral prednisolone is started initially, and then tapered gradually. Alternately, single dose of intravenous injection of methyl prednisolone—10–15 mg/Kg body weight is given to reduce the acute proptosis more rapidly.
    - ***Cytotoxic agents:*** Azathioprine or cyclophosphamide may be tried in steroid-resistant cases.
  - ***Radiotherapy:*** It is reserved for those patients who are unresponsive to steroid therapy, or have contraindication to steroids. Complications of radiotherapy include keratitis, cataract, radiation retinopathy, and localized erythema.
  - ***Surgical therapy***
    - ***Lateral tarsorrhaphy*** to prevent exposure keratitis.
    - ***Recession of levator and Muller's muscle*** to correct lid retraction.
    - ***Extraocular muscle surgery*** to correct diplopia and squint.
    - ***Blepharoplasty*** to remove excess fat and redundant skin from around the eyelids.
    - ***Orbital decompression*** should only be considered when systemic steroids

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and/or radiotherapy are ineffective. The indications are:

- Severe exposure keratitis
- Compressive optic neuropathy
- Cosmetically unacceptable exophthalmos.

Orbital decompression may be one-wall, two-wall, three-wall or four-wall decompression—depending upon the severity of proptosis.

RHABDOMYOSARCOMA (FIG. 21.11)

- It is the most common primary malignant orbital tumor in children.
- It occurs at about 7 years of age.
- Tumor arises from voluntary muscles, and presents as a rapidly-growing proptosis.
- Typically, a mass is palpable in the ***superonasal quadrant***.
- Diagnosis is confirmed by biopsy where ***cross-striations of tumor cells*** are seen.
- Three different histological types of rhabdomyosarcoma are ***embryonal, alveolar*** (most malignant variety), and ***pleomorphic*** (best prognosis).
- Ultrasonography and CT scan show, that the tumor appears to arise from the extraocular muscles.
- ***Treatment:*** Combination of radiotherapy and chemotherapy. The survival rate is 90% when the tumor is confined to the orbit. In ***unresponsive cases***, exenteration of the orbit may be required.



Fig. 21.11: Rhabdomyosarcoma

OPTIC NERVE TUMORS

These may be divided into two group:

1. Ectodermal tumor of nerve—glioma.
2. Mesodermal tumor of the optic nerve-sheath—meningioma.

Glioma of the optic nerve (Fig. 21.12)

- Most prevalent in childhood (4–8 years of age).
- Presents as unilateral proptosis with visual impairment.
- Proptosis is slow-growing and axial (Fig. 21.13).
- Marcus-Gunn pupil and optic atrophy.
- About 50% of cases, it is associated with neurofibromatosis.
- Plain X-ray of the orbit (Rhese view) shows, an uniform enlargement of the optic foramen in 90% of the cases.

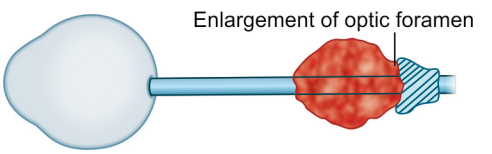


Fig. 21.12: Glioma of the optic nerve



Fig. 21.13: Axial proptosis in glioma of the optic nerve

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- Ultrasonography B scan and CT scan show fusiform or irregular enlargement of the optic nerve. Intracranial extension may be demonstrated by CT scan.
- ***Treatment:*** Local resection of tumor with preservation of the globe, by lateral orbitotomy. Radiation is helpful for tumors beyond surgical excision. Prognosis for life is excellent.

Meningioma: Meningiomas are invasive tumors which occur predominantly in middle-aged women. The ocular features are related to primary site of involvement by the tumor.

- ***Tuberculum sellae meningioma:*** It may compress the chiasma or optic nerve. The patient is having a ***junctional scotoma*** (an ipsilateral central scotoma and a contralateral upper temporal field defect).
- ***Sphenoidal-ridge meningioma (Fig. 21.14):*** It typically causes a fullness in the temporal fossa, and a slowly-growing proptosis due to hyperostosis of the roof and lateral wall of the orbit, as well as invasion into the orbit itself.
- ***Optic nerve-sheath meningioma (Fig. 21.15):*** The patient presents with a progressive impairment of vision in one eye, or a slowly-growing unilateral axial proptosis (Fig. 21.16).

The triad of long-standing visual impairment, a pale swollen optic disc, and opticociliary shunt vessels are virtually pathognomonic of optic nerve-sheath meningioma.



Fig. 21.14: Sphenoidal-ridge meningioma. Note: temporal fossa fullness on the right side

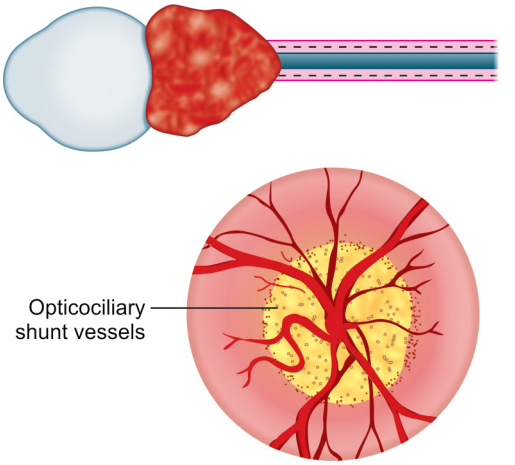


Fig. 21.15: Optic nerve-sheath meningioma—with opticociliary shunt vessels

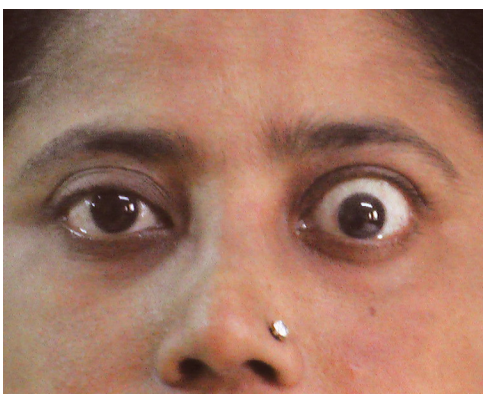


Fig. 21.16: Optic nerve-sheath meningioma—axial proptosis

Investigations

- ***Plain X-rays show:*** Hyperostosis of the roof and lateral wall of the orbit in sphenoidal-ridge meningioma.
- Computed tomography scan shows a segmental or diffuse enlargement of the optic nerve in optic nerve-sheath meningioma.

Treatment

- Surgical excision of the tumor by lateral orbitotomy. Prognosis for vision is extremely poor, but for life is good.

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- Radiotherapy may be encouraging to treat the tumor, with improved visual prognosis.

CAVERNOUS SINUS THROMBOSIS

This is an acute thrombophlebitis that originates from a purulent infection of the face, peripheral nervous system (PNS), ear or orbit that drains through the veins into the cavernous sinus.

Clinical features

- The onset in usually violent with severe pain along the ophthalmic division of the fifth nerve, fever and prostration.
- These are rapidly followed by proptosis, with congestion and edema of the lids and conjunctiva.
- Ophthalmoplegia occurs, and the lateral rectus is the first muscle to be affected.
- Edema of the mastoid region (a pathognomonic sign) indicating back-pressure in the mastoid emissary vein.
- There may be papilledema and visual loss.
- The other eye becomes similarly affected within a few hours, due to intercavernous communication.
- Death may occur from meningitis or pulmonary infarction.

Investigations: Imaging-contrast-enhanced CT scan and MRI of the orbit and/or nasal sinuses; complete hemogram, ESR blood cultures, and sinus cultures help establish and identify an infectious primary source. Lumbar puncture is necessary to rule out meningitis.

Treatment: Intensive intravenous antibiotic is necessary to control infection, and anticoagulant to prevent extension of the clot.

CAROTID-CAVERNOUS FISTULA (FIGS 21.17A AND B)

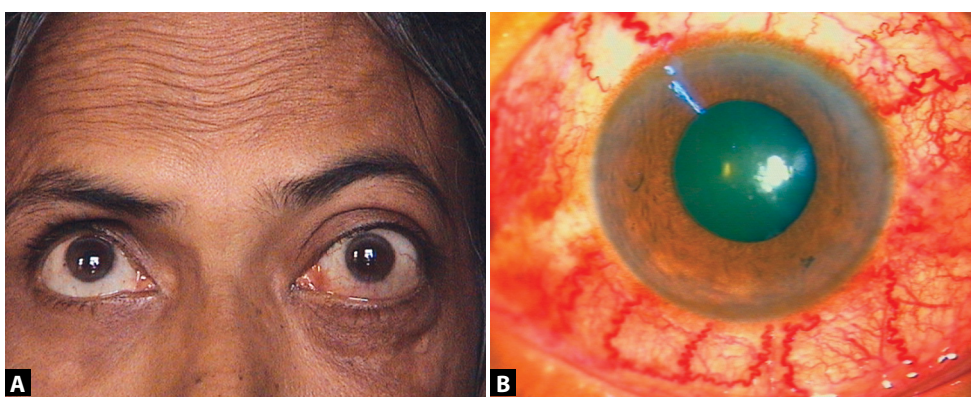
It results from an abnormal communication between the cavernous sinus and the internal carotid artery, giving the classical picture of a pulsating exophthalmos.

Etiology

- ***Trauma:*** As in fracture of the base of the skull.
- ***Spontaneous:*** Due to rupture of an intracavernous aneurysm or an atherosclerotic internal carotid artery in a hypertensive individual.

Clinical Features

- The onset is usually unilateral, sudden and dramatic.
- A marked proptosis, which is typically pulsatile, and is associated with a thrill and bruit. The pulsation can be abolished



Figs 21.17A and B: **A.** Carotid-cavernous fistula (left eye); **B.** Caput medusae of the same patient

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by compression of the ipsilateral carotid artery at the neck.

- ***Chemosis, redness*** and ***dilatation*** of the episcleral blood vessels [***caput medusae***—appearance (Fig. 21.17B)].
- ***Ophthalmoplegia*** due to involvement of the 3rd, 4th and 6th nerve (6th nerve most common).
- ***Reduced visual acuity*** due to posterior segment involvement (congestion and disc edema).
- ***Raised introcular pressure***, due to elevated episcleral venous pressure.
- ***Anterior segment ischemia*** develops in some patients. It is characterized by corneal edema, aqueous cells and flare; iris atrophy and neovascularization; and cataract formation.

Treatment

- In 5% of the cases, the fistula closes spontaneously.
- ***Surgical treatments include:*** Ligation of the common carotid or internal carotid artery, direct intracavernous surgery, and balloon catheter embolization.

FRACTURES OF THE ORBIT

Discussed in Chapter 24.

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22 CHAPTER

Diseases of the Lacrimal Apparatus

SYMPTOMS AND SIGNS

The diseases of the lacrimal apparatus are divided into two groups: Abnormalities of the lacrimal glands; defects in the drainage system.

1. ***Abnormalities of the lacrimal glands***
   - ***Excessive tear secretion:*** Reflex stimulation of the main lacrimal gland.
   - ***Deficient tear formation:*** Atrophy of the lacrimal gland and conjunctival scarring leading to occlusion of the main and accessory lacrimal glands.
   - ***Characteristic swelling with S-shaped upper lid:*** Neoplasms or inflammatory diseases of the lacrimal gland.
2. ***Defects in lacrimal drainage system***
   - Cause watering from the eye.
   - Obstructed lacrimal sac may be chronically or acutely inflamed.
     - ***Acute inflammation*** causes cellulitis of the sac and surrounding structures, and lacrimal abscess formation.
     - ***Chronic inflammation*** causes painless swelling in the sac region, with regurgitation of pus through the puncta, when pressure is applied to the sac area.
   - ***Excessive tear formation*** or poor drainage is a nuisance, since the vision is blurred by tears and discomfort caused by overflow of tears onto the cheek.
   - ***Deficient tear formation*** may cause loss of the eye and is associated with ocular surface diseases (OSD), i.e. keratinization of the cornea and conjunctiva.

DISEASES OF THE LACRIMAL GLAND

Acute Dacryoadenitis (Fig. 22.1)

- It usually accompanies a systemic disease like mumps, influenza or glandular fever.
- It is characterized by acute local pain, swelling and tenderness, and drooping of the outer part of upper lid with a characteristic S-shaped curve.
- ***Treatment:*** The infection is usually self-limiting. Antibiotics with local hot compress may be required in some cases.



Fig. 22.1: Acute dacryoadenitis right side—S-shaped right upper eyelid

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Chronic Dacryoadenitis

A proliferative inflammation of the lacrimal gland may occur as a part of generalized granulomatous disorders (e.g. syphilis, tuberculosis or sarcoidosis). It presents as a painless, firm, mobile swelling of the lacrimal gland.

Mikulicz Syndrome

It is characterized by a symmetrical swelling of the lacrimal and salivary glands, and may occur as a part of sarcoidosis, Hodgkin's disease or one of the lukemias.

Dacryops (Fig. 22.2)

It is a cystic swelling in the upper fornix due to retention of secretion owing to blockage of one of the lacrimal ducts.

Treatment is surgical excision or marsupialization of the retention cyst via the conjunctival route.

Lacrimal Gland Tumors

- They are relatively rare, and show a marked resemblance to the parotid gland tumors.
- They present in middle-aged, progress slowly, until they become palpable as hard, nodular and slightly mobile lumps.
- ***Proptosis*** occurs in downwards and medially, along with limitation of movements and diplopia.



Fig. 22.2: Dacryops of the lacrimal gland

- ***Benign mixed tumors*** form the majority, and have an excellent prognosis if they are excised completely with their capsule.
- ***Carcinomas*** have a more rapid onset, often with pain and lymph node involvement. Local hyperostosis of the orbital bone is often evident on X-ray.

Treatment

A biopsy may be needed to confirm the diagnosis. If malignant, a radical surgical removal, or a full exenteration of the orbit is often advisable.

DRY EYE SYNDROME

A dry eye syndrome is said to exist when the quantity or the quality of the precorneal tear-film is insufficient to ensure the well-being of ocular epithelial surface.

Dry Eye

Dry eye is a multifactorial disease of the tears and ocular surface that results in discomfort, visual disturbance and tears film instability with potential damage to the ocular surface. It is accompanied by increased tear osmolarity and inflammation of the ocular surface [definition given by dry eye workshop (DEWS) 2007].

In most of the situations, it results from a localized immune-mediated inflammatory response affecting both the lacrimal gland and the ocular surface.

Other commonly used terms:

- ***Tear-film dysfunction:*** It is a disturbance in the tear-film function owing to change in lipid, water or mucin component of the tears.
- ***Ocular surface disorders***
- ***Keratoconjunctivitis sicca (KCS):*** It is a deficiency of aqueous component of the tear-film.
  - ***Primary KCS:*** Involvement of the lacrimal gland alone.
  - ***Secondary KCS:*** When there are additional features of connective tissue disorders.

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- ***Sjogren syndrome (primary):*** It is an autoimmune disease, characterized by a dry eye (xerophthalmia) and a dry mouth (xerostomia), due to involvement of both lacrimal and salivary glands.
  - ***Secondary Sjogren syndrome:*** When they are associated with a connective tissue disorders, such as seropositive rheumatoid arthritis, systemic lupus erythematosus (SLE), systemic sclerosis, polymyositis, etc.
- ***Xerosis:*** It is a dryness of the conjunctiva, and presents as wrinkling, thickening, pigmentation and dryness of the conjunctiva.
- ***Xerophthalmia:*** When the same xerotic process spreads over the cornea, mainly caused by vitamin A deficiency.
- ***Keratomalacia:*** It is the softening and aseptic necrosis of the cornea, usually due to vitamin A deficiency along with malnutrition.

Symptoms

The symptoms are typically worse during nighttime, or awakening from sleep (as sleep decreases the tear production) and they include:

- Foreign body (sandy/gritty) sensation
- Transient blurring of vision
- Excessive stringy mucus
- Burning sensation
- Photophobia
- Intolerance to drafts and winds
- Constant awareness about the eye itself.

Causes

Categories	Causes
Aqueous tear deficiency	• Keratoconjunctivitis sicca • Sjogren syndrome • Riley-Day syndrome • Trauma to lacrimal gland • Surgical removal of lacrimal gland • Infiltrations by—sarcoidosis, lymphoma, amyloidosis, etc.

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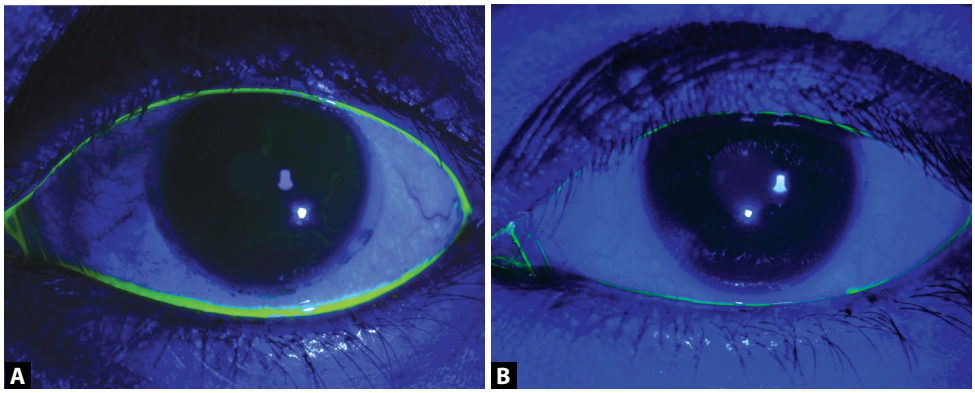
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Categories	Causes
	• Drug-induced, like antihistaminics, antimuscarinics, thiabendazole, etc. • Neuroparalytic hyposecretion
Mucin-deficiency	• Goblet cell dysfunctions, as in hypovitaminosis A • Goblet cell destruction, in alkali burn, trachoma, phemphigoid, Stevens-Johnson syndrome • ***Drug-induced:*** Beta-blockers (practolol), ecothiophate iodide
Lipid abnormalities	• Anhydrotic ectodermal dysplasia (congenital absence of Meibomian glands) • Various types of blepharitis including Meibomianitis
Lid surfacing abnormalities	• Lagophthalmos • Exposure keratitis • Entropion, ectropion • Lid-notching • Coloboma of the lid
Epitheliopathies	• Recurrent erosions • Limbal lesions • Hard contact lens • Topical anesthesia

Signs

- ***Precorneal tear-film:*** Increased amounts of mucus strands and debris in the upper and lower tear-menisci (meniscus floaters).
- ***Marginal tear-strip (tear-meniscus):*** Marginal tear-strip is reduced and it contains mucus and debris (normal lower tear meniscus height is about 1 mm) (Figs 22.3A and B).
- ***Conjunctiva***
  - Hyperemia of the conjunctiva.
  - Appearance of folds on the temporal side, especially on abduction.
  - Papillary conjunctivitis (a non-specific reaction).
- ***Cornea***
  - ***Superficial punctate keratitis (Fig. 22.4)*** involving the inferior cornea, which is stained by fluorescein and Rose-Bengal.

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Figs 22.3A and B: A. Normal tear-meniscus height after fluorescein staining. Normal lower tear-meniscus height = 0.3 mm; **B.** Diminished lower tear-meniscus height after fluorescein staining in keratoconjunctivitis sicca

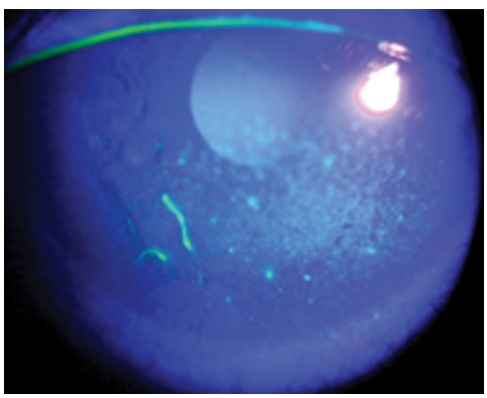


Fig. 22.4: Punctated keratopathies and corneal filaments after fluorescein stain in keratoconjunctivitis sicca

- ***Filaments (Fig. 22.5)*** are short tails that hang from the corneal surface.
  They are composed of central mucus core encased by epithelial cells and stained with the Rose-Bengal and fluorescein.
- Mucus plaques are attached loosely to the corneal surface and are also stained by Rose-Bengal dye.
- ***Dellen (Fig. 22.6)***, but not associated with limbal lesions.
- ***Thinning*** occurs in severe dry eye and sometimes with perforation.

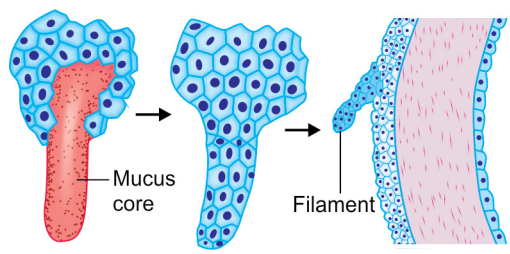


Fig. 22.5: Pathogenesis of filamentary keratopathy

Special Clinical Tests

Vital Dye Staining

- ***Fluorescein (2%) staining (Fig. 22.7):*** It stains precorneal tear-film and intercellular tissue. It does not stain the mucus and devitalized epithelial cells.
- ***Rose-Bengal (1%) staining (Figs 22.8A and B):*** It has an affinity for devitalized epithelial cells, mucus and filaments. It is very useful to detect mild cases of dry eye by staining the interpalpebral conjunctiva in the form of two triangles with their bases towards the limbus. Topical anaesthesia should not be used prior to Rose-Bengal staining, as it may induce false-positive result.
- ***Alcian blue staining:*** It is used to stain the mucus more selectively (not commonly used).

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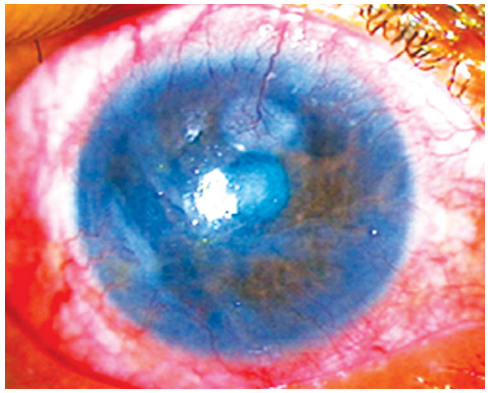


Fig. 22.6: Dellen and superficial vascularization in severe dry eye

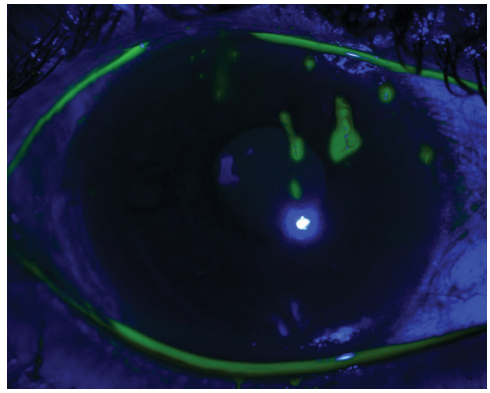
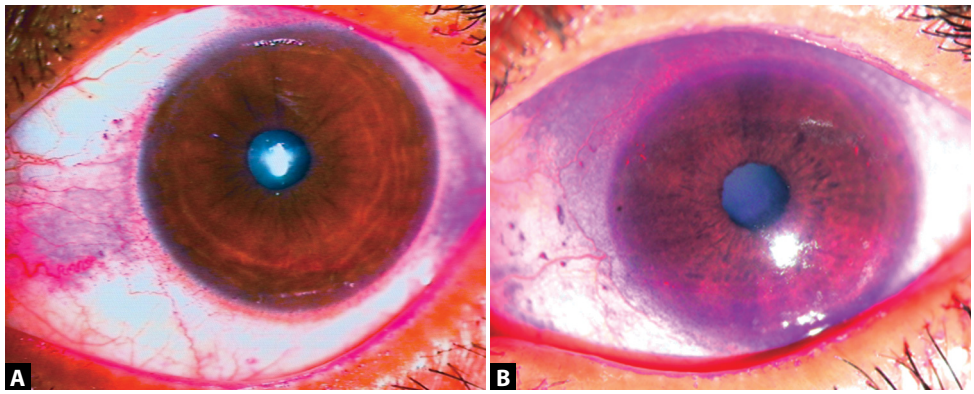


Fig. 22.7: Fluorescein stain—filamentary keratopathy and corneal staining



Figs 22.8A and B: Keratoconjunctivitis sicca typical Rose-Bengal staining. **A.** Conjunctiva; **B.** Cornea

- ***Lissamine green (Fig. 22.9):*** It is also used to stain the surface similar to Rose-Bengal stain.

Tear-Film Break-up Time

It is a simple test to assess the stability of the precorneal tear-film.

- A drop of fluorescein is instilled and the patient is asked to blink 2–3 times to distribute the dye.
- The patient is then asked not to blink while the cornea is studied by the cobalt blue filter with the slit lamp.
- The tear-film break-up time (TBUT) is the time in seconds between the last blink and the appearance of dry (black) spots on the cornea.
- A normal TBUT is more than 10 seconds (11 to 35 seconds), and a TBUT value less than 10 seconds is abnormal.

Schirmer's Test (Fig. 22.10)

- A 5 mm wide and 35 mm long special filter paper (Whatman no. 41) is placed in the lower fornix at the junction of the middle and outer-third of the eyelid after folding it at 5 mm. After 5 minutes, the amount of wetting from the fold is measured. Patient may blink or close the eyes as necessary during the test.

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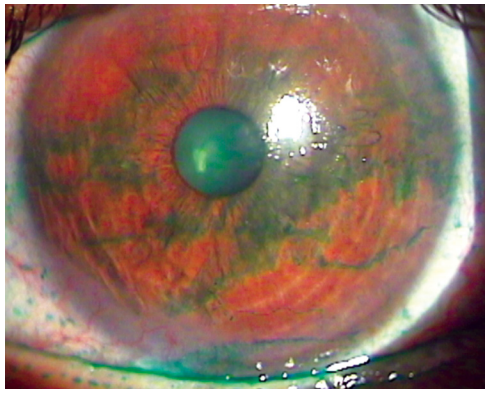


Fig. 22.9: Lissamine green staining in dry eye



Fig. 22.10: Schirmer's test

- This is ***Schirmer's test I*** and done without anesthesia. It measures the total secretion (i.e. basic plus reflex).
  - ***Values of Schirmer's test I***
    Normal value: 11–25 mm
    Borderline: 5–10 mm
    Impaired secretion: Less than 5 mm
  - Schirmer's test I may be done after topical anesthesia, which measures only the ***basic secretion***.
- ***Schirmer's test II*** measures the ***reflex secretion*** only. The procedure is same except, instillation of topical anesthesia, in the eye and irritation of ipsilateral nasal mucosa with a camel hair brush or cotton swab during the test.
  The reading is taken after 2 minutes and a measurement of less than 15 mm indicates failure of reflex secretion. This test is seldom used.

Laboratory Tests

- ***Tear lysozyme assay:*** It is estimated by placing a tear-wetted filter strip in an agar plate containing the bacteria *Micrococcus lysodeikticus,* and zone of lysis is measured after incubation at +37°C for 24 hours. In KCS there is reduction in tear lysozyme.
- ***Tear osmolarity:*** Its normal range is 290–311 mOsm/L, and it increases in KCS. Tear osmolarity value above 316 mOsmol/L is diagnostic of dry eye disease.
- ***Fluorescein dilution test:*** One drop of fluorescein is instilled in the eye and the dilution over a period of time is measured photometrically to calculate the tear volume.
- ***Tear mucin measurement:*** By measuring the hexosamine content (one of the principal moieties of mucus) of tears.
- ***Goblet-cell count of the conjunctiva:*** A biopsy specimen is taken from the inferior nasal conjunctival fornix for this. Average goblet-cell count in normal eyes is 8–15 cells/sq mm. The number is diminished in mucin deficiency states.
- ***Conjunctival impression cytology (CIC):*** The impression is taken with a cellulose acetate filter paper strip from the bulbar conjunctiva, and then stained by Papanicolaou's technique. The specimen is then analyzed for normal and abnormal cells, goblet cells and distribution of different cells. It is important in mucin deficiency states as well as in KCS.
- ***Hematological tests:*** They are important to detect etiological factors responsible for dry eye, as in collagen vascular disorders [rheumatoid factor, antinuclear factor (ANF), erythrocyte sedimentation rate (ESR) etc.].

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Levels of Severity of Dry Eye (DEWS Classification)*

Levels	Previous term	Clinical features
Level 1	Mild dry eye	Mild conjunctival signs; normal Schirmer; slightly lower TBUT; no other problems
Level 2	Moderate dry eye	Mild corneal signs, abnormal TBUT, abnormal Schirmer's value
Level 3	Severe dry eye	Marked conjunc tival and corneal sign; abnormal TBUT and Schirmer; symptomatic
Level 4	Extremely severe dry eye	More visual symptoms; severe corneal erosions; constant features

* International Dry Eye Workshop (DEWS) 2007.
Abbreviation: TBUT—Tear-film break-uptime

Treatment

There is no permanent cure for the dry eye, but there are some options available to relieve symptoms.

- ***Preservation of the existing tears***
  - Reduction of the room temperature.
  - ***Humidifiers:*** Examples are swimmer's goggles, moist-chamber goggles, etc.
  - Plenty of intake of drinking water.
  - Lid taping and tarsorrhaphy to prevent exposure keratitis.
  - ***Punctal occlusion:*** It is to preserve the natural or artificial tears in contact with the ocular surface for a longer time.
    - ***Temporary:*** By solid gelatin rods, collagen plugs, cyanoacrylate glue, '2–0' catgut pieces, or heat cautery.
    - ***Permanent:*** For severe cases of KCS when the Schirmer's value is less than 2 mm. This is done by silicone plugs, thermodynamic acrylic plugs, electrocautery or by small patch graft over the puncta.
- ***Supplementation of tears:*** Essentially four types of tear substitutes are currently available in the market. The composition of tear substitutes are cellulose (methyl cellulose, hydroxypropyl methyl cellulose, hypermellose), polyvinyl alcohol, povidone and sodium hyaluronate. They are used either alone or in combination.
  A preservative-free eye drop is more preferable for long-term use, as some of the preservatives may cause dryness, e.g. benzylkonium chloride (BAK). Nobel preservatives, like purite and perborate containing tears substitute are more important when the frequency of instillation is more than six times per day for a prolonged period.
  - ***Drops:*** Frequency of instillation may vary with the severity of dry eye 4 times/daily to half-hourly interval.
  - ***Ointments:*** Petroleum mineral oil or jelly is used at bed time as an ointment.
  - ***Slow-releasing inserts (ocusert):*** Lacrisert, formerly known as SRAT (slow-releasing artificial tear), is a small 5 mg pellet of hydroxypropyl cellulose in a cylinder form. It is placed in the lower fornix once daily. It dissolves slowly, and releases its polymer into the tear-film. Inadvertent loss of insert and blurred vision are the common problems.
  - ***Gel-tears:*** The gel is a clear synthetic polymer of acrylic acid which dissolves very slowly. It persists in the conjunctival sac for several hours after instillation. Temporary blurring of vision may be a problem.
- ***Stimulation of tears production: Bromhexine and eledosin*** are sometimes used for stimulation of the lacrimal gland to produce more tears. ***Oral Pilocarpine or Pilocarpine-like drug (Cevelamine)***

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may also be used to stimulate tears production. But, they have systemic muscarinic like toxicity.

- ***Mucolytic agent:*** Acetyl cysteine (5% or 10%) drops 4 times daily, when there is excessive production of mucus strands. It has an unpleasant sulfurous odor.
- ***Bandage soft contact lenses:*** High hydrated bandage contact lenses occupy a unique place in the treatment of dry eye. They are most valuable in filamentary keratopathy, recurrent epithelial erosions and exposure keratitis.
- ***Systemic and topical corticosteroids:*** They are useful for underlying systemic diseases, in patients with secondary KCS.
  Topical soft steroids (like loteprednol) may be used 4–6 times daily for a short period of time (pulse therapy) to reduce ocular surface inflammation in selective cases.
- ***Cyclosporine:*** Topical cyclosporine (0.05%) is now currently recommended in patient with moderate to severe dry eye due to primary or secondary KCS.
  Cyclosporine, an immunomodulator inhibits its activation of T lymphocytes and prevents T cells in releasing cytokines. It is given twice daily for at least 6 months.
- ***Major surgical procedures***
  - ***Parotid duct transplantation:*** It is rarely performed now-a-days.
  - ***Tarsorrhaphy or botulinum toxin:*** It is to reduce the exposed ocular surface area.
  - Limbal cell transplantation (autograft or allograft).
  - ***Corneal transplantation and keratoprosthesis:*** They are more heroic measures used to treat severe form of dry eye. But, they are often unsuccessful in preserving vision in severe dry eye.
- ***Treatment of associated problems:*** These include treatment of blepharitis, meibomianitis, conjunctivitis, corneal ulcer, entropion, symblepharon, etc.
- ***Change in working environment:*** This is also important especially who works in air-conditioned environment and spend a long time with the computer.
- ***Psychotherapy:*** This includes explaining to the patient about the disease and its future course. Because of its chronic nature, most patients go through periods of despondency and depression. Ophthalmologists must actively encourage these patients to continue to pursue their normal activities.

Management outline of dry eye diseases (DEWS recommendation)

Level 1

(i) Counseling, (ii) dietary modification, (iii) tears substitute (any type) 4 times/day, (iv) environmental management, (v) systemic medication review, (vi) control ocular allergy and (vii) address contact lens problems.

Level 2

(i) Steps from level one, (ii) tears supplement–preservative-free (6–8 times), (iii) oral tetracycline analogues, (iv) topical anti-inflammatory agents, (v) topical cyclosporine (0.05%): twice daily, (vi) reversible lacrimal occlusion and (vii) lid hygiene/nutritional support (omega-3 fatty acid).

Level 3

(i) Steps from level one and two, (ii) tears substitute (preservative-free) 1 hourly, (iii) autologous serum eye drop (20–50%), (iv) bandage contact lens and (v) permanent punctal occlusion.

Level 4

(i) Steps from level one, two and three, (ii) tears supplement (P/F)—1/2 hourly to 1 hourly, (iii) oral anti-inflammatory medications, (iii) oral cyclosporine, (iv) acetyl cysteine 20% eye drop, (v) tarsorrhaphy or botulinum toxin and (vi) limbal cell transplantation (auto/allograft).

WATERING FROM THE EYE

Watering is associated with blurring of vision and continuous discomfort caused by tears running down the cheek.

In all instances, it is necessary to find out, whether there is excessive production of the

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tears (lacrimation), or its defective drainage (epiphora).

Lacrimation: Excessive secretion of tears, due to reflex stimulation of lacrimal gland.

Causes

- Psychic stimulation, as in weeping or laughing.
- Irritation of the cornea or conjunctiva, by dust, fumes, chemicals, foreign body, inflammation, etc.
- In yawning, coughing, sneezing or vomiting.
- Exposure to bright lights.

Epiphora: Defective drainage of tears due to fault in the lacrimal passage.

Causes: Mechanical obstruction in the drainage system, due to:

- Congenital absence or occlusion of the puncta
- Canaliculitis, canalicular obstruction
- Congenital dacryocystitis
- Chronic or acute dacryocystitis
- Neoplasms of the lacrimal sac
- Nasal pathology, like polyps, tumors in the inferior meatus of the nose.

Lacrimal pump failure due to:

- Lower lid laxity.
- Weakness of the orbicularis oculi, as in Bell's palsy.
- Ectropion due to other causes.

Special Investigations

Special investigation of watering from the eye have been described in Table 22.1.

Evaluation of Watering from the Eye

History

- Watering due to epiphora is usually unilateral and is not associated with irritation. On the other hand, watering due to lacrimation is usually bilateral and associated with irritation, itching or photophobia.
- Past history of Bell's palsy is important, and it suggests lacrimal pump failure rather than a mechanical obstruction.
- History of medications like topical idoxuridine (IDU), latonoprost, mitomycin-C and systemic 5-FU (fluorouracil) may cause ***punctal stenosis***.

TABLE 22.1: Special investigations of watering from the eyes

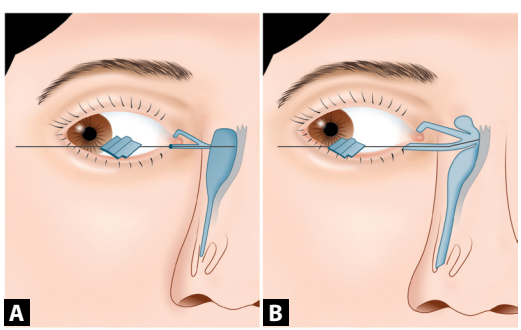
Types of test	Procedure
Saccharine, or ***quinine test*** (alternately ***chloramphenicol***)	Sweet or bitter taste after instillation of solution in conjunctival sac. One eye is to be tested at a time.
Patency test (syringing)	Irrigation of lacrimal passage is done with distilled water by lacrimal cannula. Presence of water in the nasopharynx indicates a patent nasolacrimal passage.
Dye disappearence test	Fluorescein dye disappearence from the cul-de-sac (normal 2 minutes). A prolonged retention of dye indicates inadequate lacrimal drainage.
Jones I (primary) test	Appearance of fluorescein in the nose (5 minutes), after its conjunctival instillation. If no dye is recovered the test is called ***negative***.
Jones II (secondary) test	Appearance of fluorescein in the nose after its lacrimal sac irrigation and indicates partial obstruction of nasolacrimal duct (NLD). If no dye is recovered the test is called ***negative***, and probably due to canalicular block.

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Types of test	Procedure
Probing test (Figs 22.11A and B)	After insertion of a probe or cannula, an attempt is made to touch the medial wall of the lacrimal sac and the lacrimal bone. The interpretation may be a ***hard stop*** or a ***soft stop***. • ***Hard stop:*** A firm to hard feeling caused by the probe–touching the medial wall of the sac against the lacrimal bone. It indicates that the lacrimal passage is patent up to the lacrimal sac and also indicates that the obstruction is below, i.e. at the NLD. • ***Soft stop:*** A spongy (soft) feeling as the probe presses the common canaliculus (CC) against the medial wall of the sac. It indicates either stenosis or obstruction of the canalicular system. However, an iatrogenic kinking of canalicular system during probing may also result in soft stop.
Intubation dacryocystography (DCG)	X-rays (anteroposterior and lateral view) after injection of radio-opaque dye into the lacrimal sac. It demonstrates the location of obstruction, fistula, diverticula and tumors.
Dacryoscintigraphy	Gamma camera tracing of technetium (^{99}mmTC) after instillation into cul-de-sac and its progress through the drainage system.



Figs 22.11A and B: Probing test. **A.** Hard stop; **B.** Soft stop

Examinations

Inspection

- ***Eyelids:*** For trichiasis, ectropion, lower lid laxity, blocked puncta, etc.
- ***Medial canthal (sac) area:*** For swelling (e.g. mucocele, acute dacryocystitis), fistula, etc.
- ***Orbicularis functions:*** By forcible closure of the eyelids.

Palpation

- ***Pressure over the sac area***—regurgitation of mucus or mucopurulent material in nasolacrimal duct (NLD) block (e.g. chronic dacryocystitis).
- ***Tenderness*** on palpation—in acute dacryocystitis.

Slit-lamp examination

- ***Puncta:*** Look for stenosis, foreign body, malposition, etc.
- ***Pressure over the canaliculi*** with glass rod and look for pus coming out through the puncta.
- ***Marginal tear-film strip*** for a high lower marginal tear-strip.
- Look for any corneal or conjunctival pathology.

Nasal examination

- To detect deviated nasal septum (DNS), polyps or tumor.
- To detect hypertrophy of the inferior turbinate.
- To detect the position of anterior end of the middle turbinate when dacryocystorhinostomy (DCR) is contemplated.
- To know the condition of the nasal mucosa (***atrophic rhinitis*** is a contraindication for DCR).

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A B

Figs 22.12A and B: A. Patency (syringing) test; **B.** Syringing of the lacrimal passage
CC—Common canalicular; **NLD**—Nasolacrimal duct

Patency (syringing) test (Figs 22.12A and B): A drop of 4% lignocaine is instilled into the conjunctival sac. The lower punctum is dilated with a punctum dilator. A lacrimal cannula, on a 5 mL distilled water-filled syringe is inserted into the lower canaliculus. The piston is then pushed to irrigate the lacrimal passage. The result may be:

- Water is going freely into the nasopharynx—lacrimal passage is patent.
- Water is regurgitating through the upper, and partly through the lower puncta:
  - ***Fast regurgitation:*** The fluid is clear and the regurgitation is rapid. It is due to common canalicular (CC) block.
  - ***Slow regurgitation:*** The fluid is turbid (as mixed with mucopus), and the regurgitation speed is slow. It is due to NLD block.
- Sometimes, fluid is going partially into the throat (and partly coming out through the puncta). It is due to partial NLD block.

Investigations for Surgery

- Blood for Hb%, total leukocyte count (TLC), differential leukocyte count (DLC) and erthrocyte sedimentation rate (ESR) is done.
- Bleeding time and clotting time to be checked.
- Blood sugar and blood pressure to be checked.
- General anesthesia fitness, if necessary.

DISEASES OF THE LACRIMAL PASSAGE

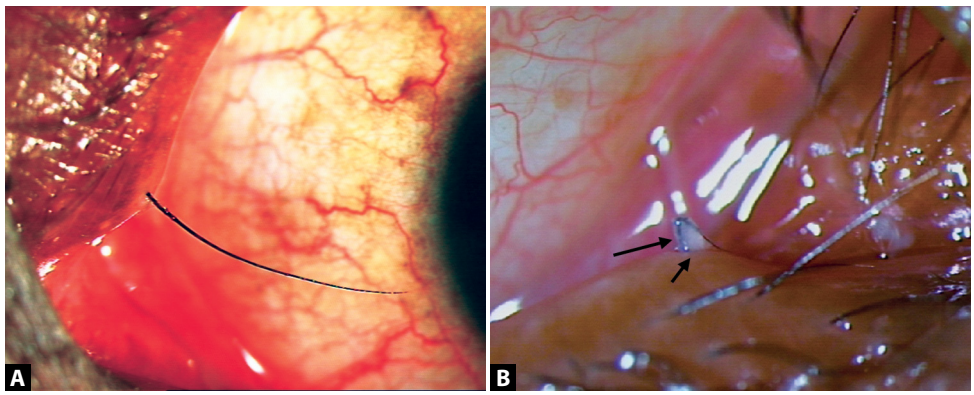
Canaliculitis

- ***Acute canaliculitis*** is usually due to herpes simplex infection and self-limiting.
- ***Chronic canaliculitis*** is most commonly caused by *Actinomyces israelii* (streptothrix).
  - ***Chronic infection causes:*** Discharge, a pouting punctum and concretions within the canaliculi.
  - It also causes stone formation (dacryolith) in the lacrimal sac.
  - A gritty foreign body sensation during probing establishes the diagnosis.
  - ***Treatment*** of dacryolith is by a linear incision into the conjunctival side of the canaliculus ***(canaliculotomy)***.

Occlusion of the puncta and canaliculus

- ***Causes***
  - Congenital closure or maldevelopment.
  - Foreign body, e.g. eyelash (Figs 22.13A and B) (most common) and concretion due to actinomycosis.
  - ***Scarring:*** It is due to burns, cosmetics, trauma or infections (trachoma and herpes simplex).
  - ***Medications:*** The examples are IDU or Phospholine iodide drop, systemic 5-FU.
- Slit-lamp examination is essential to establish the diagnosis.

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Figs 22.13A and B: Eyelash. **A.** In upper punctum; **B.** In lower punctum (arrows)

- ***Treatment*** is by removal of foreign body, or by dilating the canaliculus, and slitting it with a canaliculus knife. In case of CC block, C-DCR (conjunctivo-dacryocystorhinostomy) with a bypass tube is helpful.

Dacryocystitis

Dacryocystitis is an acute (Fig. 22.14) or chronic inflammation of the lacrimal sac. It is the sequel to obstruction of the NLD, or the sac itself, followed by bacterial infection.

Etiology

- It is commonly seen among new-born infants (congenital type) and middle-aged women (7 times more common than men).



Fig. 22.14: Acute dacryocystitis

- Left side is more commonly affected (left:right = 9:1).
- More common in lower socioeconomic status.
- ***Causes***
  - ***Lacrimal passage proper:*** NLD block due to narrowness or chronic inflammation of the sac.
  - ***Nasal factors:*** DNS, hypertrophied inferior turbinate, nasal polyps, etc.
  - Following a primary conjunctivitis.
  - Infection spreading from the nasopharynx.
- ***Organisms responsible:*** *Pneumococcus* (most common), *Streptococcus, Staphylococcus, Mycobacterium, Chlamydia,* etc.

Types

- Acute dacryocystitis
- Chronic dacryocystitis
  - Congenital type
  - Adult type.

Acute Dacryocystitis (Fig. 22.15)

- It is a suppurative inflammation of the lacrimal sac with an associated cellulitis of the overlying tissues.
- The onset is acute, and there is a painful swelling below the inner canthus, often combined with widespread cellulitis.

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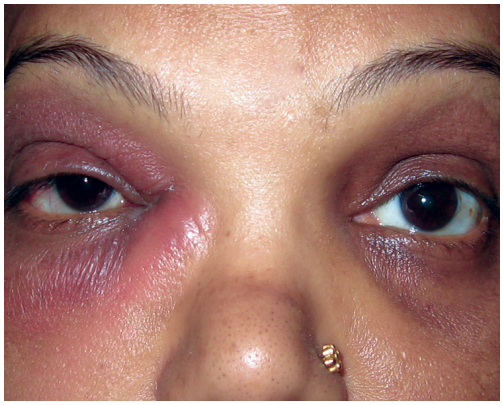


Fig. 22.15: Resolving acute dacryocystitis—right side

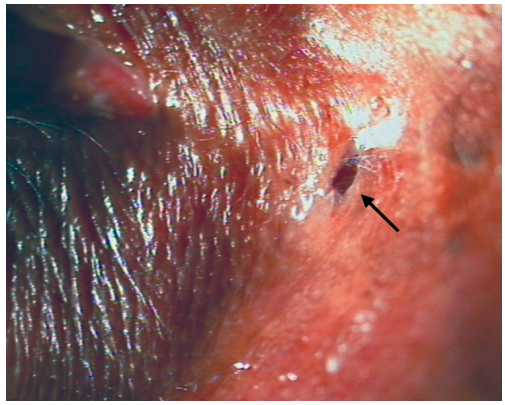


Fig. 22.16: Lacrimal fistula (arrow)

- The swelling is exquisitely tender with enlargement of the submandibular lymph node.
- Constitutional symptoms like fever, malaise, etc. are present.
- An abscess may often form, with perforation of the skin just below the medial palpebral ligament leading to formation of ***lacrimal fistula (Fig. 22.16)***.

Treatment

- Local hot compresses 3–4 times daily
- Broad spectrum systemic antibiotics
- Topical antibiotics 4–6 times daily
- Systemic analgesics with antacids
- In case of ***lacrimal abscess*** (Fig. 22.17) it is best drained by a skin incision.
  - In case of ***lacrimal fistula*** (whether formed naturally or artificially by drainage). It usually closes when the patency has been restored by DCR operation. Otherwise, the tract is excised along with dacryocystectomy (DCT) operation.
  - When everything is resolved a DCR or DCT operation has to be done as an elective procedure.

Chronic dacryocystitis (Figs 22.18 and 22.19)

- This is seen most frequently in newborns, infants and middle-aged women.
- The chief symptoms are watering and a chronic mucopurulent discharge at the inner-angle of the eye.

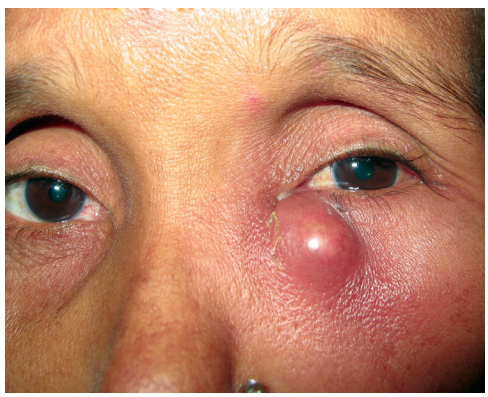


Fig. 22.17: Lacrimal abscess



Fig. 22.18: Congenital dacryocystitis (right eye)

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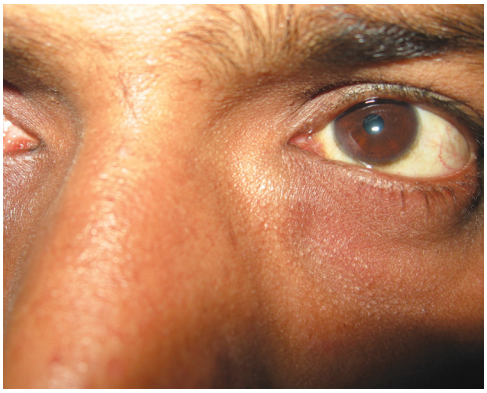


Fig. 22.19: Chronic dacryocystitis (left eye)

- There is regurgitation of pus or mucopus through the puncta, on pressure over the sac area.

Congenital chronic dacryocystitis (Fig. 22.18)

- It occurs in infants because of failure of the NLD to open into the inferior meatus of the nose, which normally occurs about third week of life.
- The initial symptom is constant watering of one eye and it is followed by the regurgitation of pus through the puncta.
- Congenital acute dacryocystitis is exceptional.
- ***Differential diagnosis***
  - ***Neonatal conjunctivitis (ophthalmia neonatorum:*** Usually bilateral, stickiness of the eyelids, no regurgitation of pus and quick response with antibiotic drops.
  - ***Congenital glaucoma (buphthalmos):*** There are associated blepharospasm and photophobia; cloudy cornea and raised intraocular tension.
- ***Management***
  - ***Hydrostatic sac massage and antibiotic drops:*** By giving downward strokes, with index or little finger (10 strokes in each time) four times daily.
    This should be followed by antibiotic or sulphacetamide (10%) drops four times a day. This treatment is to be continued until the age of 6 months (some surgeons prefer till the age of 12 months). With this conservative treatment, canalization occurs in about 95% of cases.
  - ***Probing:*** It is usually done 10–12 months of age, under general anesthesia. The success rate decreases significantly after the age of 4 years. If there is no improvement after 4 weeks the probing should be repeated.
  - ***Intubation:*** It may be done with silicone tube. If two probings fail to cure the obstruction, the tube should be kept *in situ* for 6 months.
  - ***Dacryocystorhinostomy:*** It is required only in exceptional circumstances, and wisely delayed until the child is 4 years of age.

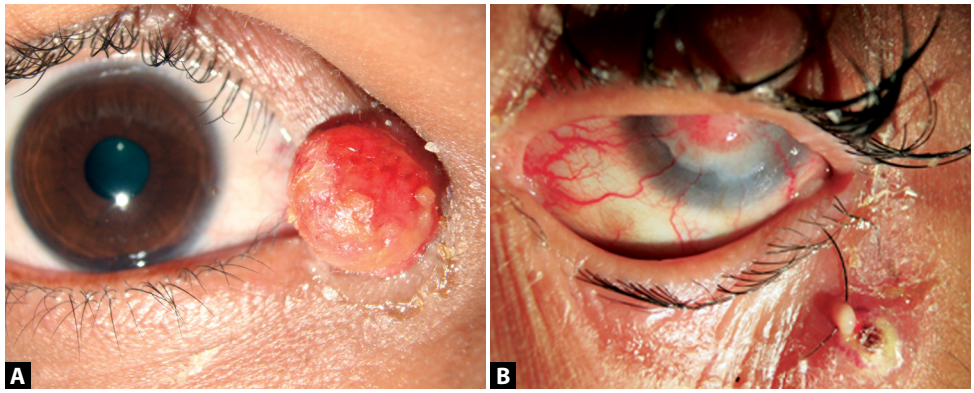
Adult chronic dacryocystitis (Fig. 22.19)

- It typically occurs in postmenopausal women and either occurs spontaneously, or follows a lacrimal sac infection.
- There may be spontaneous atresia of NLD, or weakness of the orbicularis, and it is often associated with nasal pathology.
- In long-standing cases, there is extraordinary dilatation and thinning of the lacrimal sac called ***mucocele*** or ***hydrops*** of the lacrimal sac (Fig. 22.20).



Fig. 22.20: Bilateral mucocele of the lacrimal sac

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Figs 22.21A and B: **A.** Lacrimal abscess with pyogenic granuloma at the puncta; **B.** Chronic dacryocystitis with corneal ulcer

- ***Complications***
  - Chronic conjunctivitis
  - Recurrent attacks of acute conjunctivitis
  - ***Acute-on-chronic*** dacryocystitis
  - From acute dacryocystitis, there may be chance of:
    - Lacrimal abscess pyogenic granuloma (Fig. 22.21A)
    - Lacrimal fistula
    - Orbital and facial cellulitis
    - Cavernous sinus thrombosis
    - Lacrimal osteomyelitis, etc.
  - ***Hypopyon corneal ulcer:*** Chronic dacryocystitis (Fig. 22.21B) is in fact a constant menace to the ophthalmologist or to the patient, as mild corneal abrasion due to any cause may be turned into a hypopyon corneal ulcer, which is mostly due to *Pneumococci*.

Treatment

- ***Symptomatic relief*** is often obtained by antibiotic (e.g. ciprofloxacin) and astringent (e.g. zinc sulfate) eye drops.
- ***Dacryocystorhinostomy:*** The surgical procedure of choice is DCR operation, in which a communication is established between the lacrimal sac and the middle meatus of the nose.
- ***Dacryocystectomy:*** It means simple removal of the lacrimal sac. The main idea is to eliminate the reservoir of infection and regurgitation of pus into the conjunctival sac. But watering will continue throughout the life.

  It is indicated:
  - In extreme old age.
  - When the sac is shrunken and fibrotic.
  - In presence of growth of the sac.
  - In obvious nasal pathology, like atrophic rhinitis.

  (For operative details *see* Chapter 25).

TUMORS OF THE LACRIMAL SAC (FIG. 22.22)

- Tumors of the lacrimal sac are extremely rare and represent a potentially life-threatening condition.



Fig. 22.22: Carcinoma of the lacrimal sac

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- Most neoplasms are squamous cell carcinomas, transitional cell carcinomas and adenocarcinomas.
- The triad of malignancy are:
  - A mass below the medial palpebral ligament (Fig. 22.22)
  - A chronic dacryocystitis that irrigates freely and
  - Regurgitation of bloody mucopus.
- Dacryocystography shows a filling defect.
- ***Treatment*** is by complete excision of the sac followed by irradiation.

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23 CHAPTER Ocular Motility: Squint

OCULAR MOVEMENTS

The ocular movements are of four types—***ductions, versions, vergences*** and ***supranuclear movements***.

Ductions (Fig. 23.1)

Ductions refer to the movements of one eye, and consist of adduction, abduction, supraduction (elevation), infraduction (depression), incycloduction (intorsion) and excycloduction (extorsion).

- ***Agonist:*** It is the primary muscle, moving the eye in any given direction.
- ***Synergist:*** It is a muscle which acts together with the agonist to produce a given movement.
- ***Antagonist:*** It is the muscle which acts in the opposite direction to the agonist.
  - When a person is looking towards his right side right lateral rectus is the agonist, left medial rectus is the synergist, and right medial rectus and left lateral rectus are the antagonists.
  - ***Sherrington's law of reciprocal innervation:*** Implies that increased contraction of a muscle is automatically associated with a reciprocal decrease in contraction (relaxation) of its antagonists.

Versions

Versions are binocular movements, in which the two eyes move synchronously and symmetrically in the ***same direction***.

The movements are:

- Dextroversion (right gaze), levoversion (left gaze), supraversion (up gaze), and infraversion (down gaze). These are secondary positions of gaze.
- Dextroelevation (gaze up and right), dextrodepression (gaze down and right), levoelevation (gaze up and left) and levodepression (gaze down and left). These oblique movements are tertiary positions of gaze.
- Dextrocycloversion (rotation of upper limbus of both eyes to the right) and levocycloversion (rotation to the left).

Six cardinal positions of the gaze (Fig. 23.2) are dextroversion and levoversion, dextroelevation and levoelevation, dextrodepression and levodepression.

Yoke muscles (Fig. 23.3): A muscle of one eye is paired with a muscle of the opposite eye, while moving the eyes into each of the six cardinal positions of gaze.

For example: In ***dextroversion***, two yoke muscles are right lateral rectus and left medial rectus. In ***dextroelevation***, the yoke muscles are right superior rectus and left inferior oblique.

For oblique movements the ***mnemonics***—the muscles responsible for particular gaze are ***same named rectus muscle*** (in right superior area, i.e. in dextroelevation, right superior rectus) and ***crossed-named oblique muscle*** (i.e. in right superior area, the left inferior oblique muscle).

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Fig. 23.1: Actions of extraocular muscles

RSR—Right superior rectus; **RIO**—Right inferior oblique; **RMR**—Right medial rectus; **RSO**—Right superior oblique; **RIR**—Right inferior rectus; **RLR**—Right lateral rectus; **LIO**—Left inferior oblique; **LSR**—Left superior rectus; **LLR**—Left lateral rectus; **LIR**—Left inferior rectus; **LSO**—Left superior oblique; **LMR**—Left medial rectus

Fig. 23.2: Six cardinal positions of version

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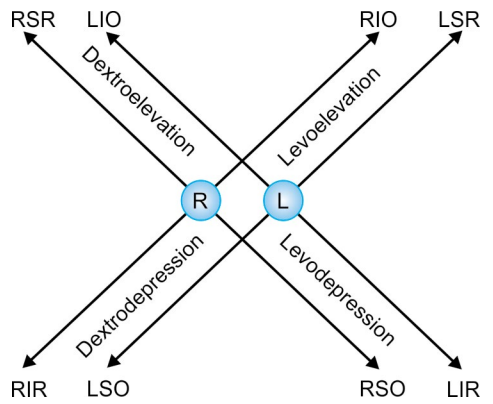


Fig. 23.3: Yoke muscles in diagonal movements
RSR—Right superior rectus; **LIO**—Left inferior oblique; **RIO**—Right inferior oblique; **LSR**—Left superior rectus; **LIR**—Left inferior rectus; **RSO**—Right inferior oblique; **LSO**—Left superior oblique; **RIR**—Right inferior rectus

Hering's law of equal innervation: States that during any conjugate eye movement, equal and simultaneous innervation flows to the yoke muscles. Hence, a paresis of one muscle is always accompanied by an apparent overaction of yoke muscle or its contralateral synergist (left inferior oblique is contralateral synergist of right superior rectus), which receives the same amount of stimulation as its lagging fellow. This is responsible for more ***secondary angle of deviation*** in paralytic squint.

Vergences

They are binocular movements, in which the two eyes move synchronously and symmetrically in the ***opposite directions***. They are ***convergence*** and ***divergence.***

Supranuclear Eye Movements

There are basic supranuclear control systems of ocular movements, which must function simultaneously. They are as follows:

- ***Saccades:*** They are the rapid eye movements, which are generally ***voluntary refixation movements***, i.e. to move the eyes quickly from one object to the another.
- ***Pursuits:*** They are ***smooth following movements*** which maintain the visual axes on any slow moving object.
- ***Vestibuloocular movements:*** They are the coordinated eye movements with respect to gravity and head positions, e.g. vestibular nystagmus (as in ***caloric test***), Doll's eye maneuver, etc.

VISUAL DEVELOPMENT

- ***At birth***, the eyes appear to move randomly, and there is no central fixation. The fovea is not fully developed, and the visual acuity is roughly 6/60 (as estimated by other means).
- ***At 6 weeks of age,*** the fixation reflex first becomes apparent, and the eyes can follow a bright light for a short distance.
- ***At 4–6 months of age,*** the re-fixation reflex develops firmly.
- ***At 6 years of age,*** the fovea develops completely with the full visual acuity of 6/6.
- If the visual developmental process is interrupted during this process, by any means (e.g. congenital cataract, high anisometropia, or squint), it results in a degree of amblyopia which becomes irreversible after few years.
- If the ***stimulus deprivation*** occurs bilaterally and severely (e.g. in bilateral congenital cataract) by the age of six months, it results in a pendular nystagmus on attempted fixation.

Binocular Single Vision

Binocular single vision (BSV) is the coordinated use of the two eyes, in order to produce a single unified image in three-dimensions.

It is not present at birth, and three factors are required for its development:

1. Reasonably clear vision in both eyes.
2. Precise coordination between the eyes for all directions of gaze.
3. Ability of the visual areas of brain to promote fusion of the two slightly dissimilar objects.

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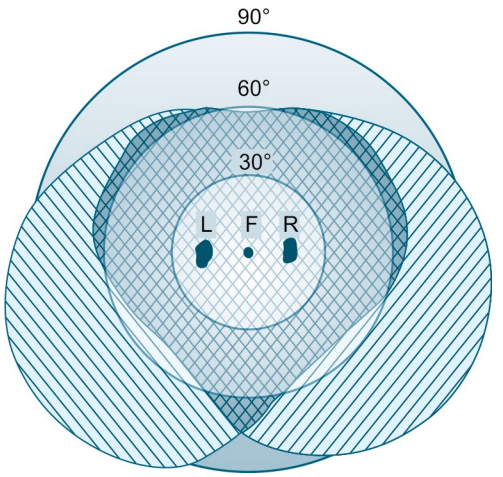


Fig. 23.4: Binocular field of vision

F—Fixation point; **R**—Blind-spot of the right eye; **L**—Blind-spot of the left eye

The three advantages of BSV:

1. An enlarged field of vision (Fig. 23.4) with compensation of each blind-spot.
2. An ability to appreciate depth with stereoscopic vision.
3. A combined binocular visual acuity is better than the uniocular visual acuity.

Three components (grades) of BSV are simultaneous macular perception (SMP), fusion and stereopsis (Fig. 23.5).

Grade-1 [Simultaneous macular perception (SMP)]: This means the ability to see two dissimilar (but not mutually antagonistic) images simultaneously, and to superimpose them.

Grade-2 (Fusion): This is the ability to see the slightly dissimilar images formed in each eye, and blended them into one.

The normal range of fusion is about 30° of convergence to 5° of divergence, and 2° deviation in vertical meridian.

Grade-3 (Stereopsis): This is the appreciation of the third dimension, allowing the perception of depth. For stereopsis to occur, disparate points must be stimulated.

Monocular Perception of Depth (Fig. 23.6)

The perception of a single eye, is in fact, two dimensional, but an 'one-eyed' person achieves a considerable power of depth perception by a number of indirect methods:

Fig. 23.5: Grades of binocular single vision—tested by synoptophore

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Fig. 23.6: Methods of monocular perception of depth

Note: The above features are apparent: overlapping of contours, relative size of houses, 'aerial perspective' evidenced in the blurring of the horizon, perspective convergence of parallel lines, shading of circular objects to give the impression of a sphere

- ***Overlapping of contours:*** They readily indicate their relative distances from the observer.
- ***Relative size of the objects:*** Objects whose size is familiar, will produce a retinal image of a size, which is inversely proportional to their distance from the observer. This distance can then be judged by the distance relative to that of another object whose size is also known. In addition, the definition of the object will be proportionately reduce. These two factors together are traditionally employed by the military in assessing their firing range.
- ***Aerial perspective:*** It is evidenced by the blurring of outlines, and the color change of the horizon, caused by intervening atmospheric scatter. It can estimate the intervening distance by the contrast of different objects.
- ***Perspective convergence of parallel lines:*** They are receding away from the observer. Similarly, when circular objects are seen obliquely, they will appear as oval, with one smaller than the other.
- ***Light and shades:*** A simple two dimensional circle, can be made into a sphere by adding the contrast of light and shades on its surface.
- ***Parallax:*** Movement of the head from side to side will cause nearer objects to move in the opposite direction, in relation to more distant objects.
- ***Accommodation:*** Within a limited range of distance, the amount of accommodative effort required to focus on an object, clearly gives an indication of the proximity of that near object.

DIPLOPIA

Diplopia (double vision) means that an object appears double.

Classification

- Physiological
- Pathological
  - Uniocular
  - Binocular
    - Homonymous or uncrossed (Fig. 23.7A)
    - Heteronymous or 'crossed' (Fig. 23.7B).

Physiological Diplopia

It is a normal phenomenon, in which objects not within the area of fixation, are seen double. It is easily demonstrated by looking at a near object with attention directed towards a distant object. Usually, it does not impinge on the consciousness.

Pathological Diplopia

Uniocular Diplopia

An object appears double even when only one eye remains open. It occurs when two images of the same objects forms on the two different parts of the retina. More commonly, there is uniocular polyopia (multiple vision) due to multiple images.

Causes

- Immature cortical cataract (due to multiple water clefts within the lens).

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Figs 23.7A and B: Pathological diplopia. **A.** Homonymous diplopia in convergent squint; **B.** Heteronymous diplopia in divergent squint (**LE**—Left eye; **RE**—Right eye)

- Subluxated clear lens (pupillary area is partly phakic and partly aphakic).
- Large peripheral iridectomy, iridodialysis or polycoria (multiple pupil).
- Retinal detachment due to dialysis when the retina becomes inverted.

Binocular Diplopia

An object appears double with both eyes remain open. This occurs when the image of an object does not fall on the corresponding points of the retina of the both eyes. Image of an object fall on the fovea of one eye, and on the extrafoveal area of the opposite eye.

Causes

- Paralysis or paresis of the extraocular muscles (most common).
- Displacement of the eyeball, by a space occupying lesion in the orbit, by fracture of orbital wall or by pressure of fingers.
- Mechanical restriction of the movements of the globe, e.g. pterygium, symblepharon, thyroid ophthalmopathy, etc.
- Deviation of rays of light in one eye, as in decenterd spectacles.
- Disparity of image size between two eyes, as in acquired high anisometropia (e.g. aphakia with spectacle correction in one eye, and the other eye being normal). Congenital high anisometropia causes amblyopia of the more ametropic eye rather than diplopid.

In diplopia, one image is distinct (***true image***), and the other is indistinct (***false image***). Binocular diplopia disappears when one eye is closed. Depending on the position of the false image in relation to midline, binocular diplopia may be ***uncrossed (homonymous),*** or ***crossed (heteronymous)***.

In a convergent squint, the diplopia is uncrossed ***(homonymous)***—that means, the false image is on the same side of the deviation.

In a divergent squint, the diplopia is crossed ***(heteronymous)***—that means, the false image is thrown across the midline.

Treatment

- Investigation to find out the cause and treat accordingly.

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- For relief of symptom:
  - Occlusion of the worse eye.
  - Prism correction if the deviation is small in paralytic squint.

AMBLYOPIA

Amblyopia denotes a unilateral or bilateral reduction in form vision, without any detectable organic ocular lesion.

The most sensitive period of developing amblyopia is in the first six months of life; and after about the age of six years, amblyopia does not usually develop.

Characteristics of an amblyopic eye:

- Reduction in visual acuity.
- ***Crowding phenomenon:*** Visual acuity is better when the test letters are viewed singly (optotype), rather than in a series (Snellen's chart).
- ***Neutral density filter test:*** Amblyopic eye shows no change in visual acuity with a neutral density filter. But a normal eye shows a drop in acuity of about one or two lines, whereas an eye with macular lesion may have a large drop in visual acuity.
- ***Color vision is unaffected***.
- ***Eccentric (extrafoveal) fixation*** is common, and the degree of amblyopia is proportionate to the distance of the eccentric point from the fovea.

Types

- ***Stimulus deprivation amblyopia (amblyopia ex anopsia):*** Results from the non-use of one eye in infancy or early childhood, and is caused by opacity in the media (cataract or leucoma), ptosis, or by occlusion (occlusion amblyopia).
- ***Strabismic amblyopia:*** Develops in a squinting eye, as a result of continued suppression, and it persists even when the deviated eye is forced to fixate.
- ***Anisometropic amblyopia:*** Occurs in the more ametropic eye, as a result of constantly blurred image of an object falling onto its fovea. This is more with high anisohypermetropia and anisoastigmatism than with anisomyopia.
- ***Ametropic amblyopia:*** It is bilateral, and associated with almost equally high refractive error of both eyes. It is also more with bilateral high hypermetropia.

Treatment

Principles of treatment

- Removal of opacity in the media.
- Full correction of refractive errors.
- Occlusion therapy

The treatment should be started as early as possible. The younger the patient is, the more rapid improvements in visual acuity will be, and the better will be the ultimate prognosis.

Occlusion therapy: The aim is to occlude the sound eye, and to force to use the amblyopic eye.

This is ***conventional occlusion***.

- It may be total (to occlude both form and light senses), or partial (to occlude only the form sense).
- If the vision in the amblyopic eye is worse than 6/18, occlusion must be total (with the help of a direct patching of the eye).
- If the vision is better than 6/18, the occlusion may be partial (with the help of cellotape or nail polish over the spectacles lens).
- Occlusion must be given in ratio (e.g. 3:1, i.e. 3 days in sound eye and 1 day in amblyopic eye) in younger children to prevent occlusion amblyopia of the sound eye.
- Occlusion therapy is to be carried out until the visual acuity develops fully, or there is no further improvement of vision after 3 months of continuous occlusion.
- ***Inverse occlusion:*** This is done when amblyopia is combined with eccentric fixation. The initial step is to return the normal foveal fixation by patching the amblyopic eye, with simultaneous stimulation of the fovea.

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After the development of central fixation in the amblyopic eye, patching of the sound eye is to be continued until maximum visual acuity is attained.

Other modes of therapy:

- ***Atropine penalization:*** An alternative to occlusion, atropine is used to blur the sound eye only (and may be with pilocarpine to the amblyopic eye).
- ***Pleoptics:*** They are the methods of reestablishment of foveal fixation and may be helpful in older children.
- ***CAM stimulator therapy:*** This consists of the viewing of rotating high contrast stripes of different sizes with the amblyopic eye.

SQUINT (STRABISMUS)

It is a condition in which only one of the visual axes is directed towards the fixation object, the other being deviated away from this point.

Normally, the visual axes of the two eyes are essentially parallel in all directions of ocular movement, except in convergence and in divergence.

Orthophoria is that ideal condition, in which the eyes are parallel without the effort of fusion faculty for distant fixation, and have the proper convergence for near vision. This in normal ocular-muscle balance. Only a few people are orthophoric, and some degree of heterophoria is almost universal.

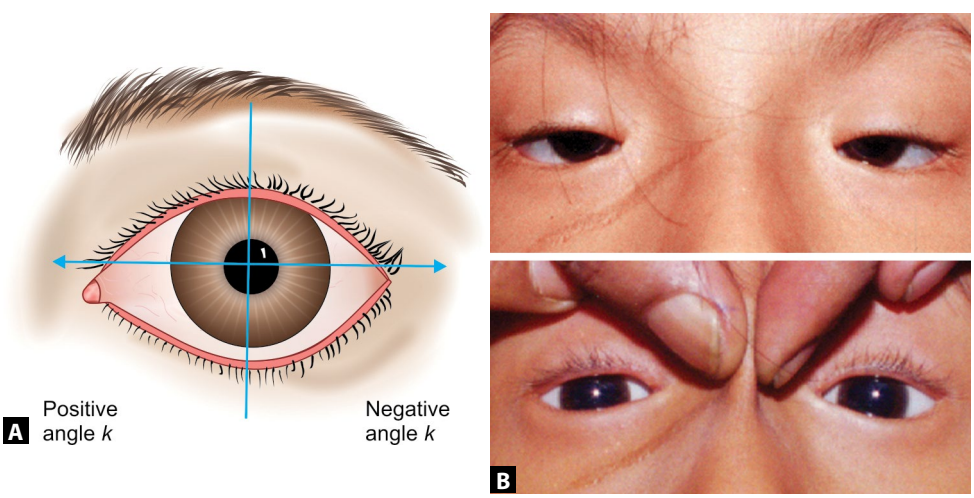
Position of rest is usually a mild divergence as in sleep, deep anaesthesia, or after death.

Apparent Squint (Pseudostrabismus) (Fig. 23.8A)

Here, the visual axes are in fact parallel, but the eyes seem to have squint.

Causes

- Prominent epicanthic folds (Fig. 23.8B) in infants—may simulate the appearance of a convergent squint.
- Hypertelorism—an apparent divergent squint.
- Hypermetropia—an apparent divergent squint due to large angle 'kappa'.
- Myopia—an apparent convergent squint due to smaller, or even negative angle 'kappa'. Squint in these conditions is easily excluded by checking the relative position of corneal light reflections, and also by other means.



Figs 23.8A and B: A. Pseudostrabismus (schematic representation); **B.** Pseudoconvergent squint in telecanthus and epicanthic folds (photographs)

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Latent Squint (Heterophoria) (Fig. 23.9)

This is a tendency for the eyes to deviate, but that is prevented by the fusion mechanism for the interest of BSV.

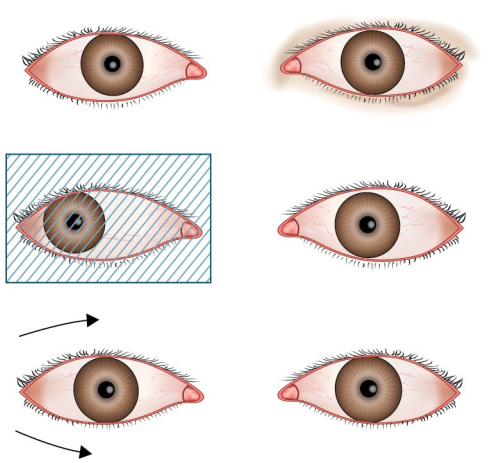


Fig. 23.9: Latent divergent squint (exophoria). Note the eye deviates under the cover

But, under condition of stress, or when the fusion is interrupted the deviation readily becomes manifest.

Types

- ***Esophoria:*** Tends to deviate inwards.
- ***Exophoria:*** Tends to deviate outwards.
- ***Hyperphoria:*** Tends to deviate upwards.
- ***Cyclophoria:*** Torsional deviation.

In general, esophoria is common among hypermetropes, and exophoria in myopes. In presbyopia, as the near point recedes, the convergence becomes weaker and there is a tendency for exophoria.

Symptoms

The smaller degrees of eso- and exophoria are extremely common, and as a rule, they do not give rise to any symptom. But when the deviation is more than 5°, there is usually some amount of distress. The most acute distress is associated with cyclophoria.

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The symptoms are:

- ***Blurring of vision***, especially at times of fatigue. Initially, with an effort of fusion, this blurring may be overcome.
- ***Headache*** and ***eyeache*** (strains) are extremely common.
- ***Intermittent diplopia*** due to exhaustion of fusional reserve intermittently.

Diagnosis

- ***Cover test:*** One eye is covered and the other eye is made to fix on an object. In presence of heterophoria, the eye under cover, deviates. When the cover is removed, the covered eye corrects the deviation, and regains its normal position quickly. Cover test should be done both for near and distant objects with or without spectacles.
- ***Maddox rod:*** It consists of a series of parallel high-power, plus cylinders, that convert a point light as a red straight line, at right angle to the axis of rod. When Maddox rod is held in front of one eye, the images of the point source of light in two eyes become dissimilar, and fusion becomes dissociated (dissimilar image test). This test is performed at a distance of 6 meters and at 33 cm.
  In heterophoria, the red line of light becomes separated from the point source of light.
- ***Maddox wing:*** It is a device to find out the amount of heterophoria for near vision (at 33 cm).
- ***Prism vergence tests:*** The strength of the involved muscles are to be tested by forcing them to a maximum effort against the prism.
- ***Synoptophore evaluation:*** To know the range of fusion.

Treatment

- Smaller degrees of heterophoria which give rise to no symptom—require no treatment.
- ***Errors of refraction must be corrected*** fully by spectacles, which should be worn constantly.
- ***Orthoptic exercises:*** With the help of a synoptophore, to increase the fusional range and muscle power.
- ***Exercises with prism (adverse prism)*** are practised against prisms with their bases turned towards the direction of deviation.
- ***Relief of symptoms with the prisms (relieving prism):*** Prisms are incorporated with the spectacles to correct the defect optically. The base of the prism is placed in the opposite side of the direction of deviation, and the power is equally divided between the two eyes.
- ***Operative treatment:*** It is rarely indicated, especially in a large deviation, and then the heterophoria is to be treated as heterotropia.

CONCOMITANT SQUINT

It is the dissociation of the eyes, wherein the angle of deviation remains the same in all directions of gaze, irrespective of eyes of fixation.

Etiological Factors

- ***Poor vision in one eye,*** e.g. uncorrected refractive errors, opacities in the media, diseases of the retina or optic nerve, etc.
- ***Disturbances of ocular muscle-balance,*** e.g. congenital malinsertion or maldevelopment of the extraocular muscles.
- ***Dissociation between accommodation convergence relationship,*** e.g. in case of hypermetropia → more accommodation is exerted to correct the error → more convergence → chance of convergent squint.
  Conversely, in myopia → minimum or no effort of accommodation to correct the error → weakness of convergence → tendency for divergent squint.

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Figs 23.10A and B: **A.** Left convergent squint; **B.** Cross-fixation in convergent squint

- ***Decompensated heterophoria*** → manifest heterotropia.
- ***Central cause:*** as in cerebral palsy or mental retardation, due to deficient development of fusion faculty.

Types

Monocular Squint

Squint is normally monocular (about 85% of cases), in the sense, that one eye habitually takes up fixation, and the other eye, therefore, becomes the squinting eye. It may be convergent, divergent, or vertical.

- ***Convergent concomitant squint (Figs 23.10A and B):*** One eye always deviated inwards, and it typically develops in early childhood before the binocular reflexes are firmly established. Amblyopia is a frequent association.

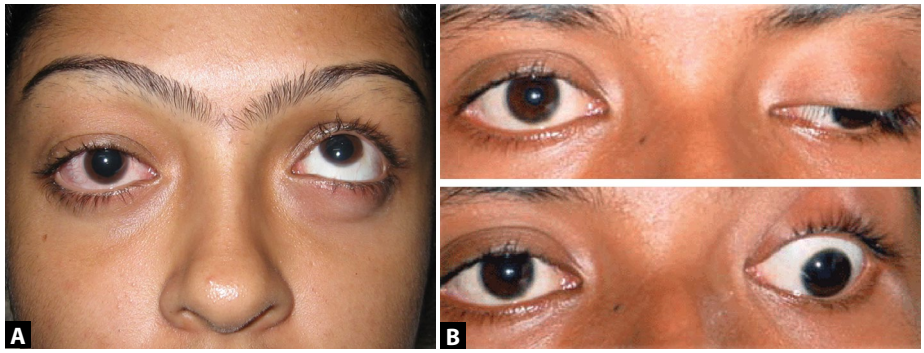
  Causes
  - ***Hypermetropia*** with excessive use of accommodation is the common cause, and frequently becomes obvious after a debilitating illness.
  - ***Congenital myopia:*** Due to excessive use of convergence for near vision.
  - ***Uncompensated esophoria.***
  - ***Anatomical factors***, e.g. orbital asymmetry, enophthalmos, etc.
- ***Divergent concomitant squint (Fig. 23.11):*** One eye always deviates outwards. It is characteristically seen in older children and adults, and less frequently associated with amblyopia.



Fig. 23.11: Right divergent squint

Causes
- ***Inherent neuromuscular incoordination:*** It is the cause of primary divergent squint in a child. It is initially intermittent, and later on, becomes constant. The angle of deviation is usually greater for distant fixation than for the near.
- ***Uncorrected myopia.***
- ***Complete loss of vision in one eye:*** As in unilateral cataract, after injury, etc. (blind eye takes the position of anatomical rest—***secondary divergent squint***).

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Figs 23.12A and B: A. Vertical squint left hypertropia; **B.** Vertical squint left hypotropia (right eye fixing)

- ***Consecutive divergent squint:*** Due to overcorrection of a convergent squint by operation.
- ***Craniofacial anomalies:*** As in hypertelorism and Crouzon syndrome, due to widely separated orbits.
- ***Decompensated exophoria***.

- ***Vertical concomitant squint (Fig. 23.12A and B):*** It is rare. Most of the cases are those of hyperphoria which become manifest.

Alternating Squint (Figs 23.13 to 23.15)

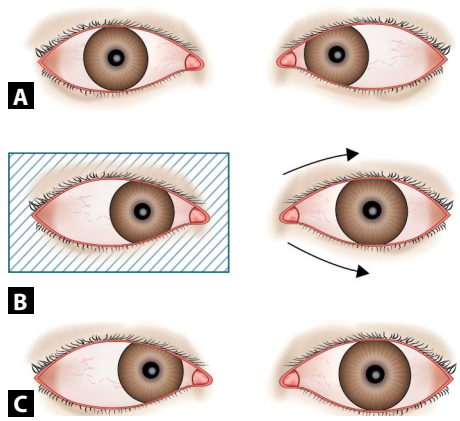
Alternating squint means when one eye fixes, the other eye deviates, and either of the eyes can adopt fixation alternately and freely. It may be convergent or divergent.

In an alternate squint, the visual acuity remains normal (or near normal) in each eye. There is no diplopia, as the image formed in the deviating eye is completely suppressed by the brain.

Alternating squint may be either, due to congenital weakness of either of lateral or medial rectus of each eye, or due to refractive errors.

Clinical Features (in General)

Usually, there is no symptom. The squinting of the eye is noticed by the parents or relatives.

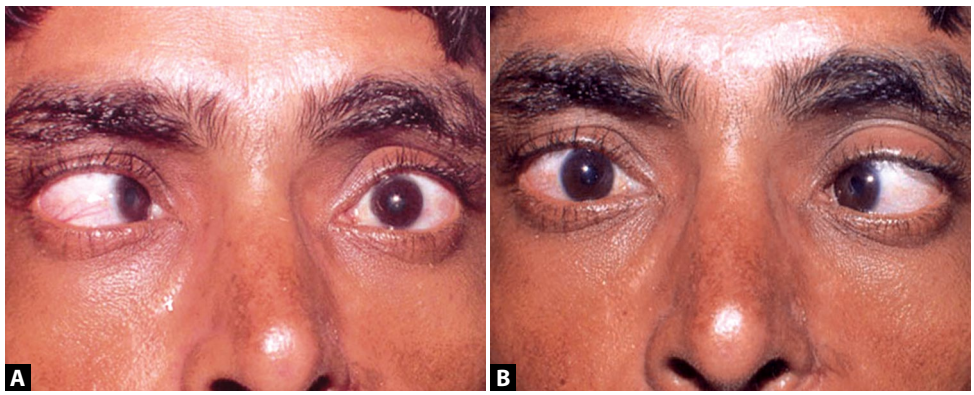


Figs 23.13A to C: Alternate convergent squint. **A.** Left eye convergent; right eye fixing; **B.** On cover test-left eye taking fixation and right eye converging; **C.** On uncover—right eye convergent; left eye fixing

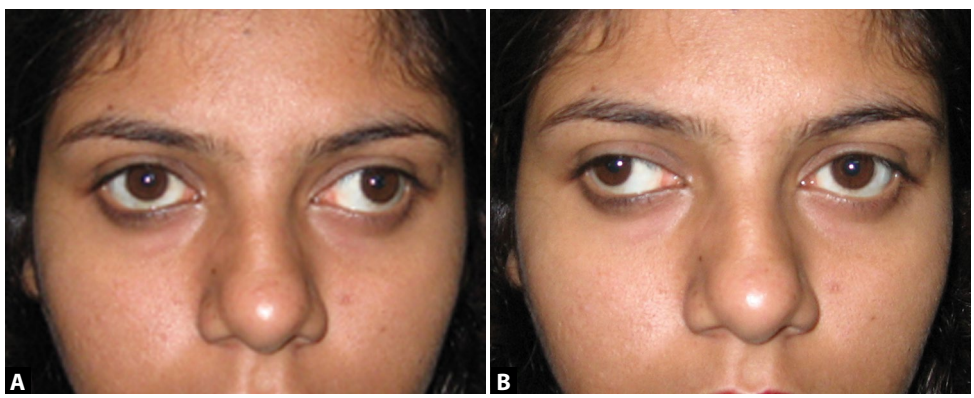
There is no diplopia, as the suppression develops easily in the young age.

- In case of uniocular squint, visual acuity in usually poor (strabismic amblyopia). This is maximum with convergent squint.
- There is no limitation of eye movements.
- Primary angle of deviation is equal to the secondary angle of deviation the primary deviation means, the angle of deviation of the squinting eye, when the other eye fixes an object, secondary deviation means, the angle of deviation of the normal eye under cover, when the squinting eye is made to fix an object by covering the normal eye.

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Figs 23.14A and B: Alternate convergent squint. **A.** Right eye convergent; left eye fixing; **B.** Left eye convergent; right eye fixing



Figs 23.15A and B: Alternate divergent squint. **A.** Left eye divergent; right eye fixing; **B.** Right eye divergent; left eye fixing

Evaluation of Concomitant Squint

History

- ***Age of onset:*** The older the age of onset and shorter the squinting period, the better is the prognosis.
- ***History of an acute illness or debility*** at the time of onset.
- ***Family history*** may often be positive.
- ***Intermittent or constant:*** The prognosis is better in the intermittent cases than in constant type, as it is likely, that some form of binocularity is still present in intermittent type.
- ***Unilateral or alternating:*** In constant unilateral squint, the presence of amblyopia should be suspected.
- ***Diplopia or head posture:*** To differentiate it, from a paralytic squint.

Examination

- ***Visual acuity testing:*** By Snellen's chart, 'illiterate E', Sheridan-Gardiner test, or by special tests, designed for younger children and mentally retards (STYCAR).
- ***Refraction under atropine:*** To find out any refractive error.
- ***Ocular motility:*** To find out any limitations of ocular movement.

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- ***Cover test:*** To find out the squint is uniocular or alternating.
- ***Fixation behavior:*** To find out foveal (centric) fixation or eccentric fixation.
- ***Anterior segment and fundus:*** To rule out any organic lesion which may be the cause of squint.
- Measurement of the angle of squint
  - ***Corneal reflection test (Hirschberg) (Fig. 23.16):*** A reasonable assessment of the angle is made by holding a fixation light at 30 cm distance from the patient, and estimating the distance of the corneal light reflex (Purkinje's first image) from the pupillary center.
    One mm decentration of reflex corresponds to about 7° of ocular deviation. For example, if the reflex is situated at the pupillary margin, the angle is about 15°, and if it is at the limbus, the angle of squint is about 45°. This test is also useful to differentiate it from pseudostrabismus.
  - ***Prism reflex test (Krimsky):*** Prisms are placed in front of the deviating eye until the corneal light reflexes become symmetrical.
  - ***Prism-bar-cover test (PBCT):*** The prism power required to negate the ocular movements, equals to the angle of deviation, and it is measured in prism diopter (Δ). Roughly, 1° of ocular deviation equals to 2 prism diopter (2Δ) deviation. It is the ***most accurate*** method of measuring the angle of deviation.
  - ***Perimeter or tangent screen:*** It is rarely used now-a-days.
  - ***Synoptophore (major amblyoscope):*** This is one of the accurate methods of estimation of the angle of squint.
- ***Worth's 'four-dot' test:*** To diagnose the presence of suppression and abnormal retinal correspondence in a manifest squint.
- ***State of binocular vision:*** All three grades of binocular vision (SMP, fusion and stereopsis) are determined by synoptophore. The alternating squint does not possess any grade of binocular vision. In intermittent squints, binocular vision is being maintained for the part of the time.

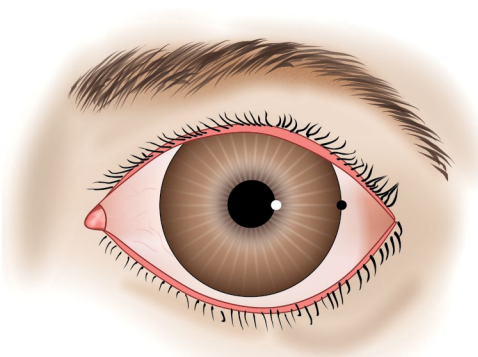


Fig. 23.16: Hirschberg's corneal reflection test

Treatment of Concomitant Squint

The aim of the treatment is to make the eyes straight, and to ensure BSV.

The treatment should be started as early as possible, so that the restoration of BSV is not delayed.

The prognosis decreases significantly after the age of 6 years and in adults the correction is purely for cosmetic reason.

Remember 4 'O's during treatment:

- ***Optical correction of refractive errors:*** By suitable spectacle. Some squints (particularly fully accommodative convergent squint) are completely controlled by spectacle correction.
- ***Occlusion therapy:*** To treat the amblyopia as already discussed.
- ***Orthoptic exercises:*** They are given with a synoptophore or prism-bar to overcome suppression, to improve the range of fusion, and to ensure easy stereopsis.
  These are initial steps to restore BSV, earlier before the surgical intervention. Fusional exercises are to be continued when the eyes become straight after the operation.

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- ***Operative measures:*** Surgical intervention should be undertaken after initial occlusion therapy, when there is no further improvement of vision in the squinting eye.

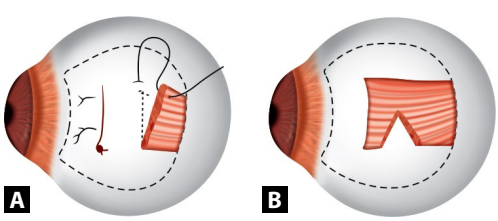
Types of Squint Operation

Weakening Procedures (Figs 23.17A and B)

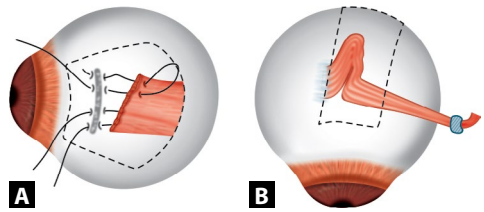
- ***Recession:*** It can be performed on any extraocular muscle. Here, the muscle insertion site is moved posteriorly towards the equator of the globe.
- ***Marginal myotomy:*** It is done by cutting partially through the muscle margin.
- ***Myotomy (tenotomy):*** It is commonly performed on an overacting inferior oblique muscle, by cutting across the muscle near its insertion, (tenotomy for superior oblique muscle).
- ***Disinsertion:*** It is almost a similar procedure, done in case of contracted rectus muscle as in thyroid ophthalmopathy.
- ***Faden procedure*** (Faden means suture) (posterior-fixation suture): It is the suturing of the muscle belly to the globe at about 12 mm behind its insertion. This will weaken its effective action.

Strengthening Procedures (Figs 23.18A and B)

- ***Resection:*** Effective pull of the muscle is enhanced by making it shorter, and is suitable only for rectus muscles.



Figs 23.17A and B: Weakening procedure.
A. Recession; **B.** Marginal myotomy



Figs 23.18A and B: Strengthening procedure.
A. Resection; **B.** Tucking of superior oblique muscle

- ***Advancement:*** The muscle is first disinserted, and then re-inserted nearer to the limbus.
- ***Tucking (plication):*** This effectively shortens the muscle, and is usually reserve for superior oblique muscle in 4th nerve palsy.

General principles of squint surgery:

- The degree of resection or recession, that is required, can not be determined mathematically. However, 1 mm recession or resection of medical rectus corrects about 3° of deviation; and for lateral rectus, it is about 2°.
- Medial rectus should not be recessed more than 5.5 mm, as this may cause convergence insufficiency. Similarly, resection of medial rectus is limited to a maximum of 5.5 mm, to prevent retraction of the globe, and narrowing of the palpebral fissure.
  The corresponding figure for the lateral rectus is 7 mm.
- It is wise to aim towards undercorrection especially in children, as a residual 5–10° may well disappear, once the stereoscopic vision is established.
- It is preferable to operate on elevators, rather than on depressors which are important in reading and walking. A 3 mm muscle recession corrects about 10° of vertical squint.

Choice of Surgery

- ***In convergent squint***
  - Medial rectus recession and lateral rectus resection of the squinting eye.

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Figs 23.19A and B: Left divergent squint. **A.** Before correction; **B.** After surgical correction

- Medial rectus recession of the both eyes (bimedial recession).

- ***In divergent squint (Figs 23.19A and B)***
  - Lateral rectus recession and medial rectus resection of the squinting eye.
  - Recession of lateral rectus of the both eyes (bilateral recession).
- ***In alternating squint:*** Treatment is only for cosmetic purpose, as fusional faculty does not develop in this type of squint. Both eyes are to be tackled to correct the deviation (e.g. bimedial recession or bilateral recession).

  Depending upon the amount of deviation, 'two', 'three' or 'four' muscles of two eyes are to be tackled.

  As there is good visual acuity in each eye, the patient must be warned that diplopia may follow the operation, and may persist for months.

PARALYTIC SQUINT

It is the misalignment of the visual axes as a result of paresis, or paralysis of one or more extraocular muscles. It is characterized by impaired movement in the field of action of the muscle(s), and thus the angle of deviation varies in different directions of gaze and with fixation of eyes.

Etiology

- ***Lesions affecting the cranial nerve supplying the muscle:***
  - ***Nuclear lesions***—e.g. cerebrovascular accidents, infection, neoplasm, aneurysm, toxin (diphtheric), etc.
  - ***Lesions of the nerve***—e.g. head injury, meningitis, cavernous sinus thrombosis, fracture of the base of the skull, pressure from a neoplasm or aneurysm.
- ***Lesions in the muscle:***
  - ***Congenital maldevelopment*** of a muscle.
  - ***Direct injury*** to the muscle.
  - ***Diseases of the muscles***—as in ocular myopathy, thyroid myopathy, myasthenia gravis, etc.

Clinical Features

- ***Diplopia:*** Paralytic squint in an adult with previously single binocular vision, causes diplopia. It is the chief symptom, and most marked when the eye is rotated into the

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field of action of the paralyzed muscle. Diplopia may be crossed, uncrossed, or may be with tilting.

- ***False orientation of the object:*** Object is projected too far in the direction of paralyzed muscle, due to increase in secondary deviation.
- ***Vertigo and nausea:*** They are partly due to diplopia, and partly due to false orientation.
- ***Secondary angle of deviation is more than*** the primary deviation (Fig. 23.20). This is because of excessive innervation is required to move and maintain the eye in primary position when the patient fixates with the paralytic eye (Hering's law of equal innervation).
- ***Restriction of ocular movements*** in the direction of action of paralyzed muscle.
- ***Compensatory head posture:*** In paralytic squint, to neutralize diplopia the chin may be elevated or depressed, the face is turned to right or left side, and the head may be tilted to the right or left shoulder (ocular torticollis). This head posture, is to neutralize the angle of deviation, or to separate the images maximally, so as to avoid diplopia.

  In ***paralysis of horizontal rectus*** muscle, the face is turned to field of action of the paralyzed muscle, but the head is not tilted. As, in right lateral rectus palsy, the patient keeps his face turned to the right.

  In case of ***cyclovertical muscle palsy***, it is more complicated, and less valuable

Fig. 23.20: Primary angle and secondary angle of deviation

In ***concomitant squint*** (right eye), the primary deviation (P) is equal to the secondary deviation (S), obtained when the non-squinting eye is occluded. In ***paralytic squint***, the secondary deviation is greater than the primary

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Difference between concomitant and paralytic squint

Concomitant squint	Paralytic squint
Mostly developmental, usually not associated with trauma/disease	Mostly acquired, usually associated with trauma/disease
Occurs in infants and children	Usually in adults
Diplopia absent	Diplopia present
No abnormal head posture	Abnormal head posture to avoid diplopia
Absence of false projection (i.e. false localization of object)	False projection is present
Angle of squint is usually constant in all directions of gaze	Angle of squint is variable in different directions of gaze
Ocular movements are normal and there is no restriction	Ocular movement is restricted in the direction of action of paralyzed muscle
Secondary angle of deviation is always equal to primary deviation	Secondary deviation is greater than primary deviation
Amblyopia is present	No amblyopia
Surgical result is usually good	Surgical result is not satisfactory, and at times, surgery may be contraindicated

diagnostically. Face turn and chin position are towards the main action of the paralysed muscle, and the head tilt is towards the hypotropic eye. As in ***superior oblique palsy***, the head is tilted on the side of the normal eye, the face is turned to normal side, and the chin is depressed.

- ***Visual acuity is normal*** in both eyes, and there is no amblyopia.

Different Types of Ocular Paralysis

Total ophthalmoplegia (Fig. 23.21): It means involvement of both extrinsic and intrinsic muscles of the eyeball. In unilateral cases, the lesion is in the cavernous sinus, or in the superior orbital fissure, and in bilateral cases, the lesion is widespread in the brainstem due to vascular or inflammatory cause.

Clinical signs

- Ptosis.
- The eyeball is slightly proptosed and divergent (due to anatomical position of rest).
- No movement of the eyeball in any direction.
- Fixed dilated pupil (no reaction to light, accommodation and convergence).
- Total loss of accommodation.

External ophthalmoplegia: It is due to paralysis of extrinsic muscles which includes six extraocular muscles and the levator. It is due to nuclear lesion without affecting the Edinger-Westphal nucleus, which supplies the intrinsic muscles.

Signs are same as total ophthalmoplegia except, that the pupillary reactions and accommodation are normal.

Third (Oculomotor) Nerve Palsy (Fig. 23.22)

- Ptosis due to weakness of levator.
- Eyeball rotates outwards (divergent) and slightly downwards due to unopposed action of the lateral rectus (N VI) and superior oblique (N IV) muscles.
- Intorsion of the eyeball on attempted down gaze, due to action of superior oblique muscle.
- Ocular movements are restricted in all direction, except outwards (due to lateral rectus).
- Pupil is dilated, and does not constrict to light or convergence.

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Fig. 23.21: Total ophthalmoplegia



Fig. 23.22: Right third nerve palsy

Fourth (Trochlear) Nerve Palsy

Trauma is the most common cause of isolated fourth nerve palsy, which may be bilateral. It causes paralysis of the superior oblique muscle. The clinical features are as follows:

- ***Abnormal head posture:*** Chin depression, head tilt and slight face turn to the opposite (normal) side.
- Diplopia is most troublesome, as it is more in downgaze.
- Eyeball deviated upwards and inwards (ipsilateral hypertropia).
- Extorsion of the globe (ex-cyclotropia).
- Restriction of the ocular movements on downwards and inwards.
- Bielschowsky's 'three-step' test is useful to diagnose a fourth nerve (superior oblique) palsy (Figs 23.23A and B).

When a healthy individual tilts their head, the superior oblique (SO) and superior rectus of the eye closest to the shoulder keep the eye level. The inferior oblique and inferior rectus keep the other eye level.

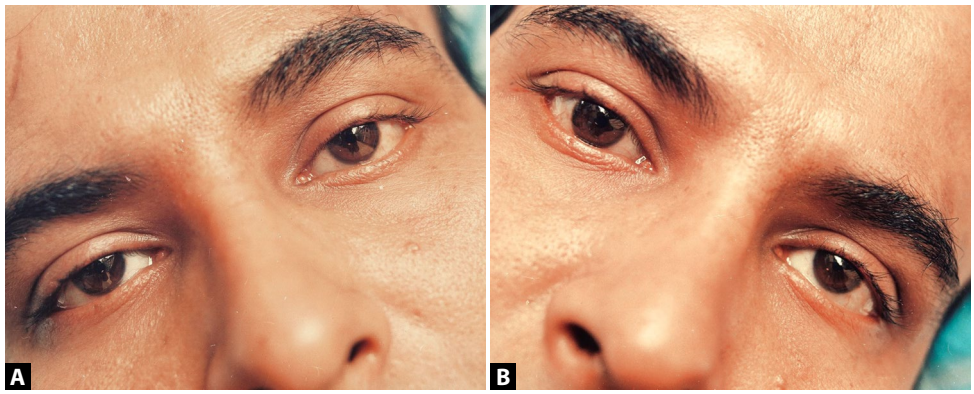
In patients with SO palsy: The SR muscle's action is not counteracted by the SO. This leads to vertical deviation of the affected eye when the head is tilted towards the effected eye.

However, there is no deviation when the head is tilted towards the unaffected eye because the SO muscle is not stimulated in the effected eye, but rather it is stimulated in the unaffected eye.

Sixth (Abducens) Nerve Palsy

This is the most common type, and commonly occurs in raised intracranial tension.

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Figs 23.23A and B: Bielschowsky's head tilt test in left superior oblique palsy

The long intracranial course, and its angulation over the petrous-tip of the sphenoid bone, make it vulnerable in raised intracranial tension. In this situation, the sixth nerve palsy is a false localizing sign.

Signs: They are due to paralysis of lateral rectus (Fig. 23.24).

- The eyeball is rotated inwards (convergent squint).
- Defective abduction of the eye, partially or completely.
- Face turn towards the field of action of paralysed muscle (e.g. a patient with right sixth nerve palsy will turn his face to the right).



Fig. 23.24: Right sixth nerve (lateral rectus) palsy. Note—absence of abduction in right eye

Investigations

- ***Diplopia charting (Fig. 23.25):*** It is done in a dark room, with red glass placed in front of right eye and green glass in front of left, to dissociate the images. A streak light is moved in different areas of binocular vision, with the head being held stationary. Position of two images are recorded on a chart with nine squares (representing nine diagnostic positions of gaze) marked on it.

  Now, note

  - The direction of gaze, where the separation of images are maximum.
  - The farther displaced image (outer image) belongs to which eye. This can be done by covering one eye of the patient, and asking him which one of the two images has disappeared. The farther displaced image belongs to the affected eye.

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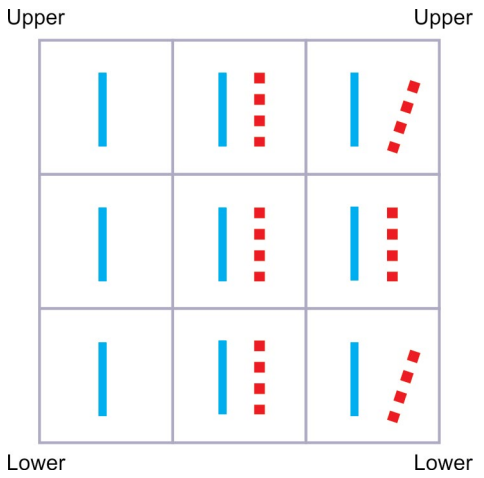


Fig. 23.25: Diplopia chart showing the position of the images in right lateral rectus palsy

The dotted lines show the positions of the false image in different parts of the field of diplopia

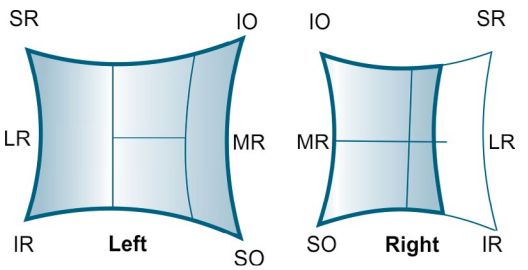


Fig. 23.26: Hess chart in right lateral rectus palsy

SR—Superior rectus; **IO**—Inferior oblique; **LR**—Lateral rectus; **MR**—Medial rectus; **IR**—Inferior rectus; **SO**—Superior oblique

- ***Hess screen (chart) (Fig. 23.26):*** The eyes are dissociated similarly with red and green goggles, and note the patients response to the dissimilar images formed by each eye while viewing a different targets (dot and line) on Hess screen.
  - The results of the test is interpreted by comparing the grids, charted for each eye, and they depend on ***Herring's law of equal innervation***.
  - In paralytic squint, the greatest restriction occurs in the direction of paretic muscle, with corresponding overaction of the contralateral synergistic muscle. (Shorter chart is of paralytic eye and the larger chart is due to overacting contralateral synergists). It also provides the accurate measurement of commitance.
- ***Worth's four-dot test:*** Patients eyes are dissociated with red glass placed in front of right eye and green glass in front of left. He then views at a box with four lights (dots); one red, two green and one white (Fig. 23.27).
- ***Forced duction test (FDT):*** This test is used to differentiate defective ocular movements due to physical restriction, from a muscle paralysis.

  After topical anesthesia, the insertion of the affected muscle is grasped with fixation forceps, and gently attempted to rotate the eyeball in the field of action of weak muscle.

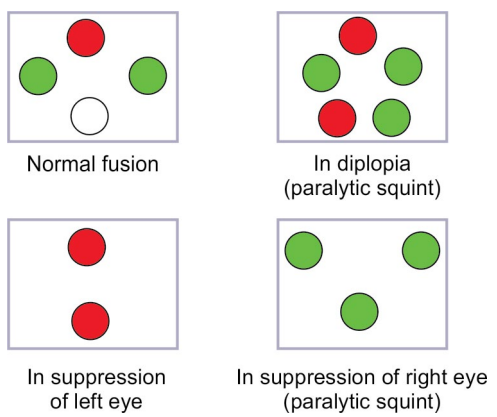


Fig. 23.27: Worth's four-dot test

The interpretations of Worth's four-dot test are as follows:

If the patient sees	Presence of
All four lights	Normal fusion
All four lights, even in presence of a concomitant manifest squint	Abnormal retinal correspondence (ARC)
Two red and three green lights	Diplopia (paralytic squint)
Two red lights	Suppression of left eye
Three green lights	Suppression of right eye
Two red or three green lights alternately	Alternate suppression

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FDT 'positive' means, it is difficult to move the globe with the forceps (e.g. contracture of muscle as in thyroid myopathy, trapped muscle in orbital floor fracture, etc.).
FDT is 'negative' in case of muscle paralysis.

- ***Investigations of find out the cause,*** e.g. neurological consultation, X-ray skull, CT scan of brain and orbit, MRI-scan, blood sugar, thyroid function tests, 'Tensilon test' for myasthenia gravis, etc.

Sequelae of extraocular muscle palsy:

- Overaction of contralateral synergist (Herring's law of equal innervation).
- Contracture of ipsilateral antagonist.
- Inhibitional (secondary) palsy of the contralateral antagonist (Sherrington's law of reciprocal innervation).

For example

- ***In right lateral rectus palsy***
  - Overaction of left medial rectus
  - Contracture of right medial rectus
  - Inhibitional palsy of left lateral rectus.
- ***In right superior oblique palsy***
  - Overaction of left inferior rectus
  - Contracture of right inferior oblique
  - Inhibitional palsy of left superior rectus.

Treatment

- Treatment must be directed towards the cause of paralysis.
- For the relief of diplopia:
  - Occlusion of affected eye temporarily.
  - Suitable prism correction for minor diplopia.
- Observation for at least 6 months, so that maximum amount of spontaneous recovery could take place.
- Recession of contralateral synergist may be done for the nerve palsy. Alternately, various type of muscle transposition operations may be undertaken.
- Botulinum toxin injection may be tried in some cases—to prevent contracture of the antagonist muscle.

DUANE'S RETRACTION SYNDROME (FIG. 23.28)

This type of non-commitant squint, occurs because of aberrant innervation which causes contraction of lateral rectus muscle instead of relaxation, when the medial rectus muscle contracts (i.e. co-contraction of medial and lateral rectus muscles during adduction).

Clinical Features

- Marked restriction, or absence of abduction.
- Slight restriction of adduction.
- Retraction of the globe on attempted adduction.
- Narrowing of the palpebral aperture on adduction, and widening on attempted abduction.
- Deficiency of convergence of the affected eye.
- 'Up-shoot' or 'down-shoot' of the eye on adduction.

This is Type-I (most common type), and the other types are:

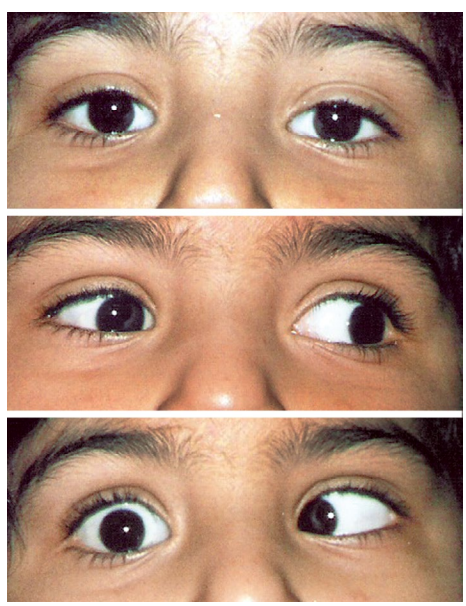


Fig. 23.28: Duane's retraction syndrome

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Type II: Limitation of adduction with relatively normal adbuction.

Type III: Limitation of the both abduction and adduction.

Girls are more commonly affected than boys. The left eye is affected more commonly than the right eye, and in few cases, it may be bilateral.

Treatment is only indicated, when there is ocular deviation in primary position. But, no method can restore a full and normal range of ocular movement.

Superior oblique Tendon-Sheath syndrome (Brown's syndrome)

It is usually congenital, but acquired Brown's syndrome may occur following trauma, tenosynovitis of the superior oblique tendon-trochlea apparatus.

Clinical features

- Limitation of elevation of the eye on adduction, simulating inferior oblique muscle palsy.
- Normal elevation on abduction.
- The eyes are straight on primary position, and there is no overaction of superior oblique muscle.
  - ***Diagnosis*** is confirmed by FDT, which is 'positive' in attempting to elevate the eye from adducted position.
  - ***Surgical intervention*** (superior oblique tenotomy) should only be considered in extreme cases.

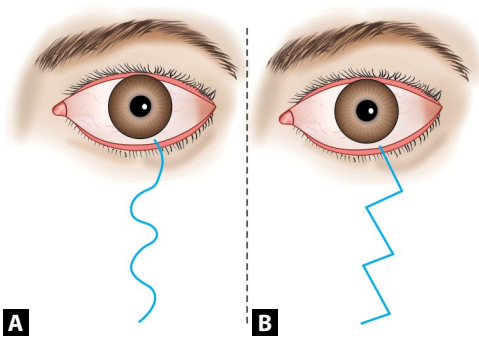
NYSTAGMUS (FIG. 23.29)

This is an involuntary, rhythmic, to-and-fro oscillation of the eyeballs.

It is usually detected by simple observation of the eyes. But when minimal, it may be observed during slit-lamp examination or ophthalmoscopy, under magnification.

On the basis of rhythm, nystagmus may be classified as:

- ***Pendular nystagmus (Fig. 23.29A):*** To-and-fro oscillation of the eyes are equal in velocity and amplitude in each direction. The movements may be horizontal, vertical, oblique or rotatory.
- ***Jerky nystagmus (Fig. 23.29B):*** It has a slow component in one direction, and a fast component in the opposite direction. The direction of nystagmus is defined by the direction of the fast component, although the slow movement reflects the basic disability.
- ***Mixed:*** Pendular nystagmus in primary position, and jerky in lateral gaze.



Figs 23.29A and B: Nystagmus. **A.** Pendular; **B.** Jerky

Clinical Types

Physiological Nystagmus

- ***End-gaze nystagmus:*** Fine jerky nystagmus in extreme gaze positions. It is common after administration of barbiturates and other tranquillizers.
- ***Optokinetic nystagmus (OKN):*** It is a jerky nystagmus induced by moving repetitive visual patterns across the visual field. It has a slow phase in the direction of moving pattern, followed by a fast phase in the opposite direction, to refixate the eye on the next target. It is also called ***railroad nystagmus***.
  - ***Clinical applications:*** For testing visual acuity in infants or young children (Catford drum test), and for detecting malingering.
- ***Vestibular nystagmus:*** It is a jerky nystagmus, caused by altered input from the vestibular nuclei to the horizontal gaze centers. Clinically, caloric test is done to induced vestibular nystagmus in suspected gaze paralysis.

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- If hot water is irrigated into right ear—the patient will develop right jerky nystagmus; and if cold water into right ear—the patient will develop left jerky nystagmus (mnemonic 'C-O-W-S', cold-opposite and warm-same).
- If both ear are stimulated—for cold water the patient develops up-beat jerky nystagmus and for warm water, down-beat jerky nystagmus (mnemonic—'C-U-W-D', cold-up and warm-down).
- In pathological vestibular nystagmus, the fast phase of jerky nystagmus is directed to the opposite side, and is associated with deafness, vertigo and tinnitus, e.g. in Meniere's disease, or labyrinthitis.

Types of nystagmus are described in Table 23.1.

Ocular (Sensory-Deprivation) Nystagmus

This is typically provoked by lowered central vision in the first few months of life, before the fixation reflexes have matured.

- Nystagmus occurs, if the lesion occurs before the age of 6 months. In later life, there is nystagmoid excursions, but after the 6 years this type of nystagmus does not occur.
- Ocular nystagmus is typically pendular and horizontal, but on looking to either side, it may break down into a jerky nystagmus.
- ***Common causes*** are—bilateral congenital cataract, albinism, aniridia, congenital high myopia, macular hypoplasia, Leber's optic atrophy, etc.

TABLE 23.1: Types of nystagmus

Types	Characteristics	Causes
Congenital nystagmus	Jerky type and horizontal, absent during sleep, and persists throughout life	Usually hereditary
Latent nystagmus	Bilateral jerky, horizontal nystagmus, elicited by covering one eye. Fast component is towards the uncovered eye. With both eyes open, patient has normal vision, but when one eye is covered, visual acuity of the open eye may be 6/60 or less	Unknown, but convergent squint is a common association
Spasmus nutans	Pendular nystagmus, abnormal head posture, and head nodding. Occurs between 4 months to 2 years of age. Spontaneously cured by 3 years of age	Unknown
Ataxic (gaze-paretic) nystagmus	Nystagmus of abducted eye	Internuclear ophthalmoplegia
Down-beat nystagmus	Jerky nystagmus, with fast phase downwards	Lesion at the cervicomedullary junction at the foramen magnum
Up-beat nystagmus	Fast phase in upward direction	Phenytoin sodium intoxication, or posterior fossa lesions
See-saw nystagmus	One eye rising and intorting, while the other eye falls and extorts	Chiasmal lesion (with bitemporal hemianopia)
Periodic alternate nystagmus (PAN)	Jerky nystagmus, with rhythmic changes in amplitude and direction.	Demyelinating disease of the brainstem

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Types	Characteristics	Causes
Nystagmus retractorius	Jerky nystagmus, with convergent movement of two eyes, and retraction of the globe. May be associated with vertical gaze palsy, lid retraction. Light-near dissociation of pupils, and accommodative spasm ***(Parinaud's syndrome)***	Lesions in the pretectal area, e.g. vascular lesions or pinealoma
Miner's nystagmus	Rotatory rapid nystagmus, defective vision at night with dancing of lights and movements of objects	Fixation difficulties in dim illuminations in coal mines
Nystagmus blockade syndrome	Occurs in some cases of congenital nystagmus in which a 'purposive esotropia' dampens the horizontal nystagmus	Compensatory phenomenon

Motor Imbalance Nystagmus

This is due to primary defect in the efferent mechanism and acquired in nature.

Treatment

Basic aim of the treatment is, ***to transfer the 'neutral point'*** (where the nystagmus is least apparent) ***from an eccentric point to a straight ahead position***. So, there is elimination of the compensatory head posture, and improvement of vision in primary gaze. This is done by:

- Prismotherapy
- Surgical treatment:
  - ***Kestenbaum's operation:*** The aim of the surgery is to shift the null (neutral) point to the primary position.
  - ***Faden*** or posterior-fixation suture operation may be better in this situation.

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24 CHAPTER

Ocular Injuries

CLASSIFICATION OF MECHANICAL INJURIES

Classification of mechanical injuries

- **Closed globe injury**
  - Contusion
  - Lamellar laceration
  - Superficial foreign body
  - Mixed

  Zone I: Conjunctiva, cornea and sclera involved
  Zone II: Anterior chamber and lens is involved
  Zone III: Posterior part is involved.
- **Open globe injury**
  - Rupture
  - Penetrating
  - Intraocular foreign bodies
  - Perforating
  - Mixed

  Zone I: Only cornea is involved
  Zone II: Cornea and sclera up to 5 mm
  Zone III: Wound extends > 5 mm of the sclera.

CONTUSIONS (BLUNT INJURY)

- Ocular injuries by blunt instruments vary in severity, from simple subconjunctival hemorrhage to rupture of the globe.
- Every part of the globe may be so injured by contusion, that may seriously cause diminished vision. Moreover, in some cases the effects are progressive or delayed. So in all cases of contusions, a guarded prognosis should be given.
- The ***mechanisms*** of ocular tissue damage are:
  - ***Direct effect*** of injury,
  - ***Indirect effect*** against bony orbit, and
  - ***Contrecoup effect*** due to propagation of wave of thrust, to and fro within the globe.

The various ocular effects resulting from contusions are briefly enumerated below.

Eyelids

- Lid lacerations
- Swelling and ecchymosis (black eye) (Fig. 24.1)
- ***Emphysema*** of the eyelids [crepitation on palpation, due to orbital fracture with involvement of peripheral nervous system (PNS) (Fig. 24.2)].



Fig. 24.1: Black eye—panda bear sign

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Fig. 24.2: Ecchymosis with emphysema of the lid

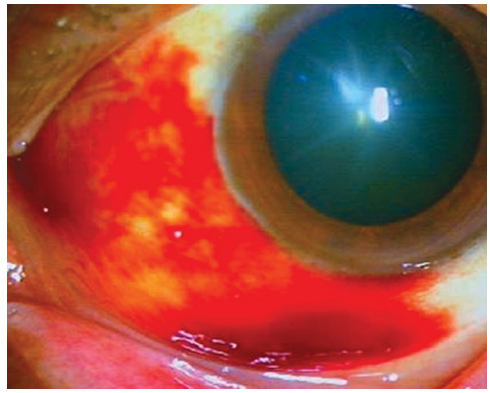
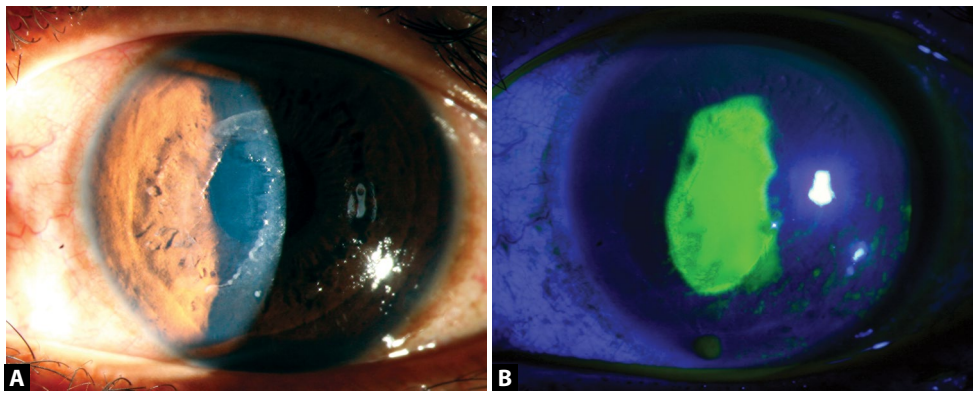


Fig. 24.3: Subconjunctival hemorrhage



Figs 24.4A and B: Corneal abrasion and with fluorescein staining

Conjunctiva

- Conjunctival lacerations and chemosis.
- Subconjunctival hemorrhage (Fig. 24.3).

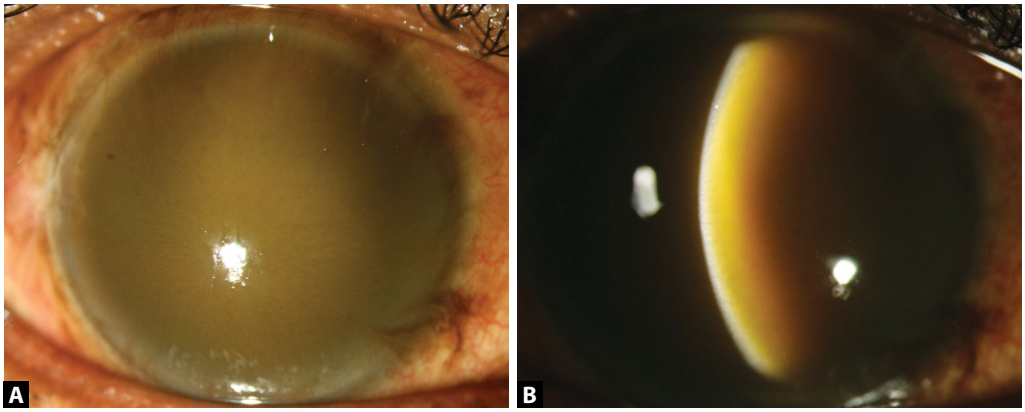
Cornea

- Simple abrasions (Figs 24.4A and B).
- Recurrent traumatic keratalgia (erosions).
- Rupture of the Descemet's membrane.
- Corneal stromal edema.
- ***Blood-staining of the cornea (Figs 24.5A and B):*** Usually it occurs after a traumatic hyphema with raised intraocular pressure and endothelial damage. The whole cornea is stained (reddish-brown or greenish). Cornea gradually and very slowly clears up from the periphery towards the center, for a period of 2 years or more. Usually, some amount of permanent visual impairment results. Penetrating keratoplasty may be necessary in future.
- ***Corneal rupture*** may also occur.

Sclera

Scleral rupture (rupture of the globe) (Fig. 24.6): The force usually comes from the direction of down and out, as there the globe is least protected by the orbital rim. The sclera gives way at its ***weakest part***, near the canal of Schlemm and the wound runs concentrically with the limbus. The conjunctiva is often intact. There may be associated uveal

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Figs 24.5A and B: Blood staining of the cornea (slit-section on right)

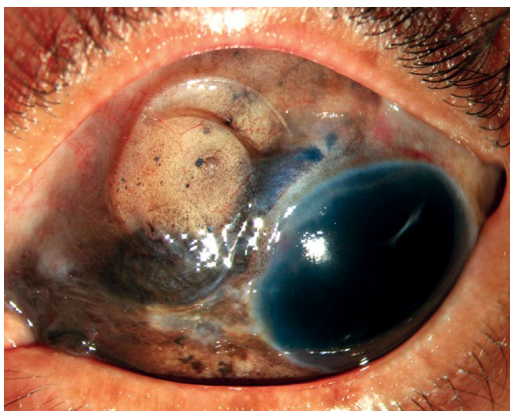


Fig. 24.6: Scleral rupture with subconjunctival dislocation of intraocular lens

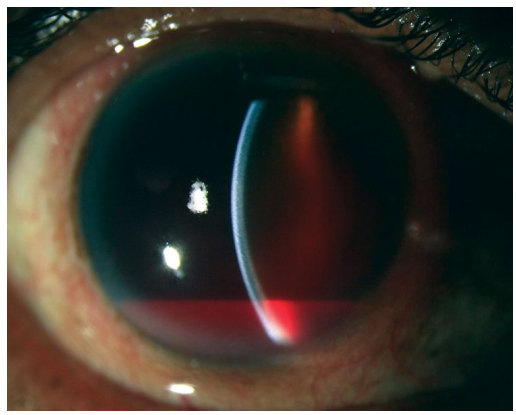


Fig. 24.7: Blood in anterior chamber—traumatic hyphema partially settled

prolapse, subconjunctival dislocation of lens, vitreous prolapse and intraocular bleeding. Prognosis is often very poor as the eye usually shrinks with total loss of vision.

Anterior Chamber

Hyphema (Blood in the Anterior Chamber) (Fig. 24.7)

Sources of Bleeding

- Small branches of the major arterial circle due to tear between longitudinal and circular fibers of the ciliary muscle.
- Capillaries of the minor arterial circle, when there is sphincteric tear.
- Radial vessels at the iris root, associated with iridodialysis.

Other Causes of Hyphema

- Intraoperative and postoperative
- Herpetic iridocyclitis
- Rubeosis iridis (as in diabetes)
- Blood dyscrasias
- Intraocular malignancies
- Idiopathic
- Juvenile xanthogranuloma (recurrent spontaneous hyphema in a young child).

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Clinical Features

- The blood usually does not get clotted, and settles at the most dependent portion of the anterior chamber, and a fluid meniscus is formed.
- Frequently, original minor hyphema is followed by a more severe bleeding within 24–48 hours of injury.
- Sometimes, the blood may be clotted, specially in presence of sphincteric tear or iridodialysis.
- ***'8' ball (or blackball) hyphema:*** When the blood gets clotted, the hyphema appears as small blackball (like no. '8' ball in billiards game). This clotted blood causes secondary glaucoma more frequently (Fig. 24.8).

Complications

- Secondary glaucoma—due to blockage of angle by red blood cells, or breakdown products of the blood.
- Secondary optic atrophy due to chronic glaucoma.
- Blood-staining of the cornea.

Treatment

- Complete bed rest with propped up position.
- Sedation
- Patching of the eyes (binocular is better).
- Local corticosteroids to minimize traumatic uveitis.

  Mild hyphema without secondary glaucoma, is usually responds with above therapy. Aspirin should not be used, as it increases the risk of secondary hemorrhage.
- Secondary glaucoma is treated with:
  - Tab acetazolamide (250 mg) = ½ to 1 tab—4 times daily.
  - Timolol (0.5%) eye drops = 2 times daily.
- ***Indications of paracentesis:***
  - Blood does not get absorbed quickly by 5–7 days.
  - Persistent high intraocular pressure (IOP) for 3–7 days depending upon the height of intraocular pressure.
  - Early signs of blood-staining of cornea.
- Limbal incision and removal of the clots may be urgently required in blackball hyphema.

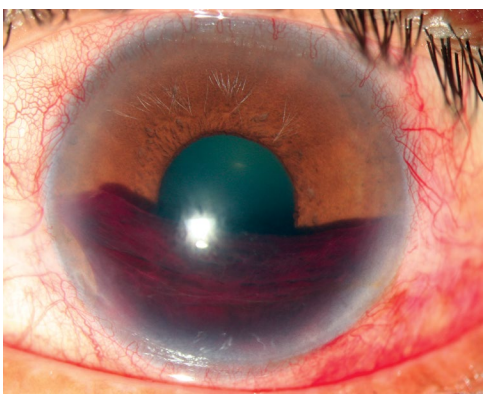


Fig. 24.8: Blunt trauma—'8' ball hyphema

Iris

- ***Iridodialysis:*** Iris is partially torn away from its ciliary attachment. A black biconvex area is seen at the periphery, and pupillary margin bulges slightly inward, causing a D-shaped pupil (Figs 24.9 and 24.10). A fundal glow is obtained through the peripheral gap. There may be uniocular diplopia.
- ***Antiflexion of the iris:*** In extensive iridodialysis, the detached portion of the iris may be rotated completely, so that pig-

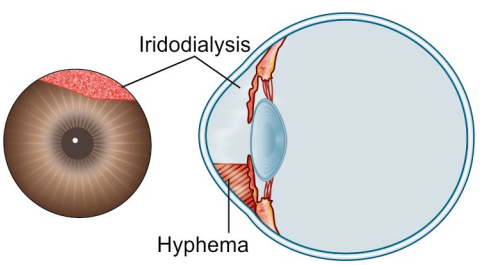
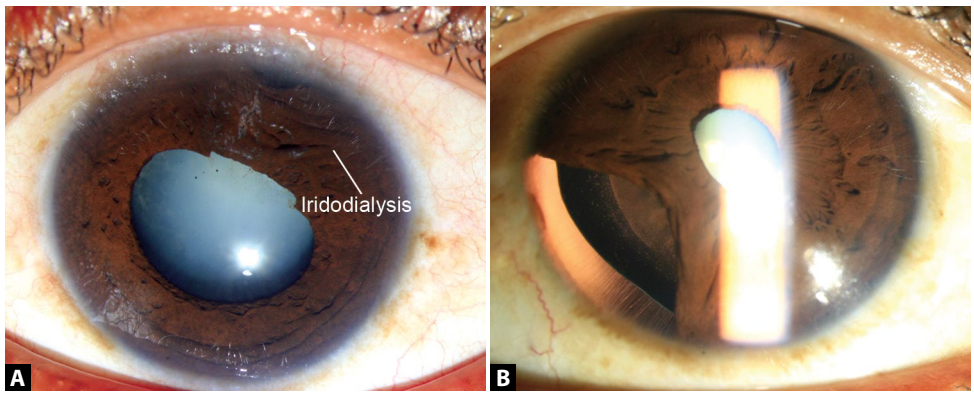


Fig. 24.9: Iridodialysis and hyphema. Note the D-shaped pupil in iridodialysis

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Figs 24.10A and B: A. Traumatic iridodialysis—D-shaped pupil; **B.** Iridodialysis—note the gap at the iris root

mented back surface of the iris faces forward. Torn peripheral edge of the iris may be sutured just behind the limbus.

- ***Traumatic aniridia (irideremia):*** The iris is completely torn away (360°) from its ciliary attachment, contracts into a small ball, and sinks at the bottom of the anterior chamber.
- ***Retroflexon (total inversion) of the iris:*** The whole iris may be doubled back into the ciliary region. When partial, it may appear as coloboma of the iris.
- ***Iridoschisis:*** Partial thickness tearing of the iris.
- ***Traumatic iridocyclitis:*** Usually mild.

Pupil

- Traumatic miosis
- Traumatic mydriasis
- D-shaped pupil (due to iridodialysis)
- Irregular pupil (due to sphincteric tear).

Ciliary Body

- Spasm of accommodation
- ***Angle recession (Fig. 24.11):*** There is longitudinal tear in the face of the ciliary body which splits the circular fibers from the longitudinal fibers of the ciliary muscle. There is deepening of the anterior chamber, widening of the ciliary band on gonioscopy (Fig. 24.12), and tendency to develop glaucoma in future.

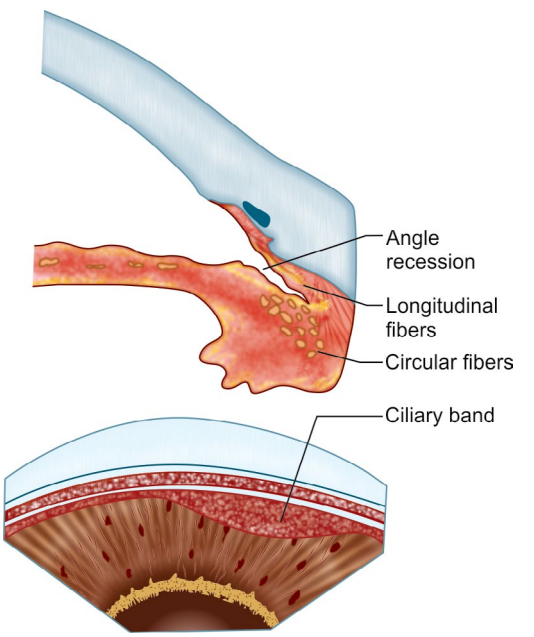


Fig. 24.11: Angle recession with irregular widening of the ciliary band

Crystalline Lens

- ***Vossius's ring:*** A circular pigmented imprint of the miosed pupil on the anterior surface of the lens.

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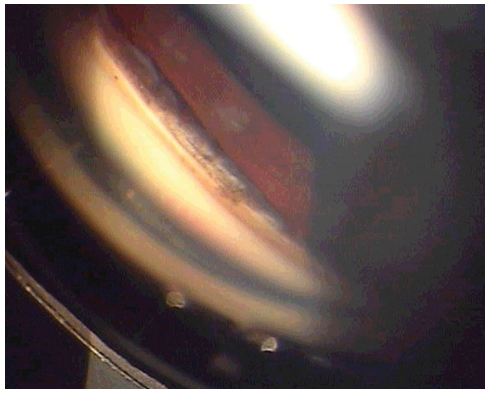


Fig. 24.12: Gonioscopic view of angle recession

- ***Concussion cataract:*** This is largely due to imbibition of aqueous through the damaged capsule, and partly due to direct mechanical effect on the lens fibers. The capsular tear commonly occurs at the posterior pole of the lens.
- ***Types of concussion cataract***
  - Minute discrete subcapsular opacities.
  - Localized cataract, often behind the iris, which may remain stationary.
  - Total cataract (in large capsular tear).
  - ***Typical rosette-shaped cataract (Figs 24.13 to 24.15):*** It is a star-shaped cataract, usually in the posterior cortex. Accumulation of fluid marks out the architectural pattern of the lens. Star-shaped cortical sutures are delineated, and radiating feathery line of opacities outlines the lens fibers. Sometimes, it remains stationary, and sometimes, it may progress into a total cataract.
  - ***Late rosette-shaped cataract:*** It is usually develops in the posterior cortex, 1–2 years after a concussion. It is smaller and more compact than early type, and its sutural extension are short.

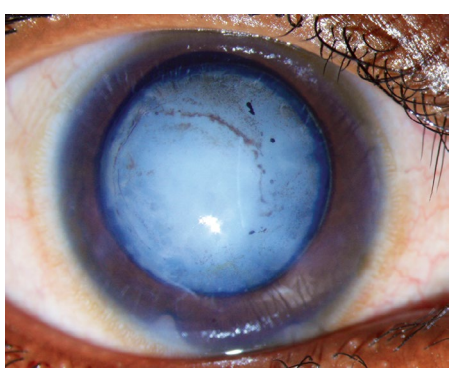
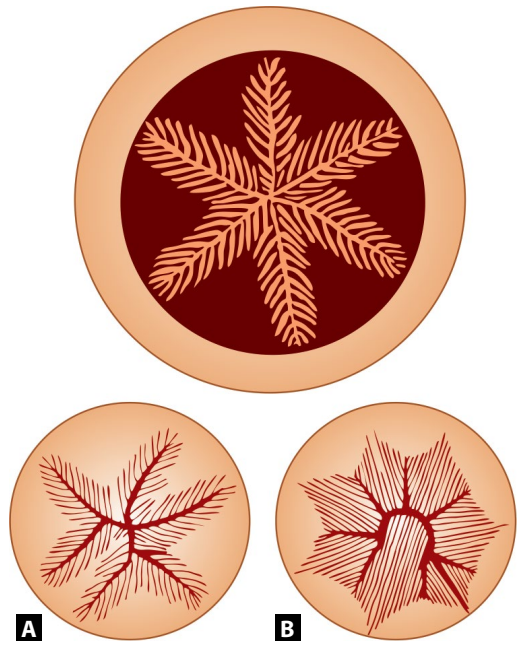


Fig. 24.13: Traumatic cataract after a blunt trauma



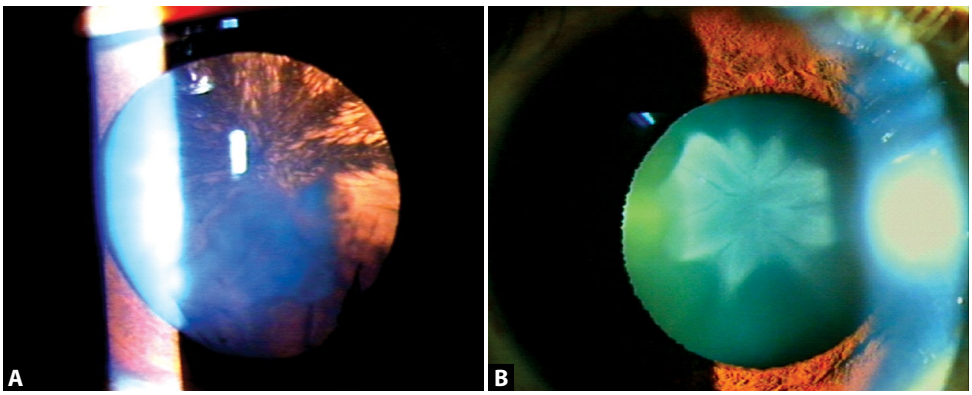
Figs 24.14: Rosette-shaped cataract. **A.** In ***early rosette***, leaves of the feathery opacities are formed by the suture acting as a backbone from which the opacities radiate outwards; **B.** In ***late rosette***, feathery opacities stream outward from which lie in the angle between neighboring leaves

- ***Subluxation of the lens***
- ***Dislocation of the lens:*** In the anterior chamber or in the vitreous cavity.

Vitreous

- Disorganization and liquefaction of the vitreous.
- Anterior and/or posterior vitreous detachment.

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Figs 24.15A and B: Rosette cataract. **A.** Early; **B.** Late

- Fine pigmentary opacities.
- ***Vitreous hemorrhage*** with its sequelae.

Choroid

- ***Choroidal rupture (Fig. 24.16):*** Immediately after the injury, choroidal rupture is not visible due to extravasation of blood. After the blood gets absorbed, the rupture is seen as two curved white lines, concentric with the disc margin and on its temporal side. These lines become rapidly pigmented along its edge.
  Simple rupture without macular involvement, causes little visual impairment.

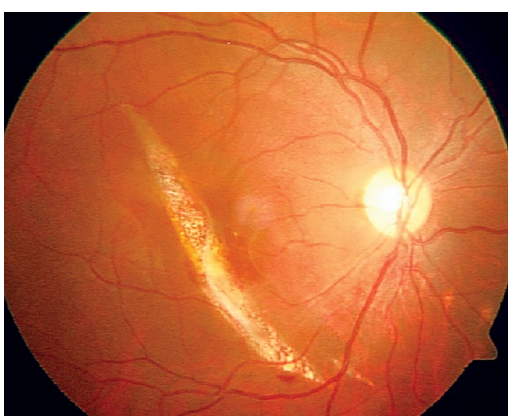


Fig. 24.16: Choroidal rupture

  Treatment is by bed rest (with upright head) and atropine ointment.
- ***Choroidal hemorrhage:*** It appears as a red dark patch underneath the retinal vessels.

Retina

- ***Commotio retinae (Berlin's edema):*** It is the milky-white cloudiness at the macular area due to edema. Since the fovea is very thin the edema is not visible, it appears more reddish (cherry-red) in the white surroundings.
- Central vision may be permanently impaired due to pigmentary deposits at the macula.
- ***Traumatic macular degeneration*** with the formation of a ***macular cyst*** and subsequently, a ***macular hole***.
- ***Retinal hemorrhage***, due to rupture of the retinal vessels.
- ***Retinal tears:*** Particularly at the retinal periphery, if the eye is already myopic or having peripheral degeneration.
- ***Retinal detachment*** following a tear in the retina.
- ***Traumatic proliferative vitreoretinopathy (Fig. 24.17)*** is usually secondary to vitreous hemorrhage, leading to fibrovascular tractional bands.

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Fig. 24.17: Traumatic proliferative vitreoretinopathy

Optic Nerve

- Optic nerve-sheath hemorrhage
- ***Avulsion of the optic nerve*** leading to optic atrophy, which may be partial or total.

Intraocular Tension

- ***Hypotony*** due to vasomotor reaction.
- ***Secondary glaucoma due to***
  - Vasomotor reaction
  - Trabecular edema
  - Hyphema
  - Subluxation or dislocation of the lens
  - Ghost cell glaucoma
  - Angle recession glaucoma.

Lacrimal Apparatus

- Traumatic subluxation of lacrimal gland.
- Injury to the lacrimal canaliculus.

Orbit

- Proptosis and displacement of the globe.
- Orbital (retrobulbar) hemorrhage.
- Orbital emphysema following orbital bone fracture involving paranasal sinuses (PNS).
- ***Fracture of the orbital bones:*** It most commonly affects the margin of the orbit. Fractures near the orbital margin are easy to diagnose from unevenness of the margin, and tenderness on pressure.

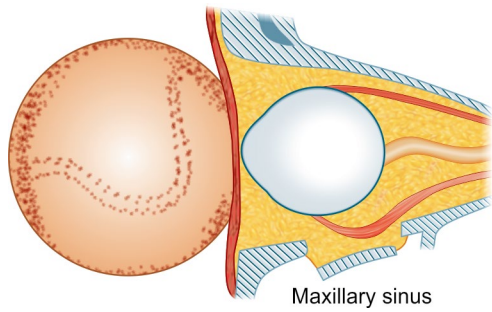


Fig. 24.18: Blow-out fracture of the orbital floor

Fracture of the superior rim of the orbit may dislocate the trochlea of superior oblique tendon, causing paresis of superior oblique muscle with diplopia.

- ***Blow-out fracture:*** Pure blow-out fracture is typically caused by a sudden rise in intraorbital pressure by a striking object which is greater than 5 cm in diameter, such as a tennis-ball or a fist. The fracture usually involves the floor of the orbit, along the thin bone covering the infraorbital canal (Figs 24.18 and 24.19).

Clinical Features

- ***Diplopia***—typically occurs in both upgaze and downgaze ***(double diplopia), due to entrapment of the inferior rectus and inferior oblique muscles***.
- ***Periocular ecchymosis and edema***.
- ***Enophthalmos with pseudoptosis*** usually appears after 10–14 days.
- ***Infraorbital nerve anesthesia*** involving the lower eyelid, side of the nose, cheek, upper lip and teeth.
- ***Nasal bleeding*** from maxillary sinus.
- ***Subconjunctival hemorrhage is common, but other ocular damage is very rare***, due to natural protection by the type of injury.

Investigations

- ***Plain X-ray (Water's view) of orbit:*** The classic finding is the presence of a polypoid mass ***(the tear-drop sign)*** protruding

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Fig. 24.19: Blow-out fracture on the left side

from the floor of the orbit into the maxillary antrum. The tear-drop represents the herniated orbital contents, periorbital fat and inferior rectus muscle.

- ***CT scan*** demonstrates the soft tissue involvement in more details.
- ***Forced duction test (FDT)*** is positive if the muscle is entrapped.

Treatment

- Small-cracks or minor fracture without herniation of the orbital content does not require any treatment.
- A blow-out fracture needs to be repaired only if:
  - There is permanent diplopia with restriction of ocular movement, and
  - There is significant enophthalmos.
- Larger fracture with herniation, should be repaired ***within 2 weeks***, to prevent secondary contracture of the muscles.
- The fracture opening is bridged with bone graft or silicone rubber sheet.

Blow-in fracture of the orbital floor is just opposite, and may be rarely caused by a sudden rise in pressure in the maxillary antrum as a result of trauma to the face with subsequent elevation of bone-fragments into the orbit.

PENETRATING (PERFORATING) INJURY

They are caused by sharp objects or foreign bodies. Perforating injuries are potentially serious, and the patient should be urgently admitted and treated promptly.

Penetrating injuries by definition penetrate into the eye but not through and through; there is no exit wound. ***Perforating injuries*** have both entry and exit wounds (a through and through injury).

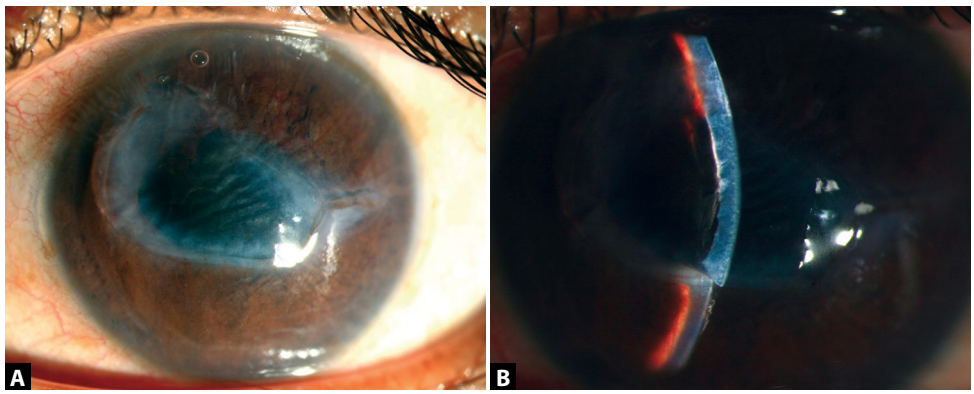
The seriousness arises from—the immediate effects of trauma, the introduction of infection, sympathetic ophthalmitis.

The Immediate Effects of Trauma

Two important signs of perforation are—wound of entry and a low intraocular tension.

- ***Wounds of the lid and conjunctiva:*** They are common, and should be stitched meticulously in three layers.
  A lid-notching is common following repair of the lids.
- ***Wounds of the cornea:*** These may be linear or lacerated. The margins soon swell-up after the injury, and become cloudy, due to imbibition of fluid. Adhesion of the iris or its prolapse is almost certain (Figs 24.20A and B).
  Treatment
  - Antibiotics and atropine drop are applied immediately, and then a pad and bandage.
  - ***The prolapsed iris must be abscised (Fig. 24.21)***.
  - The wound is repaired with 10-0 nylon under operative microscope (preferably, under general anesthesia).
- ***Wounds of the sclera:*** It is recognized by the uveal prolapse. In deep injury, vitreous may come out through the wound.
- ***Wounds of the lens***
  - If the wound is very small (e.g. with a needle), there is localized lens opacity, or after sometimes, there may be formation of rosette-shaped cataract.

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Figs 24.20A and B: Large corneal rupture without iris prolapse

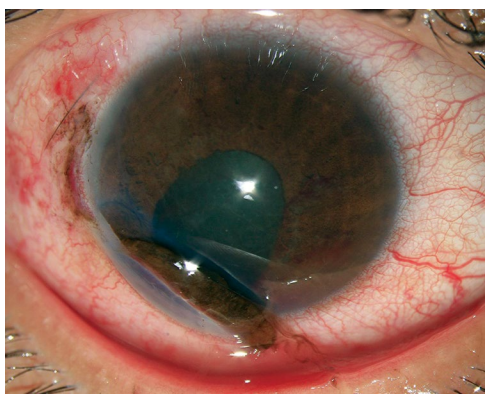


Fig. 24.21: Corneal rupture with iris prolapse

- In case of large capsular wound—the flocculent white cortical matters protrude through the capsular opening, and sometimes, the whole anterior chamber is full of white flocculi (Fig. 24.23).
- ***Treatment***
  - ***For mild injury*** of the lens, cataract is better managed as a secondary procedure.
  - ***For severe injury***—the aims of the surgery are to remove the cataract with or without posterior chamber intraocular lens implantation, to perform an anterior vitrectomy if necessary, and repair of the wound of entry at the earliest.

Signs of globe perforation
Any one or combination of the following suggests possible perforation of the globe.
- Decreased visual acuity
- Hypotony [marked decrease in intraocular pressure (IOP)]
- Shallow or flat anterior chamber
- Alteration of pupillary size, shape and location
- Focal iris tear or hole
- Injury tract in the cornea, lens or vitreous
- Marked conjunctival chemosis
- Wound leak (a positive Seidel's test) (Figs 24.22A and B).

Introduction of Infection

The organisms are introduced into the eye, by a dirty sharp object. The pyogenic organisms may lead to:
- Purulent keratitis
- Purulent iridocyclitis with hypopyon
- Endophthalmitis (Fig. 24.24) and panophthalmitis.

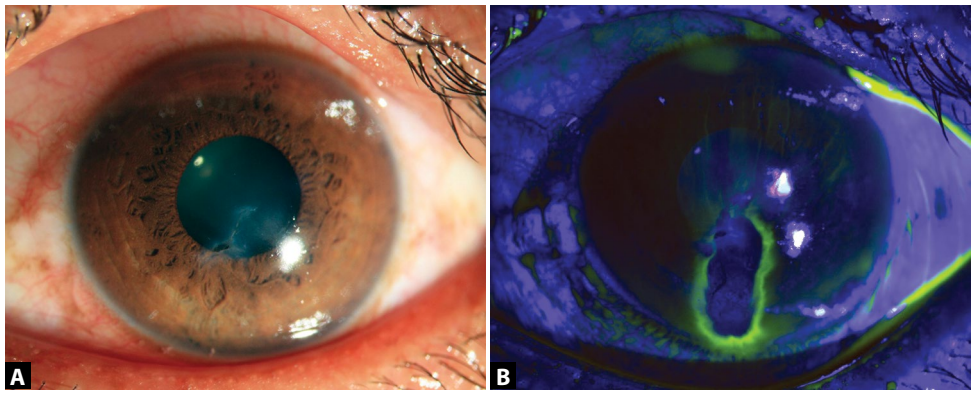
Sympathetic Ophthalmitis

One of the most dreadful complications of penetrating injury, has already discussed (*see* Chapter 14).

FOREIGN BODIES

- ***Extraocular foreign bodies (EOFB)***—corneal or conjunctival.

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Figs 24.22A and B: Small corneal perforation and Seidel's test positive

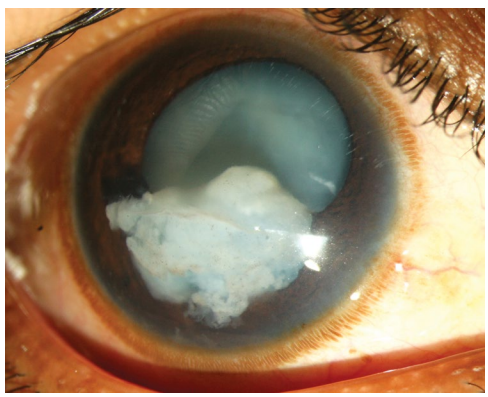


Fig. 24.23: Penetrating injury with lens capsular tear and lens matter in anterior chamber

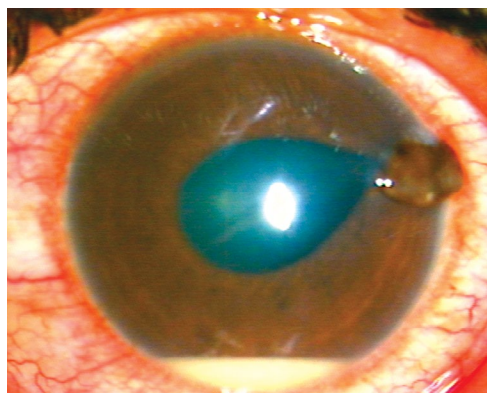


Fig. 24.24: Perforating injury—endophthalmitis

- ***Intraocular foreign bodies (IOFB)***—with a perforating injury of the eyeball.

Extraocular Foreign Bodies

Small foreign bodies, e.g. coal, dust, sand, iron particles, eyelash, wood-piece, husks of seeds, wings of insect, etc. may pitch upon the conjunctiva or the cornea.

A history of injury, and the probable character of the foreign body help in its detection and removal.

The symptoms vary from mild or no discomfort to severe pain, and watering. There may be associated photophobia and redness.

Corneal

- Patient cannot localize a foreign body on the cornea, as there is no kinesthetic sensation in the cornea. Instead, it is very often referred to the tarsal conjunctiva of the upper lid against which the foreign body rubs.
- The particle is usually embedded in the epithelium or in the anterior stroma.
- Foreign body is best localized by careful inspection of the cornea, aided by magnification with a loupe or slit-lamp.
- A fluorescein stain is also very helpful.
- Ciliary congestion may be present.

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- When the removal is delayed, there may be surrounding infiltration, or even frank ulceration of the cornea.
- In all cases of corneal foreign bodies, ***two clinical procedures*** are important:
  i. ***Eversion of the upper lid*** to check the subtarsal sulcus, and
  ii. ***Pressure over the sac area*** to exclude the presence of chronic dacryocystitis.

Conjunctival

- Most frequently, it becomes lodged at the middle of the ***upper subtarsal sulcus***.
- It may be in the upper fornix or embedded in the bulbar conjunctiva.
- Upper eyelid must be everted (sometimes double eversion is necessary), otherwise the foreign body may be overlooked.
- The foreign body in the subtarsal sulcus (Fig. 24.25) rubs against the cornea, and causes linear (vertical) corneal abrasions.

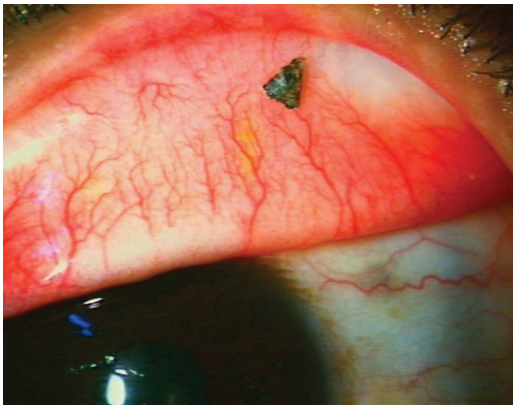


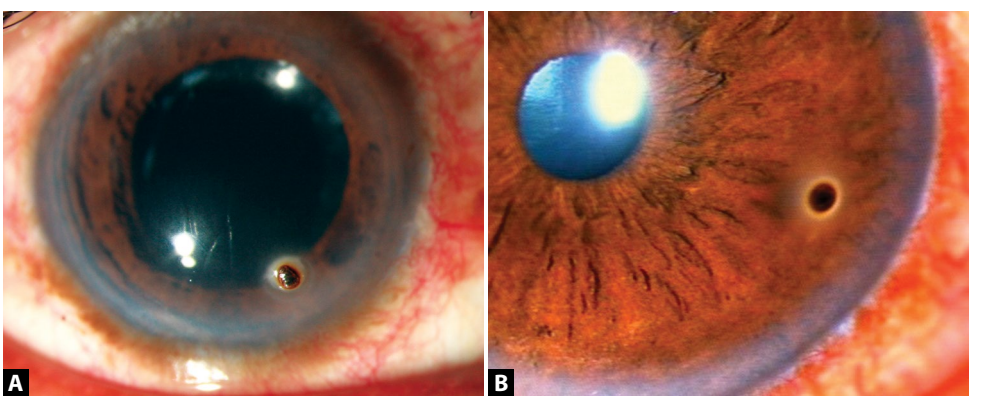
Fig. 24.25: Foreign body (subtarsal sulcus)

Removal of Extraocular

All foreign bodies should be removed as early as possible. Topical anesthesia (4% lignocaine) must be used to prevent pain and eyelid closure during removal.

- ***Corneal: Steps of removal***
  - First, an attempt should be made to remove the foreign body by means of irrigation with sterile saline solution by a syringe.
  - If it is not possible to remove by irrigation, it can be removed by a sterile cotton-tipped swab, or by a triangular piece of sterile blotting paper.
  - If not possible, the foreign body is lifted gently out of the corneal substance by means of a sterile (disposable) hypodermic 23 or 24 G needle. ***Do not scratch the cornea, and do not cross the pupillary zone of the cornea***.

    It is always wise to use some magnification during removal of corneal foreign body (Fig. 24.26A).



Figs 24.26A and B: **A.** Foreign body cornea; **B.** Foreign body cornea (rust-ring)

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- If a ferrous metal is embedded for several days, a rust-ring (Fig. 24.26B) may remain after removal of the main portion of foreign body. That is to be removed in the same manner as the foreign body.
- If the foreign body is impacted into the deeper stroma, it is to be removed under operative microscope with a sharp needle.
- After removal, antibiotic ointment and if necessary, cycloplegic (in presence of ciliary congestion, or for large foreign body) are applied. Pad and bandage is given for 24 hours.
- Since, a foreign body may introduce microorganisms into the cornea—the eye should be examined daily until the area no longer stains with fluorescein.

- ***Conjunctival***
  - Foreign bodies of the conjunctiva can easily be removed with ***irrigation, a sterile cotton-swab, or with needle or a spud***.
  - The ophthalmologist must be prepared to remove the foreign body while he everts the upper lid, because the foreign body may be dislodged and difficult to locate again, if the eyelid is released.
- ***Prophylactic measures:*** Foreign bodies in the eye are extremely common among industrial workers. Such accidents could be almost entirely prevented by the use of protective goggles.

Intraocular Foreign Bodies

The common foreign bodies which penetrate the eyes and retain, are minute chips of iron or steel (most common), stones, glass, lead-pellets, copper, spicules of wood, etc.

While chipping the stone, with an iron-chisel and a hammer, it is a chip of the chisel (from cutting edge, or from its mushroomed head) which enters the eyes, but not the chip of the stone.

The entry of the foreign body into the eye may cause ocular damage in three ways:

1. By mechanical effects.
2. By introduction of infection.
3. By specific reaction, caused by the particular type of foreign body.

Mechanical Effects

The mechanical effects largely depend upon the size and velocity of the foreign body.

They are usually small and travel at a high velocity. The effects are:

- After penetrating the cornea or sclera, it may:
  - Retain in the ***anterior chamber (Fig. 24.27)***.
  - ***Pass into, or through the lens***, either by way of the iris or pupil, and causing a ***traumatic cataract***. Thus, a hole in the iris is of great diagnostic value (Fig. 24.28).
  - Retain in the ***vitreous***, causing its liquefaction.
  - Rest onto the ***retina***; and if the media is clear, the foreign body may be seen ophthalmoscopically in the vitreous, or on the retina.
  - Rarely, pierce the coat of the eyeball and comes to rest within the orbital tissue ***(double perforation)***.

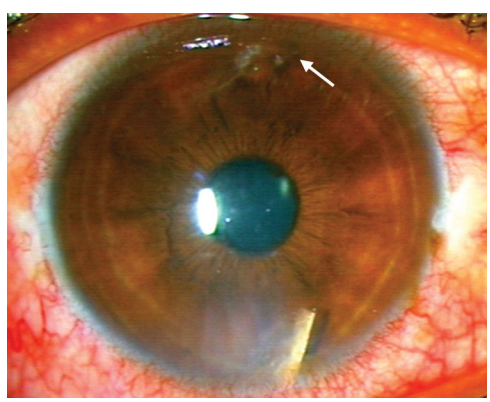


Fig. 24.27: Retained intraocular foreign bodies—angle of the anterior chamber. Arrow shows wound of entry

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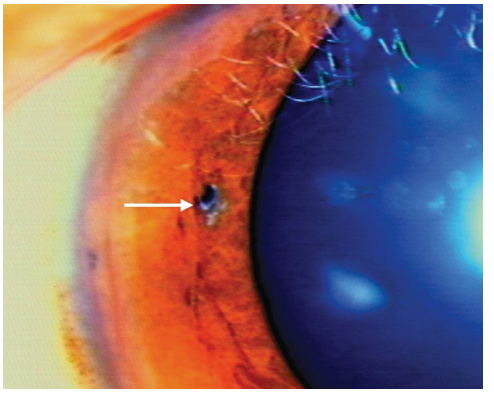


Fig. 24.28: Retained intraocular foreign bodies—iris hole (arrow)

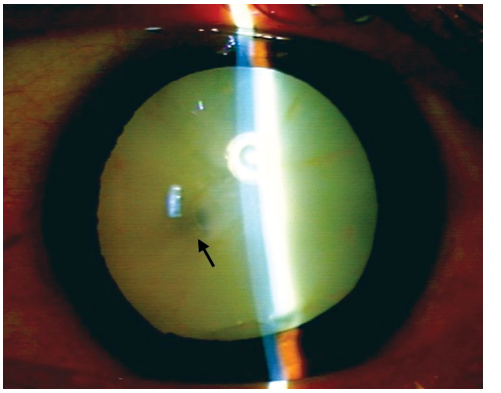


Fig. 24.29: Retained intraocular foreign bodies within the crystalline lens with cataract and siderosis (arrow)

- The lodgement of the foreign body in the posterior segment often leads to degenerative changes (apart from the changes which occur due to chemical reaction).
- The vitreous usually turns fluid, fibrous bands from along the path of foreign body, hemorrhage may be extensive, and subsequently, retinal detachment may occur.
- A particle greater than 2 mm in size usually leads to destruction of the eye.

Infection

- Small flying metallic objects are usually sterile, owing to the heat generated partly on their emission, and partly by their high velocity.
- But pieces of wood or stone, usually cause a notorious intraocular infection, leading to severe endophthalmitis or panophthalmitis.
- Despite treatment, the visual prognosis is seldom good.

Specific Reaction by a Foreign Body

It varies with the chemical nature of the foreign body, which may be ***non-organized*** **or** ***organized material***.

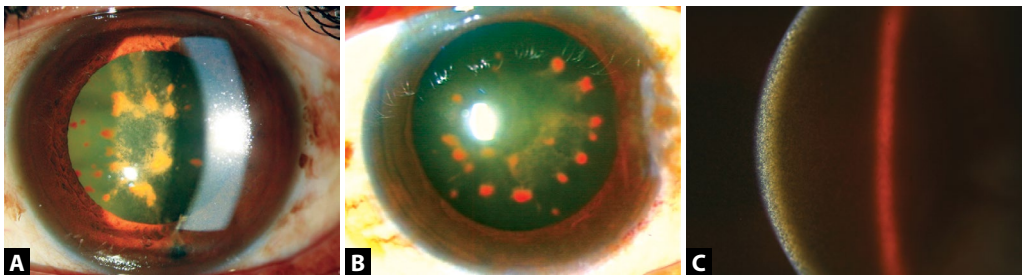
Non-organized materials cause:

- ***Minimal or no reaction***—by inert materials e.g. glass, plastics, porcelain, gold, silver, platinum or tantalum.
- ***A local irritation and often with encapsulation***, e.g. lead, aluminium.
- ***A suppurative reaction***, e.g. zinc, mercury, nickel or pure copper.
- ***Specific degenerative changes***, e.g. iron and copper undergo chemical dissociation, and are widely deposited throughout the ocular tissues, causing ***siderosis*** and ***chalcosis***, respectively (Fig. 24.29).

Siderosis Bulbi (Figs 24.30A to C)

- It is the chronic ***irreversible*** degenerative changes of the ocular tissues, caused by retained intraocular iron (and also steel, in proportion of its ferrous content) foreign body.
- Epithelial structures are preferentially affected.
- ***Characteristic signs***
  - ***Rusty deposition on the anterior lens surface*** (earliest clinical sign): Oval patches of rusty deposit are arranged radially in a ring, corresponding to the edge of the dilated pupil. This is due to deposition of iron in the anterior cubical

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Figs 24.30A to C: Siderosis bulbi **A.** Rusty deposits on the lens surface; **B.** Rusty deposits on anterior lens capsule with cataract; **C.** Rusty discoloration of the cornea

cells of the lens, and ***pathognomonic (Fig. 24.30A)***.

- ***Heterochromia of the iris:*** The iris is first greenish, and later reddish-brown.
- ***Retinal pigmentary degeneration:*** It is associated with great attenuation of the blood vessels.
- ***Rusty discoloration of the cornea*** may also occur (Fig. 24.30C).
- Secondary ***open angle glaucoma*** is a common late complication.
- Ultimately, most of these e yes go blind.

- ***Electroretinography response*** of the eye is ***reduced*** even before the appearance of clinical signs.
- ***Histochemically***, the deposition of iron is revealed by the Prussian blue reaction with Pearl's microchemical stain.

Chalcosis Bulbi

- It is the tissue reaction caused by intraocular retention of copper foreign body (in the form of an alloy). ***Unlike siderosis, it is reversible, if the particle is removed***.
- With ***pure copper***, a violent suppurative reaction ensues, which eventually results in shrinkage (phthisis) of the globe.
- ***Chalcosis, a milder reaction,*** occurs when the metal is heavily alloyed, e.g. brass (as from percussion caps), or bronze.
- ***Clinical features***
  - ***Kayser-Fleischer's (K-F) ring:*** A golden-brown ring at the level of Descemet's membrane of the cornea.

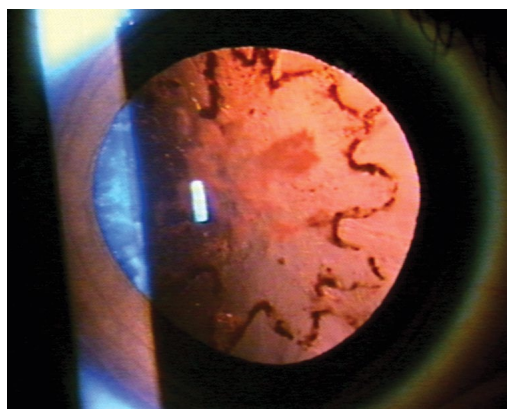


Fig. 24.31: Chalcosis bulbi—sunflower cataract

  - ***Sunflower cataract:*** A brilliant golden-green sheen in the form of petals of sunflower (with good prognosis) (Fig. 24.31).
  - ***Retinal changes:*** Lustrous golden plaques which reflect the light at posterior pole.
- As the degenerative changes do not appear, vision may remain good indefinitely.

Organized materials

- ***Wood and vegetable materials***—produce a proliferative reaction with giant cell formation.
- ***Eyelashes***—cause proliferation of the hair root epithelium leading to the formation of intraocular cysts (Fig. 24.32).
- ***Caterpillar hairs***—excite a severe iridocyclitis with granulomatous nodules ***(ophthalmia nodosa)***.

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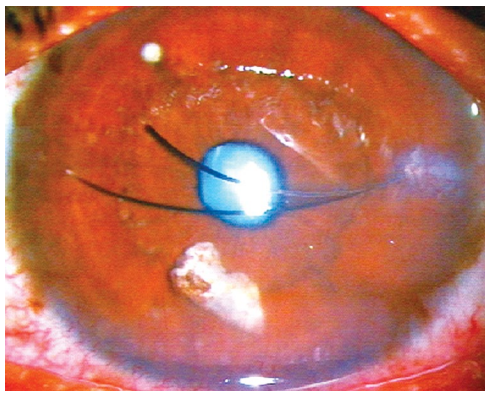


Fig. 24.32: Penetrating injury with intraocular foreign body with eyelashes

Diagnosis and Localization of Intraocular Foreign Body

- Careful history regarding the nature of injury.
- ***Slit-lamp microscopy and gonioscopy***. A hole in the iris, or an opaque-track through the lens is pathognomonic.
- If the media is clear, the entire fundus is searched under full mydriasis, with the help of direct and indirect ophthalmoscope, and if necessary, with a Goldmann's three-mirror lens.
- ***Plain X-rays*** of the orbit (anterior posterior and lateral) for radio-opaque materials.
- ***Limbal-ring X-rays*** of the orbit, for localization of the foreign body.
- ***Ultrasonography B-scan and CT-scan*** are important for radiolucent foreign bodies.
- Metal locator ***(Berman or Roper-Hall)*** to detect metallic foreign body, and to differentiate between magnetic from a non-magnetic foreign body.

Removal of an Intraocular Foreign Body

- An intraocular foreign body should be removed unless:
  - It is inert and probably sterile.
  - A little damage has been done to vision, and
  - The process of removal will almost inevitably destroy vision.

  Minute foreign bodies on the retina are typical examples to fulfil above criteria.
- In other cases, early removal is essential, because the foreign body may become enmeshed in fibrin, and depending on the site of location of the foreign body, its removal will vary.

In the anterior chamber or iris (Fig. 24.33):

- ***If it is magnetic***—removal by a point magnet or by forceps.
- ***If non-magnetic***—removal by forceps.
- ***Iridectomy*** is necessary, if it is embedded in the iris tissue.

In the lens: Removal along with extraction of the lens with or without an IOL implantation.

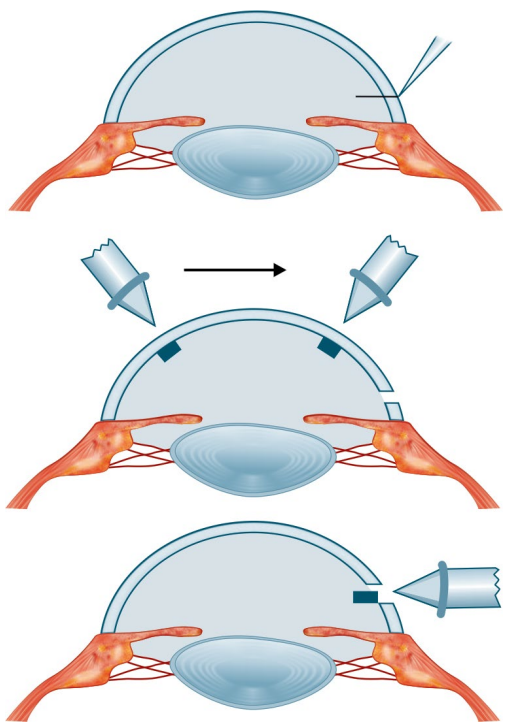


Fig. 24.33: Removal of a magnetic foreign body from the anterior chamber with hand magnet

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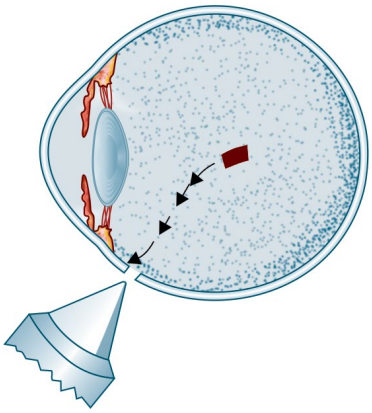


Fig. 24.34: Removal of a magnetic foreign body from the vitreous cavity via pars plana route using a giant magnet

In the vitreous or retina:

- ***For a magnetic foreign body***—the particle is removed directly through the posterior route by an electromagnet, and it is by the shortest possible route.
- ***For a non-magnetic foreign body***—via pars plana route (Fig. 24.34), by vitrectomy, and with the help of special forceps and other instruments.
- Now, because of advancement in vitreo-retinal surgery, any IOFB (either magnetic or nonmagnetic) is removed by forceps via pars plana route after doing good vitrectomy. Additional surgery of retina is performed in the same sitting if required.

CHEMICAL INJURIES (BURNS)

In most cases, chemical injuries (e.g. with household detergents, and cleaning agents) of the eye are relatively minor, and are easily treated. But occasionally, ***alkaline and acidic substances may cause severe ocular damage and permanent loss of vision***.

Alkali burns are much more dangerous than acid burns because:

- Alkalies produce a high pH (by hydroxide ion) in the tissue-water, which saponifies the fatty component of the cell membrane, leading to cell disruption.
- Alkali's cations bind to the proteins, to form gel-like soluble alkaline-protinates (unlike acids which cause instant coagulation of proteins).
- Alkali makes the collagen more susceptible to enzymatic degradation.

All these changes lead to increased penetration of alkalies deep into the tissue. Along with it, there are accelerated ptoteolytic effects (by collagenase enzyme) and defective collagen synthesis, make the damage more serious.

Whereas in acid burn, there is instant coagulation and precipitation of proteins, which are insoluble. These ***acid-protein complexes have a buffering effect***, on the further action of the acid, and so there is no penetration of the acid in the deeper tissues. Thus, most acid burns are limited and localized. But, accelerated proteolytic enzymatic activity and defective collagen synthesis may also occur in severe acid burns.

Common alkalies responsible, are liquid ammonia, caustic soda, caustic potash, lime etc., among which ammonium hydroxide is the most dangerous, and lime is the least.

Common acids are sulphuric acid (used in battery), hydrochloric acid, nitric acid, muratic or acetic acid, among which sulphuric acid is the most dangerous.

All alkalies and acids produce their effects not only by their chemical effects, but also by release of heat during the process of chemical reactions within the tissue.

Clinical Features

Severity of the injury depends on the type of chemical and its quantity, pH of the solution, duration of exposure, and the time of presentation following chemical burns. As already

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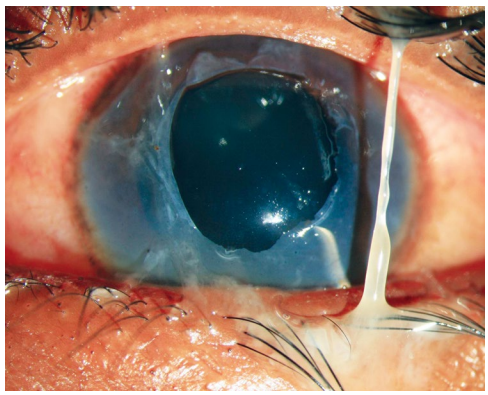


Fig. 24.35: Acid burn in acute phase

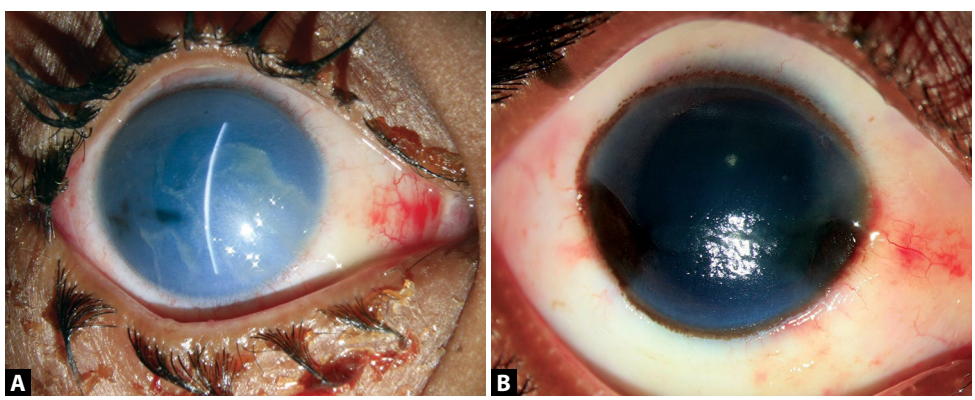
discussed, alkali burns cause more serious damage than acid burns. The effects are:

- ***Acute phase (upto 1 week) (Figs 24.35 and 36A)***
  - Conjunctival chemosis, congestion and discharge
  - Perilimbal ischemia (Fig. 24.36B)
  - Corneal epithelial defects and stromal clouding
  - Increased intraocular pressure.
- ***Early reparative phase (1–3 weeks)***
  - Conjunctival and corneal epithelia begin to regenerate
  - Corneal opacity of various degree with vascularization
  - Iridocyclitis.
- ***Late reparative phase (3 weeks to several months)***
  - Irregular scarring of the cornea.
  - Descemetocele formation or even perforation.
  - Dry eye, due to scarring of the ducts of lacrimal glands and goblet cells.
  - Posterior synechiae and cyclitic membrane formation.
  - Cataractous changes of the lens.
  - Hypotony due to atrophy of the ciliary body, followed by phthisis bulbi.
  - Development of entropion, trichiasis and symblepharon.

Acid burns produce less severe injury than alkali burns. Deeper intraocular tissue damage usually does not occur, except in burns with ***sulphuric acid*** which penetrates well inside the eye, and causes as much damage as alkali.

Treatment

- ***First and immediate treatment***
  - ***A thorough irrigation*** of the ocular surface with plain water, normal saline or distilled water, whichever is available.
  - ***No time must be spent in searching for neutralizing substances*** (e.g. 3% sodium bicarbonate solution for acids, or boric acid for alkalies), as their efficacy is doubtful.



Figs 24.36A and B: **A.** Alkali burn in acute phase; **B.** Alkali burn with 360° limbal ischemia

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TABLE 24.1: Hughes and Roper-Hall classification is useful in providing a guide to prognosis and treatment (applicable for both alkali and acid burns)

Grade	Clinical features	Prognosis
I	• Corneal epithelial damage • No limbal ischemia	Good
II	• Cornea hazy, but iris details seen • Ischemia at less than 1/3 of the limbus	Fair
III	• Total loss of corneal epithelium • Stromal haziness, blurring of iris details • Ischemia at 1/3–1/2 of the limbus	Guarded
IV	• Cornea totally opaque • No view of the iris or pupil • Ischemia at more than 1/2 of the limbus	Poor (Symblepharon and dry eye inevitable)

  - ***Removal of particles***, carefully with the help of a sterile swab or forceps.
  - Excision of necrotic conjunctival tags.
  - A careful assessment of the grade of chemical burn with the help of a slit lamp.
- ***Treatment in acute phase***
  - For grade I and II, the patient is treated at out patient department (OPD), and for Grade III and IV, the patient must be admitted (Table 24.1).
  - Antibiotics drops, cycloplegic, topical corticosteroids and antiglaucoma therapy are important.
  - A collagenase inhibitor (10–20% acetyl cysteine drop) is very effective.
  - Amniotic membrane transplantation (AMT) in acute phase is giving much better results, especially in alkali burn.
- ***Intermediate treatment***
  - Therapeutic bandage contact lens for persistent epithelial defect (Fig. 24.37).
  - A conjunctival flap may be helpful in stromal ulceration.
  - Prevention of symblepharon formation by sweeping a glass rod twice daily.
- ***Surgical rehabilitation***
  - When the eye is totally healed, the potential for useful vision in the eye, is assessed.
  - Conjunctival limbal autograft (CLAU) from the other eye if only one eye is affected with limbal stem cell deficiency. CLAU is the best type of limbal stem cell transplantation (Fig. 24.38).
  - Penetrating keratoplasty—success rate is not encouraging.
  - Keratoprosthesis in drastic cases.

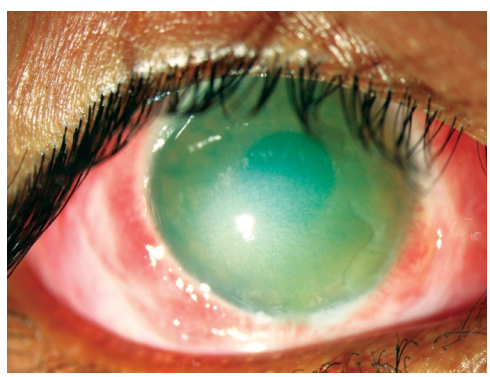


Fig. 24.37: Chemical burn in intermediate phase—persistent epithelial defect

THERMAL BURNS

- Thermal burns of the eyelids (Fig. 24.39) usually do not involve the eyeball proper, since blinking reflex provides the natural protection.
- In addition, tightly closed eyelids usually prevent involvement of eyelid margins themselves.

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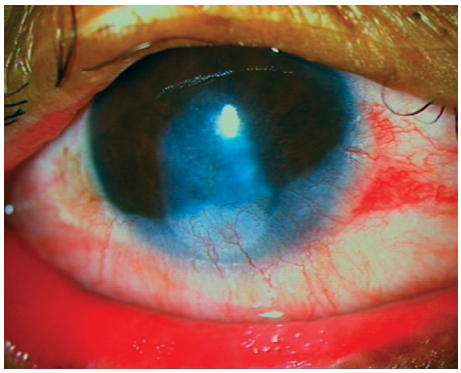


Fig. 24.38: Chemical burn—late phasecorneal opacity with limbal stem cell deficiency



Fig. 24.39: Thermal burn—eyelids

- Thermal burns of the eyelids require ***prompt care to prevent ectropion formation***. Early skin grafting in severe burns may speed up the recovery, and prevent late complications.
- Severe body burns are often associated with carbonmonoxide poisoning and asphyxia. The ocular changes then resemble those seen in ***altitudinal hypoxemia***. There are retinal hemorrhages, arterial and venous congestion, and hyperemia of the optic disc.

RADIATIONAL INJURIES

The ocular media should ideally allow maximum transmission of light, with minimum absorption and reflection.

Only energy that is absorbed, causes a reaction and injury to the ocular tissue. The effects are with:

- ***Ultraviolet (UV) rays:*** The cornea transmits the longer UV rays (300–400 nm), but its epithelium absorbs the UV rays with wavelengths below 300 nm. This results ***photokeratitis*** (UV keratitis) or snow blindness or photophthalmia.
- ***Visible spectrum (400–700 nm):*** It is perceived as light. The lens is normally transparent to all the wavelengths of visible spectrum, but becomes progressively more impervious with advancing age with nuclear sclerosis, and restricts the violet end of the spectrum in old age.

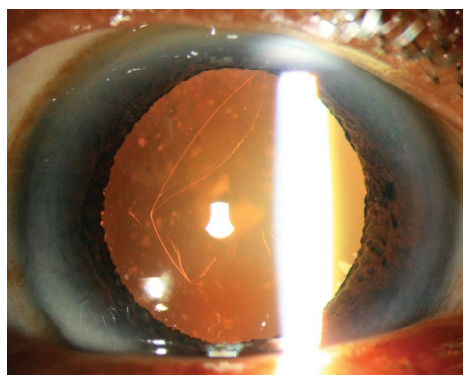


Fig. 24.40: Glass-blower's cataract—true exfoliation

- ***Infrared rays (above 700 nm):*** They are absorbed by the iris, and the resultant heat is transmitted to the lens, which becomes cataractous ***glass-blower's cataract*** (Fig. 24.40). Observation of a solar eclipse with the naked eye, causes ***solar retinopathy***. As the pupil is dilated due to low light intensity, the infrared (heat) rays are focused on the fovea centralis, causing a focal macular burn ***(eclipse burn or eclipse blindness) (Fig. 24.41)***.
- ***Electromagnetic energy of short wave lengths*** (X-rays, gamma-rays): They may damage any part of the eye, e.g. blepharoconjunctivitis, keratitis, radiation cataract (Fig. 24.42) and retinopathy.

Adequate prophylactic measures are to be taken to prevent radiational injuries.

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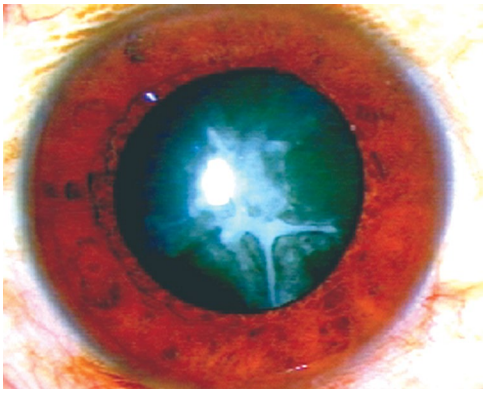


Fig. 24.41: Radiation cataract

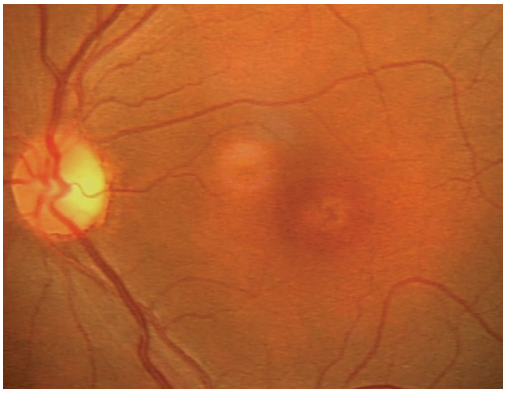


Fig. 24.42: Solar retinopathy (eclipse burn)

Different types of protective glasses are available, which can prevent lesions caused by UV and infrared rays.

Adequate and necessary protection is also to be taken during watching a solar eclipse.

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25 CHAPTER Common Eye Surgeries

GENERAL PRINCIPLES

- For eye operations which are not urgent, it is essential to have the patient in a good state of health as possible.
- There must be no septic lesions (e.g. infected teeth, or otitis media).
- Culture swab is taken from the conjunctival sac, and a broad-spectrum antibiotic drop (e.g. gatifloxacin or moxifloxacin) is started 3–4 times daily for 4 days prior to surgery.
- Lacrimal passages are irrigated, and any evidence of infection is noted, the presence of which will necessitate dacryocystorhinostomy (DCR) or dacryocystectomy (DCT) operation before intraocular surgical procedures.
- Diabetes and hypertension must be controlled.
- Cardiovascular, respiratory and renal diseases must receive medical attention.
- Hemorrhagic tendency must be investigated routinely.
- The patient should not smoke for one week before operation and about one month after it.
- The psychological state of the patient is reviewed and every effort is made to encourage a state of optimism.
- Fitness for general anesthesia is required for children, very nervous patients and for extensive operations.

PREOPERATIVE PREPARATION

Day of admission (1 day before surgery)

- Informed written consent for operation.
- Antibiotic drops are instilled frequently.
- Mild tranquillizer at bed time.
- Tab acetazolamide 1–2 tab at night (for cataract or glaucoma surgery).
- Lashes of upper and lower lids are cut short (many surgeons now prefer to leave the lashes uncut, relying on the speculum and drape to isolate them from the operative field).
- Mild purgative if necessary.

Day of operation

- Light breakfast in the morning.
- Shaving for male patients.
- The face is washed thoroughly with soap and water.
- Diabetic patient must omit the morning dose of medicine.
- Blood pressure is checked, and if necessary, antihypertensive drugs are to be administered.
- Appropriate premedication for general anesthesia.
- Mild sedative if required.
- Tab acetazolamide—2 tabs, may be given 1–2 hours before surgery if necessary.
- Injection mannitol (20%) 200–300 mL, is given intravascular at least 30 minutes before surgery (for young and selective patients with high intraocular pressure (IOP).

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- Start dilating the pupil (when necessary) with phenylephrine and tropicamide (or phenylephrine and homatropine) along with flurbiprofen or nepafenac eye drop 3–4 times 1 hour before surgery.
  Flurbiprofen or nepafenac eye drops prevent intraoperative miosis, and also decrease post-operative paid and inflammation.

POSTOPERATIVE CARE AND TREATMENT

- Avoid unnecessary jerky movements of the head and the body.
- Avoid sudden strain, e.g. sneezing, coughing, weight lifting, etc.
- While the patient is in bed, he should be made comfortable. It is preferable to place the patient at night, either supine or onto the unoperated side.
- As the wound security is excellent, prolonged bed rest is unnecessary, on the other hand, early mobility is encouraged for the elderly patients.
- Postoperative pain, which comes on half to 1 hour after the operation, must be treated with analgesics.
- A mild sedative is useful to promote sleep for some patients.
- Some disturbance of bowel rhythm is expected for a few days following major surgery. This should not cause concern unless the patient is suffering from discomfort. This can be easily tackled by mild laxatives.
- A normal, easily digestible balanced diet is re–introduced from the next day.
- A postoperative dressing is important to protect a healing wound from injury and exogenous infection to afford support and to soak up the discharge for first 24 hours.
- An eye pad is better replaced by a protective dark glasses, or a shield, after 24 hours. It may be better to use the dark glasses during night to avoid accidental trauma by finger or by a corner of a pillowcase.

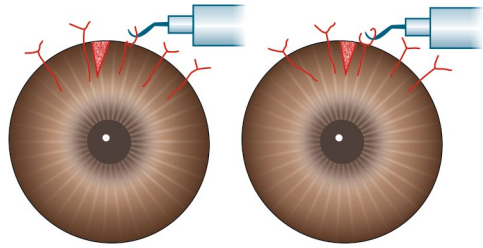


Fig. 25.1: Katzin's technique of suture removal using a bent 26 gauge disposable needle

- The patient is instructed to clean the ocular discharge daily in the morning with sterile cotton (in boiling water).
- Instillation of eye drop and application of ointment at bed time as directed by the treating surgeon.
- ***Removal of sutures***
  - ***Conjunctival sutures*** are usually removed after 5 to 7 days.
  - ***Corneal sutures***
    - Eight to zero virgin silk, is usually removed 4–6 weeks after the surgery.
    - Ten to zero sutures, when continuous or interrupted with buried knots, it is not necessary to remove them. But for exposed knots, they should be removed carefully. A bent disposable 26 gauge needle is often sharp enough for this purpose ***(Katzin's method (Fig. 25.1).***

Corneal sutures are removed either because of irritation, or as a mean of adjusting corneal astigmatism. It is to be done under magnification with full aseptic measure.

Prescription of glasses: As most of the cataract surgery are sutureless glasses are prescribed 1 to 3 weeks depending upon the type of surgery.

ANESTHESIA AND AKINESIA

A good anesthesia, either local anesthesia (LA) or general anesthesia (GA), is essential for smoother surgery to save sight and indeed a bad anesthesia may be disastrous.

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Local Anesthesia

Most eye surgeons still prefer to perform cataract and other operations under local anesthesia.

Advantages

- It is easy to administer, and there is no need of trained anesthetist.
- Patient can cooperate during surgery.
- It does not required any special and costly investigations.
- There is no major complication of GA, e.g. cardiorespiratory problems, coughing, vomiting, etc. Immediate postoperative recovery is uneventful.
- It is less expensive.

Disadvantages

- It is difficult and sometimes, hazardous for the nervous and non-cooperative patients.
- Head movements and straining, may cause serious complications.
- Cardiac shock may also occur in susceptible individual.
- Rarely, there may be globe perforation, which is sight-threatening.

General Anesthesia

The chief advantage of GA is that, control is where it should be, entirely with the surgeon and the anesthetist.

Indications of General Anesthesia in Ophthalmic Practice

In children

- Examination under anesthesia (EUA)
- Surgeries like:
  - Probing of nasolacrimal duct (NLD)
  - Cataract operations
  - Glaucoma operations
  - Enucleation as in retinoblastoma
  - Injury repair
  - Squint and DCR operation.

In adults

- Repair of perforating injury.
- Mentally retarded and non-cooperative patients.
- The patient is too nervous and apprehensive.
- Enucleation, evisceration or exenteration operations.
- Major oculoplastic surgeries.
- Major posterior segment surgeries.
- Surgeon's preference for GA.

Types of Local Anesthesia

- ***Surface (topical) anesthesia:*** This is either by instillation of 4% lignocaine, 0.5%–1% amethocaine or 0.5% proparacaine thrice at an interval of 5 minutes. It is most commonly required for the removal of corneal foreign body, syringing, tonometry or gonioscopy. Many cataract surgeons prefer to perform phacoemulsification under topical anesthesia.
- ***Infiltration (regional) anesthesia:*** There is no need to say more than a few words of encouragement and assurance, and to give a preliminary warning about the prick of the needle which injects the anesthetic solution.

The agents commonly used are:

Anesthetic agents

- ***Injection lignocaine (2%)***
  - ***Onset of action:*** 5–10 minutes
  - ***Duration of action:*** 45 minutes–2 hours
- ***Injection bupivacaine (0.50%–0.75%)***
  - ***Onset of action:*** 15–20 minutes
  - ***Duration of action:*** 5–8 hours

Adjuvants

- ***Injection adrenaline (1 in 100,000):*** It decreases the systemic absorption of the anesthetic agent by local vasoconstriction. It also reduces the chance of bleeding and prolongs the duration of action. It is contraindicated in hypertension and heart diseases.

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- ***Injection hyaluronidase:*** It enhances the diffusion of anesthetic agents through the tissues. It is used 75–150 units/10 mL of anesthetic solution.

Infiltration anesthesia is best achieved by a ***mixture*** containing 50% of injection lignocaine with adrenaline (for rapid onset) + 50% of injection bupivacaine (for longer duration of action) + injection hyaluronidase.

Types of Infiltration Anesthesia

Facial Block (Fig. 25.2)

It is to block the facial nerve or its zygomatic branch to paralyze the orbicularis oculi muscle. The aim is to prevent closure and squeezing of the eyelids during operation.

- ***O'Brien's technique:*** About 4–5 mL of Anesthetic solution is infiltrated at the neck of the mandible just infront of the tragus.
- ***Van Lint's method:*** The needle is introduced about 1 cm below and behind the lateral canthus. About 4 mL of solution is infiltrated along the superolateral and inferolateral orbital margins in a V-shaped manner.

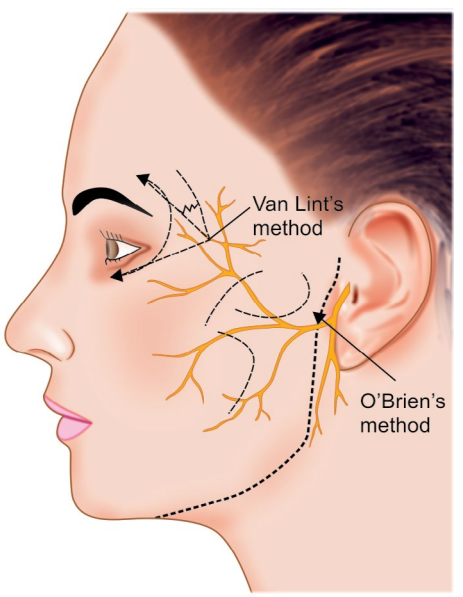


Fig. 25.2: Facial block

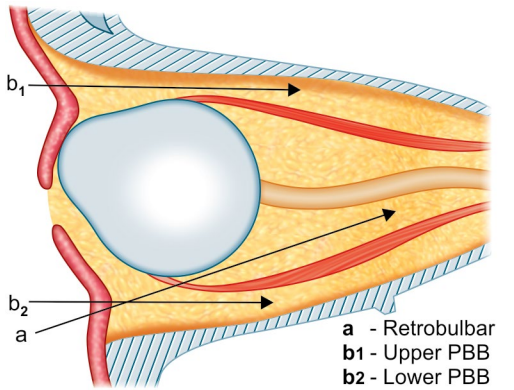


Fig. 25.3: Retrobulbar and peribulbar block (PBB)

Retrobulbar (Ciliary) Block (Fig. 25.3)

The patient is asked to look up and opposite side. A long needle (35 mm) is introduced at the junction of middle-third and lateral third along the inferior orbital margin, and then directed backwards and medially, towards the apex of the orbit. one to two mL of anesthetic solution is injected. Onset of action is indicated by mydriasis.

The aim is to block the ciliary ganglion, and to paralyze the extraocular muscles.

Effects

- Anesthesia of the deeper intraocular structures (e.g. iris)
- Akinesia of all extraocular muscles (except superior oblique)
- Mydriasis
- Hypotony is due to loss of tone of extra–ocular muscle and bulbar massage
- Proptosis, which is desirable in deep–seated and sunken eyes
- Decreased oculocardiac reflex.

Complications

- ***Retrobulbar hemorrhage (RBH):*** Immediate proptosis, subconjunctival hemorrhage and ecchymosis. Operation is to be postponed for 2–3 weeks. ***Treatment*** is

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by tight pressure bandage, (lateral cathotomy if required), tab acetazolamide and reassurance.
- Perforation of the globe (especially in myopic eyes).
- Central retinal artery occlusion.
- Injection of anesthetic solution into the subarachnoid space may cause brainstem anesthesia leading to convulsion and respiratory arrest.
- Optic nerve damage and atrophy.

Peribulbar Block

Most surgeons have abandoned the retrobulbar block in favor of peribulbar injections (Figs 25.4 and 25.5) at inferior and superior parts of peripheral space of the orbit, with a 23 or 24 gauge needle.

The ***inferior injection*** is given at the junction of middle- and outer-third of the lower orbital margin. With the patient looking up, the needle is directed towards the floor of the orbit.

An additional ***superior injection*** may be given superonasally beneath the superior orbital notch, with the needle directed towards the orbit roof.

- An intermittent pressure is given against the closed eyelids for 10 minutes with the help of fingers, ***super pinky*** or Honan's balloon. This is to achieve lowering of IOP, and reduction in orbital volume of the fluid injected.
- It has all the effects of facial, retrobulbar and surface anesthesia.



Fig. 25.4: Peribulbar injection—lower



Fig. 25.5: Peribulbar injection—upper

Advantages

- Least chance of RBH, globe perforation or optic nerve damage.
- Less painful procedure.
- No need of additional facial block and surface anesthesia.

Disadvantages

- In takes little longer time to achieve anesthesia, akinesia and hypotony.
- Chemosis of lid and conjunctiva, and subconjunctival hemorrhage are common. Injection hyaluronidase is essential for spreading the anesthetic solution.
- Rarely, additional facial or retrobulbar block is necessary.

Regional Blocks (at Other Sites)

The sites are different, for different extraocular operations, e.g. for DCT or DCR operation, for lid tumors, for ptosis, chalazion, etc.

Preparatory steps just before operation

- Periocular skin is cleaned painted with rectified spirit and povidone iodine (10%) lotion.

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- Conjunctival sac may be washed with sterile normal saline or weak (5%) povidone-iodine lotion.
- Two or three drops of antibiotics are to be instilled.
- Sterile towels are draped around the head, neck and chest. The face is covered with a mask or 'steridrape' in which an aperture has been made to get access to the eye.

CONVENTIONAL ECCE WITH PCIOL IMPLANTATION

Surgical steps: Pupil should be widely dilated preoperatively and an operative microscope (Fig. 25.6) is essential.

- ***Separation of the eyelids:*** Either by lid stitches or wire eye speculum.
- ***Superior rectus stitch (Bridle suture):*** It helps to keep the globe rotated downwards and to retract a fornix-based conjunctival flap. It also aids in lifting a deep-seated globe from the orbit.
- ***Conjunctival flap preparation (Fig. 25.7A and B):*** There are two types of conjunctival flap: fornix-based and limbal-based. The former provides better visualization of the upper limbus, and is preferred by most surgeons. Here, the conjunctiva is dissected at the upper limbus and retracted towards the fornix. Whereas, in limbal-based flap, the conjunctiva is incised 4–5 mm away from the limbus, and reflected over the cornea.
- ***Hemostasis of bleeding points:*** It is most satisfactorily done with a wet-field coagulator. Alternately it may be done with a thermocautery, which causes more tissue charring.
- ***Limbal-grooved incision:*** A partial thickness limbal-groove is made along the entire length of the intended section, with a knife (no. 11 or no. 15 blade), or with a razor blade-fragment in a blade-breaker.
- ***Anterior chamber (Fig. 25.8) is entered*** with a bent-tipped 26 gauge needle (capsulotome), fitted with balanced salt solution (BSS) (or Ringer's lactate) filled 2 mL syringe. (Alternately, anterior chamber may be entered with the tip of the knife, and then anterior chamber is filled with viscoelastic solution).

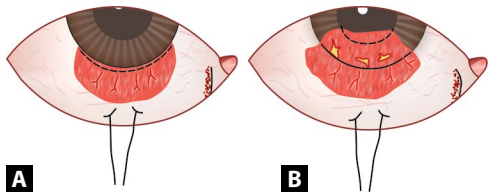


Fig. 25.7: Conjunctival flaps; **A.** Fornix-based flap; **B.** Limbal-based flap



Fig. 25.6: Operative microscope

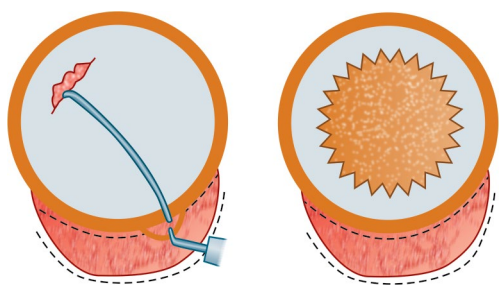


Fig. 25.8: Anterior capsulotomy with a bent tipped 26 gauge disposable needle

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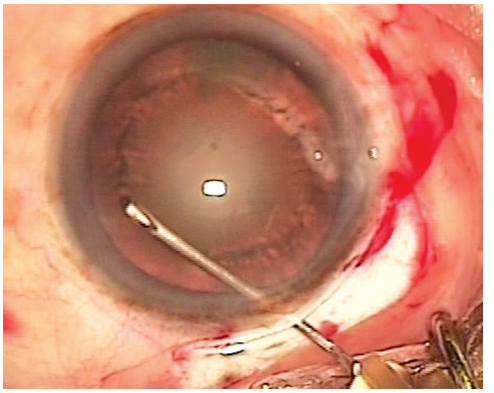


Fig. 25.9: Can-opener capsulotomy with 26 G needle cystitome

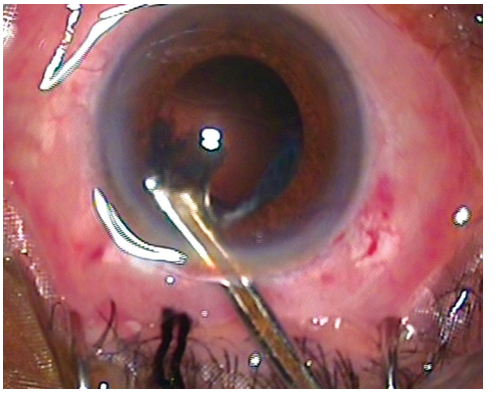
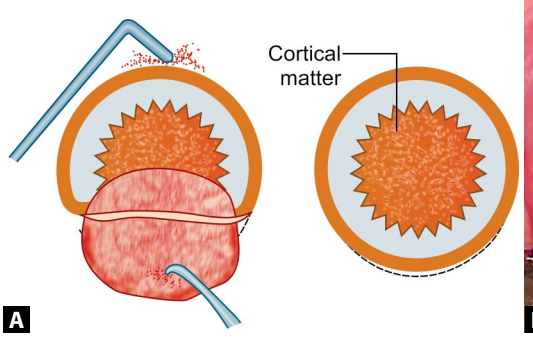
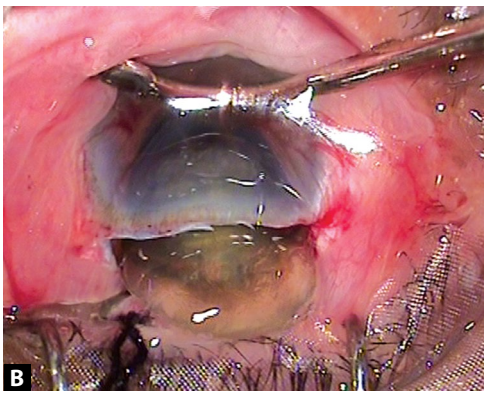


Fig. 25.11: Cortical cleaning by Simcoe I/A cannula

- ***A can-opener anterior capsulotomy (Fig. 25.9)*** is performed with the tip of the 26 gauge needle. Alternately, a ***continuous curvilinear capsulorhexis*** (CCC) is preferable for smooth edge of the anterior capsular opening which increases the strength of the capsular bag. Or, an envelope technique may be done.
- ***Limbal section is enlarged*** with corneal scissors for 10–12 mm (110°–120°), at the upper limbus.
- Anterior capsule is removed.
- ***Lens nucleus is delivered (Figs 25.10A and B)*** by scleral depression with the help of a lens hook and lens spatula, or vectis.
- ***Simultaneous irrigation and aspiration*** (I/A) with a two-way cannula (Fig. 25.11), to clear all the cortical matter of the lens. The cannula is fitted with an infusion system of Ringer's lactate, or BSS. The posterior capsule may be polished with special polisher to remove/small residual cortical plaque.
- Viscoelastic substance is injected into the capsular-bag and anterior chamber, to facilitate subsequent insertion of intraocular lens (IOL).
- ***Intraocular lens insertion:*** A posterior chamber IOL is grasped by the optic with MacPherson forceps. The anterior surface of the lens is coated with viscoelastic material. The implant is introduced into the





Figs 25.10A and B: **A.** Delivery of the lens nucleus; **B.** Nucleus delivery by sliding technique

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eye without retracting or lifting the cornea. The leading loop is placed in the inferior portion of the capsular bag or ciliary sulcus. The trailing loop is flexed towards the optic, and with slight pronation of the wrist, the loop is engaged slightly posterior to the capsular bag or in the ciliary sulcus.

- ***The implant is then dialed*** into horizontal position by engaging into the dialing holes with an IOL–dialer, and checked for correct centration of the IOL.
- ***Removal of viscoelastic substance*** by irrigation and aspiration.
- ***Constriction of the pupil*** with injection pilocarpine (0.125%) into the anterior chamber may be required and subsequently wash is given to remove pilocarpine.
- ***A peripheral iridectomy*** is not mandatory in routine cases of extracapsular cataract extraction (ECCE). It is required in complicated cataract (e.g. uveitic cataract), when combined with trabeculectomy, if posterior capsular rent occurs during surgery or in case of ACIOL if required.
- ***Closure of the wound*** with four to five interrupted 10–0 nylon sutures (Fig. 25.12) with buried knots.
- Reformation of the anterior chamber with ***BSS*** or ***Ringer's lactate solution***.

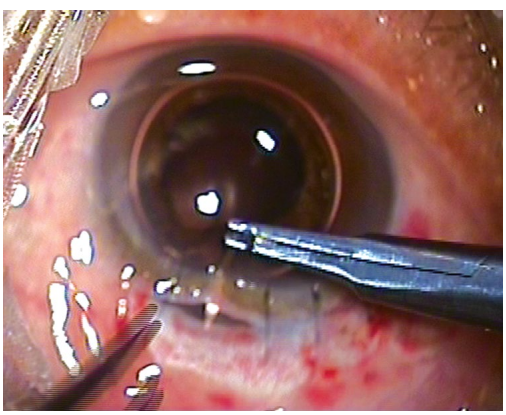


Fig. 25.12: Corneoscleral suturing by 10–0 nylon

- ***Subconjunctival injection*** of gentamycin and dexamethasone may be given.
- Topical antibiotic and steroid ointment.
- Pad and bandage.

MANUAL SMALL INCISION CATARACT SURGERY

Small incision cataract surgery (SICS) (Figs 25.13A to L) is now the method of choice of cataract surgery all over the world. Manual or non-phaco techniques are becoming popular in the developing countries. Phacoemulsification (phaco) technique is the excellent procedure of doing SICS. But, it has got its own limitations—like, lack of training facilities, cost and maintenance of the machine, and the serious complications by the beginners. Manual SICS provides the best of both worlds—the simplicity of ECCE and the excellent results of phacoemulsification procedure.

Aims

- Early ambulation and rehabilitation of the patient.
- Least complication.
- To achieve low astigmatism.

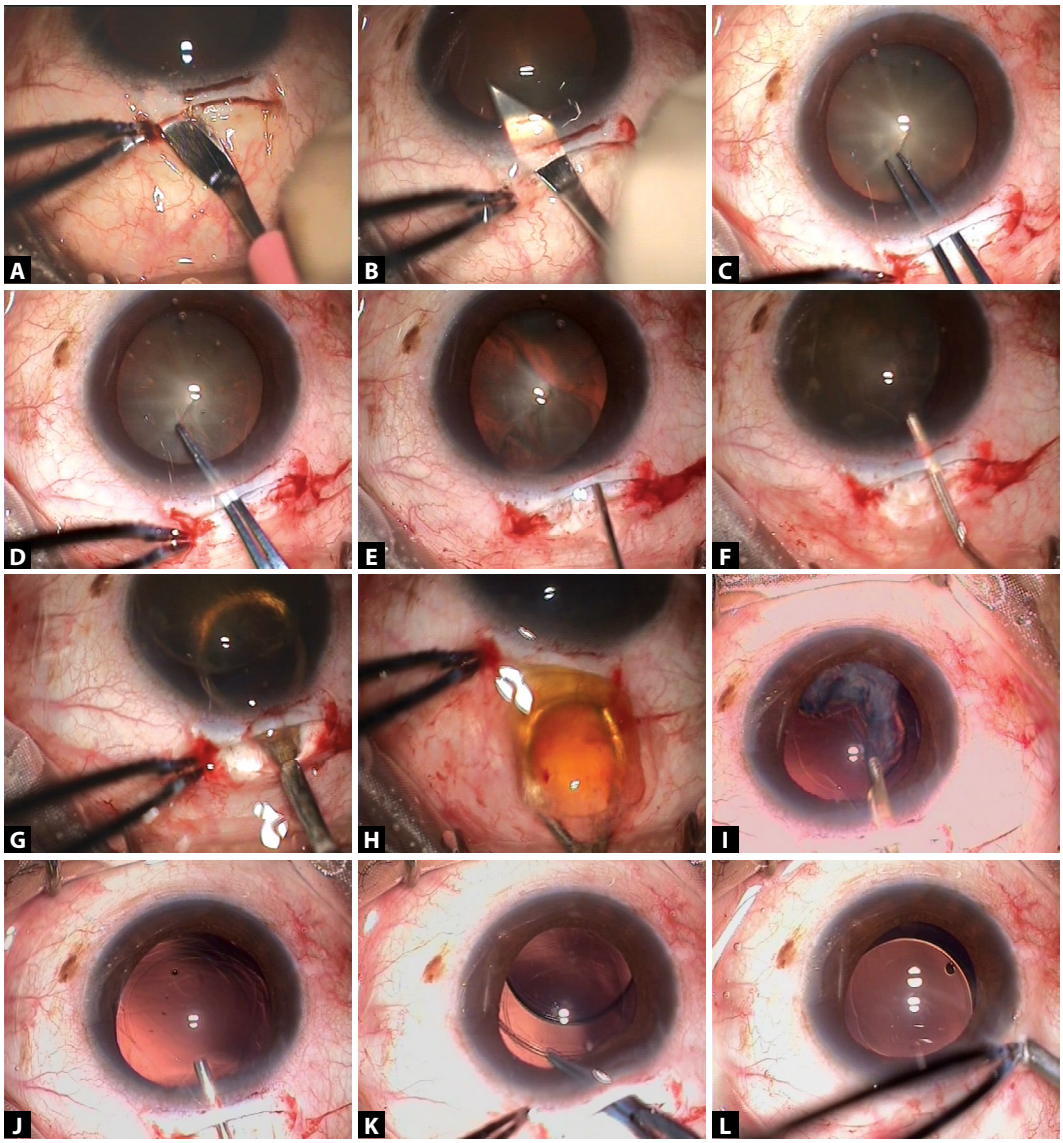
Principles

- Self-sealing sclerocorneal tunnel incision.
- Working under high IOP in close chamber.
- Working in deep anterior chamber.
- Protection of corneal endothelial layer with good viscoelastics HPMC (hydroxypropyl methylcellulose) or sodium hyaluronate throughout the procedure.

Technical Steps

- 5.5–6.5 mm sclerocorneal tunnel incision.
- Routine capsulorhexis or may be can-opener capsulotomy.
- Hydrodissection of the lens nucleus from its capsule.

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Figs 25.13A to L: Manual small incision cataract surgery (SICS). **A.** External scleral incision with tunneling; **B.** Corneal internal incision; **C.** Initiation of capsulorhexis; **D.** Completion of capsulorhexis; **E.** Hydrodissection—note the fluid wave; **F.** Nulear rotation and its prolapse into anterior chamber; **G.** Irrigating vectis behind the nucleus; **H.** Nucleus delivery by the same vectis; **I.** Cortical cleaning with Simcoe irrigation and aspiration cannula; **J.** Viscoelastic in the anterior chamber and capsular bag; **K.** Posterior chamber intraocular lens implantation in-the-bag; **L.** Wound closure after completion of surgery

- Nuclear prolapse into anterior chamber.
- Nucleus delivery by irrigating Vectis or phacosandwich or fish hook technique.
- Cortical cleaning by Simcoe I/A cannula.
- Viscoelastic agent to fill the bag and anterior chamber.

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- Intraocular lens placement in-the-bag or may be in ciliary sulcus.
- Anterior chamber wash and reformation.
- Closure of the conjunctiva.

Some surgeons use anterior chamber maintainer (ACM) during manual SICS.

Irrigating Vectis and phaco-sandwich techniques are the most popular methods for nucleus delivery in manual SICS.

Advantages

- Low cost no expensive instruments.
- Learning curve smooth and easy.
- Same results as phacoemulsification.
- Within reach of every cataract surgeon.
- Can be performed in any type of cataract (hard, black or hypermature).
- Lesser complications than phacoemulsification by the beginners.

PHACOEMULSIFICATION (FIGS 25.14 AND 25.15)

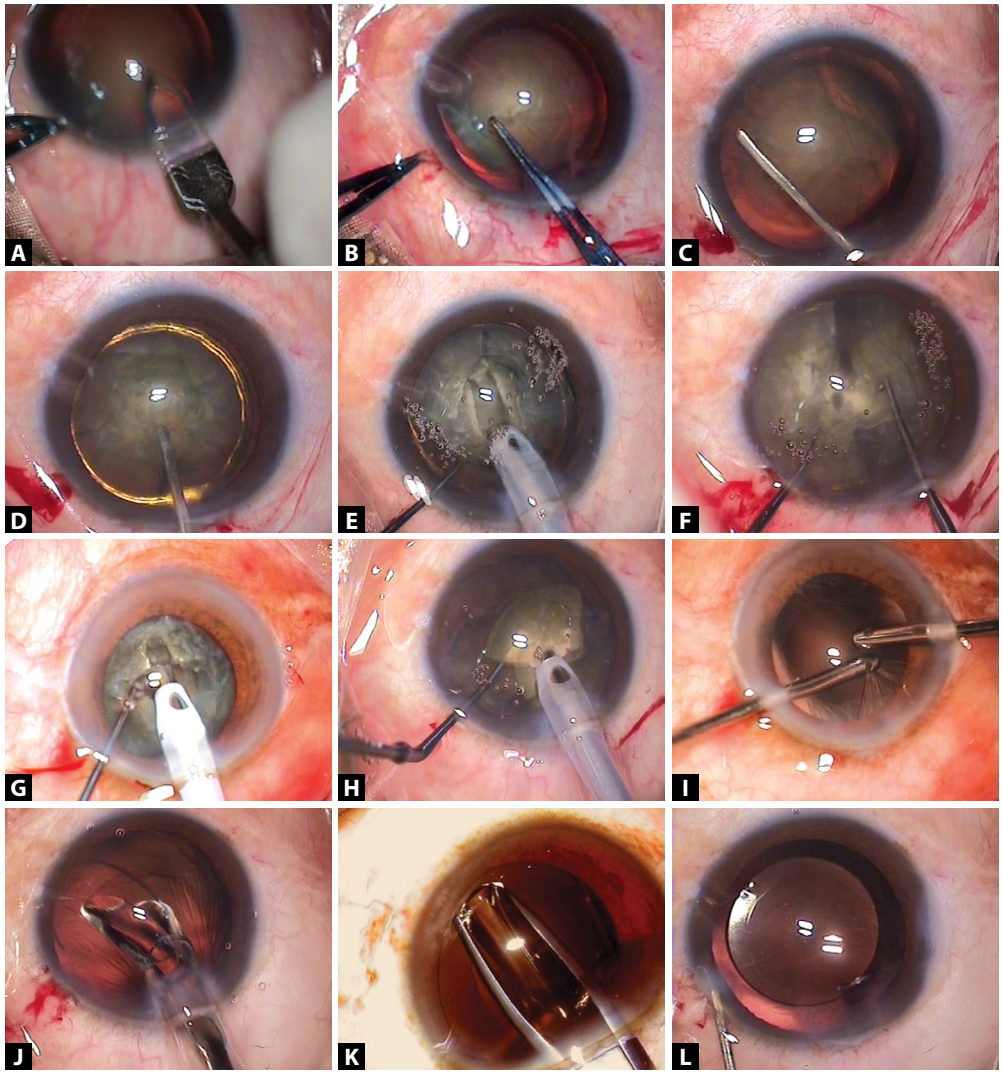
Phacoemulsification is now the standard form of extracapsular cataract extraction. The machine is known as phacoemulsifier which has three functions:

1. ***Irrigation:*** It is a gravity flow system, and the fluid BSS is allowed to flow to the handpiece (Figs 25.16A and B) being used, through foot switching.
2. ***Aspiration:*** It occurs through a peristaltic or venturi pump, and the aspiration force is also controlled by surgeon's foot, by foot–switching.
3. ***Fragmentation (emulsification):*** It occurs through a piezoelectric ultrasonic mechanism which activate a hollow 0.9 mm titanium needle, vibrating at 40,000/sec. The amplitude of vibration is also controlled by same foot switch.

Steps of surgical procedure (in brief)

- Selection of the patient (difficult in eyes with suprahard nucleus) with dilated pupil.
- Most of the surgeons preferred under topical anesthesia.
- A 2.2 mm to 2.8 mm clear corneal incision on temporal side. This is for foldable IOL. It is preferable to create two-step or three step incision (for phaco with rigid lens a 5.5 mm posterior-limbal tunnel or sclerocorneal tunnel is required; internal incision in that case will be 2.8 mm for entry of phacotip).
- Viscoelastic agent is injected into the anterior chamber.
- Continuous curvilinear capsulorhexis is performed using a bent-tipped 26 gauge needle or rhexis (utarata) forceps.
- Hydrodissection and hydrodelamination to separate lens nucleus and cortex from the capsular bag.
- Sculpting the lens nucleus from the anterior surface.
- A phaco chopper is passed into the anterior chamber through a separate stab incision.
- The chopper keeps the nucleus away from the corneal endothelium, simultaneously fragments the nucleus and feeds smaller pieces of nucleus into the ultrasonic tip, until the whole nucleus is fragmented and aspirated. The different techniques used are—chip and flip technique for soft nucleus, four quadrant divide and conquer technique stop and chop technique or direct chop technique for nucleus fragmentation and emulsification.
- Epinucleus removal by phaco hand piece.
- Lens cortex is aspirated completely from the peripheral part, using bimanual irrigation and aspiration probes.
- Polishing of the posterior capsule if necessary.
- Viscoelastic is injected to inflate the capsular bag.
- A foldable IOL is implanted within capsular bag using IOL injector system or [incision is enlarged to 5.5 mm for insertion of a rigid polymethyl methacrylate (PMMA) PCIOL in-the-bag].
- Viscoelastic material is washed thoroughly.

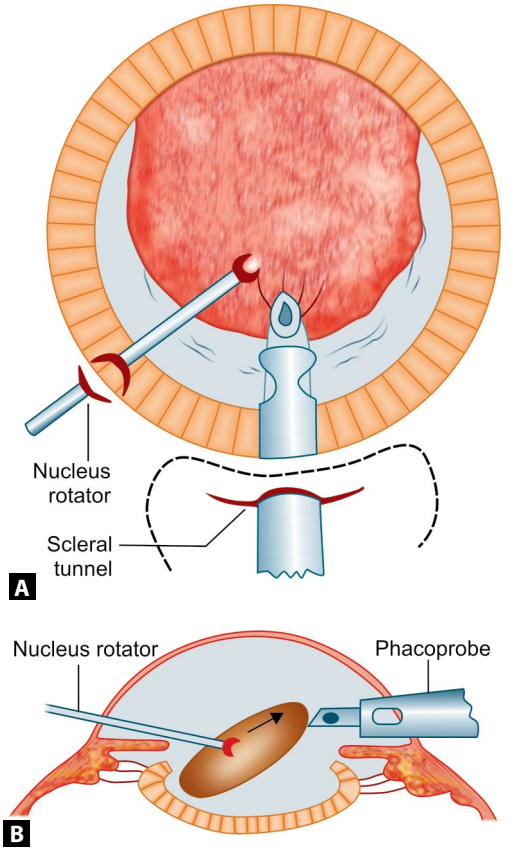
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Figs 25.14A to L: Phacoemulsification. **A.** Clear corneal tunnel incision with diamond knife; **B.** Continuous curvilinear capsulorhexis with rhexis forceps; **C.** Hydrodissection—note the fluid wave; **D.** Hydrodelamination—appearence of 'golden ring'; **E.** Deep sculpting in stop and chop method; **F.** Bimanual nuclear division (halving); **G.** Deep sculpting in divide and conquer technique; **H.** Nucleus piece emulsification and removal; **I.** Cortical cleaning by automated I/A probes; **J.** Foldable intraocular lens in-the-bag by an injector; **K.** Foldable intraocular lens in-the-bag by folder-holder; **L.** Intraocular lens in-the-bag and hydration of the wound

- Hydration of the side port(s).
- No suture (sutureless surgery) is required for foldable IOL with 2.2 to 3.0 mm incision (one suture may be required if the incision is 5.5 mm).
- Repose the conjunctival flap if required.

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Figs 25.15A and B: Phacoemulsification



B

Figs 25.16A and B: A. Phacoemulsification machine (Alcon–Infiniti®); **B.** Phaco handpiece and foot switch

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- Subconjunctival injection of gentamycin and dexamethasone.
- No pad and bandage if under topical anesthesia.

FEMTOSECOND LASER CATARACT SURGERY

It is the latest development in cataract surgery. A femtosecond-assisted cataract procedure is guided by in built anterior segment imaging (OCT or Scheimpflug) system which gives maximum precision in cataract surgery. Femtosecond laser uses in cataract surgery are for:

- A perfect centered anterior capsulorhexis.
- Phacofragmentation (by microphotolysis of the lens nucleus in multiple small fragments).
- The creation of single and multiplane incisions in the cornea.
- Astigmatic relaxing incisions when required.

Nevertheless, the need for ultrasound phaco is still there, further to emulsify the nuclear fragments and to aspirate them.

Although the cost of the procedure is at present much higher compared to phacoemulsification, femtosecond laser cataract surgery is likely to become the gold standard in the near future.

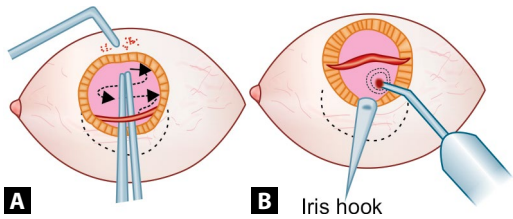
INTRACAPSULAR CATARACT EXTRACTION (FIGS 25.18A AND B)

Very rarely done now a days as in anterior dislocation of the lens, grossly subluxated lens (*ectopia lemtis*) etc.

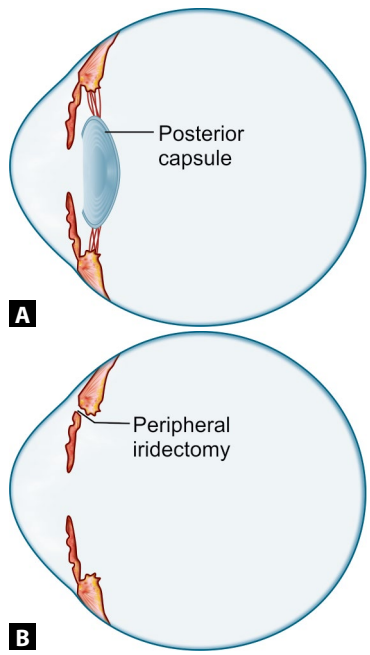
Surgical Steps

1–5. Same as conventional ECCE.

6. Anterior chamber is entered with the tip of the blade, with slow release of the aqueous.



Figs 25.17A and B: Methods of intracapsular cataract extraction. **A.** By intracapsular forceps; **B.** By cryoprobe



Figs 25.18A and B: Postoperative appearance. **A.** After extracapsular cataract extraction; **B.** After intracapsular cataract extraction

7. Limbal section is enlarged with corneal scissors about 150–180° (i.e. from 3 to 9 o'clock position).
8. A peripheral iridectomy is done at 11 o'clock to 10'clock position. This is a must for intracapsular cataract extraction (ICCE).
9. Lens (with its capsule) is extracted by either a cryoprobe (Figs 25.17A and B), or by intra-capsular forceps, or, by tumbling method.
10. Iris is reposited back.

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Differences between intracapsular cataract extraction (ICCE) and extracapsular cataract extraction (ECCE)

Features	ICCE	ECCE
Indications	• In camps or other places, where the as facilities for ECCE are not available. • Subluxated or dislocated lens.	• All types of cataract except mentioned in ICCE.
Technique	• Relatively easy	• Relatively difficult
Intraoperative complications	• Vitreous loss is more common. • Hyphema is more frequent. • Accidental capsule rupture—causes problem for cortical cleaning.	• Vitreous loss is very rare. • Hyphema is less frequent. • No such problem.
Postoperative complications	• Iridocyclitis is less common. • No question of after–cataract formation PCO, and so no need for second operation. • Incidence of retinal detachment is more. • Incidence of CME is more. • Chance of pupillary block glaucome is more.	• Iridocyclitis is more common. • Chances of after–cataract formation, and consequent need for second operation, like needling or yttrium aluminium garnet (YAG) laser capsulotomy. • Incidence of retinal detachment is less. • Incidence of CME is less. • Chance of pupillary block glaucoma is much less.
Provision for IOL	• ACIOL may be implanted, which is not popular due to its complications.	• PCIOL is usually implanted, which is an ideal IOL.
Cost	• Less costly due to less costly instrumentation.	• More costly due to more investment for operative microscope and instruments.

Abbreviations: **ICCE**—Intracapsular cataract extraction; **ECCE**—Extracapsular cataract extraction; **PCO**—Posterior capsular opacification; **CME**—Cystoid macular edema; **IOL**—Intraocular lens; **ACIOL**—Anterior chamber IOL; **PCIOL—**Posterior chamber IOL

11. Limbal section is closed with 10–0 nylon by five interrupted stitches.
12. Anterior chamber is reformed by air or BSS.
13. Subconjunctival injection of gentamycin and dexamethasone.
14. Pad and bandage are given after applying antibiotic ointment or the incision may be like SICS and the lens delivery via wire Vectis.

A very good anterior vitrectomy (triamcinolone assisted) is required in ICCE before implantation an IOL either ACIOL or scleral fixation IOL.

INTRAOCULAR LENS IMPLANTATION

Now–a–days IOL implantation has become the standard surgical procedure in the management of cataract. The main aim is to replace the function of the natural crystalline lens, and to restore the vision approximating to pre–cataractous stage.

Intraocular lens implantation is usually performed along with extraction of cataract at the same sitting (as a primary procedure), and it is called ***primary implantation*** (Fig. 25.19). But, an implantation can also be done in selected cases any time after primary cataract surgery, it is then called ***secondary implantation***.

Intraocular lens implantation was first performed by Dr Harold Ridley, on 29th November, 1949 at St. Thomas Hospital, London. It was a posterior chamber IOL.

Indications

Any type of cataract for primary implantation, except in congenital cataract before 2 years of

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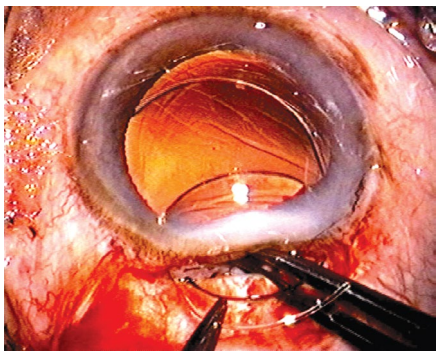


Fig. 25.19: Intraocular lens implantation in the posterior chamber

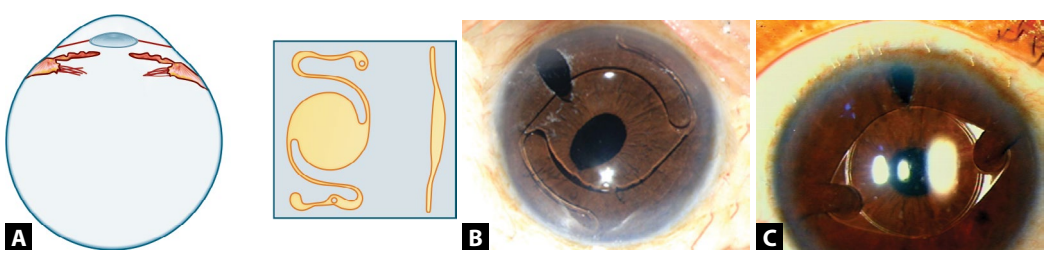
age. A secondary IOL implantation may be considered after 3 years of age.

Types

Depending upon the placement of the optic, they are of two types—anterior chamber IOL (ACIOL) and posterior chamber IOL (PCIOL).

1. ***Anterior chamber IOL (ACIOL) (Figs 25.20A to C):*** They are placed in the anterior chamber, usually following ICCE. Here, the optic of the lens lies in front of the pupil, and the haptics are either placed at the chamber-angle (angle-supported, e.g. Kelman Multiflex lens) or are fixed with the iris (iris-supported, e.g. Worst-Singh's iris claw lens).
   ACIOL implantation is practically discarded now by all surgeons as a primary procedure owing to its own complications, like:
   - Corneal decompensation leading to pseudophakic bullous keratopathy.
   - Uveitis glaucoma hyphema syndrome (a triad of uveitis, glaucoma and hyphema).
   - Iris tuck and pupillary capture, which may cause chronic irritable eye.
   - Irisfixation lens has more complications.

   It is now popularly used for secondary implantation with encouraging results, the other eye is being treated primarily with ECCE with PCIOL (Phaco or SICS).
2. ***Posterior chamber IOL (PCIOL):*** They are placed in the posterior chamber following ECCE.
   The implants are intended for placement either in the ***capsular bag*** or on the ***ciliary sulcus*** (a potential space between the posterior iris-root and ciliary processes). It is more physiological to place the IOL in-the-bag.
   A scleral fixation or iris retrofixation PCIOL may be more useful after ICCE or after complicated ECCE where the posterior capsular support is absent.
   Types of PCIOLs
   - ***Single-piece*** (both optic and haptics are in one-piece) or multipiece or three-piece (optic and two pieces of haptic) (Fig. 25.21).
   - ***Uniplaner*** (no angulation between the optic and haptics) or angular (angulation between the haptics and the plane of optic approximately 50–100).



Figs 25.20A to C: Anterior chamber intraocular lens. **A and B.** Kelman Multiflex—angle supported type; **C.** Worst-Singh iris-claw lens

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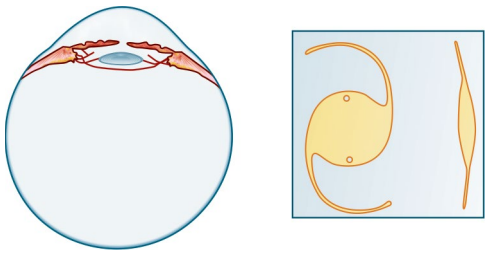


Fig. 25.21: Posterior chamber IOL (single-piece)

- With or without dialing holes.
- J-looped, C-looped, modified J-looped, cap-C haptic, plate haptic, etc.
- Rigid (conventional), disc IOL (without haptics) or foldable IOL (made of soft-materials).

Though technically difficult, PCIOL implantation gives the best visual results with relatively a fewer complications, like.

- Decentration of the lens.
- Sunset syndrome, windshield wiper syndrome
- Posterior dislocation of the IOL.
- Corneal decompensation or edema, or pseudophakic bullous keratopathy (relatively less).
- Posterior capsular opacification (PCO) or after cataract, etc.

Parts (Fig. 25.22)

An IOL has two parts—optic and haptics.

Optic

It is made of PMMA, Silicone, or acrylic materials and it is about 5.5 to 6.5 mm in diameter. Optic may be planoconvex or biconvex. There may be 2–4 minute holes at the periphery of the optic, called ***dialing holes*** (some optics do not have holes). Power of the optic varies from –5.0 to +40D.

Types of IOL

- ***Optic materials***

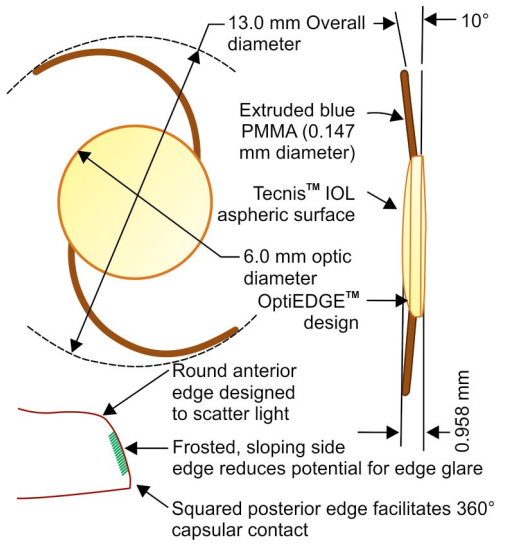


Fig. 25.22: Parts of an intraocular lens

  - ***Rigid IOL–PMMA material:*** This is time-tested over 65 years. Refractive index –1.49.
  - ***Foldable IOL***
    - Acrylic material
      i. Hydrophobic acrylic refractive index –1.55.
      ii. Hydrophilic acrylic refractive index –1.46 (Fig 25.23).
    - Silicone material refractive index –1.43.
    - Hydrogel (poly HEMA) material refractive index –1.47.
- ***Spherical or aspherical:*** Aspheric IOL has better contrast acuity.
- ***Monofocal or multifocals (Fig. 25.24):*** Multifocal IOLs may be refractive or diffractive types—provide for simultaneous viewing of both distance, intermediate and near vision.
- ***Accommodative IOLs:*** They allow for both distance vision and midrange near vision.
- ***UV-filtering; violet shielding and blue-blockers IOLs:*** Specific for light protection like human crystalline lens.
- ***Toric IOLs:*** To correct preexisting corneal astigmatism.

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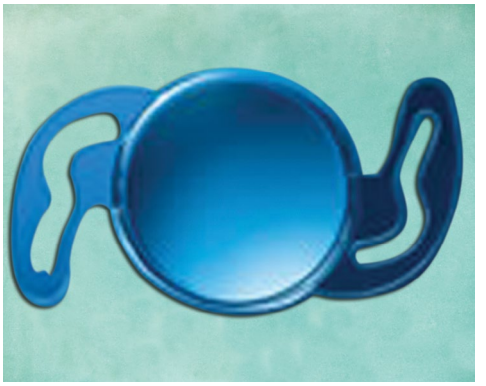


Fig. 25.23: Hydrophilic foldable intraocular lens

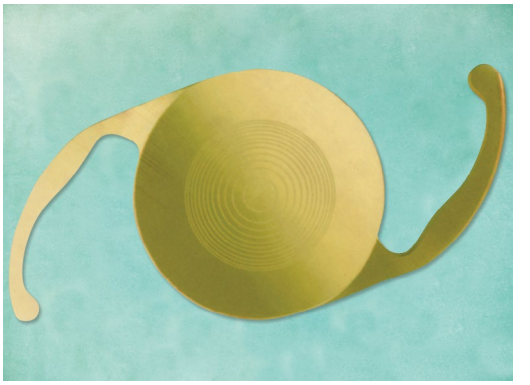


Fig. 25.24: Multifocal foldable intraocular lens

- ***Toric multifocal IOLs:*** To correct pre-existing astigmatism and to provide distance and near vision simultaneously.
- ***Square edge IOL:*** A square edge IOL is always preferable than round edge optic because of low incidence of PCO formation.
- ***Phakic IOLs:*** Mainly used to correct very high myopia in presence of crystalline lens:
  - ***Anterior chamber type:*** Either angle–fixated or iris-fixated.
  - ***Posterior chamber type:*** In the ciliary sulcus just in front of natural crystalline lens.

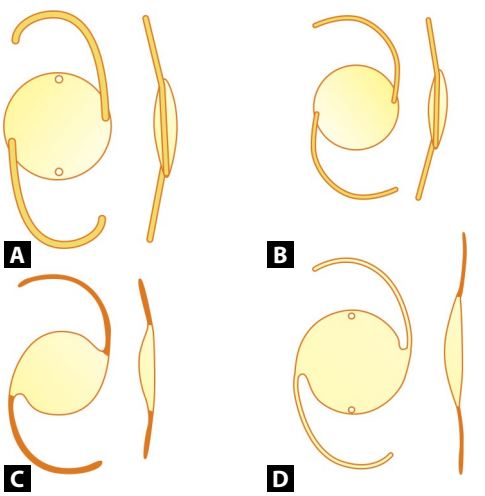
Haptics

They are made of PMMA, prolene or polypropylene or same material as IOL optic. They may be transparent (colorless) or blue in color (for better identification). The shape of the haptic varies, e.g. J-looped, modified J-looped, C-looped or S-shaped-loop (Kelman Multiflex ACIOL), etc. In multi-piece type, the haptics are fixed with optic by biological glue (Figs 25.25A to D).

The main function of the haptics is to anchor the IOL in position.

Other Specifications of Intraocular Lens

- The overall diameter = 12.0–13.5 mm



Figs 25.25A to D: Different types of PCIOL. **A.** Multi-piece, biconvex, modified 'J' loop, **B.** Multi-piece, biconvex, short 'C' loop (without hole), **C.** Single-piece, biconvex, small optic (phacolens); **D.** Single-piece, biconvex, 'C' loop

- Average weight = 20 mg in air and 3.0–3.2 mg in aqueous.
- Thickness of the optic = 1.2–2.0 mm.
- Sterilization = Presterilized by ethylene oxide. A thorough wash with BSS or Ringer's lactate is necessary before insertion of IOL into the eye.
- 'A' constant = is fixed for a particular type of IOL, like, 118.0, 118.4, 118.7, etc for PCIOL and 115.0 for ACIOL.

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Overall Complications of Cataract Surgery

Intraoperative

- ***Anesthesia-related***
  - Retrobulbar hemorrhage (RBH)
  - Injury to the optic nerve
  - Globe perforation
  - Lignocaine shock, acute anaphylaxis, and convulsion following injection into the optic nerve or its sheaths.
- ***Surgery-related (on table)***
  - Subconjunctival hematoma
  - Poor wound construction
  - Runaway rhexis
  - Iridodialysis
  - Hyphema
  - Positive vitreous pressure (upthrust)
  - Zonular dialysis
  - Subluxation or posterior dislocation of the lens
  - Posterior capsular tear (rent)
  - Vitreous prolapse (loss)
  - Nucleus drop or part of the lens drop
  - Descemet's tear and detachment
  - Corneal endothelial damage
  - Expulsive hemorrhage.

Early postoperative (within 1 week)

- Striate keratopathy (SK).
- Shallow anterior chamber due to—
  - Wound leak
  - Pupillary block
  - Choroidal detachment
  - Malignant glaucoma.
- Hyphema
- Iris prolapse
- Postoperative uveitis
- Retained lens matter (cortex or nuclear fragment).
- Postoperative secondary glaucoma
- Hypotony
- Bacterial endophthalmitis
- Toxic anterior segment syndrome (TASS).

Late postoperative (weeks to months)

- Posterior capsular opacification or after cataract
- Cystoid macular edema or lrvine-Gass syndrome
- Epithelial downgrowth
- Fibrous ingrowth
- Filtering bleb formation (cystoid cicatrix)
- Unpredictable astigmatism
- Persistent chronic uveitis
- Intraocular lens related complications
- Retinal detachment
- Corneal edema
- Bullous keratopathy (pseudophakic or aphakic).
- Late postoperative endophthalmitis.

Complications of Cataract Surgery: Specific to Type of Surgery

Conventional ECCE/PCIOL

- Complications of peribulbar or retrobulbar block (PBB/RBB)
- Positive vitreous upthrust
- Expulsive hemorrhage
- Suture-related problems
- High astigmatism
- Improper cortical cleaning.

Manual SICS

- Complications of PBB/RBB
- With sclero-corneal tunnel preparation:
  - Button-holing
  - Premature entry
  - Bleeding within the tunnel.
- Zonular dialysis during prolapsing the nucleus into AC.
- Inferior iridodialysis during nucleus delivery.
- Hyphema.

Phacoemulsification

- Posterior capsule blow-out during forcible hydrodissection in case of smaller rhexis.
- Wound burn especially with hard cataract.
- Runaway rhexis
- Iris chafing/iridodialysis
- Intermittent floppy iris syndrome (IFIS) with its problem. It is specially happens with patient taking oral alpha-1 adrenergic

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receptor antagonists (e.g. tamsulosin) for prostaterelated urinary problem.
- Zonular dehiscence
- More chances of corneal endothelial damage especially in hard cataract and with shallow AC.

Vitreous loss (prolapse): It may occur during ICCE and following accidental posterior capsular tear or zonular dehiscence during phaco or manual SICS. It is a serious complication because, it is associated with the following unpleasant sequelae:
- Updrawn pupil (hammock-shaped)
- High astigmatism
- Iris irregularities
- Uveitis
- Cystoid macular edema
- Retinal detachment
- Vitreous-touch syndrome which may lead to corneal decompensation
- Vitreous Wick syndrome which may lead to delayed endophthalmitis
- Increased risk of postoperative endophthalmitis.

Intraoperative signs of imminent vitreous loss are—creasing of the cornea along the chord of the wound, gaping of the incision, bulging of the iris diaphragm and deepening of the anterior chamber as vitreous passes into it.

Prevention: Good akinesia of the globe, bulbar massage, release of superior rectus stitch after limbal section, preoperative intravenous mannitol to shrink the vitreous, careful nucleus delivery/fragmentation and cortical cleaning during surgery.

Management: By sponge vitrectomy or by triamcinolone–assisted automated anterior vitrectomy. If the capsulorhexis margin is well preserved a sulcus-fixated PCIOL is given; otherwise a Kelman type ACIOL is implanted. A peripheral iridectomy (PI) is mandatory in case of ACIOL. Sometimes, a secondary scleral fixation IOL may be implanted later on when the eye is quiet.

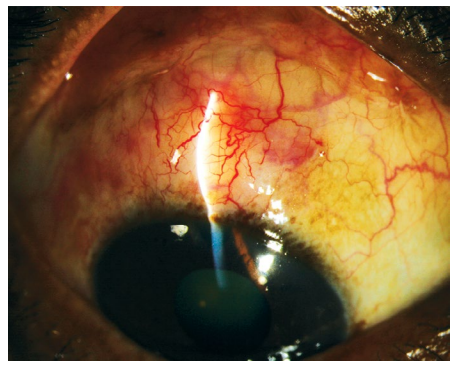


Fig. 25.26: Functioning filtering bleb with PI

GLAUCOMA OPERATIONS

Trabeculectomy

It is a ***partial thickness*** filtering operation, where the fistula is guarded by a superficial scleral flap (Fig. 25.26).

Indications

- Primary open-angle glaucoma, not controlled medically
- Chronic angle-closure glaucoma
- Secondary glaucomas
- Developmental or congenital glaucoma.

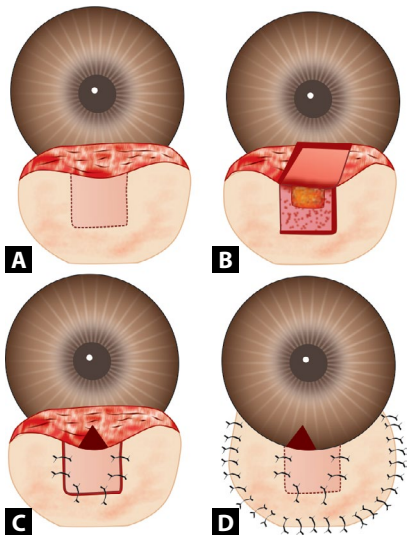
Possible Mechanisms of Action

- Filtration around the margins of the scleral flap.
- Filtration through the connective tissue substance of the scleral flap.
- Filtration through outlet channels of the scleral flap.
- Cyclodialysis, if the tissue is dissected posterior to the scleral spur, leading to increased uveoscleral outflow.
- Aqueous flow through the cut ends of the Schlemm's canal (rare).

Operative Steps (Figs 25.27A to D)

- A limbal-based conjunctival flap is made by giving an incision 10 mm posterior to the limbus.

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Figs 25.27A to D: Steps of trabeculectomy. **A.** Limbal-based conjunctival flap with marking on sclera; **B.** Scleral-flap dissection with removal of deeper sclera along with trabecular tissue; **C.** Peripheral iridectomy with suturing of scleral flap; **D.** Suturing the conjunctival flap

- An area, triangular (5 mm × 5 mm × 5 mm), or square (4 mm × 4 mm), based at limbus, is outlined on the sclera with a wet–field cautery.
- A partial thickness (half to two-third) superficial scleral flap is dissected, along the cautery mark, and hinged at the limbus.
- A deep rectangular block of sclera (1.5 mm × 3 mm) is excised anterior to the scleral spur, along with trabecular tissue.
- A peripheral iridectomy is performed.
- The superficial scleral flap is repositioned and sutured with 10–0 nylon.
- The conjunctival flap is reposed and sutured meticulously.

Scheie's Thermosclerostomy

It is a free filtering operation, where the fistula is made through the entire thickness of the sclera.

Indications: Same as trabeculectomy operation except, in developmental or congenital glaucoma.

Operative Steps

- A limbal-based conjunctival flap is made.
- A partial thickness groove, about 4 mm long, is created behind the corneolimbal junction.
- The posterior lip of the groove is cauterized.
- The incision is extended into the anterior chamber, and cautery is applied to the depth of the wound, while a dry field is maintained with a sponge.
- A peripheral iridectomy is performed.
- The conjunctiva is sutured.

Iridencleisis: A wedge of the iris is incarcerted into the limbal incision with an effort to maintain a patent channel for aqueous outflow. Endothelial cells of the iris proliferate and line the wound, and the iris stroma atrophies to act like a filtering wick. This was once a popular procedure, but is obsolete now, due to the associated higher incidence of sympathetic ophthalmitis.

Complications of Glaucoma Surgery

Early complications

- Changes in refraction
- A shallow or flat anterior chamber
- Hyphema
- Endophthalmitis
- Total loss of visual field
- Malignant glaucoma.

Late complications

- Non-functioning filtering bleb.
- Bleb infection (blebitis) which may lead to endophthalmitis.
- Cataract formation.

Full–thickness filtering procedure is more complicated, by excessive aqueous filtration leading to a flat anterior chamber associated with corneal decompensation and synechiae

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formation. In addition, the filtering blebs often become very thin and may rupture creating the danger of endophthalmitis.

Minimally Invasive Glaucoma Surgery (MIGS)

- Trabeculectomy is regarded as the gold standard in glaucoma surgery; however, there are less invasive procedures called minimally invasive glaucoma surgery (MIGS) have gained popularity in recent years among patients and surgeons.
- Minimally invasive gynecologic surgery (MIGS) the new procedures and devices are relatively safe and can easily be combined with cataract surgery.
- ***Main advantage:*** They are non penetrating and/or bleb-independent procedures with shorter surgery time. Major complications of fistulating surgery related to blebs and hypotony are also minimum.
  Minimally invasive gynecologic surgery (MIGS) procedures do not also preclude the possibility more conventional procedures, like, trabeculectomy or tube/shunt surgery.
- ***Main disadvantage:*** They are expensive may not be suitable in lowering IOP for moderate to severe glaucoma.

MIGS may be of ab interno and ab externo types:

Surgical approach

- ***Ab interno:*** Done under gonioscopic view usually through a small side port. By removing tissue or by implanting a shunt device.
- ***Ab externo:*** By using external approach to reach surgical site, like suprachoroidal space, either to remove/modify tissue or to implant device.

Internal filtration

- Trabeculotomy (excimer laser)
- Trabecular microbypass
- Suprachoroidal stent
- Intracanalicular scaffold
- Canaloplasty (canal expander)
- Suprachoroidal gold micro shunt.

External filtration: Subconjunctival implant.

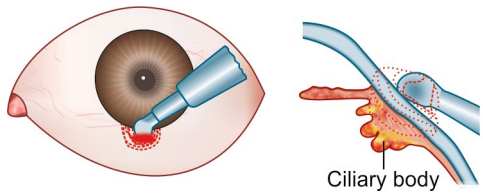


Fig. 25.28: Cyclocryotherapy

CYCLOCRYOTHERAPY (FIG. 25.28)

This is most commonly practised cyclo–destructive procedure, which involves freezing of the ciliary body, to reduce the rate of aqueous production.

Indications

- Painful absolute glaucoma
- Aphakic glaucoma
- Post-keratoplasty secondary glaucoma
- Neovascular glaucoma (may be needed along with panretinal photocoagulation).

Mechanism

Cyclocryotherapy destroys the ability of the ciliary body to produce aqueous humor.

This is by destruction of the ciliary epithelial cells, due to intracellular ice crystal formation and ischemic necrosis.

In addition, it provides relief of pain by destruction of the corneal nerves.

Steps

- Surface and retrobulbar anesthesia.
- The nearest edge of the cryoprobe (glaucoma probe) is placed 2.5 mm from the limbus.

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- Cryoprobe is applied for 60 seconds, at a temperature of –60° to –80°C. A rapid freeze, and slow unassisted thaw is desirable, as it produces maximum cell death.
- In each treatment session, 6 (six) cryo-applications are recommended over 180° of the globe.
- Subconjunctival injection of dexamethasone and atropine are preferably given.
- Two or more sessions may be required for a few patients.

Intense postoperative pain (for first 24 hours), is treated with strong analgesics. In additions, topical corticosteroids and cycloplegics are to be used from the day of surgery.

Complications: Excessive treatment may cause uveitis, hyphema, vitreous opacities, iris atrophy, pupillary distortion and hypotony (phthisis bulbi).

CYCLOPHOTOCOAGULATION

Cyclophotocoagulation (CPC) is also another way to perform cyclodestruction. It can be performed using different laser wavelengths. Previously, Nd:YAG laser was used for CPC.

But, presently, diode laser (810 nm wavelength) is preferred to perform CPC over other wavelengths since the melanin in the ciliary epithelium better absorbs this wavelength than others and therefore causes more targeted destruction with less inflammation.

Two main types of CPC: Transscleral CPC (TS-CPC) or endocylcophotocoagulation (ECP). TS-CPC requires LA (retrobulbar or peribulbar) and can often be performed in OPD setting. ECP requires local (topical with intracameral, retrobulbar/peribulbar block) or general anesthesia and is performed in operation theatre setting.

Both are performed for similar glaucoma conditions. In general, TS-CPC and ECP are indicated for refractory glaucoma, or eyes with poor visual acuity and/or visual potential.

Indications for TS-CPC or ECP:

- Elevated IOP with poor vision and/or visual potential.
- Pain relief due to very high IOP in a painful blind eye.
- Poor candidates for glaucoma filtration surgery or glaucoma drainage devices.
- Pseudophakic/aphakic glaucoma.
- Intractable pseudoexfoliation glaucoma.
- Glaucoma after penetrating keratoplasty or other corneal transplant surgery.
- Neovascular glaucoma.
- Refractory cases of pediatric glaucoma.
- Uveitic glaucoma or silicone oil induced glaucoma.
- Uncontrolled glaucoma in the presence of conjunctival scarring from previous surgery.
- Patient's medical condition preclude going to operating room.

Complications: Conjunctival burn in TS-CPC; chronic hypotony in both procedures; retreatment is required in some cases.

OPERATIONS UPON THE IRIS

Iridectomy

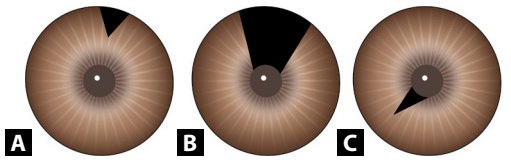
It consists of abscission of a portion of the iris, usually done in the upper part where it is covered by the upper eyelid.

There will be diplopia (in case of peripheral iridectomy) or dazzling of light (in case of complete iridectomy), if the iridectomy is done in the lower part, as the lower part of the iris is not covered by the lower lid.

Types of iridectomy with indications (Figs 25.29A to C):

- ***Peripheral button-hole iridectomy:*** It is usually done at 11 to 1 o'clock meridian.
  Indications
  - Angle-closure glaucoma.
  - As a part of trabeculectomy operation.
  - In intracapsular cataract extraction.
  - In some cases of extracapsular cataract extraction.
  - In penetrating keratoplasty operation.

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Figs 25.29A to C: Different types of iridectomy. **A.** Peripheral button–hole; **B.** Sector (complete); **C.** Optical

A peripheral iridectomy is done to prevent postoperative pupillary block glaucoma. As the aqueous can flow freely into the anterior chamber via this bypass channel. An equilibrium is thus maintained between the anterior and posterior chamber, following operations.

Steps

- Eyeball is rotated downwards with superior rectus stitch.
- A small conjunctival flap is made from 11 to 1 o'clock meridian.
- A small ***ab externo*** incision is given to open the anterior chamber.
- Slight pressure is given on the posterior lip of the wound, which causes prolapse of a knuckle of iris.
- A portion of the iris near its base is abscised, leaving the sphincter intact. Care should be taken to retain the anterior chamber during this maneuvre.
- A stitch may be required to close the wound, and then conjunctiva is repositioned.

- ***Board (complete) iridectomy:*** To facilitate the extraction of the lens when the pupil is small and rigid, or there is extensive synechiae (combined extraction of cataract). A sector of iris is abscised from the pupillary margin to the base of the iris.
- ***Optical iridectomy:*** It is rarely done now-a-days.

  Indications
  - In case of small central corneal opacity where keratoplasty is not possible.
  - In case of axial congenital cataract, if the child is mentally retarded.

  It is done in the lower pupillary margin of the iris, as in the upper part of new opening will become covered by the upper lid. The best site for optical iridectomy is judged by stenopaeic slit test, and it should correspond to the clear zone at the lower half of the cornea.
- ***Iridectomy for prolapsed iris:*** After successful abscission of the prolapsed iris, the stumps of iris retract into the anterior chamber, and should be free from the wound.
- ***Iridectomy to remove foreign bodies or small cysts or tumors of the iris.***

Iridotomy

It consists of section of the iris without abscising any portion of it.

Indications

- ***To create an artificial pupil***, when the normal pupil is closed or severely updrawn.
- ***Laser iridotomy*** for primary angle-closure glaucoma or postoperative pupillary block glaucoma. It may be carried out with an Argon laser or a Nd:YAG laser.
- ***Four-point iridotomy (quadri-puncture):*** It is done to treat secondary glaucoma in ***iris bombe***. A von Graefe's cataract knife is useful for this purpose.
- ***Division of anterior synechiae***, is also a form of iridotomy.

OPERATIONS OF THE CORNEA

Paracentesis

Paracentesis means opening the anterior chamber for the purpose of aspiration its contents, partially or completely.

Indications

Diagnostic: Aqueous humor study for:

- Intraocular malignancy
  - Cytology
  - Lactate dehydrogenase enzyme assay.
- Intraocular parasite infestation

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- Polymerase chain reaction (PCR) diagnosis in infective uveitis.

Therapeutic

- Massive hyphema to control secondary glaucoma, and to prevent blood-staining of the cornea.
- Corneal ulcer with massive hypopyon to improve corneal nutrition.
- Impending corneal perforation.
- Central retinal artery occlusion.

Steps

- Eyeball is steadily fixed with forceps.
- An incision is made at the temporal limbus within 2 mm, with the help of a keratome, cataract knife, a paracentesis needle or a sideport knife or a 26 gauge needle.
- The knife or needle is inserted in the 4. Peripheral lip is slightly depressed so that the aqueous is drained out slowly with a minimum of disturbance.

Keratoplasty

It means surgical replacement of a scarred or diseased cornea with a viable donor cornea.

Classifications

Morphological

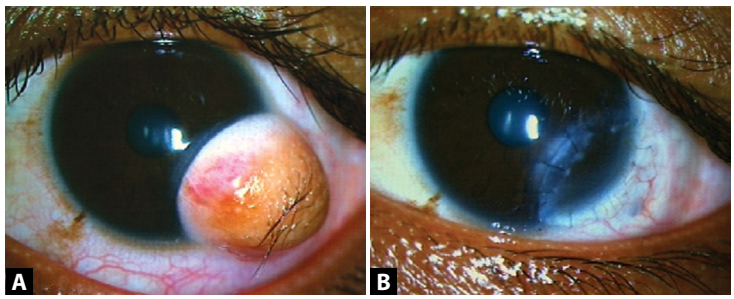
- Full thickness or penetrating keratoplasty (PK)
- Partial thickness or lamellar keratoplasty (LK) (Fig. 25.30)
  - Anterior lamellar keratoplasty (ALK) (Figs 25.31A and B)
  - Deep anterior lamellar keratoplasty (DALK) (Figs 25.32A to E)
  - Posterior lamellar keratoplasty (PLK)
    - Deep lamellar endothelial keratoplasty (DLEK)
    - Descemet stripping (automated) endothelial keratoplasty (DSEK/DSAEK)
    - Descemet membrane endothelial keratoplasty (DMEK)

According to Purpose (Table 25.1)

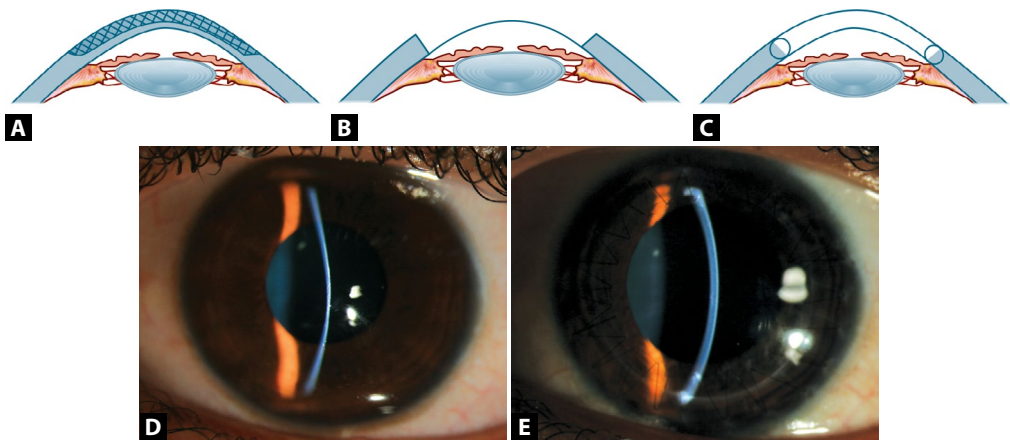
- ***Optical:*** Primary purpose is being the improvement of vision (e.g. leucoma, keratoconus, etc.).

Fig. 25.30: Level of dissection in recipient's eye in different types of keratoplasty; **ALK**—Anterior lamellar keratoplasty; **PK**—Penetrating keratoplasty; **DALK**—Deep anterior lamellar keratoplasty; **PLK**—Posterior lamellar keratoplasty; **DLEK**—Deep lamellar endothelial keratoplasty; **DS(A)EK**—Descemet stripping (automated) endothelial keratoplasty; **DMEK**—Descement membrane endothelial keratoplasty

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Figs 25.31A and B: Anterior lamellar keratoplasty in dermoid. **A.** Before operation; **B.** After operation



Figs 25.32A to E: Deep anterior lamellar keratoplasty (DALK). **A.** With anterior corneal disease; **B.** Removal of diseased part and only DM and endothelium remained; **C.** Replacement of donor cornea without DM and endothelium; **D.** In keratoconus (before surgery); **E.** In keratoconus (after surgery)—that shape and thickness like normal cornea

TABLE 25.1: Indications of keratoplasty

Indications	India Percentage	USA Percentage
Corneal scar (ulcer related)	28.1	7.9
Therapeutic penetrating keratoplasty	12.2	2.9
Pseudophakic corneal edema/bullous keratopathy	10.6	27.2
Non-Fuchs' dystrophy	7.2	9.3
Keratoconus	6.0	15.4
Fuchs' dystrophy	1.2	15.2
Failed graft	17.1	10.9
Others	17.7	2.7

- ***Tectonic:*** Restoration of altered corneal structure (e.g. thinning, perforation, etc.).
- ***Therapeutic:*** Tissue substitution for refractive corneal diseases (e.g. nonhealing corneal ulcer, pterygium, etc.).
- ***Cosmetic:*** Replacement, without the hope for visual improvement.

Frequently, several purposes are addressed when a keratoplasty is performed.

According to Donor Material Employed

- ***Homograft (allokeratoplasty):*** Donor material from the same species (human to human).

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- ***Autograft (autokeratoplasty):*** Donor material from the same individual.
- ***Isograft:*** Transplants between homozygous twin.
- ***Heterograft (xenograft):*** Graft obtained from another species.
- ***Keratoprosthesis:*** The grafted material is an optical cylinder made of PMMA and the carrier may be a donor cornea (Boston Kpro) or a canine tooth [osteo-odonto keratoprosthesis (OOKP)].

According to Type of Surgery

- ***Simple keratoplasty:*** Keratoplasty is performed alone.
- ***Combined keratoplasty:*** When combined with
  - Cataract extraction (ICCE/ECCE)
  - ECCE with PCIOL implantation (***triple procedure***)
  - Trabeculectomy
  - Vitrectomy.

Indications for Lamellar Keratoplasty

Indications

- ***Anterior lamellar keratoplasty***
  - ***Tectonic:*** For marginal thinning, e.g. Mooren's ulcer, Terrien's degeneration, etc.
  - ***Therapeutic:*** With pterygium surgery, limbal dermoids, etc.
- ***Deep anterior lamellar keratoplasty (DALK) (for optical purpose):*** Any anterior corneal disease/scar without involvement of Descemet membrane and endothelium.
  - Reis Buckler's dystrophy
  - Granular dystrophy
  - Lattice dystrophy
  - Keratoconus (most common)
  - Anterior corneal scar after an ulcer
  - Macular dystrophy (in some cases, endothelium is also involved and in that case PK is indicated).
- ***Posterior lamellar keratoplasty (DSEK/DSAEK, DMEK):*** Any disease affecting the Descemet and endothelium (endothelial dysfunctions) without any stromal scar (Figs 25.33A to E).
  - Pseudophakic corneal edema/bullous keratopathy (most common)
  - Fuch's dystrophy
  - Posterior polymorphous dystrophy
  - Post PK failed graft
  - Iridocorneal endothelial syndrome (Chandler's syndrome)
  - In some cases of congenital hereditary endothelial dystrophy.

Penetrating Keratoplasty (PK) (Figs 25.34 to 25.36)

Surgical technique

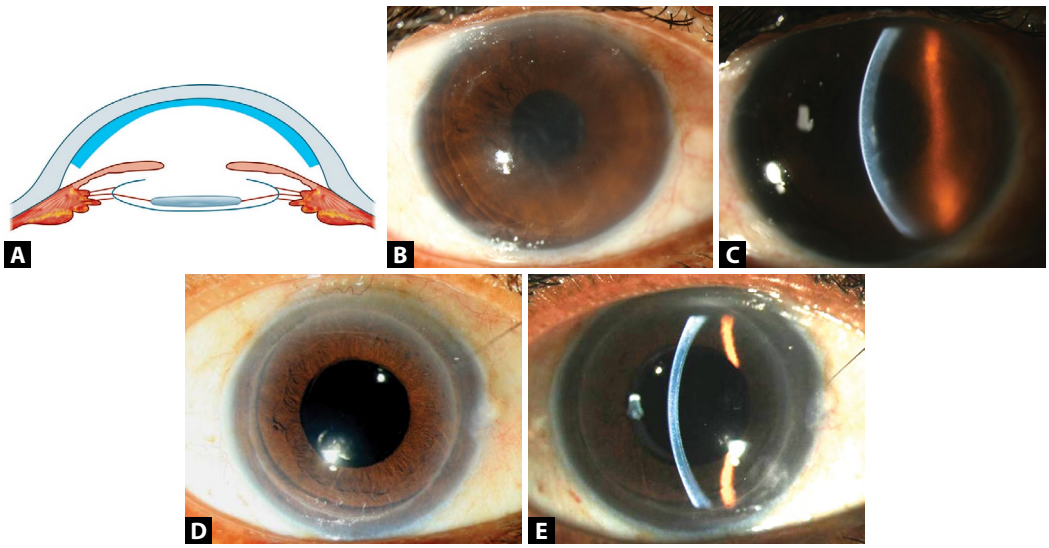
This technique aims:

- Minimal manipulation of the donor tissue.
- Protection of the intraocular structures of the recipient.
- Attaining a water-tight host-graft junction at the end.

Steps

- Preoperative miosis and hypotony
- Anesthesia and akinesia
- Exposure, globe fixation and scleral support (flieringa ring is used to prevent scleral collapse).
- ***Trephination of the donor cornea:*** It is better from the endothelial side, and should be slightly larger (0.25–0.50 mm) than the recipient corneal hole.
- ***Trephination of the recipient button:*** It is usually 7.0 to 7.5 mm diameter in size. Cutting of the recipient button is completed with fine scissors.
- Management of the iris, lens, vitreous, and IOL implantation in selected cases.
- Graft placement and maintenance of anterior chamber by viscoelastic agent.
- ***Suturing the corneal button:*** The first four sutures, placed at 12, 6, 3 and 9 o'clock meridians, are known as ***cardinal sutures***.

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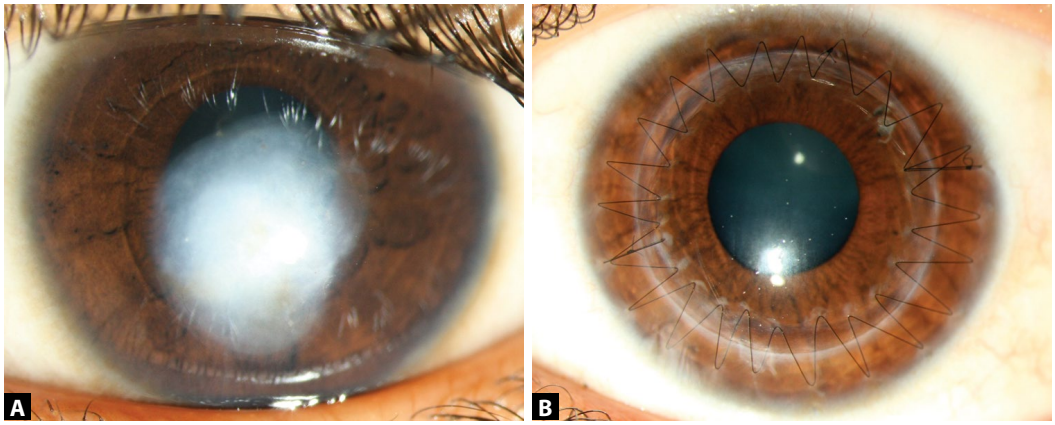
Figs 25.33A to E: **A.** Descemet stripping endothelial keratoplasty (DSEK). Here, recipient's diseased "DM and endothelium complex" is replaced with healthy donor tissue (endothelium, DM and thin layer of posterior stroma); **B.** Pseudophakic bullous keratopathy; **C.** Pseudophakic bullous keratopathy—severe corneal edema; **D and E.** Same eye after DSEK surgery in diffuse illumination and slit section

Fig. 25:34: Penetrating keratoplasty

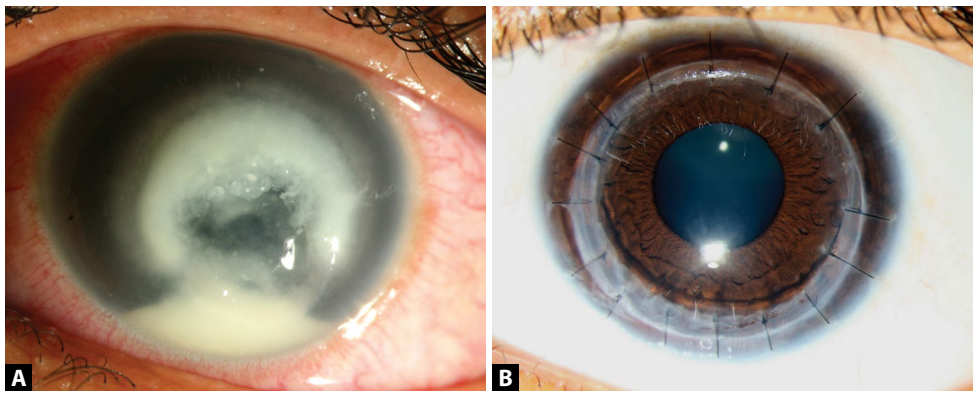
Subsequent sutures are given with 10–0 nylon (at 75–90% of depth) by continuous or interrupted fashion.

- Wound margin is checked for fluid leak and iris adherence.
- Anterior chamber is reformed.
- Subconjunctival injection of dexamethasone and gentamycin.
- Pad and bandage.

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Figs 25.35A and B: Penetrating keratoplasty. **A.** Before surgery; **B.** After surgery



Figs 25.36A and B: A. Corneal ulcer with impending perforation—before surgery; **B.** Same eye after therapeutic penetrating keratoplasty

Complications of Penetrating Keratoplasty

Early complications

- Primary graft failure
- Wound leak and iris prolapse
- Postoperative glaucoma
- Iridocyclitis
- Hyphema
- Wound infections
- Persistent epithelial defect.

Intermediate complications

- Graft rejection (immunological)
- Epithelial
- Subepithelial
- Stromal
- Endothelial (Khodadoust line)
- Graft infections
- Wound dehisence.

Late complications

- Recurrence of host disease (e.g. Herpes simplex keratitis, lattice dystrophy, Reis-Buckler's dystrophy, etc.)
- Postgraft membrane formation
- Late graft failure
- Cataract formation
- Transmission of donor disease to the host
- Post-keratoplasty astigmatism.

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EYE BANK AND DONOR CORNEA

Objectives of an Eye Bank

- Collection of donor eyes.
- Process and storage of donor cornea.
- Distribution and utilization of the highest quality of donor tissue for transplantation.
- Provide for soliciting eye donation from potential donors.
- Provide and process eye tissue for teaching or research, as needed.
- To promote public education relation system.
- To promote hospital cornea retrieval program (HCRP) to improve collection of donor eyes from hospital deaths.

Collection

Person pledges before death to donate his eyes (no living person can donate his one eye, because the law does not permit it. Similarly, eye tissue can not be sold or purchased). After getting information of death, and proper written consent from the next of kin, eye bank personnel collects the eyes. The eyeballs should be collected with 4–6 hours of death (traditional postmortem time).

It is important to know the ***age of the donor, time of death*** and ***cause of death*** before enucleation of the donor globe. Most eye banks accept eyes from donors between 6 months and 80 years of age.

Methods

There are two methods of procuring donor tissue for the eye banks:

- Voluntary donation (mostly without prior pledge)
- Hospital cornea retrieval program.

Voluntary Donation

It is the result of realization of one's social responsibility towards people with corneal blindness. However, in the moment of grief, this realization may not materialize to actual donation, because the near relatives may not be in a position to make such noble decisions.

Currently, the total donor cornea collection all over India is between 55,000 and 60,000/year. Maximum collections occur from Gujarat, Maharashtra, Tamilnadu and Andhra Pradesh. Among these, 75–80% of eye donation is from voluntary aged donors who die in their homes, hospitals, or nursing homes. Only 40–45% of the eyes from voluntary donation are utilized for transplantation.

The reasons are:

- Age of the donor
- Longer death-to-enucleation time
- Non availability of donor's complete medical history.

It is to address the non–availability of ***quality*** corneal tissue. The Eye Bank Association of India (EBAI) along with Government of India has come up with this new cornea collection strategy called HCRP. This program was initiated by Ramayamma International Eye Bank at LVP Eye Institute, Hyderabad in 1990.

Hospital Cornea Retrieval Program

Here, an ***eye donation counselor*** (EDC, or previously called ***grief counselor***) makes a timely and sensitive request to the near relatives of the bereaved in a hospital set up to make an eye donation. With several leaflets and close intimate education, he or she wins the heart of the relatives of the deceased.

If the consent is given, he organizes quick collection of donor tissue (whole eyeball or *in situ* corneoscleral rim) so that the family is not inconvenienced.

The overall conversion (request by EDC vs. actual donation) is between 5% and 35% in different hospitals. The donor cornea collection through HCRP is around 20–25% of total collection in last 2 years (i.e. approximately 15,000 cornea/year).

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Advantages of HCRP

- Access to younger and healthier tissues.
- Availability of donor medical history.
- Reduce death–to–enucleation time.
- Higher tissue utilization rate (65% or more).
- More scope of training and future research.
- Cost effective.

Choice of Hospitals

- Large multispeciality hospitals with 500 beds or more.
- Death rate = 3–4/day or >10/week.
- Should be close vicinity (within 5–15 Km) to the linked eye bank.
- Should have signed memorandum of understanding (MoU) between both.
- Help and active support is required from all level of the staffs of the hospital.

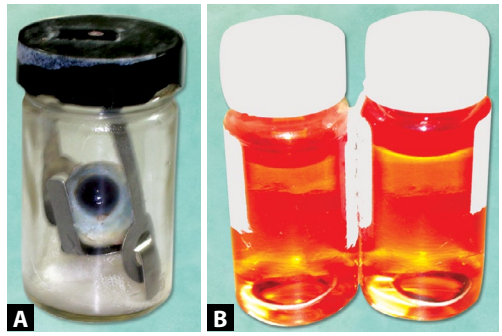
Pledged eyes can not be retrieved without the consent of the family members (next of kin). The near relatives of those who have not pledged their eye can also decide, at the time of death, to donate the eyes of the deceased on his or her behalf. However, at the time of death of near and dear one, in the midst of grief and confusion, even the best of intention may not lead to actual eye donation. HCRP (with the help of trained counselor) plays a key role in this situation.

Contraindications for donor eye collection:

- ***Medical:*** Acquired immunodeficiency syndrome (AIDS) or Humam immunodeficiency virus (HIV) seropositivity, rabies, active hepatitis B, hepatitis C, septicemia, death from unknown cause, creutzfeldt-jacob disease, leukemia, syphilis etc.
- ***Ophthalmic:*** Previous intraocular surgery, corneal pathology, retinoblastoma and malignant melanoma, iridocyclitis, endophthalmitis, etc.

Storage (preservation) of the donor eye:

- Short–term preservation (upto 96 hours)
  - ***Moist chamber method:*** Whole globe is preserved in a moist chamber at +4°C in a refrigerator for 24 hours.



Figs 25.37A and B: A. Corneal preservation—moist chamber method (whole globe); **B.** Sclero-corneal button preservation in McCarey-Kaufman medium

  - ***McCarey-Kaufman (M-K) medium (Fig. 25.37A):*** Composition: Tissue culture (TC)–199: 5%: Dextran-40; HEPES buffer to adjust pH at 7.4; Gentamycin 0–1 mg/ml; color-pink.
  - ***Corneoscleral button (Fig. 25.37B)*** is preserved in M-K medium at +4°C for upto 96 hours. It is superior than conventional moist-chamber method and practised widely.
- ***Intermediate-term preservation (upto 2 weeks):*** Optisol-GS, Cornisol, Eusol-C and Life-4C media.
- ***Long-term preservation (months to years).***
  - ***Viable:*** Organ-culture method, cryo-preservation.
  - ***Non-viable:*** Glycerine preservation.

Evaluation of the donor tissue:

- ***Gross examination*** with torch and loupe.
- ***Slit-lamp*** examination.
- ***Specular microscopy*** (for endothelial cell count and morphology).
- Vital staining (trypan blue dye).
- ***Microbial evaluation*** by culture.
- ***Serological screening*** (for HIV, hepatitis-B, hepatitis-C and syphilis).
- ***Human leukocyte antigen typing*** of the donor not done.

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TARSORRHAPHY

Aims of the Surgery

- To reduce the length of the palpebral aperture, when this is abnormal.
- To close the eye temporarily, to protect the cornea.

Types and Indications

- ***Permanent (lateral) (Figs 25.38A and B)***
  - Residual Bell's palsy
  - Abnormal length of the palpebral fissure
  - Mild degree of exophthalmos.
- ***Temporary (paramedian or lateral)***
  - Neuroparalytic keratitis
  - Moderate to severe exophthalmos
  - To assist healing of skin grafting
  - During reconstruction of a contracted socket
  - Small perforation of corneal ulcer
  - Impending perforation of corneal ulcer
  - In severe dry eye.

Steps: Apart from placement of sutures both the operations almost follow the same technique.

- Dissection of the mucous membrane from the margin of lower lid just posterior to the grey line in rectangular (5–6 mm long) fashion.
- The edge of the upper lid is similarly dissected at the corresponding position.
- A horizontal mattress suture is passed through the rubber beads and the skin, so that they come out at the posterior edge of the bare surface of both lids. Suture is then tied firmly.
- The lids will be firmly adherent within a few days (7–10 days) when the suture and rubber beads are removed.

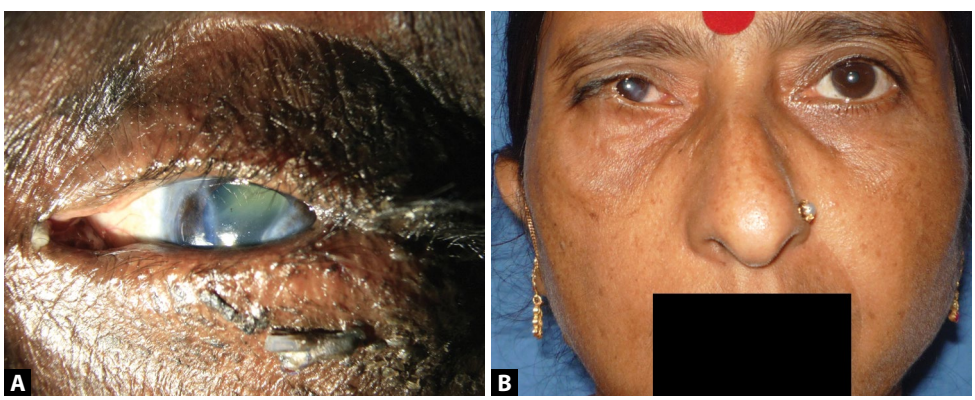
PTERYGIUM OPERATION

Anesthesia

- Surface anesthesia with 4% lignocaine.
- Infiltration anesthesia with 2% lignocaine with adrenaline into the body of the pterygium.
- Peribulbar anesthesia as cataract surgery.

Indications of Surgery

- Pterygium approaches towards the pupillary area.
- Chronic irritation due to fleshy pterygium.
- Causing much astigmatism.
- Purely cosmetic in rare situation.



Figs 25.38A and B: A. Temporary lateral tarsorrhaphy in perforated corneal ulcer; **B.** Permanent tarsorrhaphy in operated cerebellopontine angle tumor

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Bare Sclera (D' Ombrian's) Technique

- Eye speculum is inserted.
- The neck is held with a fixation forceps, and the head (apex) of the pterygium is shaved from the cornea with a no. 15 blade.
- Two diverging incisions are given along the upper and lower borders of the pterygium.
- The pterygium is lifted with a hook and rotated nasally, while the dissection is carried out subconjunctivally.
- The subepithelial thick triangular pathological tissue is removed in one piece.
- The conjunctiva over the head, neck and 2 mm of the body of the pterygium is excised to leave an area of exposed (bare) sclera.
- Conjunctiva is repositioned and sutured, the bleeding vessels of the bare area is cauterized.
- Pad and bandage.

Pterygium Operation with Limbal Conjunctival Autograft

Here, the pterygium is resected (Fig. 25.39A) first as bare sclera technique (i.e. upto step 6 as above). Then the steps are as follows:

- A rectangular piece of limbal conjunctiva is dissected from upper temporal area and taken out as graft tissue. This is usually from the same eye, but sometimes from the other eye (Fig. 25.39B).
- The tissue is placed over the bare area to cover it. Limbal side is matched carefully.
- Grafted tissue is sutured by 10–0 vicryl or 10–0 nylon. Donor site is also closed by the same suture.
  Now-a-days, graft fixation is preferably done with biological tissue (fibrin) glue. It is faster and easier. The postoperative reaction is minimal with quicker rehabilitation. The recurrence rate is also much lower.
- If required, the sutures may be removed after 3 weeks.

CHALAZION OPERATION

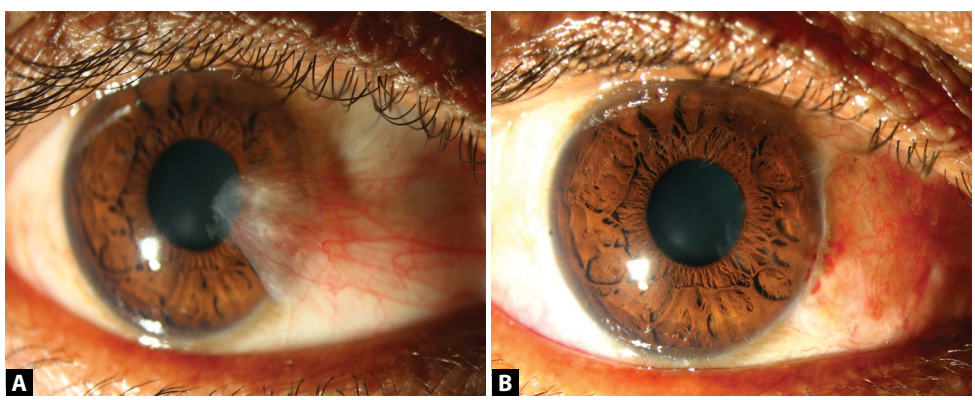
Most chalazion require incision and curettage, for very rarely does spontaneous resolution occur (Fig. 25.40).

Anesthesia

- Surface anesthesia by 4% lignocaine.
- Infiltration by 2% lignocaine with adrenaline into the skin around the chalazion.

Operative Steps

- ***Chalazion is fixed*** with chalazion clamp or forceps, the ***plate*** of which is passed



Figs 25.39A and B: **A.** Pterygium before surgery; **B.** Pterygium resection with conjunctival autograft with fibrin glue

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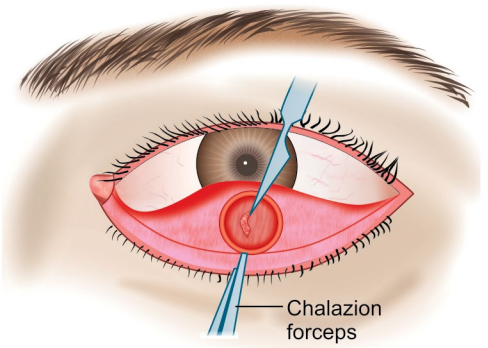


Fig. 25.40: Chalazion operation (vertical incision)

over the skin, and the ***ring*** is placed on the palpebral conjunctiva with the chalazion in its centre.
- Chalazion clamp is closed by tightening the screw, and the ***lid is everted***.
- A ***vertical incision*** is made through the tarsal conjunctiva with a sharp knife.
- Cheesy granulomatous tissue is thoroughly scooped out with the help of a chalazion scoop.
- The walls of the cavity is scraped carefully with the same scoop.
- Fibrous tissue wall may be excised entirely, if it is thick.
- Clamp is removed, and pressure is applied for 2–3 minutes to secure hemostasis.
- Pad and bandage for 4–6 hours.

DACRYOCYSTECTOMY

Anesthesia (Local)

- Two drops of 4% lignocaine into the conjunctival sac.
- ***Infiltration:*** Anesthetic agents are injected at the following sites:
  - At the junction of the inferior orbital margin with the beginning of the anterior lacrimal crest—0.5 mL solution is injected, and then along the line incision to a point 3 mm above the medial palpebral ligament.
  - Second injection is made at the above point and needle is directed posteriorly for about 8 mm, and the tissues around the fundus are injected with about 0.5 mL. The needle is then carried out further downwards to the upper half of posterior lacrimal crest, and injected with 0.5 mL solution.
- Nasal mucosa of the inferior and middle meatus is anesthetized with 4% lignocaine and adrenaline (with spray or nasal pack).

Operative Steps

- A slightly curved incision with its concavity towards the inner canthus is made (care is taken not to injure the angular vein). The incision is about 2.0 cm, and one-third of its lies above the medial palpebral ligament.
- Separation of skin and orbicularis oculi along the line of incision with lacrimal dissector, and then retracted with Muller's retractor.
- Medial palpebral ligament is then exposed, and disinserted form the anterior lacrimal crest by a rougine.
- Lacrimal sac is dissected from the floor, at the fundus, and from its connection with the canaliculi.
- After the sac is well dissected, it is grasped with a straight artery forceps upto its lower end, and twisted until it is torn off from nasolacrimal duct.
- The torn end of nasolacrimal duct is curetted and then cauterized with iodine solution.
- Skin is closed by interrupted or continuous, 5–0 or 6–0 silk.
- Pad and pressure bandage are applied for 24–48 hours. Stitches are removed after 6–7 days.

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DACRYOCYSTORHINOSTOMY (FIGS 25.41A TO F)

Anesthesia

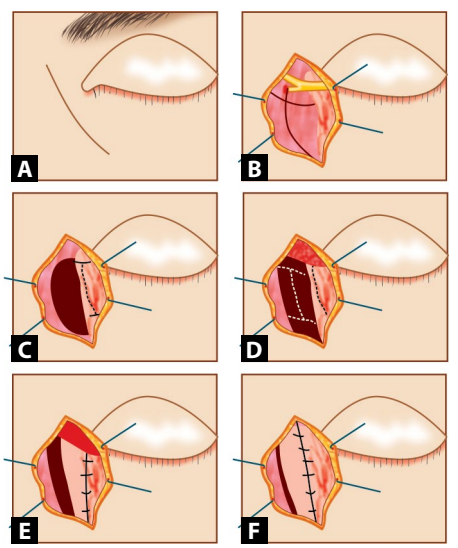
- ***In adult:*** Same as dacryocystectomy. It is better with anesthetic mixture.
- ***In children:*** General anesthesia.

Operative Steps

Nasal pack: In DCR, before starting the operation a nasal pack is given on the same side with a roller gauze, soaked with 4% lignocaine, adrenaline and hemostatic agent. This is for hemostasis during operation, and to prevent postoperative bleeding.

1–3. Same as dacryocystectomy.

4. Lacrimal sac is separated from the medical wall and floor only. Lacrimal fossa is now exposed.
5. A 10 mm bony ostium is made by cutting the lacrimal bone, part of the adjacent nasal bone, and frontal process of the maxilla. Nasal mucous membrane of the middle meatus is thus exposed.
6. Two horizontal H-shaped incisions are given on the nasal mucosa, and the medial wall of the lacrimal sac; and thereby, two anterior flaps and two posterior flaps are created.
7. First posterior, and then the anterior flaps are sutured with 6–0 or 8–0 chromic catgut or vicryl. Sometime, it is difficult to suture both the flaps. In that case, only two anterior flaps are sutured together, while the posterior flaps are excised.
8. Skin is sutured by 5–0 or 6–0 silk.
9. Nasal pack is removed after 48–72 hours.



Figs 25.41A to F: Dacryocystorhinostomy. **A.** Incision; **B.** Dissection of the lacrimal sac; **C.** Nasal mucosa being exposed after making bony ostium; **D.** H-shaped incisions on the lacrimal sac and nasal mucosa; **E.** Suturing the posterior flaps; **F.** Suturing the anterior flaps.

EVISCERATION (FIG. 25.42)

It is a destructive surgical procedure, in which intraocular contents are removed along with inner two coats, retaining the sclera and optic nerve.

Indications

- Panophthalmitis
- Very rarely expulsive hemorrhage to assist in removing the contents, if the process is incomplete.

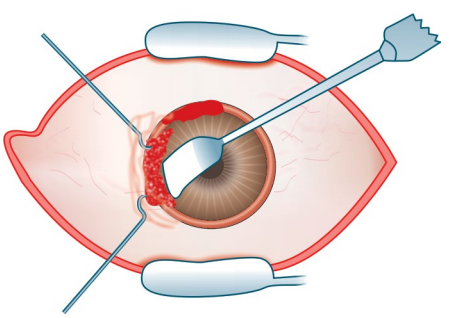


Fig. 25.42: Evisceration. Note the introduction of evisceration scoop between the sclera and uveal tract

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Special consent: An informed written consent has to be taken prior to surgery.

Anesthesia

- General anesthesia is preferable, as the procedure is painful and psychologically traumatic.
- Local anesthesia when GA is contraindicated)
  - Surface by 4% lignocaine.
  - Infiltration by 2% lignocaine with adrenaline at 4 quadrants in retrobulbar space.

Steps of Operation

- Universal eye speculum is applied.
- Conjunctiva is dissected 3600 around the limbus.
- Whole cornea is removed with a knife and scissors.
- After fixing the sclera, the evisceration scoop is introduced between the sclera and the uveal tract, and swept circumferentially 3600 to separate the uveal tissues from the sclera and optic nerve.
- Intraocular contents are scooped out thoroughly, precaution being taken to remove all the uveal tissues, specially around the vortex veins and optic nerve.
- Bleeding is stopped by applying pressure with warm saline-soaked gauze.
- Double pad and pressure bandage is applied for 48 hours.

Variations of Evisceration

- ***Frill excision:*** If the entire sclera is left out, there is considerable reaction, and delayed wound healing. These disadvantages may be avoided, by cutting the insertion of the recti muscles and excising a greater part of the sclera, leaving only a small collar of sclera around the optic nerve, so as to leave the nerve-sheath unopened.
- ***Insertion of an acrylic ball implant***
  - A small acrylic or silicone ball (about 14–16 mm in diameter) is placed within the scleral cup (pocket) after proper hemostasis.
  - Sclera is sutured over the implant.
  - Tenon's capsule and the conjunctiva are sutured separately.
  - Artificial eye (cosmetic shell) is given after 4–6 weeks.
  - Cosmetic result after this implant is very satisfactory.

ENUCLEATION

Enucleation is the surgical removal of the eyeball with a portion of the optic nerve from the orbit.

Indications

- ***Absolute indications:*** When there is risk of life or risk to the other eye of the patient
  - Retinoblastoma in children
  - Malignant melanoma in adults
  - Severely traumatized eye with no PL to prevent sympathetic ophthalmitis of contraindication.
- ***Panophthalmitis:*** Because the infection can spread via the cut ends of optic nerve sheath, causing meningitis.
- ***Special consent:*** An informed written consent has to be taken prior to surgery.
- ***Anesthesia:*** Same as evisceration.

Steps of Operation

- Universal eye speculum is applied.
- Conjunctiva is dissected all around the limbus, and Tenon's capsule is separated from the sclera upto the insertion of the extraocular muscles.
- Each rectus muscle is hooked with a squint hook and cut with scissors, in the order of superior, lateral, inferior and medial rectus muscle (SLIM). For insertion of the implant, transfix each muscle with 5–0 chromic catgut suture.

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- Speculum is depressed, and the eyeball is made to be luxated out.
- Enucleation scissors are introduced with closed-tip along the lateral side of the globe. Scissors are opened, and optic nerve is severed.

In case of malignant intraocular tumor, it is essential to ***cut the optic nerve at least 10 mm*** behind the globe, near the optic foramen.

To do this, the eyeball is rotated laterally by a traction suture in the medial rectus tendon, and the optic nerve is tensioned. Enucleation spoon with optic nerve guide is introduced from the lateral side and optic nerve is then transected with one bold cut with a pair of ***straight enucleation scissors*** from the medial side of the globe.

- Oblique muscles are severed lastly with the same scissors.
- Bleeding is stopped by applying pressure within the socket.
- Tenon's capsule and conjunctiva are sutured separately.
- Pressure bandage is applied for 48 hours.

Enucleation with Implants

Implants are inserted within the Tenon's capsule, and the aims are:

- To provide a mobile base for the prosthesis to pivot upon.
- To prevent bony deformity of the orbital wall (i.e. contracted socket) in children.
- To maintain orbital socket in adults.

Types of Implants

- ***Buried implants***
  - ***Non-integrated:*** It is an acrylic sphere, which is inserted within the Tenon's capsule to maintain shape of the eyeball. The recti muscles are tied in a cruciate manner over the implant.
  - ***Semi-integrated:*** It has holes or tunnels in which the four recti muscles are tied separately. It moves with the movement of the opposite eye.
- ***Semiburied implants (integrated):*** It is partially buried and partially expose implant. In its exposed part, the posterior surface of the prosthesis could be exactly fitted. Though it is capable of affording the best movement of the prosthesis, in the majority of the cases, there is infection and extrusion of the implants. It is not used now-a-days.
- ***Hydroxyapatite implant:*** It is from natural source ***coral***. It is a costly type of intregrated implant in which an artificial eye is pegged. It gives the best cosmetic appearance with excellent movements of an artificial eye.

A ***prosthesis (or an artificial eye)*** may be fitted in the 3rd or 4th week after the operation. The person who is responsible for making and fitting of an artificial eye, or prosthesis is called an ***ocularist***.

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26 CHAPTER

Blindness and its Prevention

BLINDNESS

"Too blind to perform work for which eyesight is essential." *—Parsons*

The term ***blindness*** means inability to perceive light. There are at least 65 definitions of blindness throughout the world and they differ widely.

Uniform Definition of Blindness (WHO)

"Visual acuity in the better eye is less than 3/60 (Snellen's) or its equivalent" or ***in the absence of visual acuity chart***.

"Inability to count fingers in daylight at a distance of 3 meters" (to indicate less than 3/60 or its equivalent).

Categories of Visual Impairment (WHO) (Tables 26.1 and 26.2)

Levels of visual impairment, recommended by World Health Organization (WHO), has been incorporated in International Classification of Diseases (ICD).

Definition of Blindness (National Program for Control of Blindness)

Simple definition: Inability of a person to count fingers from a distance of 6 meters or 20 feet.

Technical definition: Vision 6/60 or less with the best possible spectacle correction and/or diminution of field vision to 20° or less in better eye.

TABLE 26.1: Categories of visual impairment (World Health Organization)

Categories of visual impairment	Visual acuity with best correction (better eye)	
	Maximum vision (less than)	Minimum vision (equal to or more than)
Low vision	• 6/18 • 6/60	6/60 3/60
Blindness	• 3/60 (finger counting at 3 meters) or, visual field constricted to < 10° • 1/60 (finger counting at 1 meter) or visual field constricted to < 5° • No perception of light (no PL)	1/60 (finger counting at 1 meter) PL (perception of light)

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TABLE 26.2: Visual impairment disability categories based on its severity and proposed disability percentages (Government of India)

All with best corrections			
Categories	**Better eye**	**Worse eye**	**Percentage impairment**
Category 0	6/9–6/18	6/24 to 6/36	20%
Category I	6/18–6/36	6/60 to no PL	40%
Category II	6/60–4/60 or Field of vision 10–20°	3/60 to no PL	75%
Category III	3/60 to 1/60 or Field of vision <10°	FC at 1 feet to no PL	100%
Category IV	FC at 1 feet to no PL or Field of vision less than 10°	FC at 1 feet to no PL or Field of vision less than 10°	100%
One-eyed person	6/6	FC at 1 feet to No PL	30%
Category I to IV = Visually handicapped (challenged)			

Abbreviations: **PL**—Perception of light; **FC**—Finger counting

Few More Definitions

- ***Legal blindness:*** Visual acuity in the better eye is less than 3/60, and/or, visual field constricted to less than 10° (in India).
- ***Economical blindness:*** Visual acuity in the better eye is less than 6/60, and/or, visual field constricted to less than 20° (in India).
- ***Visually handicapped:*** Visual acuity in the better eye is less than 6/18 (in India).
- ***Avoidable blindness:*** This includes both preventable blindness and curable blindness. 85% of the blindness is estimated to be avoidable.
  - ***Preventable blindness:*** The blindness which can be easily prevented by attacking the causative factors, e.g. corneal blindness (vitamin A deficiency, trachoma, infectious keratitis, etc.), industrial blindness (by improving occupational safety conditions), retinopathy of prematurity (ROP), diabetic retinopathy, etc.
  - ***Curable blindness:*** It is almost synonymous with cataract blindness. Others are glaucoma, inflammation of ocular tissues, etc.

In the World, 82% of people, 50 years and older are blind. The major causes of visual impairment are uncorrected refractive errors (43%) and cataract (33%); the first cause of blindness is cataract (51%) (Table 26.3 and Fig. 26.1).

Prevalence of blindness in India
Vision <6/60 in better eye
1986-9 – 1.49%
2001-4 – 1.10%
2007 (RAAB/NPCB) – 1.05% and 0.62% (3/60)
Increased in prevalence is due to population explosion and more old-aged patient.
RAAB = Rapid Assessment of Avoidable Blindness/
NPCB = National Program for Control of Blindness

Blindness Data of India

Prevalence of visual impairment and blindness in different age-group stastical data have been described in Tables 26.4 and 26.5.

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TABLE 26.3: Causes of blindness in World and India

Causes	World (Vn < 3/60)	India (Vn < 6/60)
Cataract	51%	62%
Refractive errors	3%	20%
Glaucoma	8%	6%
Corneal opacity	4%	0.9%
Trachoma	3%	0
Posterior segment diseases	–	4.7%
• AMD	5%	–
• DR	1%	–
PCO		0.9%
Surgical complication		1.2%
Childhood blindness	4%	–
Misc/undetermined	21%	4.5%

Abbreviations: **Vn**—Vision; **AMD**—Age-related macular degeneration; **DR**—Diabetic retinopathy; **PCO**—Posterior capsular opacification

NATIONAL PROGRAM FOR THE CONTROL OF BLINDNESS (NPCB)

One of the basic human rights, is the right to see. We have to ensure that no citizen goes blind needlessly, or being blind does not remain so, if, by reasonable deployment of skill and resources, his sight can be prevented from deteriorating, or if already lost, can be restored.

–National policy pronounced in 1975

Overall Objectives of NPCB (1976)

- Provision of comprehensive eye care facilities for ***primary, secondary and tertiary levels*** of eye-health care.
- Substantial reduction in the prevalence of eye diseases in general and ***reduction in the prevalence of blindness from 1.40% to 0.3% by 2000 AD.***
- National Program for the Control of Blindness was launched by the Government of India, Ministry of Health and Family Welfare in the year 1976 and is a 100% centrally sponsored scheme/program with the goal of reducing the prevalence of blindness.
- To reduce the backlog of blindness through identification of treatment of blind and visually handicapped.
  - To develop eye care facilities in every district.
  - To develop human resources for providing eye care services.
  - To improve the quality of service delivery.

Fig. 26.1: Causes of blindness in World and India

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TABLE 26.4: Prevalence of visual impairment (WHO-2010)

Blindness	(BCVA < 3/60 in better eye)	Low vision (BCVA <6/18 in better eye)	Total visual impairment
World	39 million	246 million	285 million
India	8 million	54.5 million	62.5 million

Abbreviation: **BCVA**—Best corrected visual acuity

TABLE 26.5: Blindness in different age-groups

Age-groups	Causes of blindness
Infants and preschool-age	• Congenital cataract • Congenital glaucoma • Optic atrophy • Xerophthalmia • Rare congenital • Anomalies • Trauma • Retinoblastoma • ROP
School-age	• Congenital/developmental anomalies • Neurological (optic neuritis, papilloedema) • Trauma
Adult life (20–50 years)	• Pathological myopia • Neurological diseases • Industrial injuries • Diabetes • Infectious diseases
Late adult life (51–70 years)	• Cataract • Glaucoma • Diabetes • Retinal vasculopathies • Trachoma
Elderly life (above 70 years)	• Cataract • Glaucoma • Age-related macular degeneration • Diabetes • Retinal vasculopathies • Trachoma

- To secure participation of voluntary organization in eye care.
- To enhance community awareness of eye care.

Current Infrastructure (2010–11)

• Apex Institute	1
• Regional Institute of Ophthalmology	18
• Upgraded Medical College	150
• District Hospitals equipped	550
• District Blindness Control Society (DBCS)	575
• Central Mobile Ophthalmic Units	155
• District Mobile Ophthalmic Units	250
• Ophthalmic Assistant training center	100
• Vision Center in Primary Health Center	3000
• Eye Bank and Eye Donation Centers	650

- ***Ophthalmologists:*** Approximately 20,000 (about one-third in Government sector).
- ***Paramedical ophthalmic assistant (PMOA)*** 18,000.
- ***Need:*** One Ophthalmologist and one ophthalmic assistant per 50,000 population.
- Cataract operation performed in the year 2014–5: 6.33 million 6.29 million.
- Donor eye collection in the year 2014–5.

Source: NPCB, Ministry of Health and Family Welfare, Govt of India

Methods of Intervention (Fig. 26.2)

Peripheral (Primary) Sector

- Community health worker
- Ophthalmic Assistant at Primary Health Center (PHC), Block Primary Health Center (BPHC) and rural hospitals.

Service: Primary eye-health care

- Treatment of common eye aliments, e.g. acute conjunctivitis, trachoma, superficial foreign body, etc.
- Treatment of xerophthalmia
- Vitamin A prophylaxis
- Correction of refractive errors
- Screening for cataract.
- Teleophthalmology services via vision center.

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Fig. 26.2: Community eye-health care

Intermediate (Secondary) Sector

- Sub-divisional hospitals
- District hospitals.

Service: Secondary eye-health care (in addition to primary eye care).

- Treatment of common blinding conditions e.g. cataract, glaucoma, trichiasis, entropion, ocular trauma, etc.
- Mobile ophthalmic units (MOU)
- Eye camp via outreach program.

Mobile ophthalmic unit

It provides a comprehensive eye-health care to the people in remote rural areas. Each unit organizes camps in a group of village in five adjoining districts to provide:

Contd...

Contd...

- Free ophthalmic service (medical/ surgical)
- Vision testing service
- Prescription of spectacles
- Survey of eye diseases
- Education on prevention and rehabilitation.

Central (Tertiary) Sector

- Medical colleges
- State Eye Hospitals/Institutes
- Regional Institutes of Ophthalmology (RIOs)
- National (Apex) Eye Institute.

Service: Tertiary eye-health care (in addition to secondary eye care).

- Sophisticated eye care, e.g. retinal detachment, corneal grafting and other complex surgeries.

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- To provide eye bank services.
- To provide specialized training programs for all staffs.
- To arrange continuing medical education program for the ophthalmologists.
- To undertake and promote research activities in ophthalmology.

Apex center (National Eye Institute) is to function as a center of excellence to provide overall leadership, supervision and guidance in technical matters in planning and implementation of the program.

Dr Rajendra Prasad Center for Ophthalmic Sciences, AIIMS, New Delhi, is identified for this purpose.

Specific National Programs

- ***Trachoma control program:*** Early diagnosis and treatment can cure trachoma. This program launched in 1963, and has merged with NPCB in 1976.
- ***Vitamin A prophylaxis:*** Two lakh IU of vitamin A as oil are given orally at 6 months interval between the age of 1 and 5 years.
- ***School eye-health services:*** It is estimated that 5–7% of children aged 5–14 years have problems with their eye-sight affecting their participation and learning at the school. This can be easily corrected by a pair of spectacles.
  Other diseases, like, vitamin A deficiency, squint, amblyopia, congenital anomalies, etc. can be detected earlier for further and better management.

The school eye screening program seeks to:

- Screen all children in the age group of 5–14 years for refractive errors (difficulty in clearly seeing distant or near objects).
- Train teachers, preferably with science background, female teachers and those wearing spectacles for identifying children with eye problems.
- Refer children with suspected refractive errors, to the Ophthalmic Assistant at the PHC for refraction.
- Prescription of spectacles.
- Provide children from poor families with free glasses.
- Screening should be on an annual basis. In 2014–5, more than 700,000 free spectacles has been distributed among school children.
- ***Occupational eye-health services:*** This is to prevent and treat eye hazards in industries. Education on the prevention of occupational eye hazards and use of protective devices in some occupations are important.

District Blindness Control Society (DBCS)

Features

- Autonomous society to implement NPCB
- Representation consists of Government, Non-Governmental Organization and Private Sectors
- Decentralized planning, management and monitoring
- Direct funding from Government of India
- Empowered to utilize and raise funds
- Forum for community participation.

Composition of DBCS

Chairman	District Magistrate/ District Collector/ Deputy Commissioner
Vice Chairman	Chief Medical Officer (CMO)
Technical Advisor	Head, Deptt of Ophthalmology of Medical College/District Ophthalmic Surgeon
Member Secretary	Deputy CMO or any Government Officer of equal rank or higher
Members	Representative of Non-governmental organization (NGO), Private Sector and Media.

Functions of DBCS

- ***Planning:*** Preparation of the district microplan based on magnitude and distribution

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of blind persons and resources available for eye care.
- ***Implementation of program*** though utilization of government facilities, involvement of NGOs and community participation.
- ***Monitoring of program*** activities and quality control.
- ***Financial and material management***.
- ***Social mobilization and public awareness***.
- ***Orientation of various functionaries of health*** and other sectors, formal and non-formal leaders.
- ***Procurement*** of good quality refraction sets, drugs, consumable, cataract sets, etc. and maintenance of the equipments.
- ***Arrangement*** for screening camps, intraocular lens (IOL), spectacles, generator sets, etc.
- ***Monitoring and financial assistance*** to eye banks and eye donation centers.

Manpower Development

Technical manpower development is an important priority for effective implementation of its services and time-bound targets achievement.

The priority training programs are:
- Training of ophthalmic assistants.
- Training of mid-level ophthalmic personnels (MLOPs).
- Orientation training to the medical officers of PHC, BPHC and rural hospitals about the need of the NPCB.
- Refresher training course to the district ophthalmologists and ophthalmic assistants.
- Fellowship training to ophthalmologists and teachers of medical colleges under the prevention of blindness program.

Voluntary organizations

Voluntary or non-Government Organizations (NGOs) have played a vital role in the control of blindness in India. They are active in the field of educative, preventive, rehabilitative and surgical services to control blindness for many years.

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Some of the active organisations
- Vision-2020 India with its members across the country
- The Royal Commonwealth Society for the Blind (sight savers)
- Lions International (sight first)
- Rotary International and its branches
- International Agency for Prevention of Blindness (IAPB)
- Helen Keller International
- ORBIS International and its branches
- Helpage India, etc.
- SightLife International

Other assistances
- WHO assistance
- Danish assistance
- Indo-UK collaboration
- World Bank assistance.

NUTRITIONAL BLINDNESS VITAMIN A DEFICIENCY

Vitamin A deficiency (VAD) along with protein energy malnutrition (PEM) causes severe blinding corneal destruction.

Most of the time, VAD is precipitated by PEM and other diseases which precipitate malnutrition (e.g. measles, diarrhea, malaria or other acute illness in children).

Again malnutrition is caused by VAD. Together, this blindness is termed as ***nutritional blindness***, though the main factor is ***VAD***.

Vitamin A deficiency is a systemic disease that affects cells and organs throughout the body.

The resultant changes in epithelial architecture are termed as ***keratinizing metaplasia***.

The characteristic ocular manifestations of VAD ranging ***from night blindness to corneal melting*** are termed as ***xerophthalmia (Figs 26.3 and 26.4)***.

Approximately, one-third of preschool-age population of the world is estimated to be vitamin A deficient, with just less than 1% being night blind at any given time.

Vitamin A deficiency is largely limited to the first 4–6 years of life and is especially frequent among those 6 months to 3 years.

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Fig. 26.3: Xerophthalmia

In India about 15,000–20,000 preschool children suffer from VAD at any point of time.

Major Signs and Symptoms of Xerophthalmia (WHO Classification)

XN	=	Night blindness
XIA	=	Conjunctival xerosis
XIB	=	Bitot's spot
X2	=	Corneal xerosis
X3A	=	Corneal ulceration/keratomalacia (<1/3 corneal surface)
X3B	=	Corneal ulceration/keratomalacia (>1/3 corneal surface)
XS	=	Corneal scar
XF	=	Xerophthalmic fundus.

- ***XN (night blindness):*** It is usually the ***earliest manifestation*** of VAD; sometimes termed as ***chicken eyes*** (chicken lack rods and are thus night blind).
  Night blindness responds rapidly (within 24–48 hours) to vitamin A therapy.
- ***X1A and X1B (conjunctival xerosis and Bitot's spot):*** The conjunctival epithelium in VAD, is transformed from normal columnar to stratified squamous with a resultant loss of goblet cells, formation of a granular cell layer and keratinization of the surface.
  Conjunctival xerosis (Fig. 26.4A) first appears at the temporal side as an isolated oval or triangular patch, adjacent to the limbus in the interpalpebral fissure. It is almost always present in both eyes.
  In some cases, keratin and saprophytic bacilli accumulate on the xerotic surface, giving it a foamy or cheesy appearance. These lesions are known as ***Bitot's spots (Fig. 26.4B)***.
  Isolated, usually temporal, patches of conjunctival xerosis or Bitot's spots are some times encountered in absence of active VAD (Fig. 26.5).
  In most instances, these patches represent persistent areas of squamous metaplasia induced during an earlier episode of VAD.
  The only certain means to distinguish an active lesion from an inactive lesion, is to observe its response to vitamin A therapy.
  Active conjunctival xerosis and Bitot's spot begin to resolve within 2–5 days and mostly disappear by 2 weeks.
- ***X2 (corneal xerosis):*** A hazy, lustureless dry appearance of the cornea, is first seen

A B

Figs 26.4A and B: Bilateral conjunctival xerosis and Bitot's spot (X1A and 1B)

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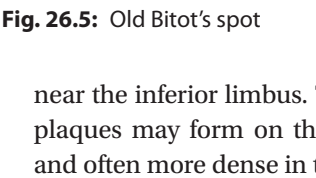


Fig. 26.5: Old Bitot's spot

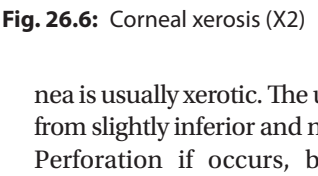


Fig. 26.6: Corneal xerosis (X2)

near the inferior limbus. Thick, keratinized plaques may form on the corneal surface and often more dense in the interpalpebral zone (Fig. 26.6).

Corneal xerosis responds within 2–5 days to vitamin A therapy and it returns to normal appearance by 1–2 weeks.

- ***X3A and X3B (corneal ulceration/keratomalacia) (Fig. 26.7):*** They indicate permanent destruction of a part, or all the corneal stroma, resulting in permanent structural alteration.

  These ulcers are classically oval or round punched out defects. The surrounding cornea is usually xerotic. The ulcer usually starts from slightly inferior and nasal aspects.

  Perforation if occurs, becomes plugged with iris, thereby preserving the anterior chamber.

  With therapy, superficial ulcers often heal with surprisingly little scarring, but deeper ulcers with perforation forms adherent leucoma. Sometimes, a bigger perforation results with extrusion of intraocular contents leading to phthisis bulbi or pseudocornea formation leading to anterior staphyloma (Fig. 26.8).

  An emergency vitamin A therapy may still save the child's eye to some extent.

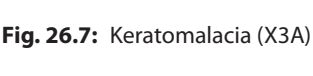


Fig. 26.7: Keratomalacia (X3A) (arrows)

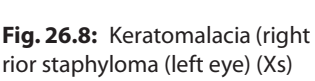


Fig. 26.8: Keratomalacia (right eye) (X3B) and anterior staphyloma (left eye) (Xs)

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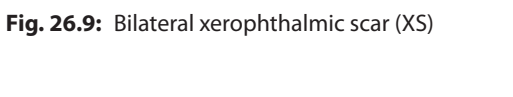


Fig. 26.9: Bilateral xerophthalmic scar (XS)

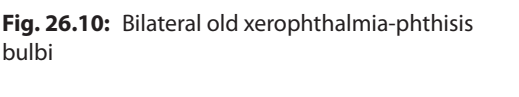


Fig. 26.10: Bilateral old xerophthalmia-phthisis bulbi

- ***XS (xerophthalmic scar):*** They are usually bilateral and indicate healed sequelae of prior corneal disease related to VAD (Fig. 26.9).
  They include ***nebula, macula, leucoma, adherent leucoma, anterior staphyloma or phthisis bulbi (Fig. 26.10)***.
- ***XF [xerophthalmic (Uyemura's) fundus]:*** Small white lesions on the retina, seen in some cases of VAD, are only of academic interest. They may be associated with constriction of the visual fields.

Treatment

Xerophthalmia is a medical emergency as it carries a high risk of corneal destruction and blindness and/or sepsis and death.

Effective therapy is immediate administration of massive doses of vitamin A with concomitant treatment of underlying systemic illness and PEM, and prevention of any recurrence.

Treatment Schedule for Xerophthalmia

Vitamin A: (WHO recommendation)

- Immediately upon diagnosis: 200,000 IU vitamin A orally.
- Next day: 200,000 IU vitamin A orally.
- Within 1–4 weeks: 200,000 IU orally.
- Children between 6 and 11 months old, or less than 8 kg—half the above dose.
- Children less than 6 months old-one-quarter of the above dose.

Intramuscular injection of vitamin A (water-soluble) 100,000 IU is usually given, when—

- The children cannot swallow,
- In case of persistent vomiting,
- In severe malabsorption, or
- Where the compliance is poor.

Oral administration is preferred, as it is safe, cheap and highly effective even in presence of mild diarrhea (as it is also helpful for the intestinal epithelium).

Medical status and diet: Proper treatment includes rehydration, frequent feeding with easily digestible and protein-rich food, and general supportive care. Concurrent illness, e.g. respiratory infection, diarrhea, worm infestation, etc. should also be treated.

Eye care: In case of corneal involvement:

- Board-spectrum antibiotic ointment, e.g. chloramphenicol or ciprofloxacin: 4 times daily.
- Atropine eye ointment: 2 times daily.
- If secondary infection is present—second antibiotic is added. Subconjunctival injec-

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tion of gentamycin and atropine may also be given.
- Artificial tears (PVA or Povidone iodine or both) eye drop—4 to 8 times daily.

Preventing recurrence: This is for the vulnerable children.

By inexpensive, readily available vitamin A rich diet.

Periodic administration of a large dose of vitamin A at an interval of 4–6 months to ensure adequate vitamin A store in children (*see* below).

Vitamin A Prophylaxis

Three main intervention strategies are as:
1. Increasing the dietary intake of foods rich in vitamin A and pro-vitamin A.
2. Periodic administration of large doses of oral vitamin A.
3. Administration of commonly consumable fortified food items (vitamin A fortification).

Increased Intake of Dietary Vitamin A

Dark-green leafy vegetables are usually the least expensive and most widely available source of vitamin A.

The daily requirement of vitamin A is obtained from a handful of fresh spinach (65 gm) as from a small portion of liver (65 gm), 4 medium-sized hen's eggs, 1.7 liter of whole cow's milk or 6 kg of mutton.
The dark-green leafy vegetables should be boiled, shredded (mashed or sieved for the infants), and should be combined with a small amount of edible oil to improve vitamin A absorption.

Sources of vitamin A
- ***Vegetable sources:*** Dark-green leafy vegetables, spinach, carrot, tomato, pumpkin, etc.
- ***Animal sources:*** Liver, meat, cod-liver oil, shark-liver oil, egg-yolk, etc.
- ***Fortified food items:*** Vitamin A enriched commercially available food items.

Daily requirements
- ***School children, adolescent and adults:*** 2250 IU (750 μg).
- ***Children (0–4 years):*** 1000–2000 IU (300–400 μg).
- ***Pregnancy and lactation:*** 3000–3500 IU (750 μg + 300 μg).

Periodic Supplementation

Oral administration of 200,000 IU vitamin A and half this dose for children age 6–11 months, every 4–6 months will protect majority of the recipients.

This prophylaxis may be for entire neighborhood ***(mass prophylaxis)***, or for high risk group ***(selective prophylaxis)***.

The high-risk conditions are as follows:
- Children with severe PEM
- Children with measles
- Children with diarrhea, lower respiratory tract infection, or other acute infection (e.g. malaria, chickenpox, etc.).

They should received a dose of 200,000 IU vitamin A orally at the time of diagnosis by the pediatrician.

Children below 1 year should received half dose.

Fortification of Dietary Items

Fortification, the addition of selected nutrients to common dietary items, is a successful means of protecting nutritional status of the children.

Dalda, milk, sugar, tea, cereal grains, bread, butter, margarine, etc. may be fortified with vitamin A.

The fortified foods should be inexpensive, stable and virtually undetectable in food-vehicle selected, and they should be acceptable to the community.

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Ocular Findings in Other Vitamin Deficiencies

Vitamins	Ocular manifestations
Vitamin B_1 (Thiamine)	• Retrobulbar neuritis • Corneal hyposthesia • Corneal and conjunctival degenerative changes
Vitamin B_2 (Riboflavine)	• Corneal vascularization Punctate epithelial keratitis • Blepharoconjunctivitis
Vitamin B_{12}	• Retrobulbar neuritis
Vitamin C	• Delayed wound healing • Hemorrhage in the conjunctiva, retina or orbit. • Keratoconjunctivitis
Vitamin D	• Zonular cataract • Papilloedema
Vitamin K	• Hemorrhages in different ocular tissues.

VISION 2020: THE RIGHT TO SIGHT

This is the global initiative program of WHO for the elimination of avoidable (curable or preventable) blindness by the year 2020 AD.

Aim

To intensify and accelerate present prevention of blindness activities so as to achieve the goal of eliminating avoidable blindness by the year 2020.

Present Position

- Upto 80% of global blindness is avoidable.
- 180 million people worldwide are visually disabled.
- 40–45 million people are totally blind.
- Sixty percent of blind population reside in sub-Saharan Africa, China and India.

Blindness will continue to increase by 2 million cases/year—unless more aggressive intervention is taken.

Action needed

- Disease control and eye care
- Human resource development
- Provision of appropriate technology and infrastructures.

Vision 2020 will be implemented through 4-five-year plans. The first three has already expired in the year 2015. This last phase will be completed by the year 2020.

Five conditions have identified as immediate priorities within the framework of VISION 2020 program.

1. Cataract
2. Trachoma
3. Onchocerciasis
4. Childhood blindness and
5. Refractive errors and low vision.

In India, as there is no onchocerciasis, and trachoma is declining ***two*** more diseases have been included recently in ***Vision-2020 India program***.

These are ***diabetic retinopathy*** and ***glaucoma***. They need special attention by all levels of eye health care personnel.

Cataract

Problem

- 20 million cataract backlog
- 8 million cataract operation/year globally
- Worldwide problem.

Action

- To eliminate backlog by 2020.
- To provide good qualities of surgery:
  - Accessible and affordable to all people.
  - High success rate with IOL for all.
- Global cataract operation ***target:***
  - 2000 AD – 12 million
  - 2010 AD – 20 million
  - 2020 AD – 32 million.

Trachoma

Problem

- 146 million with active disease

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- 10 million trichiasis
- 6 million blind
- Mostly in Africa, South-East Asia, Western Pacific, Eastern Mediterranean.

Action

- Implementation of SAFE in all endemic countries.
- 5 million trichiasis surgery (by 2010).
- Treatment of 60 million people (by 2010).
- Total elimination by 2020.

SAFE strategy for trachoma:

- ***S***urgery for trichiasis—the immediate precursos to corneal blindness.
- ***A***ntibiotics—to treat active diseases—oral azithromycin or 1% tetracycline ointment.
- ***F***acial cleanliness—to reduce person to person transmission.
- ***E***nvironmental improvement—to affect the determinants of vulnerability.

Onchocerciasis

Problem

- 17 million people infected
- 0.3–0.6 million blind
- Mainly in African countries.

Action

- Implementation of control program in endemic countries
- Community-directive distribution of ***ivermectin***
- Surveillance
- Elimination by 2010 AD
- In 2015, Colombia, Ecuador and Mexico are the three countries declared free of onchocerciasis.

Dr Satoshi Ōmurao and Dr William C Campbell have been awarded Nobel Prize in Medicine, 2015 for the development of "Ivermectin".

Childhood Blindness

Problem

- 1.5 million blind children in the world:
  - Asia—1 million
  - Africa—0.3 million.
- 5 million visually disabled.

Action

- Promotion of primary eye care by:
  - Elimination of vitamin A deficiency
  - Measles immunization
  - Prevention of newborn conjunctivitis.
- Development of pediatric eye care services:
  - Surgery for squint, congenital cataract, congenital glaucoma and retinopathy of prematurity.
  - Low visual aids.

Refractive Errors and Low Vision

Problem

- ***Refractive errors***
  - Magnitude not actually known
  - Varies from country to country.
- ***Low vision***
  - 35 million in need of low visual aids
  - Will rapidly escalate because of aging and retinal problems.

Action

- Introduction of refractive services at the primary eye care level
- Vision screening
- Low cost production of glasses and low visual optical devices.

Glaucoma

Glaucoma has been declared to be the second most common cause of blindness in adult population in India. Glaucoma accounts for 8–13% of the blind and 12 million have estimated to have glaucoma. Studies have estimated that this number will increase to 31.6 million by 2020.

Primary angle-closure glaucoma (PACG) is more common than primary open angle glaucoma (POAG) (2–4 times) in Indian population. The high rate of blindness in Indian population is due to high proportion of undiagnosed glaucoma. Glaucoma was undetected in more than 90% of individuals in the population studied.

Diabetes

India has the highest number of people with diabetes in the world with 31.7 million in 2000. This figure is estimated to rise to 79.4 million by 2030.

The prevalence of diabetic retinopathy in general population was 3.5%. The prevalence of diabetic retinopathy in the population with diabetes mellitus was 18.0%.

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Human Resource Development

- Number of ophthalmologists per million population has to be increased especially in Africa and Asia.
- Equal distribution of eye care services for urban and rural population.
- ***Training of eye care supporting staffs***
  - Ophthalmic medical assistants
  - Ophthalmic nurses
  - Refractionists.
- ***Management training***
  - For ophthalmologist
  - For paramedical staffs.
- Ophthalmic technician training.

Mission of VISION 2020 Program

To eliminate the main cause of blindness in order to give all people in the world, particularly the millions of needlessly blind, the right to sight (Fig. 26.11).

National Apex Center 1

Centers of excellence 20:

- Profession leadership
- Strategy development
- Continued medical education (CME)
- Laying of standards and quality assurance
- Research.

Training Centers 200

- Tertiary eye care including retinal surgery, corneal transplantation, glaucoma surgery, etc.
- Training and CME.

Fig. 26.11: The proposed structures for vision 2020 program

Service Centers 2000

- Cataract surgery
- Other common eye surgeries
- Facilities for refraction
- Referral services.

Vision Centers 20,000

- Refraction and prescription of glasses
- Primary eye care
- School eye screening program
- Screening and referral services.
- Teleophthalmology services with the training centers.

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27 CHAPTER Surgical Instruments

EYE SPECULUMS

The types are—(1) universal eye speculum, (2) guarded eye speculum, (3) wire speculum.

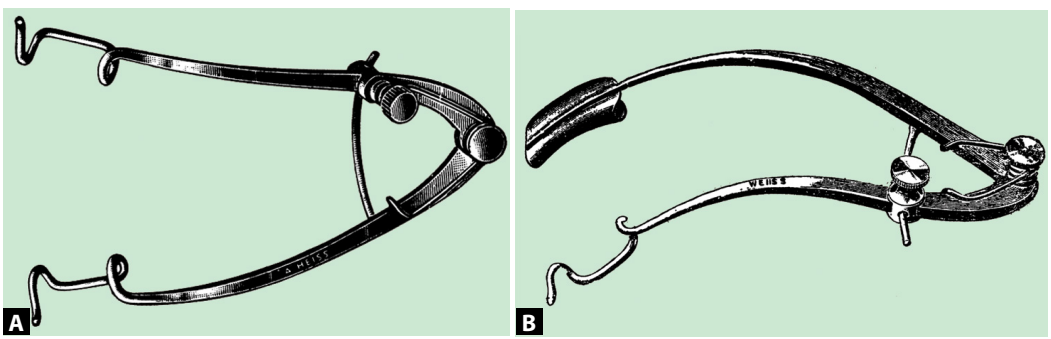
Universal Eye Speculum (Fig. 27.1A)

Identification: It has a spring and two limbs, and a screw to adjust the limbs. It is called universal because it can be used on either side.

Method: It is fixed to the eyelids in such a way that screw should face outward and forward.

Uses: It is used to separate both eyelids for good exposure of the eyeball mainly during extraocular operations, e.g.

- Pterygium excision
- Squint operation
- Evisceration and enucleation
- Debridement and cautery of corneal ulcer
- To give subconjunctival or sub-Tenon's injection
- Removal of corneal foreign body



Figs 27.1A to C: **A.** Universal eye speculum; **B.** Guarded eye speculum (left sided); **C.** Wire speculum

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- Removal of conjunctival cyst or mass
- During suture removal.

It is not used routinely in intraocular operations as it gives pressure over the globe. This causes rise in intraocular pressure (IOP) during operation. One can put a cotton pellet or assistant can lift the instrument during intraocular operation to avoid rise in IOP. Otherwise, stay sutures to the lids are commonly used for intraocular operation.

Disadvantages

- Since it has no guard, eyelashes of upper lid (larger) come in the field of operation.
- Not used in intraocular operation because of more vitreous-upthrust and risk of vitreous loss by direct pressure on the globe.

The speculum should not be confused with Muller's retractor, which has got two or three right-angle sharp pointed pins (teeth) underneath to engage the skin flap.

Guarded Eye Speculum (Fig. 27.1B)

Identification: The upper limb of the speculum is having a guard plate which keeps the eyelashes of the upper lid away from the field of operation. So two instruments are required, one for the right eye and the other for the left eye.

Uses: Same as universal eye speculum. It is specially useful in squint operation in children and in pterygium operation.

Disadvantages

- Operating field is reduced.
- It is heavier, hence gives more pressure on the globe.

Wire Speculum (Fig. 27.1C)

It is made up of a stainless steel wire and there is no screw. It is of universal type.

It is very light and hence gives little pressure on the eyeball. So, it can be used safely during intraocular operations as well as extraocular operation.

SILCOCK'S NEEDLE HOLDER (FIG. 27.2A)

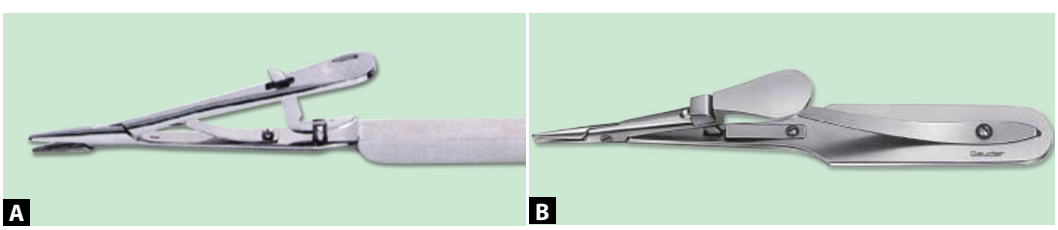
Identification: This stout instrument has two limbs (one short and one long) and a catch. The short limb has a concave impression at its end for giving pressure with thumb. This is to hold the needle, and an upwards stroke with thumb on short limb will automatically release the needle.

ARRUGA'S NEEDLE HOLDER (FIG. 27.2B)

Identification: This stout instrument has also two limbs and a catch similar to Silcock's type. But its long limb is having a broad platform, and thumb rest on short limb is wider without any impression on it.

Uses: It is used to hold the needle (larger needles).

- During suturing the skin and conjunctiva.
- To pass stay sutures through the skin, and underneath the superior rectus muscle during intraocular operations.



Figs 27.2A and B: **A.** Silcock's needle holder; **B.** Arruga's needle holder

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Fig. 27.3: Artery forceps (hemostat)

- To pass stay sutures underneath the rectus muscles in detachment surgery. Needles of 6–0 suture, 8–0 suture or more fine sutures should not be held with these needle holders.

ARTERY FORCEPS (HEMOSTAT) (FIG. 27.3)

Identification: This is a medium-sized, fully serrated forceps with catch. It may be straight or curved. The curved forceps are used more frequently.

Uses

- To hold lid stitches and superior rectus stitch, and then to fix the suture ends with head towel.
- To crush lateral canthus in lateral canthotomy.
- For hemostasis during dacryocystorhinostomy (DCR) or dacryocystectomy (DCT), specially if the angular vein is damaged.
- Fasanella-Servat operation in ptosis (to clamp conjunctiva, tarsal plate, Muller's muscle and levator muscle).
- To hold muscle stump during enucleation.
- To make irrigating cystitome from a 26 gauge needle.
- To hold whole lacrimal sac prior to excision in DCT.

FIXATION FORCEPS (EYEBALL FIXATION FORCEPS) (FIG. 27.4)

Identification: It is a medium-size forceps with 2:3 teeth (may be 3:4) at its tip.

Method: The conjunctiva and episcleral tissue are firmly held at 6 o'clock position within 3–4 mm of limbus with left hand. Because, the conjunctiva is firmly adhere to episcleral tissue and sclera around the limbus, and it is 4–5 layers thick. It may be used at other part around the limbus (e.g. 3 o'clock, 9 o'clock, etc.).

Uses

- To fix the eyeball during operative procedures, like:
  - Cataract surgery
  - Paracentasis
  - Pterygium surgery
  - Needling, etc.
- To catch superior rectus muscle to pass bridle suture.
- To catch other tissues, e.g. skin, Tenon's capsule, etc.
- To lift bulbar conjunctiva during subconjunctival or sub-Tenon injection.
- Used in forced duction test (FDT) in case of squint to know any mechanical restriction is present or not.
- To hold the sponge piece, gauze piece or cotton ball for swabbing in depth.

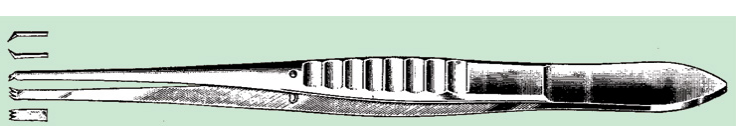


Fig. 27.4: Fixation forceps

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VON GRAEFE'S CATARACT KNIFE (FIGS 27.5A AND B)

Identification: It has a thin straight blade with sharp pointed tip and a cutting edge, the other edge is blunt. The blade may be thin or thick. It may have sliding case arrangement where the sharp blade end is kept in a cover to protect its tip when it is not in use.

Method: It is used for ***abinterno*** corneo-scleral section in cataract operation (***abinterno*** means—the section is given from inside to outside).

Steps of knife section:

- Lid sutures and superior rectus suture.
- Fix the eyeball with fixation forceps at 6 o'clock position.
- For right eye, hold the knife with right hand and for left eye ideally with the left hand (otherwise surgeon has to move to the left temporal side, if he uses his right hand).
- Hold the knife as pen holding position.
- The tip is introduced into anterior chamber from the temporal limbus, then make the knife horizontal.
- Quickly cross over the iris, pupil and opposite iris.
- When the tip reaches near the opposite limbus, counter-puncture the cornea just 1 mm inside the limbus (because of refraction of the tip of the blade in aqueous, the tip appears to be displaced).
- When the tip is pierced out the opposite limbus, knife is moved as see saw movement towards 12 o'clock position and the section is completed at upper half of cornea.
- It is preferable to make a triangular flap of conjunctiva along with section at its completion.

Now-a-days this knife is not used for routine cataract surgery. It was mainly used in eye camps because:

- It is a quicker procedure.
- Bleeding is minimum.
- Uniform fine section.

Disadvantages

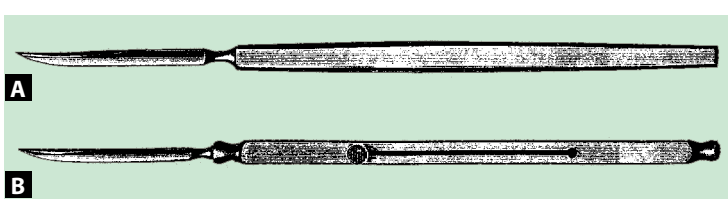
- This type of section is not 'gaurded', i.e. sudden release of aqueous—leads to sudden decompression of the globe which may cause uveal prolapse or effusion in subchoroidal space.
- Not 'stepped', i.e. uniplaner section, so wound closure may not be well secured.

Complications may occur during knife section:

- Loss of anterior chamber and incomplete section. In that case, the section is completed with the help of corneal scissors.
- Involuntary iridectomy or iridotomy.
- Injury to the lens or its capsule.
- Scleral section which may lead to bleeding.
- Subluxation or dislocation of hypermature cataractous lens.
- Conjunctival tear by the fixation forceps.

Test of sharpness of the tip:

- ***Drum test:*** Touch a mini drum (commercially available previously) with the tip—it will break immediately.
- Sweep a cotton ball along the blade—if there is any catch in cotton fiber—knife tip is not that sharp.



Figs 27.5A and B: Von Graefe's cataract Knief; **B.** Knife in sliding-case

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Uses

- ***Abinterno*** section in case of cataract operation
- Optical iridectomy
- Paracentesis
- Glaucoma iridectomy
- Suture removal after cataract operation
- In pterygium operation to dissect the head, or in chalazion operation to give incision
- Four-point iridotomy in iris bombe (***quadri-puncture***).

SCALPEL HANDLE (BARD-PARKER HANDLE) (FIG. 27.6)

Identification: It is a flat handle with a short grooved neck. Handle no. 3 is used in ophthalmic surgeries. The blades are fitted with the neck is no. 11 or no. 15 (no. 11 is triangular blade and no. 15 has curved elliptical tip).

Method: In cataract operation.

- The knife is held in pen holding position.
- Eyeball is fixed with fixation forceps.
- A groove (partial thickness) is made at the limbus with the knife (preferable with no. 15 blade).
- A puncture is made at 11–12 o'clock position to enter into the anterior chamber (preferably with 'no. 11' blade).

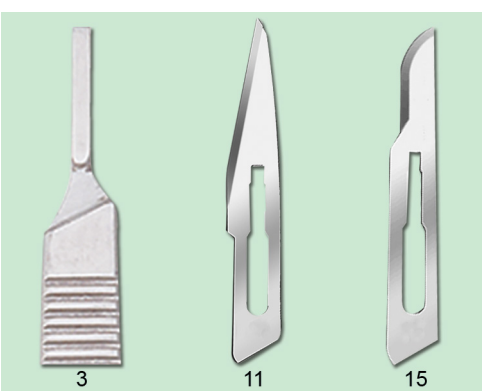


Fig. 27.6: Scalpel handle and blades

- Posterior or scleral lip is depressed to drain aqueous very slowly.
- Then the section is enlarged with the corneal scissors.

Uses

- ***Abexterno*** corneoscleral section for cataract surgery.
- Trabeculectomy operation.
- Skin incision as in DCR, ptosis or other lid surgeries.
- To dissect the pterygium head from the cornea.
- To give incision in chalazion operation.
- For suture removal after cataract operation or keratoplasty.

Advantages in cataract operation

- It is a 'guarded' section. As there is slow release of aqueous, the chance of quick decompression of the globe is almost nil.
- It is 'stepped', i.e. biplaner or triplaner incision—so wound security is better.
- Easier for the beginners.

Disadvantages

- Conjunctival flap is usually necessary before giving section.
- It is time consuming.

BLADE BREAKER

Identification: It is an instrument to break the razor blade (must be a carbon-steel blade, e.g. 'Bharat' blade). It is of medium size, with two jaw-like plates at its tip to catch the razor blade firmly. The other end of the instrument is having catch for better grip of the blade. At least eight blade fragments can be made from a single razor blade (Figs 27.7A to C).

Advantages

- It is cheaper than any other knife-blade.
- It is sharper than no. '11' or no. '15' blade.

Disadvantages: As only the breakable carbon-steel razor blade is used—rust formation is a problem.

Uses: Blade breaker with razor blade fragments is having the same uses as Bard-Parker handle with knife.

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Figs 27.7A to C: A and B. Carbon-steel blade; **C.** Blade breaker

Instruments used for corneoscleral or clear corneal incision are: (a) Cataract knife, (b) Bard-Parker knife, (c) Blade breaker with razor blade, (d) Disposable super-blades, (e) Keratome, (f) Diamond knife, (g) Laser (femtosecond).

THERMOCAUTERY (HEAT CAUTERY) (FIG. 27.8)

Identification: It is a metallic ball with pointed tip, attached to the end of a handle. The ball is made up of steel or brass (***brass retains heat for a longer time***).

Method: The metallic ball is heated over the flame of a spirit lamp before cauterization. When it is sufficiently hot, the pointed tip is applied to the bleeding points for hemostasis.

Uses

- To cauterize any superficial bleeding points during operation on the eyeball (e.g. in cataract operation and glaucoma operation after making the conjunctival flap).
- To cauterize the bare scleral area in pterygium surgery.
- To cauterize the margin of a progressive corneal ulcer.
- To cauterize small iris prolapse.
- Temporary punctal occlusion.

Advantages: It is cheap and easily available.

Disadvantages

- As the application of heat is not uniform—there may be more tissue charring at the beginning of cautery.
- More tissue charring may excite more tissue reaction.

The best method of hemostasis is by diathermy, or wet field cautery (***hemostatic eraser***), where the hemostasis is uniform and tissue reaction is minimal.



Fig. 27.8: Thermocautery

CONJUNCTIVAL SCISSORS (FIG. 27.9)

Identification: It is a straight fine scissors with pointed tips. It may be curved.

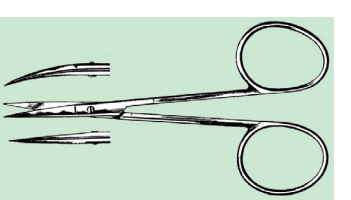


Fig. 27.9: Conjunctival scissors

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Uses

- It is used to dissect the bulbar conjunctiva in some operations:
  - Conjunctival flap in cataract operation. Here the flap may be limbal-based or fornix-based. In ***limbal-based flap***, the conjunctiva is cut few mm away from the limbus and then it is reflected over the cornea from its attached base at the limbus.

    In ***fornix-based flap,*** conjunctiva is cut at the limbus and retracted towards fornix. Fornix-based flap may be better than limbal-based flap in some operations.
  - Conjunctival flap in trabeculectomy operation.
  - Squint, retinal detachment, enucleation operation, etc.
- It may be used to cut the suture ends.
- It is used to cut other tissues, e.g. Tenon's capsule, pterygium, skin edges, etc.

PLAIN DISSECTING FORCEPS (FIG. 27.10)

Identification: It is an ordinary straight forceps without any teeth at its tips. The ends are little blunt, and on their inner surface, there are fine serrations for better grip.

Uses

- To hold the bulbar conjunctiva during its dissection.
- To hold episcleral tissue during its dissection.
- It is also used during suturing of conjunctiva.
- To hold conjunctiva during subconjunctival injection.
- It may be used to hold small sponge swabs.

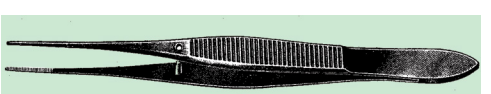


Fig. 27.10: Plain dissecting forceps

CORNEAL SPRING SCISSORS (UNIVERSAL) (FIG. 27.11A)

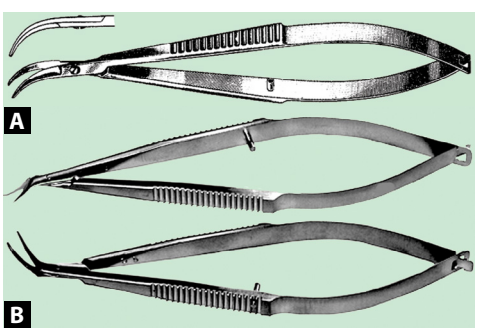
Identification: It is a small curved spring scissors with sharp small blade. It is called universal, as it is used to cut both right and left half of the section (right half means temporal half of right cornea and nasal half left cornea, left half means temporal half of left cornea and nasal half right cornea).

Note: Right sided corneal scissors means to cut right half of the section and left sided corneal scissors for left half of the section (Fig. 27.11B). In case of universal scissors to cut the right half of the section, surgeon must use his left hand and to cut the left half, use right hand, to avoid any inconvenience. But those who are no proficient with the left hand—they use right and left corneal scissors, and both are operated by right hand.

Method: In case of cataract surgery, after making corneoscleral groove and entering into anterior chamber, one blade of the scissors is introduced into the anterior chamber, the other blade is being outside. Scissors are held in pen holding position, and with small snips, the section is enlarged both on temporal and nasal side upto 120°–160° in upper half. Left hand is used for making right half of the section.

Uses

- To extend ***abexterno*** section in cataract operation.



Figs 27.11A and B: A. Corneal spring scissors (universal); **B.** Corneal scissors—right and left sided

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- To cut the corneal button from the donor and recipient eyes in case of keratoplasty.
- It may be use to cut the 8-0 suture ends.
- It may be used to cut the iris for iridectomy.
- To make conjunctival flap in cataract or trabeculectomy operation.

IRIS FORCEPS (FIG. 27.12)

Identification: It is a small light-weight forceps with 1:2 teeth on the inner side of its tips. If the limbs are closed, teeth can not be seen or felt. The shape of the forceps may be straight, curved or angular.

Method of iridectomy

- Hold the instrument in such a way that its tips face toward 12 o'clock position and angle towards 6 o'clock position.
- Now enter into the anterior chamber with close tips and touch the peripheral iris.
- Open the forceps a little to catch a pinch of iris tissue and take out as a triangular fold of iris outside the section.
- Cut a small triangular piece of iris tissue radially with de-Wecker's scissors. Always check the severed iris tissue by rubbing it over face mask.

This is to see the pigmented layer has cut or not, i.e. if there is release of pigment—it means iridectomy has done in full thickness.

Uses: To grasp the iris-tissue in various types of iridectomies.

Iridectomy: It is usually done in upper part as it is covered by the upper lid. There will be diplopia (in case of peripheral iridectomy) or dazzling (in case of complete iridectomy) of light, if the iridectomy is done in the lower part, as the lower part of iris is not covered by the lower lid.

There is usually ***no bleeding during iridectomy*** as iris vessels are highly contractile. Iridectomy is avoided in case of diabetic cataract and rubeosis iridis as there may be chance of bleeding.

Types of iridectomy with indications (Figs 27.13A to C)

- ***Peripheral button-hole iridectomy:*** (At 11 and/or 1 o'clock position) one full thickness iridectomy is equally effective as two iridectomies. The indications are:
  - In intracapsular cataract extraction
  - Angle closure glaucoma
  - As a part of trabeculectomy operation
  - Sometimes, in penetrating keratoplasty operation.

  It is not mandatory in extracapsular cataract extraction. Peripheral iridectomy is done to prevent pupillary block glaucoma and consequent iris prolapse in case of intracapsular cataract extraction (ICCE). As the aqueous can flow into the anterior chamber via this bypass channel, equilibrium between the anterior chamber and posterior chamber is maintained after ICCE.
- ***Optical iridectomy:*** In case of central corneal opacity where facilities for keratoplasty are not available and rarely in polar cataracts.

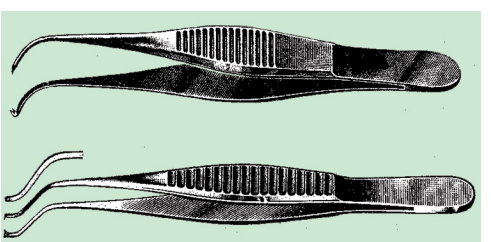
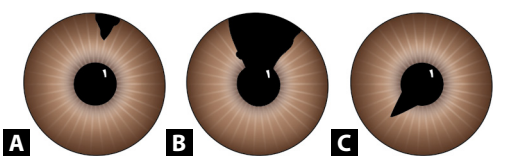


Fig. 27.12: Iris forceps



Figs 27.13A to C: Different types of iridectomy. **A.** Peripheral button-hole; **B.** Sector (complete); **C.** Optical

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It is done in the lower part of the iris, as in upper part, the new opening will be covered by upper lid. The best site for optical iridectomy is judged by stenopaeic slit test, and it must be at the clearest zone. Customarily, it is said that the site of election will be down and in for the literates, and down and out for illiterates.

- ***Broad or complete iridectomy:*** To facilitate the extraction of lens (combined extraction of cataract) when pupil is small and rigid, or if there is extensive synechiae, and if there is vitreous loss after cataract extraction.
- ***Iridectomy for prolapsed iris.***
- ***Iridectomy to remove foreign body on the iris.***
- ***Iridectomy to remove tumor or cyst of the iris.***
- ***Inferior iridectomy*** in cases of vitreo-retinal surgery with silicon oil injection.
- ***Yttrium aluminium garnet (YAG) laser peripheral iridectomy*** is better in acute attack of angle closure glaucoma as it is a non-invasive (so no chance of infection) out patient department (OPD) procedure. It is also less expensive for the patient.

Iridotomy: Puncture of the iris without abscission of any portion.

- To create an artificial pupil when the true pupil is closed or severely up drawn.
- ***Four point iridotomy*** is done by a von Graefe's cataract knife to treat secondary glaucoma in iris bombe.

DE-WECKER'S IRIS SCISSORS (FIG. 27.14)

Identification: It is a butterfly-shaped spring scissors with the cutting blades bent at an angle of 60° with the handle. On the handle, there are two wings for index finger and thumb. Its one blade has pointed tip and the other blade has rounded tip.

Method: Scissors is held in such a way that the plane of blade lies at the same plane as of iris, whereas the handle is almost vertical.

Uses

- It is used to cut a piece of iris tissue in iridectomy.
- It is used for anterior or open-sky vitrectomy, if there is any vitreous loss during cataract extraction.
- It is used to cut the trabecular tissue with a part of sclera in trabeculectomy operation.
- It may be used to cut the suture ends (8–0 or 10–0 sutures) during corneascleral suturing.



Fig. 27.14: De-Wecker's iris scissors

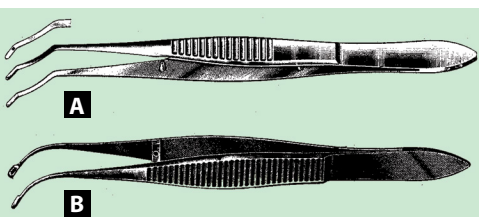
INTRACAPSULAR FORCEPS

Identification: There are two types of intracapsular forceps.

1. ***Elschnig's:*** It has no tooth, and is double curved with blunt tips (Fig. 27.15A).
2. ***Arruga's:*** It has also no tooth, and is single curved. Its tips are more blunt (knob-like) and have cups on the their inner surfaces (Fig. 27.15B).

Methods

- ***Lower pole delivery (Arruga's method)***
  - Here midperipheral lens capsule is grasped at 6 o'clock position by intracapsular forceps carefully, without catching the iris.



Figs 27.15A and B: Intracapsular forceps.
A. Elschnig's; **B.** Arruga's

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  - Side-to-side movement is made and simultaneous pressure over the zonules is given by the lens expressor (taken in the left hand) from outside to tear them.
  - Cataractous lens is taken out by tumbling (i.e. lower pole is delivered first).
- ***Upper pole delivery (Kirbi's methods)***
  - Here midperipheral lens capsule is grasped at 12 o'clock position after retracting the iris (sometimes, a complete iridectomy is done to facilitate upper pole delivery).
  - (Same as above) pressure from outside at limbus is given by lens expressor to break the zonules.
  - Lens is delivered by sliding (i.e. upper pole is delivered first).

Lower pole delivery (i.e. tumbling) is better as tumbling itself gives support to the anterior vitreous face, so there is less chance of vitreous loss.

Uses

- Intracapsular cataract extraction
- To remove capsular tags in extracapsular cataract extraction (ECCE)
- As a suture tier
- To hold suture ends during suture removal,
- To remove clots from the anterior chamber as in clotted hyphema
- To remove caterpillar hair.

Advantages of intracapsular forceps in cataract extraction:

- No assistant is required
- It is cheap and quick procedure—useful for camp surgery.

Disadvantages

- It is not preferred in intumescent or hypermature morgagnian cataract, where the capsule may be torn.
- It is a difficult procedure for the beginners.

Complications of Intracapsular Forceps Extraction

- Iris may be caught—leading to iridodialysis and hyphema.
- Capsule may be torn—leading to accidental ECCE.
- Lens may get dislocated into the vitreous due to excessive pressure.
- Corneal endothelial damage by the tip of the forceps.
- Capsular grasp may open up in the midway of the procedure.

Cryoextraction of Cataract (Fig. 27.16)

It is a very safe procedure and useful for the beginners. The gases used for the formation of iceball are: (i) Nitrous oxide and (ii) Freon.

Temperature at cryoprobe tip is about –40°C to –60°C.

Here a pencil-tipped insulated cryoprobe is applied over the anterior surface of the lens while retracting the iris. The assistant lifts the cornea during the whole procedure. When the iceball forms (i.e. when the adhesion of cryoprobe with the lens capsule and cortex is good) little side-to-side movement is made to tear the zonules. Thereby cataractous lens is extracted by sliding.

Advantages

- It is useful in all types of cataract, e.g. immature, mature and hypermature.
- Safe technique—for the beginners.

Disadvantages

- Accidental cryotouch to the cornea or iris may cause damage.
- Mechanical failure of the instrument.
- More costly and sometimes not portable.

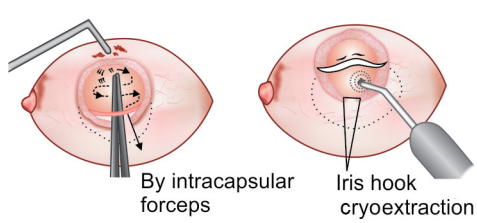


Fig. 27.16: Methods of intracapsular cataract extraction

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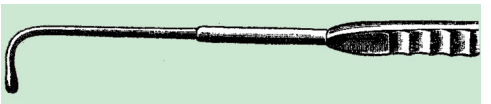


Fig. 27.17: Lens expressor (hook)

LENS EXPRESSOR (LENS HOOK) (FIG. 27.17)

Identification: It has a flat corrugated handle with a curved, olive-pointed blunt tip. The plane of the flatness of the handle is perpendicular to the plane of curvature of the limb.

Uses

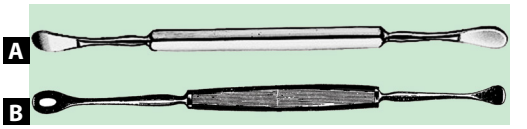
- During intracapsular-extraction of the lens, either by intracapsular forceps or by Smith-Indian technique.
  Pressure is applied from out side at the limbus and adjacent part of the cornea at 6 o'clock position. This is to facilitate zonular tear. Then a gradual support is given at the lower pole of the lens.
- In case of extracapsular cataract operation—to deliver the nucleus.
- Milking out the cortical materials with the knee or bend of the instrument in ECCE.
- It may be used to hook the extraocular muscles.
- It may also be used as a fine tissue retractor, particularly in DCR operation to retract the sac during punching or breaking the bone.

McNAMARA'S SPOON (LENS SPATULA)

Identification: It is a straight long instrument with two flat spoons or spatula at the two ends. One of the spoons may be perforated at its center (Figs 27.18A and B).

Uses

In intracapsular cataract extraction: Along with lens hook, the lens is delivered by pressure and counter-pressure (Smith-Indian technique). Spoon is in the left hand outside the section and lens expressor is in the right hand at the lower limbus. By giving pressure and counter-pressure, lens is delivered out by tumbling or by sliding.



Figs 27.18A and B: **A.** Lens spatula; **B.** McNamara's spoon

Advantages

- No instrument is introduced into the anterior chamber—so endothelial damage is less.
- Useful in camp surgery—as this method works best in hypermature cataract (in camp number of hypermature cataract is more).
- No assistant is needed.

Disadvantages

- Not a very good method for immature cataract.
- Chance of vitreous loss is more.
- A difficult technique.
  - To deliver the nucleus in extracapsular cataract operation.
  - The perforated spoon end may be used as a vectis to deliver the subluxated or dislocated lens.

Erysiphake extraction of ICCE: It is an instrument that uses vaccum or negative pressure to grasp the lens (anterior lens capsule and cortex) in intracapsular cataract extraction.

It is not used now-a-days. It was used previously to deliver the immature cataractous lens where intracapsular forceps could not be used.

VECTIS (WIRE VECTIS) (FIG. 27.19)

Identification: It is a ring of wire (round or oval) at the end of a narrow limb, attached to a handle (like a large platinum loop). It may be little curved like a spoon.

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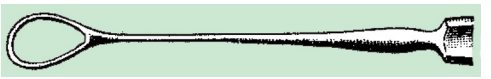


Fig. 27.19: Wire vectis

Method: Pupil must be fully dilated. Never pass it blindly. The lens edge should be visible. Carefully pass the vectis behind the lens. Lift the lens and then take the lens out. Vitreous loss is inevitable in vectis delivery. Sometimes, a sector iridectomy is needed instead of peripheral iridectomy. Open-sky vitrectomy is done after lens delivery.

Uses

- To remove a subluxated or dislocated lens.
- To deliver the nucleus in ECCE with irrigating vectis.
  If the vectis is not available, the instruments may be used as vectis:
  - Perforated end of McNamara's spoon.
  - Intracapsular forceps—here apply forceps with closed tips, carefully pass it behind the lens and then open the tips little bit so that lens sit well on it. Now, lift the lens and remove it.

IRIS REPOSITOR (FIG. 27.20)

Identification: It is an elongated 'S' or 'Z'–shaped instrument with a stout handle and two long narrow flattened extrimities. Both the edges and tips are blunt. Its one end may be curved and other end may be angulated.

Method: A few strokes over the peripheral iris on its upper part is required to reposit the iris. When the iris properly repositioned the pupil becomes perfectly circular and central.

Uses

- To reposit the iris after the delivery of lens in ICCE or after nucleus delivery in ECCE.
  Infact, the assistant must be ready with this instrument when the surgeon is delivering the lens or nucleus.
- To reposit the iris after iridectomy.
- To retract the iris during cryoprobe application over the anterior surface of the lens in ICCE.
- To bring air from posterior chamber to anterior chamber during intracapsular cataract surgery.
- To break the adhesions in synechiae (synechiolysis).
- To bring back the folded conjunctiva to cover the wound.
- Lamellar dissection of the cornea in lamellar keratoplasty.

Precaution: Iridectomy site should be avoided during repositioning of the iris. If reposition is not done properly there may be:

- Iris incarceration at the wound
- Prolapse of the iris
- Chance of infection
- Gaping of the wound
- Non-reformation of the anterior chamber.

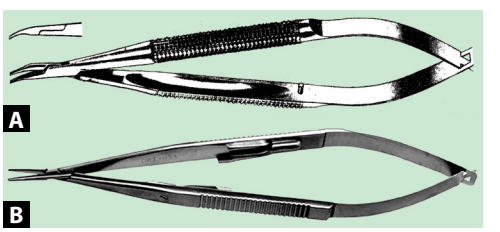


Fig. 27.20: Iris repositor

NEEDLE HOLDER (BARRAQUER'S AND CASTREVEIJO'S) (FIG. 27.21)

Identification: It is a medium-sized spring needle holder with two narrow and fine curved jaws. It may be available with or without a catch.

Uses: It is used to hold the fine needles (of 6-0, 8-0 and 10-0 sutures) for:



Figs 27.21A and B: Needle holder. **A.** Barraquer's; **B.** Castreveijo's

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- Corneoscleral suturing after cataract operation.
- Scleral suturing in squint, detachment and trabeculectomy operations.
- Corneal suturing in penetrating injury or keratoplasty.
- Suturing the mucosal flaps in DCR operation.
- Sometimes in conjunctival suturing.

Castroveijo's needle holder is mainly required to hold slightly larges needles (of 4–0 to 6–0 sutures) for conjunctival suturing as in squint, glaucoma, pterygium or retinal detachment.

COLIBRI FORCEPS (FIG. 27.22A)

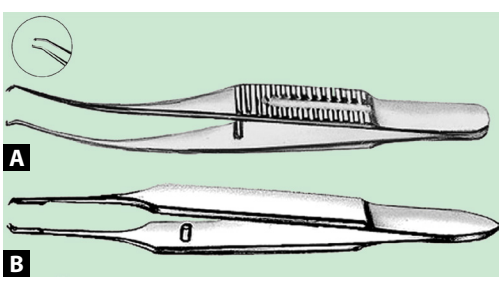
Identification: It is a curved or angular forceps with fine limbs, having 1:2 teeth at its tip. It is thicker and stout.

ST. MARTIN'S FORCEPS (FIG. 27.22B)

Identification: It is a straight small but stout forceps with 1:2 teeth at its fine tip.

Uses: Both instruments have similar uses:

- To hold the cornea and scleral lips during corneoscleral suturing after cataract surgery, and corneal lips in repair of penetrating injury or wound closure in keratoplasty.



Figs 27.22A and B: **A.** Colibri forceps; **B.** St. Martin's forceps

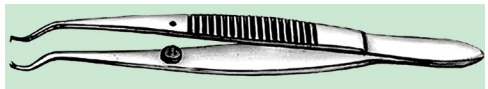


Fig. 27.23: Superior rectus holding forceps

- It is also used to hold the scleral lip for dissection and scleral suturing (as in trabeculectomy operation).
- It is used to hold the corneal lip in cataract operation to extend the section with corneal scissors.
- To lift the cornea upwards during application of the cryoprobe.
- It may be used to catch the iris for iridectomy.

SUPERIOR RECTUS HOLDING FORCEPS

Identification: It is a stout forceps with double curvature (S-shaped) at its ends. It has 1: 2 teeth at its tip. Its curvature at the tip is to fit with the curvature of the globe (Fig. 27.23).

Uses: It is used to catch the superior rectus muscle belly for passing stay (bridle) sutures, so that the eyeball can be rotated and fixed downwards in cataract, glaucoma or other surgery. It is also used to catch the inferior rectus muscle as in keratoplasty.

SUTURE TIER FORCEPS (FIG. 27.24)

Identification: It is a small straight or curved forceps with long fine limbs. It does not have any tooth at its tip. The tips are made stout by extra thick platform.

Uses

- To hold the suture ends during tying the sutures after proper tightening.

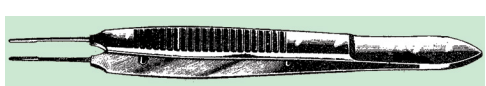


Fig. 27.24: Suture tier forceps

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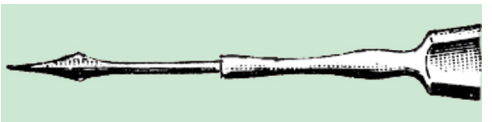


Fig. 27.25: Bowman's discission needle

- To hold the cut ends of the suture during its removal.
- May be used to catch the margin of incised conjunctiva during suturing.
- To remove caterpiller hair.

BOWMAN'S DISCISSION NEEDLE (FIG. 27.25)

Identification: It is a straight fine needle, attached to a handle. Its tip is very sharp, pointed and diamond-shaped. There may be a guard at the middle of the needle to prevent deeper penetration into the eye.

Uses

- Discission or needling of congenital and childhood-traumatic cataract.
- Needling of 'after-cataract' to create an aperture in the pupillary area.

ZEIGLER'S KNIFE (FIG. 27.26)

Identification: It is a tiny curved or sickle-shaped, sharp-pointed knife-blade at the end of narrow limb, attached to a handle.

Uses

- Discission of 'after-cataract' to create a gap in the pupillary area.
- In discission or needling of congenital or traumatic cataract.
- To incise the iris, in up-drawn pupil complicated by cataract surgery.

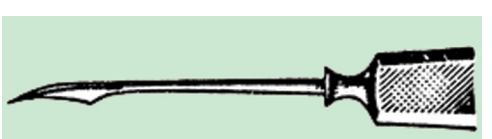


Fig. 27.26: Zeigler's knife

- To cut and separate the iris in adherent leucoma.

Surgery in congenital, developmental or childhood-traumatic cataract: Operation should be done as early as possible to prevent amblyopia (may be within 6 weeks after birth).

- ***Needling and aspiration***
  - ***Single stage:*** As in total cataract.
  - ***Two stages:*** As in partial cataract.
    Not used now-a-days because repeated surgeries are needed due to quick posterior capsule opacification (PCO) or after-cataract formation in almost all cases.
- ***Planned extracapsular cataract extraction (ECCE):*** As in adult ECCE. Posterior chamber intraocular lens (PCIOL) implantation may be considered along with ECCE if the child is more than 3 years.
- Extracapsular cataract extraction with PCIOL with primary posterior curvilinear capsulorhexis.
- Pars plana lensectomy with anterior vitrectomy is also an alternative choice.

LASER TREATMENT FOR POSTERIOR CAPSULE OPACIFICATION

This is specially useful in PCO following ECCE with PCIOL implantation. It is known as ***YAG laser capsulotomy***. It is done with Nd-YAG laser beam by a slit-lamp delivery system (Nd means ***neodynium***; YAG means ***yittrium aluminium garnet***).

This laser acts by photodisruption of the tissues.

Advantages

- It is a non-invasive procedure, chance of infection is nil.
- It is an OPD procedure.
- It is safe and painless.

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Disadvantages

- Not widely available in district or sub-divisional level as it is costly.
- Retina check up is essential after the procedure.
- It is very difficult in children and non-cooperative patient.

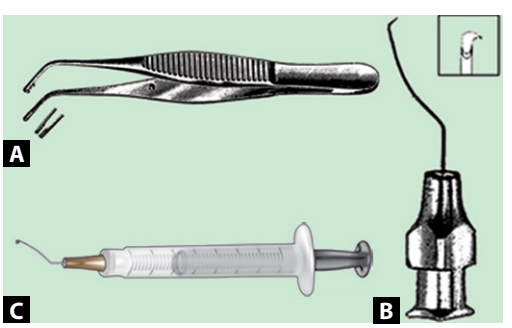
CAPSULOTOMY FORCEPS

Identification: It is a small curved forceps having 3:4 teeth hanging downwards from the end of the curved limbs (Fig. 27.27A).

Uses: To tear the anterior capsule in extracapsular cataract operation.

It is not used now-a-days as the capsular opening is not perfect and there may be damage to the corneal endothelium. It was done by open method, i.e. after limbal section.

Now-a-days a bent tipped 26 gauze needle is used as a capsulotome or cystitome (Fig. 27.27B) which is fitted with a 2 mL syringe. Here, the capsulotomy is done by a close method after making limbal groove. The syringe is filled with balanced salt solution (BSS) (Fig. 27.27C) or Ringer's lactate to keep the anterior chamber well formed during needle manipulation. The capsule is cut with the bent tip of the needle in a continuous circular fashion capsulorhexis, or by interrupted multiple puncture by can-opener technique or stamp-hole technique. After withdrawing the needle, the limbal section is extended and loose circular anterior capsule is removed.



Figs 27.27A to C: A. Capsulotomy forceps, **B.** Cystitome, **C.** Balanced salt solution filled 2 cc syringe with needle (26 G) Cystitome



Fig. 27.28: Capsulorhexis forceps (Utrata)

CAPSULORHEXIS FORCEPS (UTRATA FORCEPS) (FIG. 27.28)

Identification: It is a fine long forceps with curved sharp tip without any tooth.

Uses: To perform continuous curvilinear capsulorhexis (CCC) in case of manual SICS or in phacoemulsification.

The initiation of rhexis is started with a 26 G needle, and then completed with the rhexis forceps, or it may be with the rhexis forceps alone.

It may be used to remove small capsular tag.

IRRIGATING VECTIS (FIG. 27.29)

Identification: It is a hollow vectis fitted with a needle base. Its tip has 3–5 small openings for free passage of irrigating solution. The maximum horizontal diameter of the vectis is 5.0–5.5 mm.

Use: The vectis is connected with the tubing system of a Ringer's lactate or BSS bottle, or it may be fitted with a Ringer's lactate or BSS filled 2 cc syringe during nucleus delivery.

The irrigating fluid creates hydrostatic pressure and at the same time mechanical pull by the vectis is to deliver the nucleus out. Posterior scleral lip depression and simultaneous counter balancing force by

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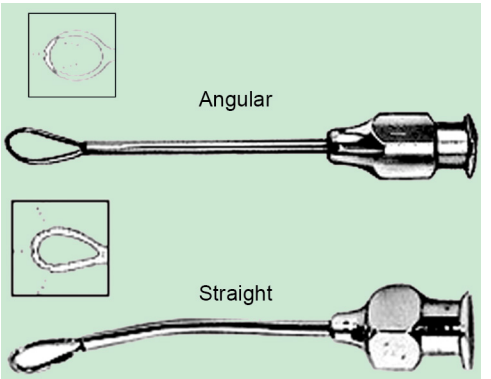


Fig. 27.29: Irrigating vectis

superior rectus bridle suture are the important steps during nucleus delivery.

IRRIGATION-ASPIRATION TWO-WAY CANNULA (SIMCOE CANNULA)

Identification: It is a two-way cannula, one end of which is attached to a blunt needle via a silicone tube. Normally, this end is fitted with an irrigation or infusion system and the two-way cannula itself is fitted with a 2 mL or 5 mL syringe for aspiration (Fig. 27.30).

Method: After delivery of the nucleus in ECCE, the cannula in introduced into the anterior chamber. The infusion is slowly started either from a hanging Ringer's lactate or BSS bottle (or, alternately assistant may irrigate via BSS filled 10 mL syringe). As the anterior chamber is formed, the surgeon starts aspirating cortical material slowly till the posterior capsule is cleaned (as appreciated by brilliant fundal glow under co-axial illumination of the operative microscope).

Uses

- For simultaneous irrigation and aspiration of cortical materials in case of ECCE or ECCE with PCIOL.
- To remove viscoelastic material after insertion of IOL.
- May be used to remove blood in hyphema.

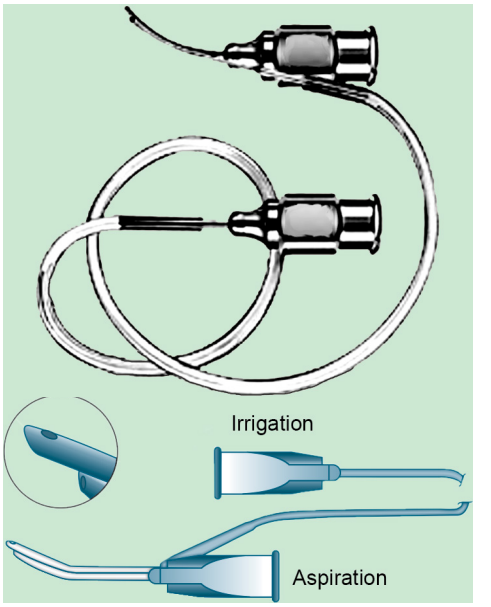


Fig. 27.30: Irrigation-aspiration two-way canula (Simcoe cannula)

McPHERSON'S FORCEPS (FIG. 27.31)

Identification: It is a fine medium-size toothless forceps with an angulation at about 7–8 mm from its tip.

Uses

- It is used to hold the intraocular lens during its placement in the posterior chamber.
- It is used to catch and remove the loose anterior capsule after completion of capsulotomy.
- It may be used as a suture tier during suturing with 10–0 nylon.

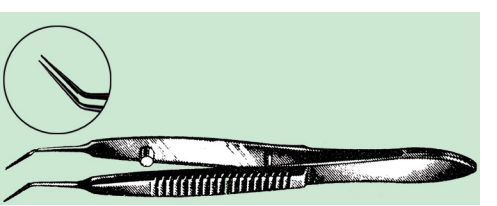


Fig. 27.31: McPherson's forceps

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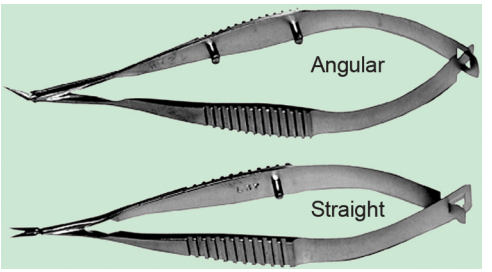


Fig. 27.32: Vannas' scissors

VANNAS' SCISSORS (FIG. 27.32)

Identification: It is much smaller scissors with spring action. It may be straight, curved or angular.

Uses

- To prepare conjunctival flap.
- To cut 10–0 sutures in cataract or keratoplasty operation.
- To cut trabecular flap in trabeculectomy.
- To cut anterior capsular tags in ECCE.
- To cut vitreous in open-sky vitrectomy, as in vitreous loss during ICCE or ECCE.
- To cut corneal button after trephination in corneal grafting.
- To cut iris in different types of iridectomy.

INTRAOCULAR LENS DIALLER (SINSKY'S HOOK) (FIG. 27.33)

Identification: It is an angular fine hook attached to a long round solid handle.

Methods and uses

- It is used to dial the IOL for the purpose of centration and bringing the IOL-haptics in horizontal position. The hook is positioned in the dialling holes of the optic of IOL, and then to rotate in clockwise manner.



Fig. 27.33: Intraocular lens dialler (Sinsky's hook)



Fig. 27.34: Sclerocorneal splitter

- It may be used as a left hand instrument during phacoemulsification:
  - To rotate the lens nucleus.
  - To crack the lens nucleus after doing quadrant trenching.
  - To chop the nucleus.
- It is used to break the posterior synechiae (synichiolysis) during ECCE.

SCLEROCORNEAL SPLITTER (CRESCENT KNIFE) (FIG. 27.34)

Identification: It has a thin cresecntic blade, at the end of a neck and attached to a polycarbonate handle. The cutting edge blade may be beveled-up or beveled-down. The blade has a forward angulation of 45° for better and parallel movement during dissection.

It is available as a disposable blade in pre-sterile pack. It may be reused for 3–4 times after ETO sterilization.

Uses

- Used to make sclerocorneal tunnel in manual SICS or phacoemulsification.
- Used for lamellar dissection of the cornea in lamellar keratoplasty.

ANGULAR KERATOME

Identification: It has a thin triangular blade, at the end of a neck and attached to a polycarbonate handle. The cutting edge blade may be beveled-up or beveled-down. The blade has a forward angulation of 45° for better and parallel movement during dissection. The width of the blade may be 2.8–3.0 mm diameter (for phacoemulsification). or 4.5–5.5 mm for manual SICS (Fig. 27.35).

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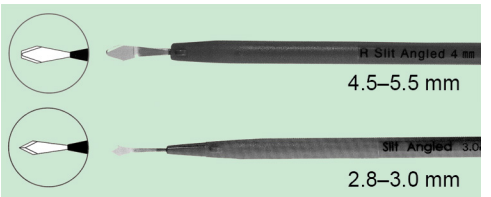


Fig. 27.35: Angular keratome

It is also available as a disposable blade in presterile pack. It is advisable not to reuse these disposable blades.

Uses

- Used to enter into the anterior chamber after making sclerocorneal tunnel in manual SICS or phacoemulsification.
- It may be used directly for making corneal section in phacoemulsification.
- 4.5–5.5 mm diameter keratome is used to enlarge the tunnel.

SIDE-PORT BLADE (FIG. 27.36)

Identification: It has a thin, long straight blade, at the end of a neck and attached to a polycarbonate handle. Sometimes, it has got 15° angulation. The maximum width of the blade is to pass a 20 G needle.

It is also available as a disposable blade in presterile pack which may be reused.

Uses

- To make the side-port (to enter into the anterior chamber from the side of main incision) in cataract surgery.
  Indication of side-port
  - For paracentesis
  - To inject viscoelastic substance
  - To inject trypan blue dye
  - To clean subincisional lens cortex by irrigation aspiration cannula
  - To manipulate second instrument (like phaco-chopper, Sinsky hook, etc.) in case of phacoemulsification. Some times, two side-ports are given on either side of main port for bimanual irrigation and aspiration
  - To insert the anterior chamber maintainer.
- It is also used for paracentesis to drain blood in hyphema.
- To remove small intracameral foreign body.



Fig. 27.36: Side-port blade

EPILATION FORCEPS (CILIA FORCEPS)

Identification: It is a small stout forceps with blunt flat ends. Near the tip, the inner surfaces are reinforced by extra thick platform which may be rhomboidal or elliptical (Figs 27.37A and B).

Uses

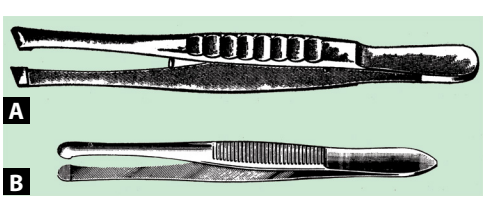
- To remove the offending, misdirected eyelashes in trichiasis.
- To remove the involved eyelash in stye.

No anethesia is required for epilation. It is not an ideal technique as it is not a permanent procedure. As the root is not destroyed, eyelash grows again within 6–8 weeks and causes same irritation.

Ideal method should be permanent where the hair-root is destroyed.

Ideal methods are:

- ***Electrolysis:*** Anesthesia is required. A fine needle (the negative pole) is introduced into the hair follicle and a current of 2 mAmp is passed. End point is noted by:



Figs 27.37A and B: **A.** Rhomboidal tip; **B.** Eliptical tip

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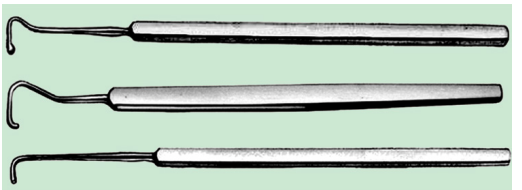


Fig. 27.38: Strabismus hooks

- Appearence of foam due to liberation of nescent hydrogen and saponification, at the root.
- Eyelash can be easily lifted (no pulling against resistance).

- ***Electrodiathermy:*** A current of 30 mAmp is applied for 10 seconds.
- ***Cryosurgery:*** Pigmentation is a problem.
- If more than one-third lid margin is involved with trichiasis—partial entropion correction operation is better choice.

STRABISMUS HOOK (MUSCLE HOOK OR SQUINT HOOK) (FIG. 27.38)

Identification: It has a solid handle with a long narrow limb with 90° bent at its tip. The tip may be sharp or knobbed. The handle is not corrugated and the plane of curvature of the limb is same as the plane of handle.

Uses

- It is used to hook the extraocular muscles during:
  - Squint operation
  - Retinal detachment operation
  - Enucleation operation.
- Occasionally, it may be used as tissue retractor.

LID RETRACTOR (DESMARRE'S) (FIG. 27.39)

Identification: It is a saddle-shaped folded instrument at the end of a metallic handle. It is of varying size depending upon the size of the palpebral aperture. It is not self-retaining.

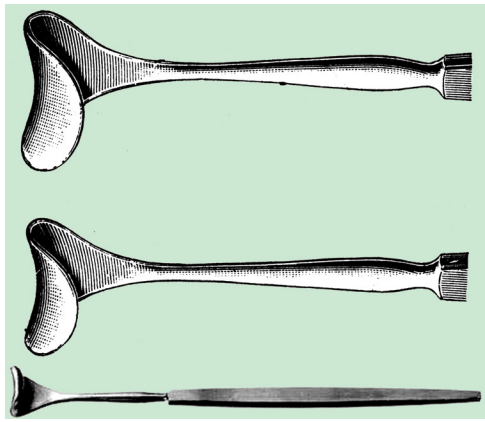


Fig. 27.39: Desmarre's lid retractor

Method (for children)

- Patient is made to lie on the table and an assistant must fix his head.
- Lignocaine (4%) eye drop is instilled.
- Lid retractor is introduced in the eye from the temporal side sliding against the eyeball.
- Upper lid is retracted and the eye is examined.
- As the lower lid can be easily retracted by finger normally, lower lid retractor is not necessary. But in some cases of severe blepharospasm, two retractors are needed.

Uses

- It is used for examination of conjunctiva and anterior segment of the eye—to find out any abrasion, ulcer, foreign body on the cornea or any abnormality:
  - In children and non co-operative patient.
  - In case of severe blepharospasm and photophobia.
  - In case of lid edema.
- For removal of corneoscleral stiches (better than eye speculum) after cataract surgery.
- To examine upper fornix by double eversion of the lid.
- It may be used as a tissue retractor (e.g. to retract conjunctiva and Tenon's capsule in squint and detachment operation).

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FOREIGN BODY SPUD AND NEEDLE (FIGS 27.40A AND B)

Identification: The spud has a straight narrow limb with blunt tip with a handle. One surface of the limb is flat and the other is convex. The needle is tiny scoop with sharp pointed tip on a narrow limb with a handle. These two instruments may be available with one handle—one on either end.

Methods and uses

- The spud is used to remove the superficial corneal foreign body. With the convex smooth surface of the spud, foreign body is removed by gentle stroke.
- The needle is used to remove the embeded foreign body from the cornea.

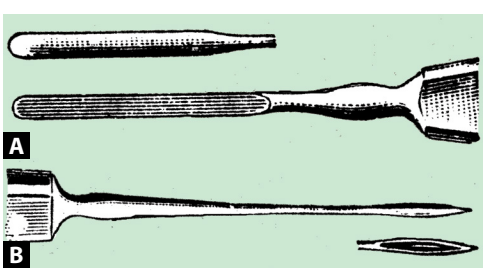
In both the cases, the direction of the stroke is from the central part towards the periphery. Otherwise, the central part of the cornea may be damaged.

Both the instruments are not used now-a-days because of their broader tip which may cause more damage to the cornea.

Steps for removal of foreign body:

- Search for the foreign body with a good torch light and if necessary with a loupe (a slit-lamp is much better).
- Evert the upper lid to find out any foreign body in the subtarsal sulcus. ***This is the most common site of lodgement of a foreign body.*** If it is there, remove it with a sterile cotton swab or pellet, even without anesthesia.
- If the foreign body is on the cornea, put 4% lignocaine or 1% proparacaine drop to anesthetize the ocular surface.
- If it is superficial, remove the foreign body with a sterile cotton swab or a sterile triangular blotting paper simply by touching it.
- If it is not possible to remove it, take a disposable sterile hypodermic needle of any size and remove the foreign body by lifting. It will cause only a point damage. Do not scratch and cross the pupillary zone of the cornea.
- If the foreign body is embedded into the deeper stroma of the cornea, take the patient to the operation theater and remove the foreign body with a needle under operative microscope.
- In all cases apply antibiotic ointment and if necessary cycloplegic drop. Put a pad and bandage for 24 hours. Examine the patient on the next day, and frequent antibiotic drops for the next few days.
- In case of old iron foreign body, there is formation of rust-ring around the foreign body. It is also to be removed carefully with a needle.
- In all cases of corneal foreign body, always examine the lacrimal sac by giving pressure over the sac region to rule out any chronic dacryocystitis.

If it is present, the puncta is temporarily occluded by thermocautery and antibiotic drops and ointment are to be applied frequently after removal of foreign body.



Figs 27.40A and B: A. Foreign body spud; **B.** Foreign body needle

TOOKE'S KNIFE (SCLERACORNEAL SPLITTER) (FIG. 27.41)

Identification: It is a short, flat blade with semicircular cutting edge at its tip. It is bevelled on both sides at its distal end. It is

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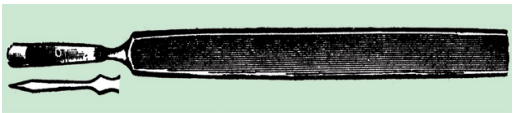


Fig. 27.41: Tooke's knife

attached to a stout handle with short neck.
Note: Rougine has a long neck, the blade is more flat and bevelled on one side.

Uses

- For splitting the sclerocorneal junction during trephining operation in glaucoma.
- Used to clear the sclerocorneal junction or episcleral tissue in cataract or trabeculectomy.
- May be used for lamellar dissection of the sclera.

ELLIOT'S SCLERACORNEAL TREPHINE IDENTIFICATION (FIG. 27.42)

- Fine cylindrical blades with sharp cutting edge and
- A stout aluminium cylindrical handle.

These two portions can be detached. The diameter of the blade may be 1.0 mm, 1.5 mm, 1.75 mm or 2.0 mm.
Use: To cut the scleracorneal disc in a circular fashion in trephining operation for glaucoma.
Trephine means—circular blade. Larger diameters of trephine (6.0 to 10.0 mm) is widely used in keratoplasty operation to make corneal button both from the donor and the recipient.

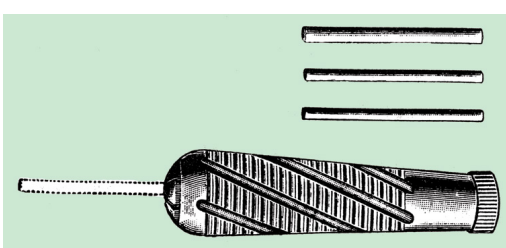


Fig. 27.42: Elliot's trephine with handle

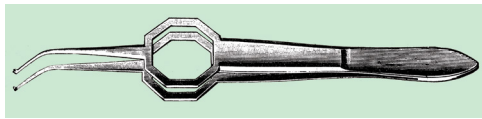


Fig. 27.43: Disc-holding forceps

DISC-HOLDING FORCEPS (FIG. 27.43)

Identification: It is a fine forceps with 1:2 teeth on the undersurface at the tip the limbs. The blade of the forceps has large hexagonal hole for holding.
Use: It is used to hold the sclerocorneal disc, made by the trephine during trephining operation for glaucoma. After holding the disc, it is cut by scissors.
Tooke's knife, Elliot's trephine and disc-holding forceps are not used now-a-days, and Elliot's sclerocorneal trephining operation for glaucoma is obsolete. The standard procedure for glaucoma filtration operation is trabeculectomy for the last 20 years.

CHALAZION FORCEPS (CLAMP) (FIG. 27.44)

Identification: It is a forceps with a large screw for fixing or tightening the limbs like a clamp. One limb has got a solid disc-shaped plate and the other limb has a ring at its end. It is hemostatic and self-retaining.
Method: The solid plate is applied on the skin surface of the lid and the ring side is applied on the tarsal conjunctiva, encircling the chalazion. The screw is tightened and the lid is everted and then chalazion is exposed for incision.

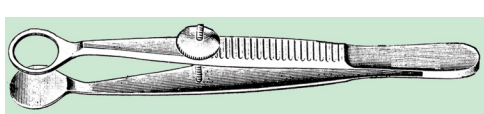


Fig. 27.44: Chalazion forceps

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The functions of the screw are:
- Fixation of the lid
- Hemostasis by means of tightening.

Uses
- To fix the chalazion for surgery and also to ensure hemostasis.
- To give intralesional injection of steroids in chalazion after fixing it with forceps.
- Excision of a small granuloma or papilloma of the lid.

BEER'S KNIFE (FIG. 27.45)

Identification: It is a large triangular knife with a sharp pointing cutting edge.

Method and uses
- It is used to give incision on palpebral conjunctival side of the chalazion after fixing it with forceps. The Beer's knife is not used routinely. Now-a-days the incision is given by a no. 11 blade attached to the Bard-Parker handle or by side-port blade.
- Beer's knife may be used to give incision over skin in any lid surgery.

The incision is vertical to avoid injury to the meibomian glands and ducts as they are arranged vertically.

Sometimes, when the pus is pointing towards the skin surface the fixation plate and ring of the chalazion clamp is reversed. Then a horizontal incision is given on skin surface. This reverse type of incision is given because:
- Vascular arcades are running horizontally so less chance of bleeding.
- The scar mark will be along the natural crease of lid skin.

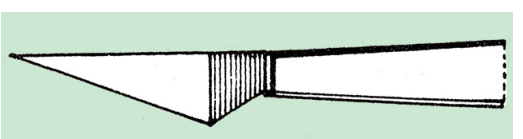


Fig. 27.45: Beer's knife

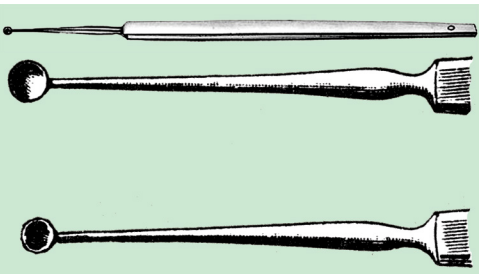


Fig. 27.46: Chalazion scoop

CHALAZION SCOOP (CURETTE) (FIG. 27.46)

Identification: It is a small scoop with sharp edge, attached to a handle. The size of the scoop may vary. Two different sized scoops may be attached to the two ends of same handle for convenience.

Uses: To scoop out the granulation tissue after giving incision on chalazion. Scooping of the chalazion must be complete, otherwise there may be chance of recurrence. After removal of the chalazion forceps, the bleeding is controlled by pressure over the lid, against the incised area. A pressure bandage is applied for few hours with antibiotic ointment.

PUNCTUM DILATOR (NETTLESHIP'S) (FIG. 27.47)

Identification: It is a long narrow solid cylindrical instrument with a smooth conical pointed tip. Its body is corrugated for better gripping with thumb and index finger.

Method of syringing
- Anesthetize the eye with 4% lignocaine or 1% proparacaine drop.
- Pull the lower lid and identify the lower punctum in bright light.



Fig. 27.47: Punctum dilator

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- Hold the punctum dilator vertically by right index and thumb and place it on the punctal opening.
- Twist it with light pressure and introduce into the punctum.
- Then hold the punctum dilator horizontally and push it medially by rotatory movement—following the course of the canaliculus (first vertically then horizontally). Then withdraw it.
- Take the lacrimal cannula, fitted in a syringe, filled with distilled water and introduce it in the same direction.
- Push the piston of the syringe to inject water into the canaliculus and ask the patient whether water has reached the throat or not. Alternatively, note the swallowing movement of the neck.

Uses

- To dilate the punctum and part of the canaliculus before introducing lacrimal cannula for syringing.
- To dilate the punctum for probing in case of congenital dacryocystitis.
- To dialate the punctum and then probing to identify lacrimal sac during DCR operation.
- For dilatation of the punctum in congenital or acquired punctal stenosis.
- Before dacryocystography (DCG).
- May be used as a marker (by dipping the pointed tip in gention violet) in squint or retinal detachment operation.

LACRIMAL CANNULA (FIG. 27.48)

Identification: It is a small straight angulated or curved canula with blunt tip. It can be fitted to any hypodermic syringe.

Method: Same as punctum dilator.

Uses

- Syringing or patency test of the lacrimal passage.
- For dacryocystography (DCG).

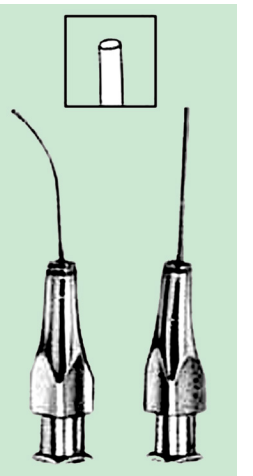


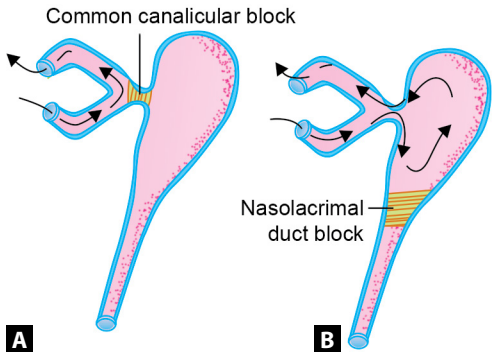
Fig. 27.48: Lacrimal cannula

- During probing and syringing in congenital dacryocystoitis.
- Postoperatively, it is used to check the patency after DCR operation.

In patency test or syringing, the interpretation may be:

- Water is going freely into the throat—lacrimal passage is patent.
- Water is not going into the throat, instead it is regurgitating through the upper punctum and partly through the same punctum.
  - ***Fast regurgitation:*** The fluid coming out through the upper puncta, is clear and it is rapid. It is due to common canalicular block (CC block) (Fig. 27.49A).
  - ***Slow regurgitation:*** It is turbid fluid (i.e. water is mixed up with pus, mucopus or mucoid material) and the regurgitation speed is slow. It is due to nasolacrimal duct (NLD) block (Fig. 27.49B).
- If little water is going into the throat after forced syringing (some part is coming out through punctum). It is partial NLD block. In case of NLD block—the treatment of choice is DCR operation. But in case of CC block the treatment of choice is CDCR (canaliculo-DCR.)

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Figs 27.49A and B: Patency test. **A.** Fast regurgitation; **B.** Slow regurgitation

LACRIMAL DISSECTOR WITH SCOOP (LANG'S) (FIG. 27.50)

Identification: It is a narrow long instrument with a stout pointed dissector at one end, and the other end is having an elongated scoop.

Uses

- ***Dissector end***
  - It is used for blunt dissection of lacrimal sac after giving skin incision (both in DCT and DCR operation).
  - It is used to clean the bone pieces from the punch, while punching or cutting the bone of DCR.
- ***Scoop end:*** To scoop out the tissue remnants by scraping from the upper end of bony NLD after excision of the lacrimal sac (only in DCT).

ROUGINE (FIG. 27.51)

Identification: It is rectangular blade attached by a narrow neck to a corrugated handle. The blade is sharp at its distal end which is beveled on one surface, the other surface being plane. It is basically a small periosteal elevator.



Fig. 27.50: Lacrimal dissector with scoop



Fig. 27.51: Rougine

Uses

- To dissect and disinsert the medial palpebral ligament to find out cleavage between lacrimal sac and the lacrimal fossa.
- Dissection of the lacrimal sac from the medial wall and the floor of the lacrimal fossa, by separating the periosteal tissue surrounding the sac.

  It is used both in DCT and DCR operation.

MULLER'S (SELF-RETAINING ADJUSTABLE HEMOSTATIC) RETRACTOR (FIG. 27.52)

Identification: It is made up of two limbs with a screw to fix the limbs in a retracted position. Each limb has got two or three right-angled curved pointed hooks (pins) for engaging the incised edges of the skin and deeper tissues.

Uses: To retract the incised edges of the skin and deeper tissues during dacryocystectomy and dacryocystorhinostomy operations.

Its hemostatic effect is due to angled hooks causing compression of the blood vessels by the spring action of the retractor.

This instrument has disadvantage of reducing field of operation. Instead, one can pass 3 deep stay sutures on each skin-edge of incision to achieve retraction and hemostasis.

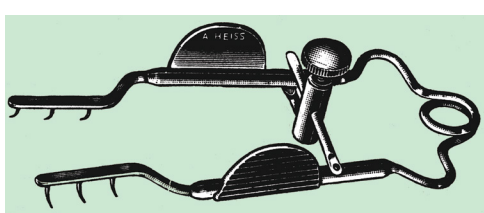


Fig. 27.52: Muller's retractor

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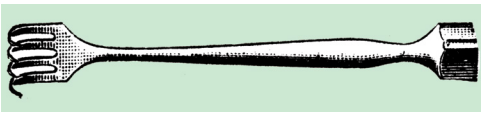


Fig. 27.53: Cat's paw retractor

The complications may occur during giving incision to the skin are:

- Injury to the angular vein
- Cutting the medial palpebral ligament and
- Injury to the lacrimal sac inadvertently.

The structures cut to reach the lacrimal sac proper are:

- Skin
- Subcutaneous tissue
- Few fibers of orbicularis oculi
- Medial palpebral ligament and
- Anterior lacrimal fascia.

CAT'S PAW RETRACTOR (FIG. 27.53)

Identification: This instrument is just like a small dinner fork. The ends of the fork are bend downwards.

Uses

- It is used to retract the skin, subcutaneous tissues and ligament during lacrimal sac surgery.
- It may also be used in other plastic operations of the eyelid.

BONE PUNCH (FIG. 27.54)

Identification: It is a large instrument, which consists of a spring handle and two long blades. The upper blade has a hole with sharp cutting edge and the lower blade has a cup like depression with sharp edge.

Use: It is used to punch or break the bones (lacrimal bone, adjacent nasal bone and frontal process of the maxilla) in DCR operation to create a bony ostium. Ideally, two different sized bone punches are required to make a round bony opening of about 10 mm diameter.

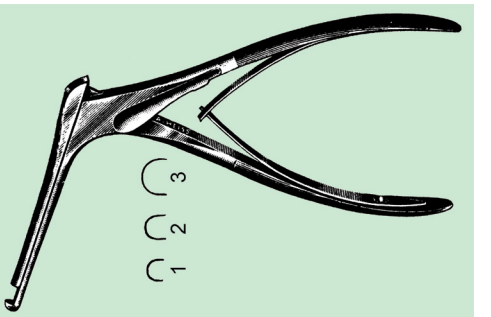
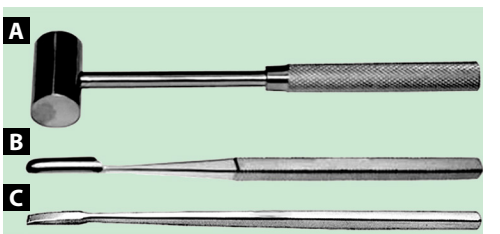


Fig. 27.54: Bone punch



Figs 27.55A to C: **A.** Hammer; **B.** Bone gouge; **C.** Chiesel

HAMMER, CHIESEL AND BONE GOUGE (FIG. 27.55)

These stout and heavy instruments are not routinely used during DCR operation. But when the bones of the lacrimal fossa are seem to be hard, these instruments are then used for making a bony ostium (osteotomy) in DCR surgery.

Usually, first the lacrimal bone is broken with a lens hook or blunt dissector, then the bony window is made with help of a bone punch.

EVISCERATION SPOON OR SCOOP (FIG. 27.56)

Identification: It has a large rectangular or oval shallow spoon attached to a handle. Its shape is almost like a spade.

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Fig. 27.56: Evisceration scoop

Uses: It is used to scoop out the intraocular contents during evisceration. Evisceration is the removal of the contents of the globe with inner two coats while leaving the sclera and the optic nerve intact.

The structures scooped out are:

- Lens
- Vitreous
- Whole uvea and
- Whole retina.

Indications of evisceration:

- Panophthalmitis,
- In case of expulsive hemorrhage—to assist auto-evisceration if the process is incomplete—rarely performed.

Method: Steps of operation *(see* Chapter 10*).*

In case of painful blind eye or disfigured eye-evisceration is relatively contraindicated—as the remnant of healthy uveal tissue may excite sympathetic ophthalmia. In case of malignancy, evisceration may cause spread of the tumor cells via the bloodstream.

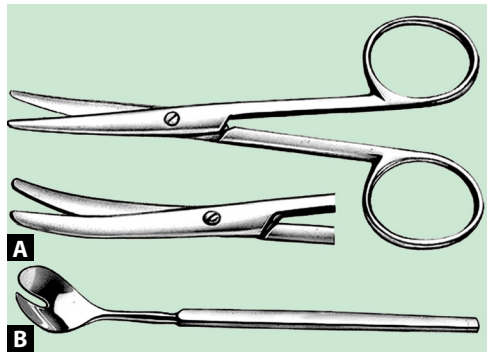
But in case of panophthalmitis as the uveal tissue is dead and necrotic, there is no antigenecity of the uveal tissue, so there is no chance of sympathetic ophthalmia.

ENUCLEATION

Scissors (Fig. 27.57A)

Identification: It is a broad, stout scissors having uniformly curved blades with blunt tips. The scissors is curved to fit the curvature of the globe.

Use: It is used to cut the optic nerve with its sheaths during enucleation operation. Enucleation is the surgical removal of the globe and a portion of the optic nerve from the orbit.



Figs 27.57A and B: Enucleation, **A.** Scissors; **B.** Spoon with optic nerve guard

Spoon (Fig. 27.57B)

Use: After severing all the recti muscles, it is passed from the lateral side deep inside the orbit to get hold the optic nerve. Then, the scissors is used to cut the optic nerve.

Indications of enucleation

- ***Absolute indications:*** When there is risk of life, or risk to the other eye due to the disease.
  - Malignant melanoma in adult
  - Retinoblastoma in children
  - Non repairable severely injured eye to prevent sympathetic ophthalmia in the other eye.
- ***Relative indications:*** Enucleation may be done in case of:
  - Painful blind eye due to absolute glaucoma, chronic iridocyclitis, intraocular hemorrhage, etc.
  - Phthisis bulbi with calcification
  - Disfigured eye, e.g. anterior staphyloma, ciliary staphyloma, etc.
  - Sympathetic ophthalmia.
- ***To collect donor eyes from the cadaver for eye bank for keratoplasty:*** By and large, this is the most common indication of enucleation in ophthalmic practice.

Method: Steps of operation (*see* Chapter 10).

The structures cut by enucleation scissors are:

- Optic nerve with its meningeal sheaths
- Central retinal artery and vein

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- Two long posterior ciliary arteries
- Two long ciliary veins
- Short posterior ciliary vessels and nerves
- Lastly, two oblique muscles.

In case of malignant tumors of the eyeball, at least more than 10 mm optic nerve stump is to be cut behind the globe. This is because the central retinal artery enters the optic nerve at this distance. So when the extraocular vascular spread occurs—it may be detected by histopathological examination of the optic nerve. Thereby subsequent treatment by radiation or exenteration may be considered.

In panophthalmitis, enucleation is contraindicated because infection may spread via the meningeal sheaths or their spaces into the brain leading to meningitis or encephalitis.

The cosmetic appearence of the artificial eye is better with evisceration than with simple enucleation. Because the muscle attachment are still present to the scleral shell (later form a ball like structure) in case of evisceration, the movements of the artificial eye is well maintained. But enucleation with an orbital implant has a better cosmetic result.

KERATOME (FIG. 27.58)

Identification: It has a thin triangular blade, at the end of a neck attached to a handle. The blade has a sharp apex with two sharp cutting edges. Keratome may be angular or straight.

Uses

- It is used to make small limbal section for cataract surgery. Then the section is enlarged with a corneal scissors.

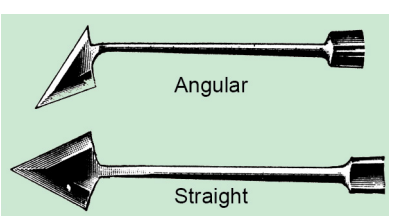


Fig. 27.58: Keratome

- To make small limbal section for glaucoma iridectomy or optical iridectomy.
- Newer disposable angular keratomes are widely used in phacoemulsification, for making sclerocorneal or corneal tunnel incisions.
- May be used for paracentesis.

LACRIMAL PROBE (BOWMAN'S) (FIG. 27.59)

Identification: It is a thin, long probe with round lip. It is made up of stainless steel or silver. They are of different diameters (size 00 to 08). Each instrument has two different size probes attached centrally with a flat handle.

Uses: To probe nasolacrimal passage in:

- Congenital punctal stenosis.
- Congenital dacryocystitis—if probing fails to achieve the patency of nasolacrimal passage then one should go for DCR.
- During DCR to identify the exact position of lacrimal sac for giving incision on the sac wall.

Method (probing and syringing):

- Under general anesthesia.
- Probing is usually done through the upper punctum.
- Start with a smaller size probe and gradually increase the size.
- Always follow the direction of nasolacrimal passage during probing.
- Syringing is done 2–3 minutes after the probing.

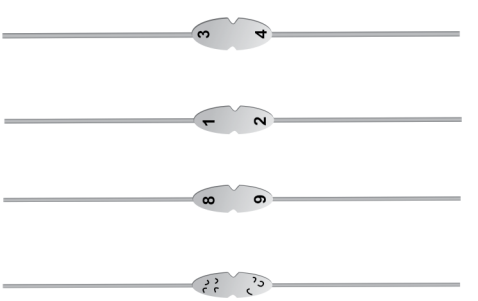


Fig. 27.59: Lacrimal probes (Bowman's) of different size

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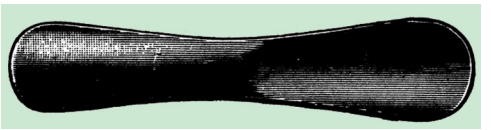


Fig. 27.60: Lid plate (spatula)

Complications of probing:
- False passage
- Nasal bleeding
- Orbital cellulitis.

LID SPATULA (PLATE) (FIG. 27.60)

Identification: It is a flat solid instrument of about 10 cm long. Its both ends are round and convex. It is little curved or grooved at right angle to its long axis, near its end.

Method: Before its application, eyeball is smeared with antibiotic ointment to prevent corneal abrasion. It is introduced under the lid to provide a solid support during lid surgery. Three stay sutures are usually passed along the lid margins so that the assistant can hold the sutures together along with lid plate for better support during dissection.

Use: It is used in various lid surgeries, e.g. ptosis, entropion, etc. It gives more exposure of operative field than a clamp, but it does not help in hemostasis.

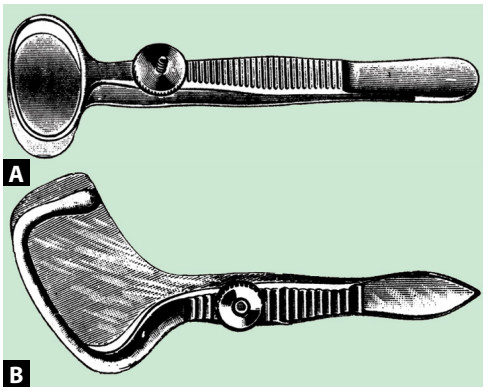
ENTROPION

Forceps (Fig. 27.61A)

Identification: It is just like chalazion forceps except its shape is horizontally oval, and it is bigger in size. It is used for both sides.

Clamp (Fig. 27.61B)

Identification: It is a stout clamp with two limbs, and one screw for fixation. One limb has got a solid semilunar plate and the other limb has U-shaped curved rim corresponding to the semilunar plate. Right and left sided clamps are different.



Figs 27.61A and B: Entropion. **A.** Forceps; **B.** Clamp

Method: Same for both instruments. Eye is smeared adequately with antibiotic ointment before its application to prevent corneal abrasion. Its solid plate is applied on the tarsal conjunctival side and the fenestrated or rim-side is on the skin surface (in reverse order of chalazion forceps). The screw is tightened for fixation.

Use: Same for both. It is used to fix the lid during entropion operation. By tightening the screw, it helps in hemostatis during operation. But it reduces the area of operative field.

CALLIPER (CASTROVEIJO'S) (FIG. 27.62)

Identification: It is a measuring calliper in which the measurement (in mm) is adjusted by spring action of a screw. The measuring ends are pointed like a compass, and the scale is fixed to the opposite end.

Methods: The exact measurement is taken by adjusting the spring action of the screw which is indicated by the pointed end on the scale.

Uses
- To measure the size of the cornea, as in buphthalmos, congenital hereditary

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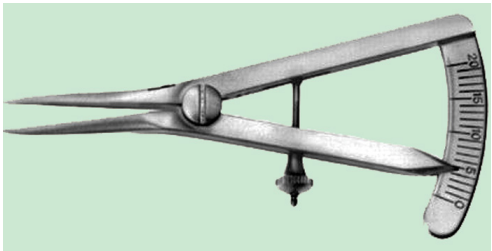


Fig. 27.62: Calliper

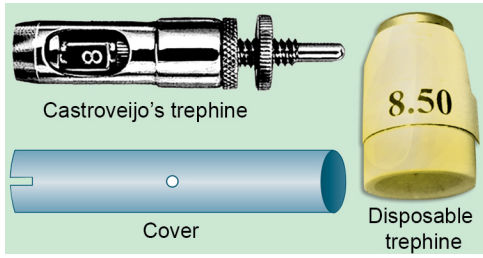


Fig. 27.63: Parts of corneal trephine

endothelial dystrophy (CHED), megalocornea, microcornea, etc.

- ***To use in various surgeries:***
  - Phacoemulsification or small incision cataract surgery—length of incision
  - Trabeculectomy—length of scleral flap
  - Squint operation—amount of resection or recession of the muscle
  - Keratoplasty—to determine the size of the donor and recipient corneal button.
  - Retinal detachment surgery—to measure the distance for passing encircling band or to make port at pars plana
  - Intraocular foreign body (IOFB) removal—to measure the site of incision for IOFB removal
  - Ptosis surgery—to measure the amount of lipopolysaccharide (LPS) to be resected.
  - To measure the length for any purpose.

CORNEAL TREPHINE (CASTROVEIJO'S)

Identification: It is a cylindrical instrument which has three parts (Fig. 27.63):

1. A circular blade
2. An adjustable inner core or 'obturator'
3. A cover to protect the sharpness of the blade.

It is available in different diameters (like 6.0, 6.5,7.0, 7.5 ... 10, 10.5, etc. in mm). The obturator has a scale (marking 0, 2, 4, 6, etc. in 1/10th of mm) which helps the surgeon to select exact depth of the cornea to be cut. This is important in lamellar keratoplasty.

Uses: To cut the 'donor' and 'recipient' corneal button in penetrating and lamellar keratoplasty.

Now-a-days disposable corneal trephine is used.

PIN-HOLE (PH) (FIG. 27.64)

Identification: It is a black disc with a small central hole, attached to a small handle.

Principle of pin-hole: When it is held in front of eye, only a small pencil of rays get through, which passes through the axis of dioptric system of the eye, and is therefore, unaffected by it.

It follows that if the hole is small enough, all refraction would be eliminated and a clear image thus be formed in the same manner as seen in a pin-hole camera.

Method: Patient complaining of dimness of vision, is asked to look at the Snellen's chart. Then a pin-hole is placed in front of his eye, the other eye is being closed. He is

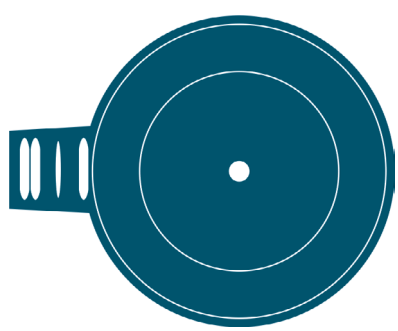


Fig. 27.64: Pin-hole

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asked whether his vision is better or worse, or unchanged.

Uses

- To differentiate dimness of vision is due to refractive error or due to organic diseases of the media; or due to macular or neuro-ophthalmologic disease.
  - In case of refractive error or in minor opacity in the media; there will be a substantial improvement of vision.
  - In case of macular or neurological disease, and major opacity in the medial—there will be no improvement, rather some worsening may occur.
- During prescription of the glasses in refractive error, achieved by proper lenses or not.
- During follow-up after cataract surgery or keratoplasty, the potential visual acuity can be judged.

Note: Best visual acuity obtainable with a pin-hole is approximately 6/7.5 (20/25), not 6/6 (20/20); due to defraction caused by pin-hole aperture.

STENOPAEIC SLIT (FIG. 27.65)

Identification: It is black disc with a large slit-like opening at the center. It has small handle.

Method: Stenopaeic slit is placed in front of the eye by means of a spectacle frame within the slot. It can be properly positioned by rotating it within the frame.



Fig. 27.65: Stenopaeic slit

Uses

- Detection of the axis for prescribing cylindrical lens for astigmatic correction.
- Fincham's test to differentiate between glaucomatous halos from cataract halos.
- To find out the best meridian for optical iridectomy.
- As a low visual aid.
- Used as a part of Maddox wing.

The stenopaeic slit is essentially an elongated pin-hole. When placed in front of the eye, it allows only the rays of light in the particular meridian to enter into the eye. As for example, if the slit is placed horizontally, the vertical focal line passes through the pin-hole and reduced to almost point image.

OPHTHALMIC LENSES

Types of lens used in ophthalmic practice are (Fig. 27.66).

- ***Spherical lenses:***
  - Convex and
  - Concave
- ***Cylindrical lenses:***
  - Convex and
  - Concave

Methods of identification of a given lens: Close left eye. Hold the lens by index finger and thumb. Look any object through the lens with the right eye for magnification or minification.

Then hold the lens close to the right eye with thumb and index finger, left eye being closed. ***Concentrate on a distant object*** (some figure or a line) through the lens. Now move the lens in:

- Horizontal direction
- Vertical direction
- Rotatory fashion.

Note the following:

- Object is moving in the same direction with the movement of the lens, or in the opposite direction.

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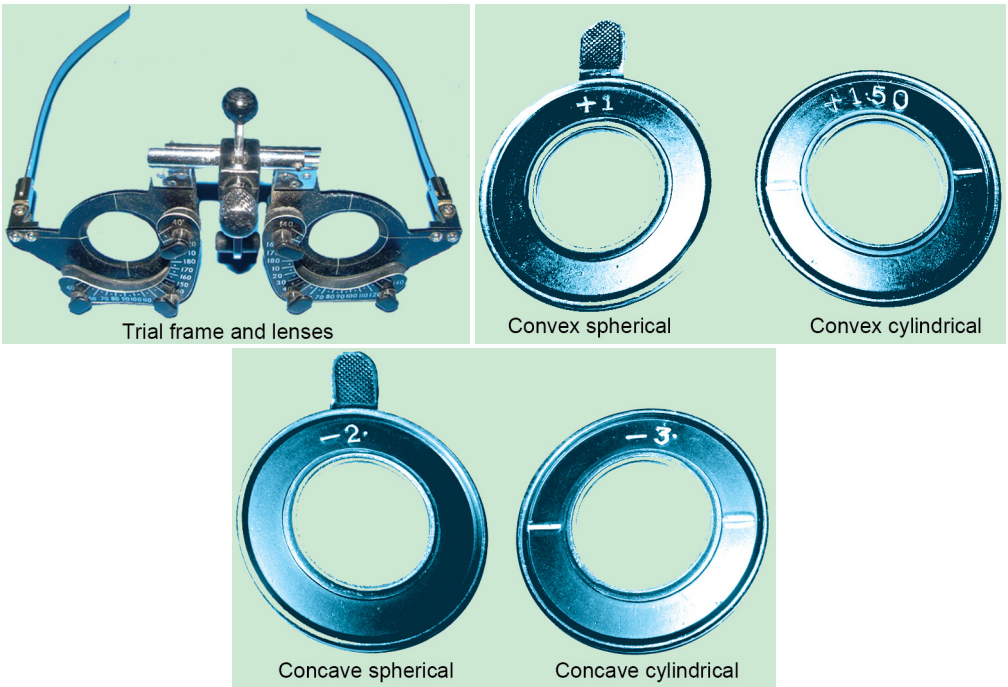


Fig. 27.66: Ophthalmic lenses

- Any distortion of the object while moving the lens in a rotatory fashion.

Now identification of the lens:

- ***Spherical***
  - The object appears to move in both directions, i.e. horizontal and vertical,
  - No distortion of the image of the object in rotatory movement.
- ***Cylindrical***
  - Object appears to move in one direction only,
  - There is distortion of the image when the lens is in rotatory movement.

Then identify whether the lens is convex or concave:

- ***Convex lens (plus lens)***
  - Magnification of the image,
  - The objects appears to move in the ***opposite direction*** as that of the lens.
- ***Concave lens (minus lens)***
  - Minification of the image,
  - Objects appears to move in the same direction as that of the lens.

So a given lens may be either:

- ***Convex spherical***
  - Object appears to move in both meridians and in the ***opposite direction***.
  - No distortion of the image.
  - Magnification of the image.
- ***Concave spherical***
  - Object appears to move in both meridians and in the ***same direction***.
  - No distortion of the image.
  - Minification of the image.
- ***Convex cylindrical***
  - Object appears to move in one meridian and in the ***opposite direction***.
  - Distortion of the image.
  - Magnification of the image.
- ***Concave cylindrical***
  - Object appears to move in one meridian and in the ***same direction***.
  - Distortion of the image.
  - Minification of the image.

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Note: In a plane glass (plano): ***Lens with 'no' power***—no movement of the object, no distortion of the image, size remains same.

Some hints for identification of a given lens:

- ***Spherical***
  - It has got a handle with the rim and
  - No axis marking on it.
- ***Cylindrical***
  - Perfectly circular with no handle and
  - Axis mark on the metallic or plastic rim in one side only.

Uses

- ***Convex spherical***
  - ***Correction of refractive status:***
    - Hypermetropia
    - Aphakia
    - Presbyopia
    - As a low visual aid (magnifier).
  - ***Instrumental uses:***
    - Direct ophthalmoscope
    - Indirect ophthalmoscope
    - Microscope
    - Synaptophore
    - Corneal loupe or telescopic loupe.
  - ***Diagnostic uses:***
    - Volk +90D lens, or +78D lens—to see the fundus
    - Placido's disc (+3.0D)
    - Malingering (with high plus lens)
    - Different condensing lenses for laser therapy.
- ***Concave spherical***
  - ***In refractive error:*** Myopia.
  - ***Instrumental use:***
    - Direct ophthalmoscope
    - Telescopic loupe (Gallelian system).
  - ***Diagnostic use:***
    - Hruby lens (–55D)
    - Fundus contact lens (–45D)
    - Central lens of gonioscope
    - Malingering test with high minus lens.
- ***Convex cylinder:*** Regular hypermetropic astigmatism.

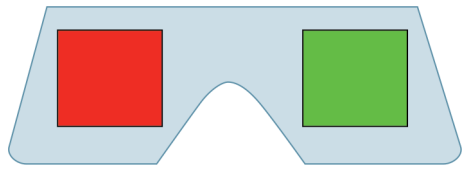


Fig. 27.67: Red-green goggles

- ***Concave cylinder:*** Regular myopic astigmatism.

In case of irregular astigmatism—one should use contact lens.

RED-GREEN GOGGLES (FIG. 27.67)

Identification: It is like a toy goggles with an elastic band. At one side, it is red and on other side, it is green.

Procedure: Conventionally, the red side of the goggles has to be placed in front of right eye (red for right, i.e. R for R).

Use: It is used to dissociate binocular single vision in various tests:

- Worth's four dot test
- Hess screen test
- Diplopia charting.

PLACIDO'S DISC (FIG. 27.68)

Identification: It is a medium-size circular disc with a central hole and attached to a handle. On one surface of the disc, there are alternate black and white circle like Bull's eye.

Procedure: Placido's disc is held in front of the patient's eye while examiner looks through the hole in center of the disc. The examiner then observe the corneal image of the disc as reflected from the light behind the patient.

Use: It is used to assess the corneal anterior surface and anterior corneal curvature (astigmatism):

- A loss in sharpness of image denotes a loss of normal smoothness of the anterior corneal surface, e.g. corneal abrasion, dry eye, etc.

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Fig. 27.68: Placido's disc

- Elliptical image is seen in regular astigmatism.
- Irregular image is seen in keratoconus.

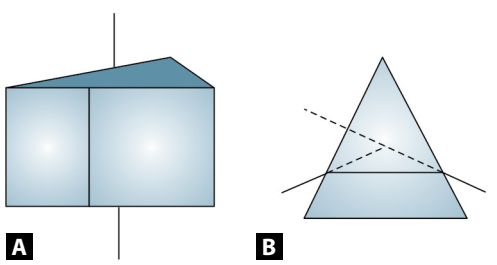
PRISM (FIGS 27.69A AND B)

Identification: It is wedge-shaped refracting material (glass or plastic) with thin edge at one side and thick edge on opposite side. It is triangular in cross-section having an apex and a base.

Refraction through a prism: There is total deviation of light towards the base of the prism. The image is displaced towards the apex of the prism.

Positions

- ***Frontal position:*** Plastic prism—parallel to infraorbital margin.



Figs 27.69A and B: **A.** Prism; **B.** Refraction through a prism

- ***Prentice position:*** Glass prism—posterior face of the prism is perpendicular to the line of sight.

One prism diopter power produces a linear apparent displacement of 1 cm of an object situated at 1 meter.

Uses of Prisms in Ophthalmology

Diagnostic

Assessment of squint and heterophoria:

- Objective measurement of angle by prism cover test, prism reflex test of Krimsky.
- Subjective measurement of angle by Maddox rod.
- Assess likelihood of diplopia after squint surgery in adults.
- Measurement of fusional reserve.
- Dioptre base out test for microtropia.
- To detect malingering.

Therapeutic

- Convergence insufficiency—synaptophore.
- To relieve diplopia—decompensated heterophorias, small vertical squints and some paralytic squints due to some surgical contraindications.

Forms of therapeutic prisms:

- ***Temporary wear:*** Clip on spectacle prisms (Fresnel prisms).
- ***Permanent wear:*** Incorporation into the spectacles by decentring the spherical lenses.

Instruments

- Goldmann applanation tonometer
- Indirect ophthalmoscope
- Slit-lamp microscope
- Keratometer
- Operating microscope
- Synaptophore
- Haidinger's brush
- Koeppe's goniolens.

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Forms of instrumental prisms

- Right angle prism—angle of deviation 90°.
- Porro prism—angle of deviation is 180°, image is inverted but not transposed.
- Dove prism—no deviation, image is inverted but not transposed.

Miscellaneous

- Recumbent spectacles
- Hemianopic spectacles—8 diopter prism with base towards blind side
- Low visual aid (LVA)—base in prisms incorporated into binocular magnifier.

RETINOSCOPE (FIG. 27.70)

Identification: This is a circular plain mirror with a central aperture in a plastic frame with handle. Sometimes, a concave mirror of 2.0D may be attached with other end in a dumble-shaped frame.

Uses

- It is used for determination of refraction.
- It may be used for distant direct ophthalmoscopy (at 22 cm). This is—
  - To know any opacity in the media
  - To discover the edge of a subluxated or dislocated lens
  - To recognize a retinal detachment or tumor.
  - To confirm the results found by external examination.

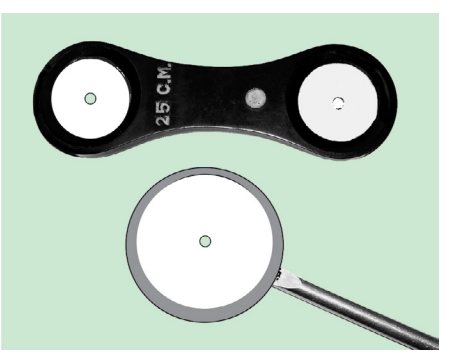


Fig. 27.70: Retinoscope

Optics of Retinoscopy

It entails a study of the movements of the retinal image produced by a beam of light that sweeps across the pupil.

The observer then watches this illuminated retinal image by looking down the path of incident light, through a hole in the center of a mirror (retinoscope).

- ***If the eye is emmetropic:*** Parallel rays of light come to a point focus on retina, so they are emerging in the same pathway.
- ***If the eye is hypermetropic:*** Parallel rays converge behind the retina and hence, the emerging rays are divergent.
- ***If the eye is myopic:*** Parallel rays converge infront of the retina and hence, the emerging rays are convergent.

The principle of retinoscopy is to make every observing eye emmetropic, so that the emerging rays should form a parallel beam.

Procedure

- ***Cycloplegia:*** It is only required in children and young patients. In adults and old patients, it is usually not necessary.
  - ***If the patient is less than 5 years:*** Atropine eye ointment (1%) is to be applied three times daily for 3 days.
  - ***If the patient is between 5 and 15 years:*** 1% cyclopentolate, or 2% homatropine eye drop is instilled for 3 times, about 1 hour before examination.
  - ***If the patient is between 15 and 20:*** The same procedure may be undertaken.

  The refraction under cycloplegia is always pathological because the shape of the lens has been altered. A ***post-cycloplegic test (PCT)*** is therefore advisable after 3 days to 3 weeks depending upon the cycloplegic used.
- ***Dark-room test:*** Retinoscopy should preferably be conducted in dark room.
  - Examiner sits at 1 metre away from the patient (point of reversal is at 1.0D).

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It is even more convenient to sit at arm's length, i.e. two-third metre away, so that the trial lenses can be held in the other hand while the light beam is passed. The point of reversal is then be 1.5D.

- The patient is normally seated and looking towards the far end of the room (relaxed eye).
- Source of light is from behind the patient.
- The surgeon looks through a ***plane mirror*** with central perforation, and light is reflected into the patients's eye.
- The mirror is slowly moved from side to side in different meridians, and movement of the shadow is noted.
- In hypermetropia, emmetropia and myopia <1.0D the reflex moves in the same direction.
  In myopia of –1.0D = there is no movement of shadow.
  In myopia of >1.0D = the shadow moves in the opposite direction.
- Increasing convex (if the movement is on same side) or concave (if opposite side) lenses are placed before the eye until the ***point of reversal*** is reached.
  At this point there will be no movement of the shadow, and pupil will be brightly illuminated.
- The procedure is done in each meridian separately.
  - In simple spherical refractive error—the movement and the point of reversal will be same in both meridians.
  - In astigmatism, they are different. If the axes are oblique, the shadow themselves will seem to move obliquely and the mirror is then tilted accordingly.

- ***Calculation:*** Refraction of patient's eye—lens required to reach end point = –1D (myopia). Since the surgeon is sitting at 1 metre distance, and if he is at $^{2}/_{3}$ metre, it will be –1.5D (myopic). So the refraction of the eye = –1.0D + lens.

Examples:

- ***If the end point is with +4.0D lens:***
  Refraction = –1.0D + 4.0D = +3.0D.
- ***Similarly if, with –4.0D lens:***
  Refraction = –1.0D – 4.0D = –5.0D.
- ***If the end point is with +1.0D lens:***
  Refraction = –1.0D = 0, i.e. the patient is emmetropic.
- In case of astigmatism, each meridian is to be calculated separately.

Streak retinoscopy: Instead of circular light as obtained by a plain mirror, a self-illuminated streak of light is used. Here, the appearences of the shadow are more dramatic. Axis of the astigmatism is easily determined. It has certain other advantages:

- Can be done in any position of the patient.
- Can be done in difficult patients, e.g. in children or non-cooperative patient.
- Can be used per-operatively.

MAGNIFYING LOUPE

Identification: It is a strong biconvex lens, fitted in a metal-case (uniocular); or in a plastic frame (binocular) with forehead band (Figs 27.71A and B).

Uses: It is used for examination of the anterior segment of the eye with oblique illumination. It is useful to search minute corneal foreign bodies, keratic precipitates (KPs), superficial punctate keratitis (SPK), caterpillar's hairs, etc.

Magnification

- Uniocular loupe—10 times
- Binocular loupe—3–4 times

Dioptric power of the loupe

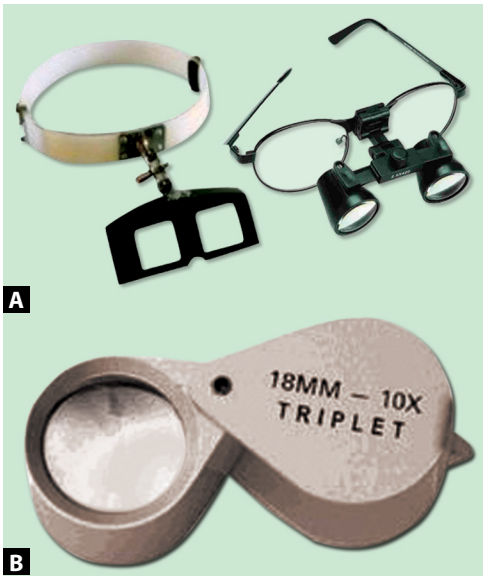
- Uniocular = +40 D
- Binocular = +12–16D.

Magnification = power in diopter/4

JACKSON'S CROSS CYLINDER (FIG. 27.72)

Identification: It is a sphero-cylindrical combination of lens in which the spherical

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Figs 27.71A and B: Magnifying loupe. **A.** Binocular; **B.** Uniocular

component is half the power but of the opposite sign of the cylindrical component. ***The most convenient form*** is a combination of a –0.25D spherical with +0.50D cylinder.
Method: The cylindrical axis of the cross-cylinder is first placed in the same direction as the axis of the cylinder in the trial frame and then perpendicular to it. In the first position, the cylindrical correction is enhanced by 0.5D, and in the second, it is diminished by the same amount. If the visual acuity is not improved in either of these positions, the cylinder in the trial frame is correct.

If the visual acuity is improved, a corresponding change should be made in the correction, and the new combination is verified by running through the cycle again.
Use: It is used to check the power and axis of the cylinder in refractive correction.

MADDOX ROD (FIG. 27.73)

Identification: It consists of a series of parallel high-power, plus cylinders (rods) of red glass placed side by side in a supporting disc. It can convert a point light into a red streak light at right angle to the axis of rods.



Fig. 27.72: Jackson's cross cylinder

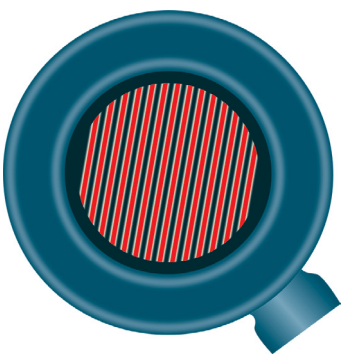


Fig. 27.73: Maddox rod

Methods: When a Maddox rod is held in a front of one eye, the image of a point source of light becomes dissimilar between the two eyes, and fusion becomes dissociated (dissimilar image test). This test is performed at 6 metres and at 33 cm from a bright spot light in a dark room.
Uses
- To test the latent squint for distance.
- For orthoptic exercise in cyclophoria.
- To test macular function, in presence of opaque media.

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METHODS OF STERILIZATION

The methods of sterilization commonly practiced in ophthalmic surgery are: (1) Autoclave, (2) Boiling, (3) Dry heat, (4) Chemicals, (5) Gamma-rays and (6) Ultraviolet (UV) rays.

Autoclave

Steam under pressure is biocidal. A temperature of 134°C (321 lbs/sq inch pressure) for 3 minutes, or 121°C (15 lbs/sq inch pressure) for 15 minutes is used. It is used to sterilize towels, gowns, masks, sutures, dressings, irrigating solutions and blunt instruments. Sharp instruments should not be autoclaved.

Boiling

A traditional method of sterilization is still used routinely, not only in eye camps, but also in many hospitals. Boiling for 30 minutes usually kills most bacteria, but some spores may withstand boiling for this period. It is preferable to use distilled water to prevent salt deposition. Sharp instruments are better not to be sterilized by boiling.

Dry Heat

Dry heat kills bacteria by oxidative destruction of their protoplasm. In dry heat, a temperature of 150°C is used for 90 minutes in the hot air oven. It is better not to sterilize sharp instruments by dry heat.

Chemicals

- ***Ethylene oxide:*** The vapor of ethylene oxide can destroy all micro-organisms, including spores. As undiluted ethylene oxide is highly explosive, it is diluted with an inert gas like carbon di oxide or freon (12% ethylene oxide and 88% freon). Sharp instruments, plastic cryoprobes, IOLs, indirect opthalmoscopy lens, diathermy wires, etc. can be sterilized by this method.
- ***Glutaraldehyde solution (cidex):*** Two percent glutaraldehyde kills all micro-organisms including spores. Its full potency persists for 2 weeks, then it is to be discarded. Cidex does not reduce the sharpness of the cutting instruments. In 10 minutes all micro-organisms are killed and in 3 hours all spores are destroyed. The instruments after removed from cidex, are thoroughly rinsed with sterile water and hollow instruments are to be flushed. All metallic instruments including the sharp-cutting instruments may be sterilized by this method.
- ***Alcohol (rectified spirit):*** Sharp cutting instruments and sutures may be dipped in 90% alcohol for 30 minutes for sterilization. It is not an excellent method.
- ***Isopropyl alcohol (70%):*** This is also used for sterilization of all kinds of metallic instruments. Dip the instruments for 15 minutes and then wash it with sterile water or saline before use.
- ***Formalin vapor:*** Keep the instruments in formalin vapor in a chamber (vapor released from formalin tablets) for overnight. Rinse thoroughly before use. It is carcinogenic and now banned in many countries.

Gamma-Irradiation

Gamma rays from cobalt-60. This type of sterilization is used commercially, for disposable needles, disposable syringes, infusion sets, etc.

Ultraviolet Rays

Ultraviolet (UV) radiation is used in the disinfection of drinking water, operating room air and contact lenses. Bacteria and viruses are more easily killed by UV light than the bacterial spores.

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28 CHAPTER

Miscellaneous

LASER IN OPHTHALMOLOGY

Laser is the acronym of light amplification by stimulated emission of radiation.

Physics

Certain substances have the property to 'lase', i.e. to absorb energy in one form, and to emit a new form of light. On pumping these 'lasing' substances are able to transfer electrons from a lower orbit to the higher metastable orbit ***(Bohr's theory)***. The excited atoms in turn decay back to their original orbit to lower energy, thus emitting a new form of photons. This decaying can be spontaneous, or induced ***(stimulated emission)***. These photons are then passed through a close system of mirrors, which leads to ***light amplification***, and delivered as ***laser***.

Common Types of Laser

Common types of laser used in ophthalmology has been described in Table 28.1.

TABLE 28.1: Common types of laser

Types of laser	Wavelengths
Ruby laser (first laser)	550 nm
Argon laser	
• Blue-green	488 nm
• Pure green	518 nm
Krypton laser	647 nm
Nd-YAG laser (Neodymium: Yttrium-Aluminium Garnet)	
• Single frequency	1064 nm
• Double frequency (pulsed)	532 nm
Diode laser Produced from semiconductor crystals Lasing substance: Gallium-aluminium-arsenate	810 nm
Excimer (excited dimer) laser Lasing substance: Argon fluoride	193 nm
Femtosecond laser Nd: Glass	1053 nm

Properties of Laser

- It is always ***monochromatic*** (has got one wavelength of light).
- It is ***collimated*** (all photons run parallel).
- It is ***coherent*** (always in the same phase).
- It is ***polarized*** at high energy level.

Laser Effects on Ocular Tissues

- ***Photocoagulation (controlled burn):*** Argon laser, diode laser, krypton laser.
- ***Photovaporization:*** Argon laser, diode laser.
- ***Photodisruption (optical breakdown to disrupt tissues by the formation of plasma):*** Nd-YAG laser, femtosecond laser.
- ***Photoablation (ablation of corneal tissue without thermal damage):*** Excimer laser.

Indications of Laser (in General)

Common Indications

- Iridotomy for angle-closure glaucoma
- Pupilloplasty

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- Argon-laser trabeculoplasty (ALT) in primary open-angle glaucoma
- Yttrium aluminum garnet (YAG) capsulotomy for posterior capsular opacification following extracapsular cataract extraction (ECCE)
- To create chorioretinal adhesions around the retinal breaks
- Panretinal photocoagulation (PRP) for proliferative retinal diseases and rubeosis iridis, e.g. diabetes, central retinal vein occlusion (CRVO)
- Central serous retinopathy (CSR)
- Direct photocoagulation to treat the vascular abnormalities
- 'Macular-grid' photocoagulation to treat macular lesions
- Intraocular tumors and malignancy
- Photorefractive keratoplasty (PRK) in refractive errors, by excimer laser
- Laser assisted *in situ* kerotomelieusis or LASIK for the treatment of refractive errors (mainly myopia)
- Phototherapeutic keratoplasty (PTK) with excimer laser for superficial corneal scars
- Creation of LASIK flaps and trephination of donor and recipient button in penetrating and lamellar keratoplasty
- Femtosecond laser cataract surgery.

Miscellaneous

- Trichiasis (laser cilia-ablation)
- Suture removal (laser suturolysis)
- Pterygium (to treat the bare area)
- Cyclophotocoagulation in glaucoma
- ***Diagnostic:*** Laser-interferometry with helium-neon (He-Ne) laser to test the potential visual acuity in presence of opacity in the media.

Femtosecond Laser in Ophthalmology

Femtosecond laser (FS) is an infrared laser (wavelength = 1,053 nm) with ultrashort pulse duration (10–15 sec). This laser has the ability to deliver laser energy with minimal collateral damage to the adjacent tissue. Thermal damage to neighboring tissue in the cornea has been measured to be around 1 μm.

- Femtosecond laser works by photodisruption like Nd-YAG laser, a process in which small volumes of tissue are vaporized resulting in the formation of cavitation gas (CO_2 and water) bubbles.
- Furthermore, the unique FS laser can be focused anywhere within or behind the cornea and is capable of passing through optically hazy media, such as an edematous cornea or cataractous lens. It may be applied in multiple geometric patterns including vertical, horizontal, spiral or zig-zag cuts.
- There are multiple commercially available femtosecond laser models in the market.

Applications in Ophthalmology

Femtosecond Laser in Refractive Surgery

- ***Flap creation in LASIK:*** Corneal flap creation in LASIK is the most common use of FS laser. It has many advantages over mechanical microkeratome flap, like—fewer flap-related complications, thinner flap, improved thickness predictability and less induced higher order aberrations.
- ***Femtosecond lenticule extraction (FLEx):*** It is a new procedure to correct myopia and myopic astigmatism. This procedure involves making 2 cuts (anterior and posterior) that intersect in the periphery, creating a lenticule which is ultimately removed.
- ***Small incision lenticule extraction (SMILE):*** A procedure in which the corneal flap is eliminated and instead a small incision is created in the mid-periphery of the cornea and the lenticule is removed through this self-sealing incision. SMILE has less tendency to induce post-refractive surgery dry eye, ectasia and least flap complications.

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Intracorneal Ring Segments (INTACS)

- Intracorneal ring segments are arcuate, polymethyl methacrylate (PMMA) implants for intrastromal insertion in mid-peripheral cornea for correction of milder cases of keratoconus with upto –3.50D of myopia and without central scarring.
- Femtosecond laser creates arcuate tunnels at 70% corneal depth. This method is easier and safer than manual dissection.

Femtosecond Laser for Presbyopia Correction

Femtosecond laser can be used to create intrastromal pockets for the implantation of INTRACOR or the Kamra small-aperture corneal inlay which help to correct presbyopia.

Femtosecond Laser in Corneal Transplantation

- ***Penetrating keratoplasty (PK):*** Femtosecond laser creates complex pattern trephination cuts for enhanced wound integrity of the graft-host junction. Theoretically it may decrease suture-induced astigmatism and allow for faster visual recovery. The cuts include top-hat (larger diameter cut posteriorly), mushroom (larger diameter anteriorly), zig-zag and christmas tree pattern. Mushroom cut may be advantageous in keratoconus by providing a larger anterior refractive surface while the top-hat may be useful in endothelial disorders by replacing more endothelial cells.
- ***Anterior lamellar keratoplasty (ALK):*** Femtosecond-assisted sutureless anterior lamellar keratoplasty can be used to treat anterior corneal pathologies with less irregular astigmatism and faster visual rehabilitation. Femtosecond-assisted DALK (deep anterior lamellar keratoplasty) is also a promising development.
- ***DSAEK (Descemet's stripping automated endothelial keratoplasty):*** Femtosecond laser can be used to create the lenticule for insertion in DSAEK procedure. The results are equal to or better than microkeratome assisted DSAEK.
- ***DMEK (Descemet's membrane endothelial keratoplasty):*** Femtosecond laser can be used in the donor preparation in DMEK and also for the creation of smooth stromal interface.

Femtosecond Laser and Astigmatic Keratotomy and Arcuate Wedge Resection

Femtosecond laser may be used in astigmatic keratotomy (AK) and/or arcuate wedge resection for the correction of high astigmatism following penetrating keratoplasty (PK) or cataract surgery. This laser procedure is easier, more precise and carries less risk of perforation than diamond blade method.

Femtosecond Laser and Cataract Surgery

- Femtosecond laser are now used routinely in cataract surgery to create capsulotomy, clear corneal incision, limbal relaxing incisions and lens fragmentation.
- Incisions (main incision and paracentesis) created by the laser are more precise, stable and with faster wound healing.
- The capsulorrhexis created by the laser is more precisely centered and measured leading to better outcomes in premium intraocular cataract lenses (IOLs) like multifocal and toric IOLs.
- Lens fragmentation with the laser leads to lesser phacoenergy being used and thus lesser endothelial damage.
- Visual recovery is also faster.
- Cystoid macular edema may be less in FS laser as compared to conventional phacoemulsification.

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CRYOTHERAPY IN OPHTHALMOLOGY

Cryotherapy is a technique to injure, or to adhere tissues by the application of intense cold.

- This is achieved by a ***cryoprobe,*** a pencil-like instrument held by the surgeon, and it is cooled at a temperature of –40°C to –100°C by a freezer-gas under pressure. The gas-flow to the cryoprobe is controlled by a foot-switch.
- The ***gasses used*** are nitrous oxide (liquid nitrogen), freon or solid carbon-dioxide.

Principle

***Joule-Thomson effect*—**when a gas (cryogen) is released under high pressure through a micro-orifice, the gas expands to reach at its boiling point to reduce the temperature (e.g. liquid nitrogen boils at –195.6°C, and is an excellent cryogen).

Mechanism

When the freezing source is applied to a tissue, there is formation of hemispherical ice-ball which has different temperature zones.

- The temperature at any point within the ice-ball depends upon:
  - The distance from the cryogen.
  - The rate and duration of freezing.
- ***Two basic mechanisms*** involved in cryotherapy are:
  i. ***Cryoadhesion*** (as in ICCE or retinal breaks).
  ii. ***Cryoinjury*** *(in vivo* cell death), which has two phases:
    a. ***Initial phase:*** (i) Extracellular ice-crystal formation → dehydration → cell death. (ii) Intracellular ice-crystal formation → cell death.
    b. ***Late phase:*** Damage to the micro-circulation of the cells → ischemic necrosis.

Maximum cell death is best achieved with a 'rapid freeze' and a 'slow thaw'.
The three different probes are:

1. ***Cataract probe:*** –40°C to –50°C
2. ***Retinal probe:*** –50°C to –70°C
3. ***Glaucoma probe:*** –60°C to –80°C

Use of Cryo in Ophthalmology

- ***Eyelids:*** Papilloma, molluscum contagiosum, trichiasis, basal cell carcinoma, xanthelasma, etc.
- ***Conjunctiva:*** Cystoid cicatrix, giant papillae in vernal conjunctivitis
- ***Cornea:*** Herpes simplex keratitis (epithelial or stromal), epithelial downgrowth, carcinoma *in situ*
- ***Iris and ciliary body:*** Prolapsed iris, cyclocryopexy
- ***Lens:*** Intracapsular cataract extraction
- ***Retina***
  - To seal the peripheral retinal breaks (cryoretinopexy).
  - To flatten retinoschisis.
  - To treat retinal neovascularization (when the media is opaque).
  - To destroy retinal neoplasm, either simple (angioma), or malignant (retinoblastoma).

FLUORESCEIN DYE IN OPHTHALMOLOGY

Sodium fluorescein is a non-toxic, stable highly fluorescent dye. It does not penetrate the cell membrane, but remains on its surface.

When administered parenterally, the dye binds with albumin in the bloodstream, and circulates into the oclar tissues and intra-ocular fluids.

The dye absorbs light in the ***blue*** range of visible spectrum, with an ***absorption peak*** at 465 to 490 nm, and after excitation it emits light (fluoresce) in the ***green*** range of visible spectrum, with an ***emission peak*** at 520 to 530 nm. This color-change can be detected

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even at a dilution of 1 in 1,000,000 by using ultraviolet light.

After intravenous (IV) injection, fluorescein dye is eliminated from the body through the kidneys and liver within 24–36 hours.

Dosages

- ***For topical uses***—2% solution—1 drop.
- ***Fluorescein paper strip*** (available in sterile pouch) convenient to stain cornea and to assess tear film dysfunction.
- ***For fundus fluorescein angiography:*** 5 mL of 10% solution (most commonly), 10 mL of 5% solution, or 3 mL of 20% solution—as intravenous (IV) bolus injection.

Adverse Effects of Intravenous Fluorescein Dye

- Nausea and vomiting
- Vasovagal (syncopal) attack
- Painful local extravasation and phlebitis
- Pruritus and urticaria
- Anaphylaxis and bronchospasm (acute emergency).

Uses of Fluorescein Dye

Topical uses

- Staining corneal abrasions or ulcer
- Hard contact lens fitting
- Tear-film break-up-time (TBUT)
- To detect wound leak (Siedel's test)
- Applanation tonometry
- Patency of the lacrimal passage:
  - Dye disappearance test (DDT)
  - Jones test (I and II).
- To marks the radial cuts in radial karatotomy, etc.

Intravenous uses

- Fundus fluorescein angiography.
- Fluorescein fluorophotometry to study aqueous outflow or corneal endothelial cell function.
- Fluorescein iridography to study iris vascular pattern (e.g. in rubeosis iridis or angioma).
- As an antidote for aniline dye poisoning.

Fundus Fluorescein Angiography

It is a procedure of recording serial photographs (black and white) of retinal and choroidal circulation, and their integrity, after IV injection of fluorescein dye.

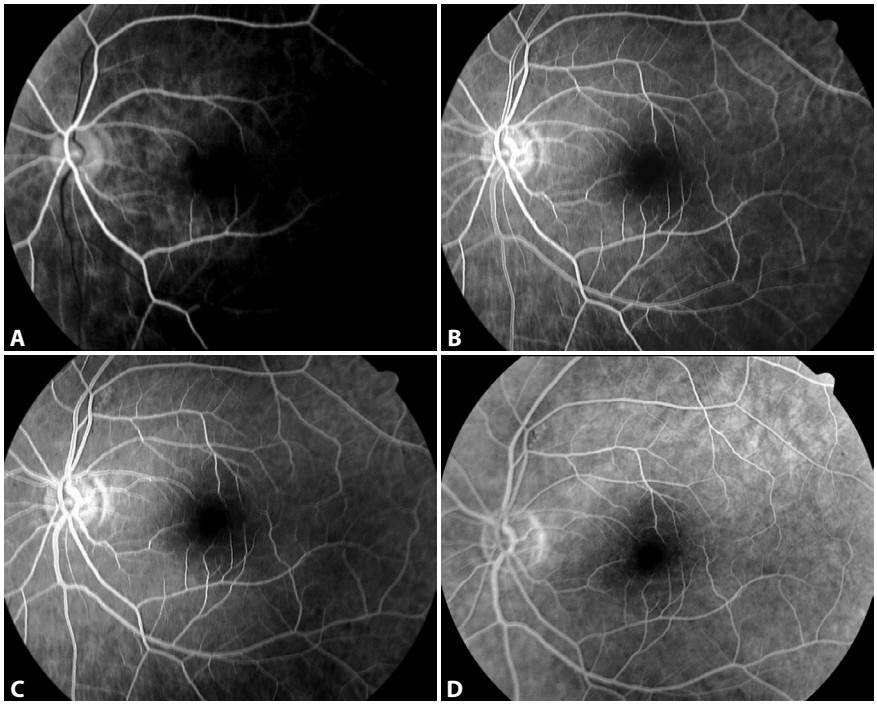
Normal Angiogram

The dye first appears at the choroid, then the retina, and finally it is drained via the central retinal vein. Normal ***'arm-to-retina'*** circulation is about 8–12 seconds ***(average—10 seconds)***. In the choroid, the dye appears about 1 second before the retina.

Fundus fluorescein angiography can be divided into following phases:

- ***Choroidal (pre-arterial) phase:*** When the dye enters into choroidal circulation, a bright glow is seen, known as ***choroidal flush.*** If a ***cilioretinal artery*** is present, it fills during this phase.
- ***Arterial phase (Fig. 28.1A):*** The dye appears in the retina through the central retinal artery. The superior and inferior branches fill quickly, and then the whole retinal arterial-tree becomes brightly displayed.
- ***Arteriovenous (capillary) phase:*** The dye is seen both in the retinal artery and vein. The visualization of capillaries is at its best, during the venous phase.
- ***Early venous phase (Fig. 28.1B):*** This phase is marked by ***laminar flow*** which is due to presence of dye on the lateral wall of vein.
- ***Late venous phase (Fig. 28.1C):*** Soon the laminar flow reverses, and the entire blood column in the vein is mixed with the dye.
- ***Recirculation phase (Fig. 28.1D):*** This phase is marked by the ***eventual fade of the dye.*** If the blood-retinal barrier has been broken, the dye leaks out and stains the abnormal vessels, or pools into the tissue.

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Figs 28.1A to D: Phases of fundus fluorescein angiography. **A.** Arterial; **B.** Early venous; **C.** Late venous; **D.** Recirculation phase

- ***Normal macula:*** It appears as a small hypofluorescent area. The perifoveal capillary arcade is seen during venous phase.
  Dark appearance of macula is due to:
  - Macular-RPE (retinal pigment epithelium) cells are taller and more pigmented
  - Presence of xanthophyll pigment
  - Foveal avascular zone (FAZ)
- ***Optic disc:*** The capillary fluorescence is seen at different stages, and valuable in differentiating pseudopapilledema, from papilledema and papillitis.

After 10 minutes, the fundus resembles that of pre-angiographic state, and any deviation from this is abnormal.

Abnormal Angiogram

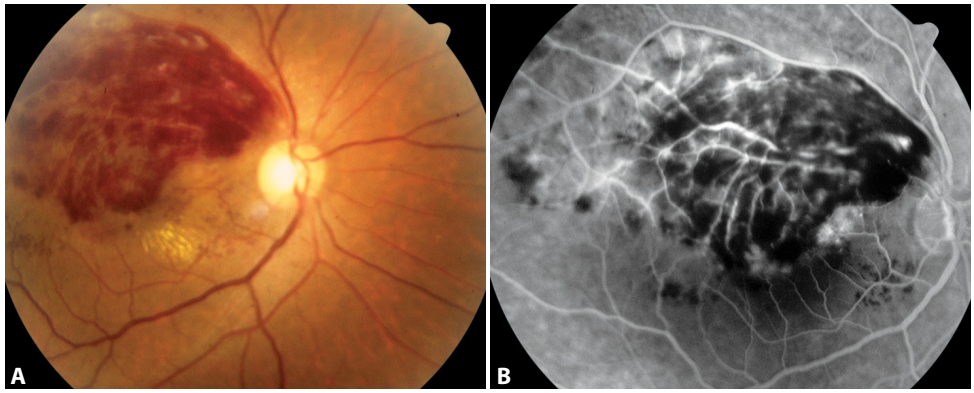
It is based on two important features:
- The blood-retinal barrier and
- The barrier between choroid and RPE.

1. ***Hypofluorescence (Figs 28.2A and B):*** It means a reduction or absence of normal fluorescence. It appears as dark area on the positive print of an angiogram.

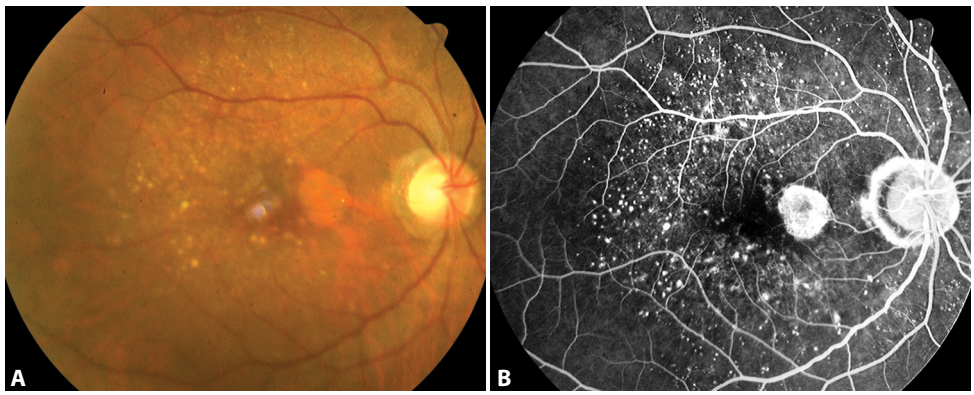
Two possible causes of hypofluorescence	
Causes	**Example of lesions**
Blocked fluorescence	• ***Pigments***—melanin, hemoglobin, xanthophyll • ***Exudates***—hard, soft • ***Hemorrhages***—subhyaloid retinal • Abnormal tissues materials—foreign body scar tissue
Vascular filling defects (dropout areas)	• Retinal artery, vein and capillaries, e.g. vascular occlusion • Choriocapillaries, e.g. sclerosis

2. ***Hyperfluorescene (Figs 28.3 and 28.4):*** It constitutes the appearance of an area that is more fluorescent than the surrounding

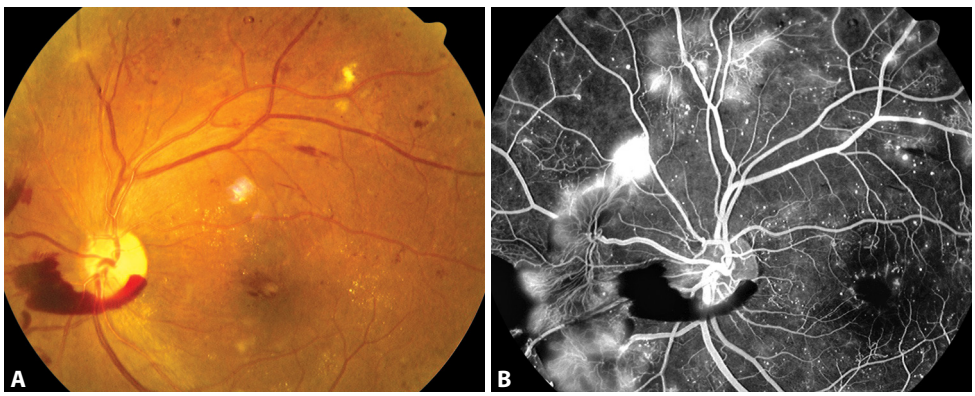
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Figs 28.2A and B: A. Branch retinal vein occlusion—at the disc margin; **B.** Fundus fluorescein angiography—same eye showing hypofluorescence



Figs 28.3A and B: A. Hard and calcified drusen; **B.** Fundus fluorescein angiography—same eye showing hyperfluorescence



Figs 28.4A and B: A. Proliferative diabetic retinopathy; **B.** Fundus fluorescein angiography—both same eye showing hypo and hyperfluorescence

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area, or that stays fluorescent for a longer period of time.

Three possible causes of hyperfluorescence	
Causes	**Example of lesions**
Increased transmission	• ***Atrophic:*** RPE—drusen, RPE-window defect
Leak	• ***Pooling*** (in spaces) – ***Retinal:*** CME – ***Subretinal:*** CSR, RD • ***Staining*** (of tissues) – ***Retinal***—soft exudates – ***Subretinal***—drusen, scar
Abnormal vessels	• ***Retinal***—neovascularization, aneurysms, shunts, collaterals, telangiectasias • ***Subretinal***—SRNVMs, vessels in scar • ***Tumors*** – ***Retinal***—retinoblastoma – ***Subretinal***—malignant melanoma, choroidal hemangioma

Abbreviations: **RPE**—Retinal pigmented epithelium; **CME**—Cystoid macular edema; **CSR**—Central serous retinopathy; **RD**—Retinal detachment; **SRNVMs**—Subretinal neovascular membranes

Fundus fluorescein angiography (FFA) is useful as an important diagnostic tool for certain clinical conditions. It is also important to monitor laser photocoagulation treatment, in certain retina and choroidal diseases.

The common indications are:

- Diabetic retinopathy
- Retinal venous occlusions (CRVO or BRVO)
- Eales' disease
- Central serous retinopathy (CSR)
- Age-related macular degeneration
- Tumors of the retina and choroid
- Vascular anomalies of the retina and choroid
- Papilledema

Fundus Autofluorescence

- Fundus autofluorescence (FAF) is the emission of fluorescent light from ocular structures in the absence of sodium fluorescein. Pseudofluorescence occurs when the blue exciter and green barrier filters overlap.
- Retinal pigmented epithelium (RPE) is a monolayer which plays a critical role in phagocytosis and lysosomal breakdown of pigmented outer segments of photoreceptors. With aging, incomplete or partial breakdown of these segments in the postmitotic RPE cells causes the accumulation of lipofuscin. Excessive lipofuscin deposition is considered pathologic and is associated with visual loss.
- Because of its diverse composition, lipofuscin has a broadspectrum of excitation (300–600 nm) and emission (480–800 nm). Thus, it can be excited by many wavelengths of visible light in the blue and green portion of spectrum. Autofluorescence uses the fluorescent properties of lipofuscin to generate images.
- The unique autofluorescent properties of RPE lipofuscin can be detected and quantified using imaging devices like, confocal scanning laser ophthalmoscope (cSLO) and FAF camera systems.
- Fundus autofluorescence imaging has potential role to provide useful information in conditions where the health of the RPE plays a key role (Figs 28.5A to D).

Reduced fundus autofluorescence:

- Retinal pigmented epithelium loss or atrophy
- Intraretinal fluid
- Reduction in RPE lipofuscin density
- Fibrosis
- Presence of luteal pigment.

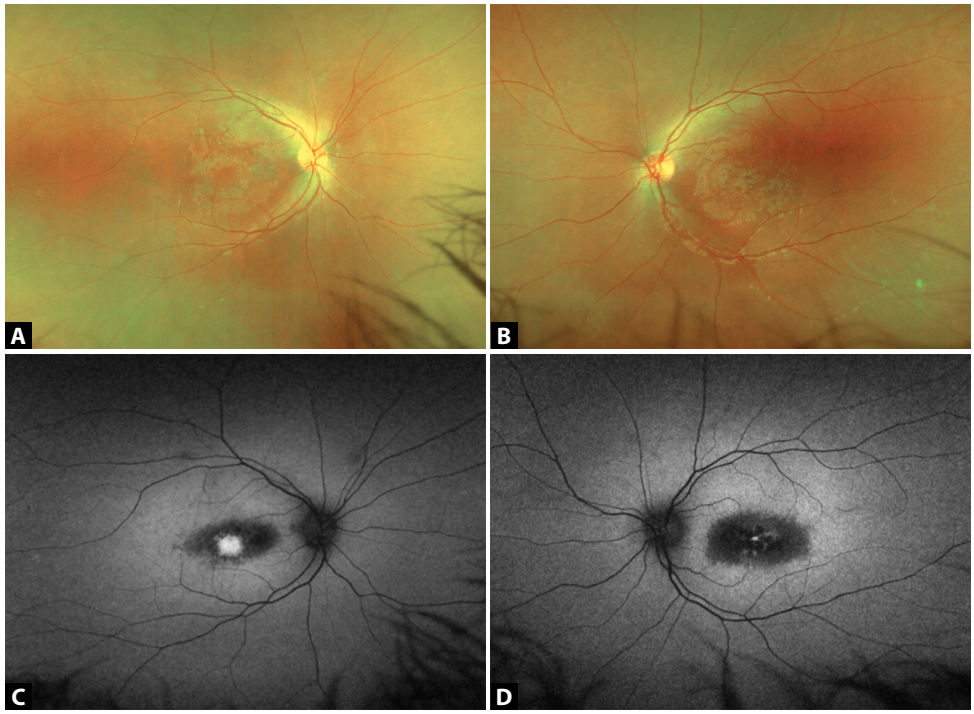
Increased fundus autofluorescence:

- Drusen
- Excessive RPE lipofuscin accumulation
- Age-related macular degeneration.

Fundus autofluorescence is useful in the diagnosis of:

- Age-related macular degeneration
- Retinitis pigmentosa
- White dot syndromes
- Central serous chorioretinopathy

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Figs 28.5A to D: Fundus autofluorescence showing heredomacular dystrophy. **A and B.** Fundus as seen in normal digital fundus photography; **C and D.** Same eyes with FAF camera system

- Macular dystrophies like—Stargardt disease, cone dystrophies and Best's disease
- Chloroquine maculopathy.

Indocyanine Green Angiography

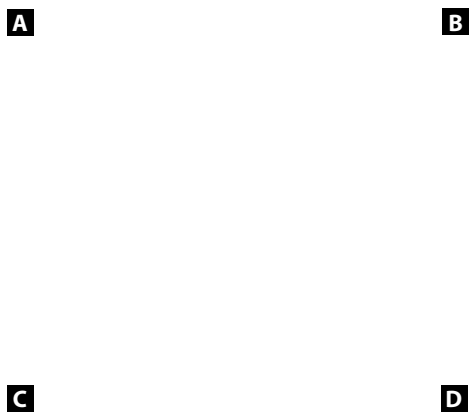
Indocyanine green angiography (ICGA) is a diagnostic technique that uses ICG dye's infrared fluorescence to provide details about the choroidal circulation and its disorders (Figs 28.6A to D).

- Indocyanine green angiography is an amphiphilic tricarbocyanine dye and the lyophilized powder is dissolved in water for injection. It is 98% bound to proteins and remains longer in large blood vessels with a lesser tendency to diffuse in the interstitial space. Hence, it is ideal for diseases affecting the choroidal tissues.
- Plasma half-life of ICG is 2–4 minutes. Maximal peak of absorption is at 790–805 nm and a peak emission of 835 nm.
- There are 2 types of ICG systems—modified fundus cameras and SLO-based systems. Dosage of dye varies from 20–50 mg dissolved in 2–4 mL of aqueous solvent. Preferred technique is to inject 25 mg dye in 5 mL of water slowly.
- Some iodine is present in the dye and crossover allergy can occur. Contraindications are prior anaphylactic reactions to iodine-based preparations, hepatic insufficiency, uremia and pregnancy.

Phases

- ***Early phase:*** It occurs 2 seconds after injection of ICG, choroidal arteries and choriocapillaris fills up, 3–5 seconds after injection, larger choroidal veins begin to fill.
- ***Middle phase:*** It occurs at 3–15 minutes after injection and marked by continuous fading of choroidal and retinal vessels.

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Figs 28.6A to D: Indocyanine green angiography (ICGA). **A.** Digital fundus photograph of a case of occult choroidal neovascular membrane (CNVM); **B.** Same lesion as visible under autofluorescence; **C.** Same lesion as seen in fundus fluorescence angiography; **D.** Same lesion best visible in ICGA (white arrows)

- ***Late phase:*** It occurs at 15–60 minutes after injection.

Abnormal areas can be described as hypocyanescence or hypercyanescence.

Hypocyanescence

Blockage–blood/serous fluid/pigment/exudates/impaired choroidal perfusion.

Hypercyanescence

- Lack of overlying tissue—RPE dropout/lacquer cracks
- Leakage from surrounding vessels
- Leakage from abnormal blood vessels
  - Hot spots and plaques are used to define areas of intense hyperfluorescence during the middle and late phases.
  - Hot spots are defined as <1DD (disc diameter) in size. Plaques are >1DD in size and reveal less obvious leakage.

ULTRASONOGRAPHY IN OPHTHALMOLOGY

Sound frequencies between 16 and 20,000 Hz are audible, and frequencies above 20,000 Hz, are called ultrasound.

The ultrasound is reflected towards its source, when it encounters a change in elasticity or density of the medium through which it is passing. This refleced vibration ***(echo)*** is converted into an electric potential by a ***piezoelectrical crystal,*** and displayed on an oscilloscope.

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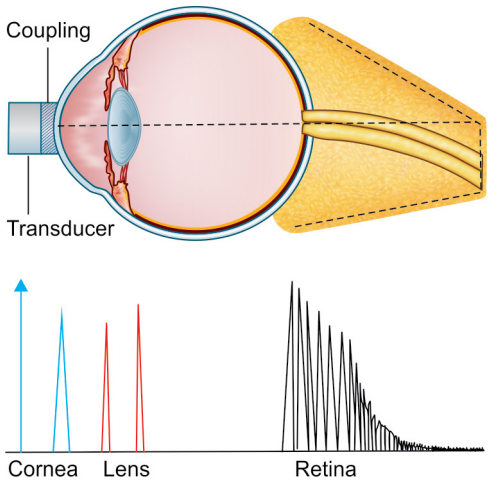


Fig. 28.7: A-scan ultrasonography

Ultrasonic frequencies in the range of 10 MHz are used in ophthalmic ultrasonography (USG). ***Two types of USG are used: A-scan and B-scan.***

Ultrasonography A-scan (Fig. 28.7)

A transducer is positioned so that the ultrasonic beam passes through a chosen ocular meridian. The tracing records a series of spikes, at sites of change in intraocular impedance. The ***height of each spike*** depends on the acoustic density of the tissue, which varies with cellular composition. ***The distance*** between the spikes, gives a measure of the distance from the transducer, as well as distance between intraocular structures. A-scan is ***one-dimensional,*** and hence, amplitude modulated display provides the information. A-scan may be axial or non-axial ***(di-scleral)*** (Figs 28.8A and B).

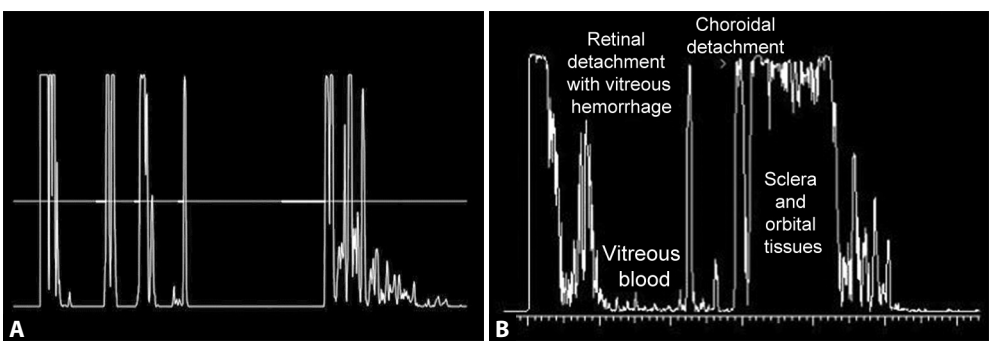
Uses of A-scan

- Axial length measurement for intraocular lens power calculation.
- Measurement of anterior chamber depth, lens thickness, or depth of a lesion.
- To detect pathological lesions preoperatively, in presence of opacities in the ocular media.
- To differentiate a preretinal membrane from a retinal detachment ***(quantitative echography)***.
- To differentiate between benign and malignant intraocular lesions.
- To measure corneal thickness ***(ultrasonic pachymetry)*** before radial keratotomy operation, or PRK or LASIK.

Ultrasonography B-scan (Fig. 28.9)

The testing transducer is moved in a linear fashion across the eye, to build up a ***two-dimensional*** picture of the intraocular structures, and the orbit.

Echoes are plotted as ***dots*** instead of spikes, and the brightness of the dot indicates



Figs 28.8A and B: Ultrasonography A-scan. **A.** Normal spikes in normal eye; **B.** Abnormal spikes in vitreous chamber indicating vitreous hemorrhage with retino-choroidal detachment

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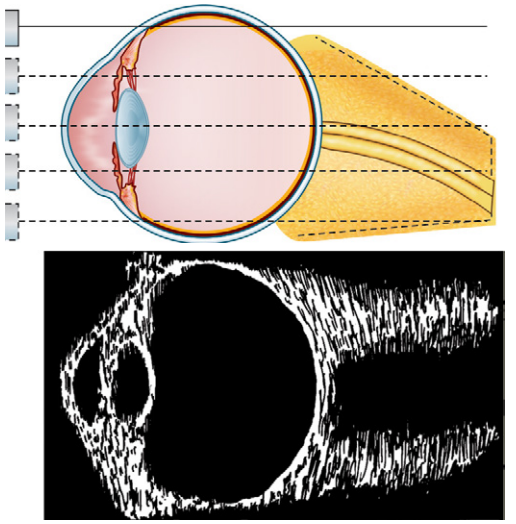


Fig. 28.9: B-scan ultrasonography

the size of the received echoes. The resulting B-scan picture is comparable to a histological cross-section through the eye and the orbit. B-scan may be taken on the sagittal, horizontal or oblique planes of the eye.

Uses of B-scan (Figs 28.10A and B)

- To differentiate the space-occupying lesions within the eye and the orbit.
- To determine the vitreoretinal pathology (e.g. retinal detachment, or vitreous hemorrhage) in presence of opacity in the ocular media.
- To localize the intraocular foreign body, especially, if it is not radio-opaque.
- To study the muscle thickness, e.g. in thyroid ophthalmopathy or orbital pseudotumor.
- To study the vitreous hemorrage and its complications (e.g. vitreous bands) prior to vitreous surgery.
- To differentiate the types of retinal detachment, (e.g. rhegmatogenous, exudative or tractional).

ULTRASOUND BIOMICROSCOPY (UBM)

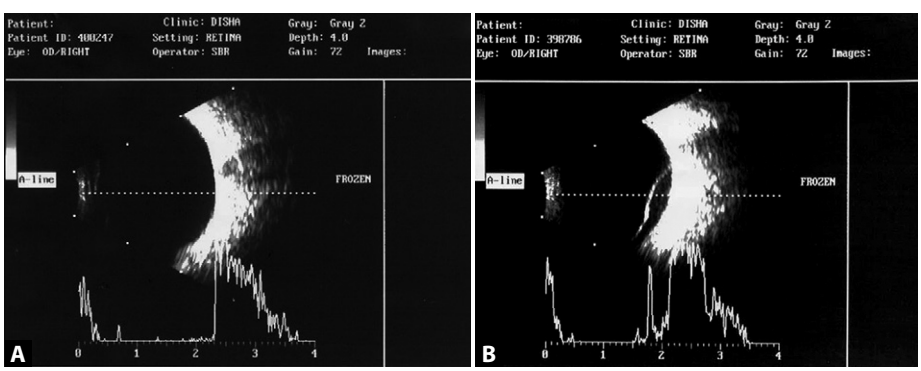
It is a new ultrasound-guided diagnostic tool which allows detailed imaging of the anterior segment of the eye ***(like slit-lamp biomicroscopy)***.

High-frequency, high-resolution ultrasound biomicroscopy is similar in principle to conventional USG B-scan.

In general, the resolution and depth of penetration of ultrasound waves are affected by transducer frequency.

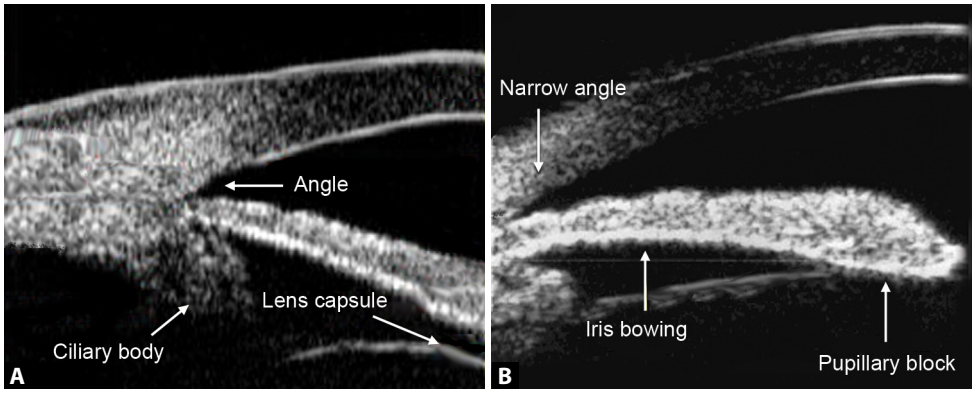
The ocular USG B-scan uses a 10 MHz transducer, with approximately 150 μm resolution.

Whereas, an UBM is performed with a ***high frequency (50 MHz)*** transducer with a tissue resolution of 50 μm. But this is at the expense of decreasing tissue penetration depth of 4–5



Figs 28.10A and B: A. Normal USG-B scan; **B.** Abnormal membrane (spikes) in front of lower retina indicating inferior retinal detachment

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Figs 28.11A and B: A. Normal human eye; **B.** Pupillary block

mm which is sufficient to image the anterior segment (Figs 28.11A and B).

Ultrasound scanning of the cornea is possible with a 100 MHz transducer that increases the tissue resolution to less than 20 μm.

Uses

- High frequency UBM allows detailed imaging of the:
  - Anterior segment of the eye
  - Angle of the anterior chamber (Fig. 28.11)
  - Ciliary body (e.g. tumor, etc.)
  - Pars plana and peripheral vitreous (e.g. pars planitis).
- It also helps to identify position of intraocular lens and its haptics.

Advantage of UBM: To assess the above structures in patients with media opacity—like, edematous or scarred corneal tissue.

RADIOLOGY IN OPHTHALMOLOGY

Each orbit is a four-sided pyramidal cavity with apex aimed posteromedially and the base towards the face. It is made up of ***parts of seven bones*** and divided into the roof, medial wall, lateral wall and the floor. Radiological examination of the eye and orbit is useful for evaluating trauma, foreign body and tumors.

X-ray

X-ray studies of the orbits are difficult because of superimposition of the other bones of the skull. The patients is placed on the radiographic table, usually in the prone position.

Several techniques are used to localize lesions at a particular depth and side:

- ***Caldwell's view*** is a PA projection of the orbit. This is important for orbital rim, roof and superior orbital fissure.
- ***Water's view*** is useful for orbital floor.
- ***Rheese view*** is useful for optic canal.
- ***Oblique view*** is especially useful for orbital rim fracture.
- ***Lateral view*** is useful for localization of foreign bodies.
- ***X-ray orbit with limbal marker*** to localize intraocular metallic foreign body more precisely in relation to the limbus.
- ***Dacryocystography*** is an X-ray evaluation of the drainage system of lacrimal apparatus in an attempt to localize the precise site of obstruction.
- ***Orbital venography*** is an X-ray evaluation of the orbital veins with a contrast dye to diagnose orbital varix in case of proptosis.

CT Scan

The CT scan is the study of choice for soft tissue and bony abnormalities. The main

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diagnosis possible with CT scan is the orbital tumors. The other diagnosis possible with CT scan includes:

- Ruptured globe
- Intraocular or intraorbital foreign body
- Ocular tumors
- Fracture of any wall including, blow-out fracture
- Secondary sinus involvement
- Neuro-ophthalmological diseases.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is the procedure of choice for soft tissue anatomy and pathology of vascularized lesions of ocular, orbital and neuro-ophthalmic structures from the orbit to the brain. MRI is also useful in early diagnosis of multiple sclerosis.

Magnetic Resonance Angiography

It is a non-invasive method for imaging the carotid and major cerebral arteries, helpful for neuro-ophthalmologic diagnosis, like carotid artery occlusion/stenosis, any aneurysm, arteriovenous malformation, vascular mass, etc.

OCULAR EMERGENCIES

Ocular emergencies are those situations that requires immediate attention and management. The following situations need emergency care of the patients:

- ***Chemical burns:*** Alkali, acid or organic solvents in the eye.
- ***Sudden painless total loss of vision:*** This problem suggests an acute vascular occlusion and could cause permanent loss of vision if not treated immediately, especially in central retinal arterial occlusion (CRAO).

Above two conditions need real emergency management without any delay.

Others are:

- ***Open globe*** injury of the eyeball.
- ***Severe blunt trauma*** such as a forceful blow to the eyeball with a fist or by a cricket or tennis ball.
- ***A foreign body*** in the eye.
- ***Corneal abrasion*** caused by a foreign body or by trauma.
- ***Sudden painless severe dimness of vision***—as in optic neuritis or retinal detachment.
- ***Acute painful red eye with loss of vision,*** e.g. acute attack of angle closure glaucoma, lens-induced glaucoma, etc.
- ***Any emergency referral*** from other physician or ophthalmologist.

Ocular Urgent Situations

The urgent situation requires the patient should be seen within 24–48 hours. The patients with urgent conditions may report with a variety of symptoms, some of which may suggest an emergency situation, while the others, although not serious, but may deserve prompt attention. Sometimes, it is difficult to distinguish an urgent situation from an emergency.

The following situations are generally considered to be urgent:

- Subacute or gradual loss of vision over a few days to a week
- Sudden onset of diplopia
- Recent onset of flashes and floaters especially in myopes
- Acute red eye with or without discharge and loss of vision
- Acute and progressively increasing ocular pain
- Sudden onset of photophobia
- Loss or breakage of glasses or contact lenses needed for work, driving or studies (though not all physicians consider losing or breaking glasses as urgent).

SUBCONJUNCTIVAL INJECTION

Subconjunctival injections are most useful for attaining effective concentration of drugs in

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the aqueous. Though, in most cases they can be replaced by frequent topical application of drugs.

Routes of absorption of drugs after subconjunctival injection:

- Part of the effects of a subconjunctival injection is due to gradual reflux of the drug along with the needle track, with subsequent ***transcorneal*** absorption.
- Partly, via the sclera adjacent to the injection site.

Disadvantages

- These injections may be painful, specially in case of inflamed eyes.
- Conjunctiva tends to become scarred.
- Accidental globe perforation may occur which is dangerous.

Indications

- Severe ocular infections, e.g. in bacterial corneal ulcers (mainly antibiotic and atropine).
- Severe intraocular inflammation, e.g. iridocyclitis (steroids and atropine) or endophthalmitis (antibiotics and atropine).
- At the end of any intraocular surgery, e.g. after cataract operation or penetrating keratoplasty (gentamycin and dexamethasone).
- To give subconjunctival anesthesia in certain operations, e.g. pterygium resection, or to remove a conjunctival cyst or mass.

Procedure

i. Topical anesthesia by 4% lignocaine or 0.5% proparacaine. A small cotton-palette soaked in anesthetic agent is held on the area for 2 minutes—very effective.
ii. Patient is lying on a bed.
iii. Care has to taken to minimize patient's apprehension.
iv. Wire speculum is applied.
v. Patient is asked to look upward or opposite side away from the site of injection.
vi. 0.5-1 mL of drug is taken in a 1 mL or 2 mL syringe with 26 gauge needle or (in an insulin syringe).
vii. Gently hold a fold of conjunctiva in a tent fashion, 3 mm away from the limbus with a plain forceps, avoiding the blood vessels.
viii. The needle tip with beveled up, is slid underneath the bulbar conjunctiva. The plunger of the syringe is withdrawn to ensure against any intravascular penetration.
ix. Inject about 0.5 mL solution at a time.
x. Gently withdraw the needle.
xi. Cover the eye with a pad for 1 hour.

SUB-TENON INJECTION

Here, the main route of absorption is via the transscleral route near the injection site. Posterior sub-Tenon injection is mostly preferred.

This is the most effective method of continuous drug delivery, specially in inflamed eyes. The most popular drug used for sub-Tenon injection is depot steroids, e.g. triamcinolone or methylprednisolone with or without atropine and antibiotic.

Indication: Chronic uveitis, especially in pars planitis and posterior uveitis.

Best site of injection: Upper temporal quadrant. Alternately, the inferotemporal quadrant may be selected for injection.

Procedure

Step (i) to (v) same as subconjunctival injection.

- Patient is instructed to look down and in, towards his nose.
- With the beveled side towards the globe, the needle is introduced into the superior bulbar conjunctival area avoiding the conjunctival blood vessels and moved from side to side and injected deep into the posterior sub-Tenon's space.

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Disadvantages

- It is technically difficult.
- Chance of globe perforation is more than subconjunctival injection.

LABORATORY DIAGNOSIS OF OCULAR INFECTIONS

Conjunctival Swab

Laboratory diagnosis in bacterial conjunctivitis is not a routine. However, the ***indications*** of conjunctival scraping for microscopic examination are:

- Neonatal conjunctivitis
- Hyperacute purulent conjunctivitis
- Chronic recalcitrant conjunctivitis.

Conjunctival swab is a routine procedure before any intraocular operation (e.g. cataract operation, glaucoma operation) in some places. But it is not a mandatory.

Methods

- Conjunctival scrapings should be taken prior to the use of topical anesthetics, as these agents and their preservatives may mask the recovery of certain bacteriae.
- Cultures are taken by moistering a sterile alginate (not cotton) swab with saline and wiping the inner lid margin or conjunctival cul-de-sac.
- They are then inoculated in solid or liquid culture media (blood agar and/or chocolate agar).
- Simultaneous smear may be prepared for staining with Gram or Gimsa-stain. This is to identify the organisms and inflammatory cell types. The cytologic features of each type of conjunctivitis are different and helpful in diagnosis.

Corneal Scraping

This is the most important step in early diagnosis of a corneal ulcer, for prompt and rigorous initiation of therapy.

- The scraping is performed after instillation of topical preservative-free anesthetic agents (e.g. proparacaine 0.5% or lignocaine 2–4%).
- The procedure should obtain as much material as feasible, particularly from the deeper areas (floor) and the margins of the ulcer.
- Scraping material is usually taken with a no. 15 blade or a Kimura's spatula.
- Smear is prepared for microscopic examination with KOH mount, Gram or Gimsa stain. In 30-40% cases in results may be negative for bacteria even if infection is present.
- Culture should be done on blood agar plate (at room temperature) and Sabouraud's media (for fungus) and into Page's medium (for acanthamoeba), if suspected.

Aqueous or Vitreous Tap

In suspected cases of infective endophthalmitis—to find out the causative agent.

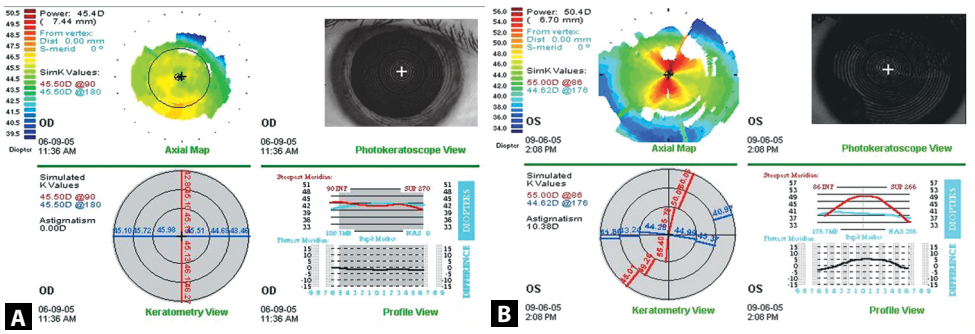
CORNEAL TOPOGRAPHY (FIGS 28.12A AND B)

It is a method of qualitative and quantitative evaluation of the corneal surface by the reflected light.

- In 1619, Father Scheiner first found that corneal surface can be studied from reflection of a window on it ***(window reflex)***.
- In 1880, Placido introduced a disk ***(Placido's disc)*** with alternate white and dark concentric circles with central aperture for viewing. These circles were reflected on the cornea. A qualitative assessment was made.

The invention of videography has been incorporated into the Placido image system. The image thus captured on a CCD camera and analyzed by computer software on the basis of relative distances between the rings. 'Shorter the distance-steeper is the cornea' and vice versa. Normal cornea is a prolate surface, i.e. steeper in the center and flatter in the periphery.

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Figs 28.12A and B: **A.** Normal corneal topography; **B.** Corneal topography in keratoconus

The computer adds chosen color to the image and produces a color-coded map of the whole cornea. By standardization the:

- ***Warm (hot) colors*** such as red, yellow represent high corneal power ***(steeper)***.
- ***Cool colors*** like violet, blue represent low corneal power ***(flatter)***.
- ***Yellow-green spectrum*** represents the most normal corneas.

A scale is given alongside the color-coded map which gives the numerical values in diopters to the specific colors.

Uses

- The curvature of the whole cornea can be studied.
- Early diagnosis of corneal ectatic disorders such as keratoconus, pellucid degeneration or secondary corneal ectasia, etc.
- Study of changes in corneal a stigmatism following surgical procedures-like, keratoplasty, cataract surgery, refractive surgeries, etc.
- Recent corneal topography machines, like Pentacam Oculyzer and Orbscan II are used to study both the anterior and posterior corneal surfaces as well as the thickness of the cornea.

SPECULAR MICROSCOPY

The ophthalmologist can visualize the corneal endothelium directly by performing specular reflection method of slit-lamp microscope. But, this is not enough to get sufficient information about cell density and morphology of the corneal endothelium.

Specular microscope is a useful instrument to visualize the corneal endothelium (at 500 x) to the ophthalmologists, especially the corneal surgeons.

It yields information that guides the physician in decision making of certain corneal disorders and anterior segment surgeries. It also captures the pictures that are useful for documentation and monitoring a patient over a period of time.

The corneal endothelium is a hexagonal monolayer that does not divide in human being.

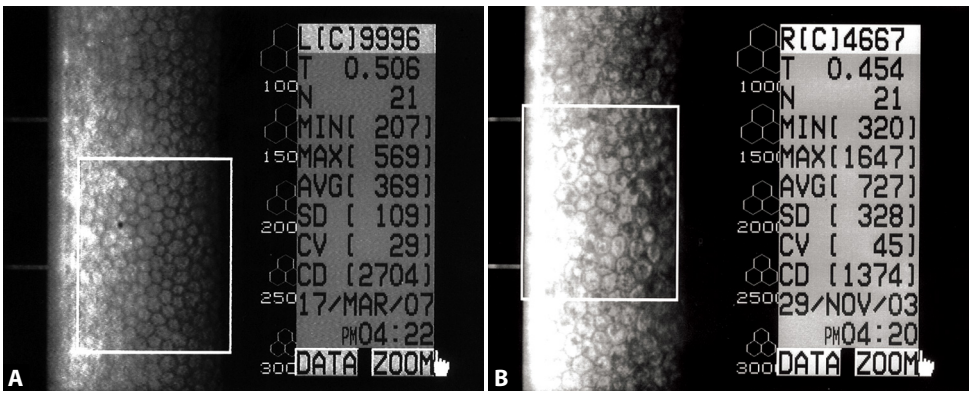
The cell count of an infant is around 3500–5000 cells/sq mm. With age, the cell count declines. The adults typically have 2000–2500 cell/sq mm of endothelium. The cell morphology is also changed with ages. In young adult, the percentage of hexagonal cells are more (>60%).

The critical ***endothelial cell*** count is around 500 cell/sq mm. If the cell count is <500 cells/sq mm, there may be chance of corneal decompensation and corneal edema (Figs 28.13A and B).

Specular Microscopy Analysis

- ***Cell density (CD):*** Number of endothelial cells/sq mm.
- ***Hexagonality:*** Percentage of hexagonal cells among total endothelial cells.

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Figs 28.13A and B: A. Normal specular microscopic photograph; **B.** Corneal guttata with low endothelial cell count in early Fuchs' dystrophy

- ***Cell size and shape:*** Percentage of cells that show polymegathism (different cell-sizes) and pleomorphism (different cell-shapes).
- ***Coefficient of variation of cell size:*** An automatic statistical index of polymegathism.
- ***Corneal thickness (CT):*** It gives the measurement of corneal thickness in μm in a noncontact way.

The additional software algorithm can analyze in depth the morphological changes of the corneal endothelium and can compare the results with other values.

Both contact and noncontact specular microscope are available. But practically, noncontact specular is more safe and popular.

Two types of specular microscopes:

1. ***Clinical specular:*** To study the endothelium in a clinical set-up.
2. ***Eye bank specular:*** To study the sclerocorneal donor button for keratoplasty purpose.

Uses

- To study the corneal endothelial diseases.
- To take decision about corneal surgery.
- Before cataract operation when there is any suspicion during slit-lamp examination (*see* Chapter 15).
- To study the safety of certain surgical procedures.
- To study the safety of certain drugs used intracamerally during surgery.
- During follow-up of keratoplasty and refractive surgery patients.

SCANNING LASER POLARIMETRY (GDx VCC) (FIGS 28.14A AND B)

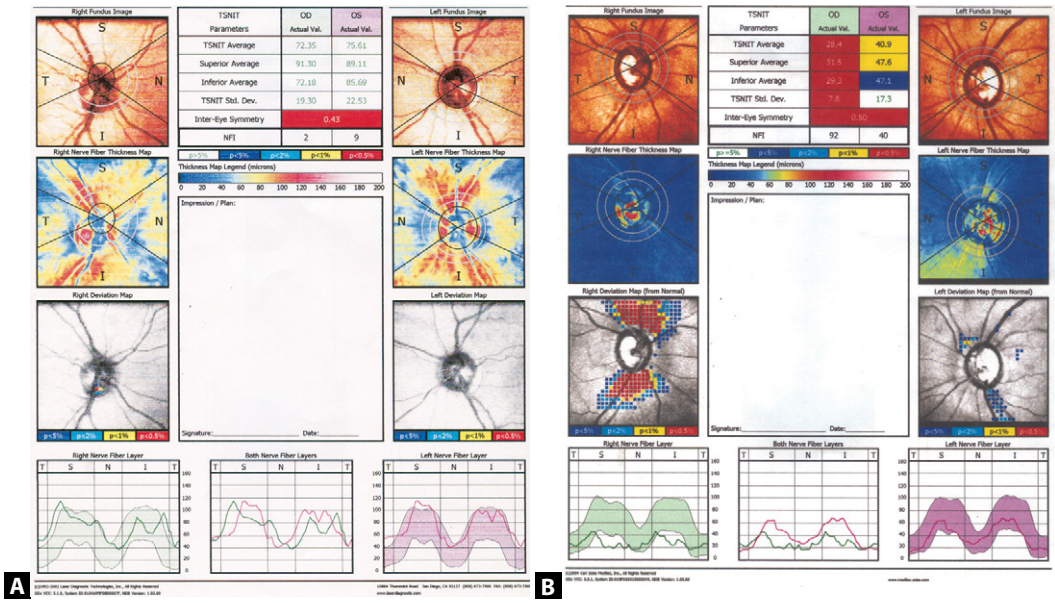
Glaucoma detection with variable corneal compensation (GDx VCC) is a scanning laser polarimeter which helps to assess the thickness of the retinal nerve fiber layer (RNFL) without dilatation.

Patients with early glaucoma having normal optic nerves and visual fields may have RNFL defects and thus, GDx VCC helps in diagnosing glaucoma in ***preperimetric stage***.

Principle: When polarized light passes through the RNFL, the birefringent properties of the axons causes polarized light to undergo a phase shift. The amount of phase shift or retardation is measured, and is proportional to the thickness of RNFL. This technique is known as ***scanning laser polarimetry***.

Using a near infrared ***diode laser*** (780 nm) RNFL thickness is measured in a 15° × 15° grid centered on the optic nerve head.

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Figs 28.14A and B: GDx VCC pictures. **A.** Normal appearance of RNFL with normal thickness; **B.** Thinning of RNFL—more in right eye with advanced glaucoma and inferior part in left eye with early glaucoma

The readings obtained are compared to a normative database.

The colored printout includes images and various parameters along with the probability of deviation of such values in compare to the normal database. RNFL thickness is represented using a color scale, with ***dark blue being the thinnest*** and the ***red being thickest***.

Uses

- It is a noninvasive quick technique for diagnosis and monitoring of early glaucoma.
- It may also be used as a screening tool for the ***'glaucoma-suspect'*** patients.

OPTICAL COHERENCE TOMOGRAPHY (OCT) (FIG. 28.15)

It is a new diagnostic tool mainly for the posterior segment disorders. It rapidly provides a quantifiable two (or three) dimensional cross-section (tomography) of the retina.

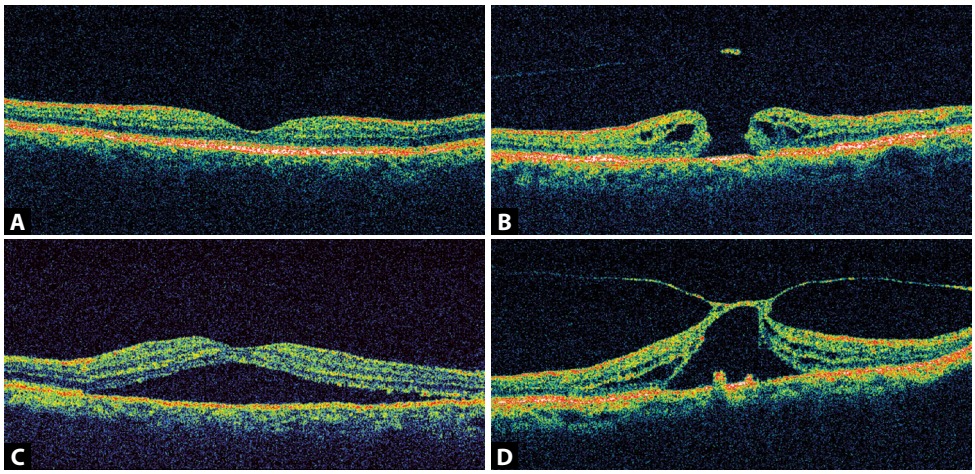
Ultrasonography B-scan mechanism has a similarity with OCT. But instead of dynamic echoes of sound used in USG, OCT relies on differential reflections of light. Spatial resolution of OCT is approximately 5 μm, whereas that of USG B-scan is approximately 150 μm.

Optical coherence tomography is based on the principle of low coherence interferometry. It uses an infrared incident beam of approximately 830 nm in wavelengths. The beam is focused on retina by a +78D lens.

Optical coherence tomography unit employs a Michaelson interferometer to compare the magnitude of back scattered and reflected light from the retina and demonstrates it as a false color-coded image in two/three dimensions by software manipulations.

Different types of OCT imaging have been developed: Time-domain and Fourier-domain techniques. The main advantages of Fourier-domain OCT are higher signal acquisition rates and higher sensitivity than time-domain OCT.

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Figs 28.15A to D: **A.** Normal optical coherence tomography; **B.** Macular hole with detached; **C.** Central serous retinopathy; **D.** Vitreomacular traction

Uses of Optical Coherence Tomography

- ***Macular imaging:*** In today's clinical ophthalmology, OCT is used mainly for imaging in macular diseases, like:
  - Diabetic macular edema
  - Macular edema related to vaso-occlusive diseases
  - Cystoid macular edema
  - Epiretinal membrane (ERM), with or without vitreomacular traction (Fig. 28.15D)
  - Macular hole or pseudohole (Fig. 28.15B)
  - Choroidal neovascular membranes (CNVM)
  - Central serous chorioretionopathy and pigment epithelial detachment (Fig. 28.15C).
- Anterior segment evaluation by anterior segment OCT.
- Quantification of peripapillary RNFL analysis.

Anterior Segment Optical Coherence Tomography

Optical coherence tomography is a non-contact optical device that provides cross-sectional images and quantitative analysis of the ocular tissues in the anterior and posterior segment.

Anterior segment OCT systems are categorized by the wavelength of the light sources.

- Dedicated systems using 1310 nm, e.g. Zeiss Visante, Heidelberg SL-OCT, Tomey CASIA.
- Systems converted from a retinal scanner using 830 nm, e.g Optovue, Zeiss Cirrus, Heidelberg Spectralis.

A shorter wavelength (830 nm, near infrared) provides a higher axial resolution, but its imaging depth is limited. A longer wavelength (1310 nm) provides deeper penetration.

Uses

- ***Refractive surgery and ectatic disorders:*** Used in visualization of flap thickness, flap interface and flap displacement post LASIK.
- ***Assessment of Descemet membrane:*** ASOCT can help in prognosis, e.g. planar/nonplanar, local/extensive detachment, and rupture of DM. Also useful in knowing the status of DM post PK, DMEK, DSAEK, e.g. lenticule detachment, fluid in the interface and status after rebubbling.

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- ***Deep anterior lamellar keratoplasty (DALK), descemet stripping endothelial keratoplasty (DSEK), Descemet's membrane endothelial keratoplasty (DMEK):*** Live intraoperative OCT is useful while performing DALK, DSEK and DMEK, e.g. dissection depth, insertion depth of cannula for Big bubble technique, lenticule insertion in DMEK and its attachment and interface quality.
- ***Corneal deposits:*** Useful in diagnosis of corneal dystrophies. Useful in the planning of PTK for superficial opacities, as in granular dystrophy.
- ***Keratitis:*** Useful in knowing the depth of the infiltrate and can help in the planning of therapeutic PK vs therapeutic DALK.
- ***Tumors and OSSN:*** It has a supplementary role in some nonpigmented tumors.
- ***Dry eye:*** Useful in tear meniscus measurement viz tear meniscus height (TMH), tear meniscus depth (TMD) and tear meniscus area (TMA).
- ***Cataract surgery:*** Useful in femtosecond assisted cataract surgery where a live image of the cornea and lens is available for detailed planning of main port, side ports, relaxing incisions and capsulorrhexis.

Optical Coherence Tomography in Glaucoma

- ***Angle assessment:*** Anterior segment OCT can provide important parameters about the anterior chamber, like the anterior chamber angle (ACA), angle opening distance (AOD 500 and 750 μm) and trabecular-iris space area (TISA).
  These parameters help in—angle assessment especially in angle closure screening, structural cause of angle closure, evaluation of efficacy of a laser iridotomy procedure and analysis of iris configuration.
- ***Filtering bleb and tube:*** ASOCT can reveal morphology of filtering blebs, the recently developed 3D ASOCT technique allows for a detailed evaluation of internal morphology of blebs. It can help determining which blebs are suitable for needling and for visualizing tube erosion in glaucoma drainage devices.
- ***Glaucoma diagnosis and progression:*** ASOCT allows for *in vivo* cross-sectional imaging of the optic nerve head (ONH) and retina.

Spectral domain optical coherence tomography (SDOCT) with 3D images helps in glaucoma diagnosis by studying the RNFL thickness, rim area and total macular thickness.

Ganglion cell layer with inner plexiform layer (GCIPL) is the most sensitive parameter for the diagnosis of glaucoma.

SDOCT is useful in detecting RNFL changes in glaucoma progression. SDOCT glaucoma progression algorithms measure changes based on either event-based or trend-based analysis.

HEADACHE IN OPHTHALMOLOGY

- ***'Headache'*** is defined by the dictionary, as continuous or prolonged dull pain in the region of the head.
- It is the ***most frequent and most ill-understood clinical symptom,*** which depends to a great deal on the emotional background and pain threshold of the subject.
- Headache is usually mild, vague, brief-lasting, and often innocuous, ***but sometimes,*** it is severe, persistent, or frequent, and could be a symptom of more serious conditions, in an apparently healthy individual.
- So, a patient presenting with headache as a primary symptom to an ophthalmologist, often presents a diagnostic challenge.

Causes of Ocular Headache

- Associated with refractive errors and ocular muscle imbalance.
- Secondary to organic diseases of the eye.
- Due to systemic disorders, but have prominent ocular symptoms.

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Headache associated with refractive errors and ocular-muscle imbalance:

- Majority of the patients with ocular headache belong to this category, and the pain is dull, boring and often associated with eye-strain.
- Headaches are usually experienced towards the afternoon, and typically, experienced on undertaking tasks which entail binocular vision, e.g. reading, writing, knitting, etc.
- ***Conditions responsible***
  - ***Astigmatism and hypermetropia:*** Low grades of error cause more headache, because the ciliary muscle tries to overcome the defect by spasm. But this element is lacking in higher errors, which present as visual deficit than headache.
  - ***Presbyopia*** (mainly on near work)
  - ***Heterophoria (latent squint)***
  - ***Convergence insufficiency***
- These headaches are relieved by proper glasses and orthoptic exercises.

Headache secondary to organic eye-diseases:

- These headaches are usually unilateral, acute, continuous and located in and around the eyeball, and often aggravated by light.
- The pain is characteristically radiated along the sensory distribution of trigeminal nerve.
- The patients often have sudden blurring of vision.
- ***The important causes:***
  - Angle-closure glaucoma
  - Acute iridocyclitis
  - Scleritis
  - Acute keratitis due to infection, contact lens over-wear, or photokeratitis.
- Treatment is directed towards the cause.

Ocular headache related to systemic diseases: This category includes the more serious diseases:

- Raised intracranial tension
- Malignant hypertension
- Migraine
- Temporal arteritis
- Psychogenic (e.g. anxiety neurosis).

Ophthalmic Evaluation of Headache

- ***Refraction under cycloplegia***
- Ocular movements
- ***Orthoptic check-up***
- Slit-lamp examination—to detect abrasion, ciliary congestion, KPs, etc.
- Ophthalmoscopy to check ***papilledema***
- Cranial nerve examinations
- Intraocular pressure measurement
- Gonioscopy to know the angle-status
- Palpation of the temporal artery for tenderness
- Automated perimetry for visual field.

Ophthalmologist's Approach

- The ophthalmologists are responsible for the management of headache due to refractive errors and ocular muscle imbalance, and those secondary to organic eye diseases.
- But, for the headaches related to systemic disease, his basic responsibility in early diagnosis (may be of an intracranial neoplasm by detecting the papilledema) and quick referral to other colleagues.
- On the other hand, every case of headache must undergo a through ophthalmological check-up, and general practitioner can play an important role to select such cases for proper referral.

MIGRAINE (TABLE 28.2)

It is a familial disorder characterized by:

- Recurrent attacks of headache, widely varied in intensity, frequency and duration.
- Attacks are commonly unilateral, and usually associated with anorexia, nausea or vomiting.
- In some cases, they are preceded by, or associated with neurological and mood disturbances.

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TABLE 28.2: Features of different types of migraine

Types	Characteristic features
Classical migraine	• Severe hemicranial throbbing pain • Preceded by an aura for 15 to 20 minutes • Visual symptoms are common, e.g. scintillating scotoma. In the dark field, zig-zag rays of various color are often seen, called 'fortification spectra' ***(teichopsia)*** • Sensory symptoms include paresthesia and numbness
Common migraine	• More common • Severe headache lasts for hours to 1–2 days • Associated with nausea, vomiting, and anorexia • No prodrome, no aura
Basilar migraine	• Bilateral visual symptoms with headache • Associated features of vertebrobasilar insufficiency (e.g. tinnitus, ataxia, vertigo) • More common in adolescent girls
Ophthalmoplegic migraine	• Common in children below 10 years of age • Ocular cranial nerve palsy (3rd most common) • Spontaneous recovery is common
Complicated migraine	• Associated with neurological deficit lasting for more than 24 hours after the headache
Hemiplegic migraine	• Associated with hemipiegia which outlasts headache
Retinal migraine	• Headache associated with transient unilateral visual loss
Migraine equivalent	• Auras occurring in isolation without headache • May be associated with abdominal pain • A variant of migraine
Cluster headache	• Severe unilateral headache in clusters, several times daily for several days or weeks • It is worse at night, and aggravated by alcohol ingestion

  - ***Incidence:*** About 5–10%.
  - ***Age and sex:*** It usually begins in childhood or early adult life, and is more common in women.
  - ***Pathophysiology:*** Migraine represents a disorder of cerebrovascular regulation. In a typical migraine, there is cerebral vasoconstriction → decrease in O_2 supply → brain cell hypoxia → biochemical and vascular changes → reactive extracranial vasodilatation leading to typical headache.

  Vasoconstriction initiates the prodromal symptoms including the neurological manifestations, and is due to:
  - Release of serotonin and thromboxane A_2 from platelets ***(vascular theory)***.
  - Substance P, a neurotransmitter ***(neurogenic theory)***.
- ***Trigger factors:*** Migraine is often precipitated by some extrinsic or intrinsic factors, e.g. alcohol, cheese, chocolate, dairy products, change in weather, oral contraceptive pill, menstruation, change in sleep habit, stress, fatigue, depression, etc.

Treatment of Migraine

- ***Treatment for an acute attack:*** It may include analgesics, antiemetics, sedatives, ergot-derivatives and sometimes systemic steroids. Ergots should be used cautiously. New drug, Sumatriptan, a 5-HT-receptor agonist is highly effective for the treatment of an acute attack, with fewer side effects.
- ***Prophylactic therapy:*** Prevention of future attacks is only considered with the frequency of attacks is more than two per months.

  The agents are beta-blockers (e.g. propranolol), cyproheptadine, anti-depressants, calcium-channel blockers (e.g. flunarizine), methysergide, etc.
- ***Reduction of migraine trigger-factors:*** Try to avoid all the 'trigger-factors' which may precipitate a migraine attack.

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