CONTENTS

Anatomy and Physiology of Eye

SQs

1) Visual fields [13, 09]

Ans.

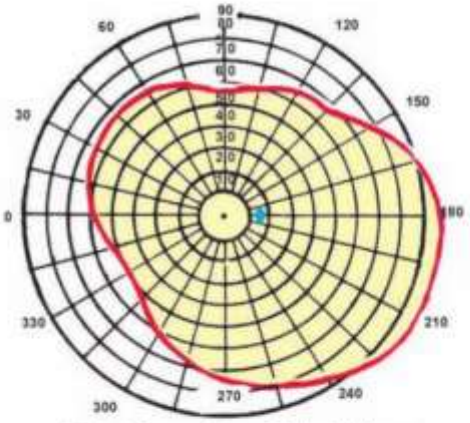
Visual field is a 3-D area of subject's surroundings that can be seen at any one time around an object of fixation

⇒ It is described as **'island of vision surrounded by a sea of blindness'**.

⇒ The extent of normal visual field with a 5 mm white colour object is superiorly 50°, nasally 60°, inferiorly 70° and temporally 90°.

⇒ The visual field can be divided into central & peripheral field:

- Central field includes an area from the fixation point to a circle 30° away. The central zone contains physiologic blind spot on the temporal side.
- Peripheral field of vision refers to the rest of the area beyond 30° to outer extent of the field of vision.



Extent of normal visual field of right eye

⇒ **PERIMETRY** is used to evaluate both central and peripheral visual fields using targets of various sizes and colours.

KINETIC PERIMETRY: In this, the stimulus of known luminance is moved from a peripheral non-seeing point towards the centre till it is perceived. **Ex:** confrontation method, Lister's perimetry, scotometry & Goldmann's perimetry.

STATIC PERIMETRY: In this, the stimulus is at fixed position with varying luminance in the field of vision.

Ex: Goldmann perimetry, automated perimetry.

MANUAL PERIMETRY: confrontation method, Lister's perimetry, scotometry & Goldmann's perimetry.

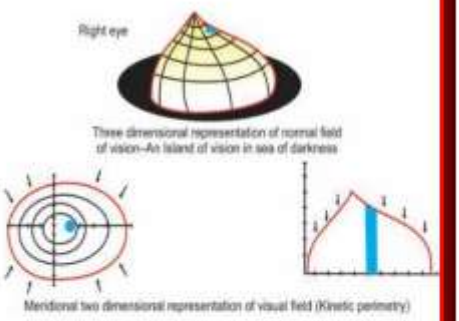
AUTOMATED PERIMETRY: Automated perimeters are computer assisted and test visual fields by a static method. Ex: Humphrey's Field Analyzer

Advantages of automated perimetry over manual perimetry are:

- Flexibility & level of precision
- Data storage capability, ease of operation, well controlled fixation, menu driven software
- Can compare results statistically with normal individuals of the same age group and with previous tests of the same individual.
- Examiner bias is eliminated.

⇒ ***Uses of perimetry***: Charting of the visual fields is useful in the diagnosis of many diseases like –

- Glaucoma
- Retinal diseases e.g., retinitis pigmentosa
- Follow up of laser treatment for diabetic retinopathy
- Neurological disorders, e.g., brain tumours, head injury, multiple sclerosis, cerebral thrombosis, aneurysms.



Three dimensional representation of normal field of vision–An island of vision in sea of darkness

Meridional two dimensional representation of visual field (Kinetic perimetry)

VSQs

1) Rods and Cones [17]

Ans.

- Sensory organs {photoreceptors}
- Present in Outer nuclear layer of retina

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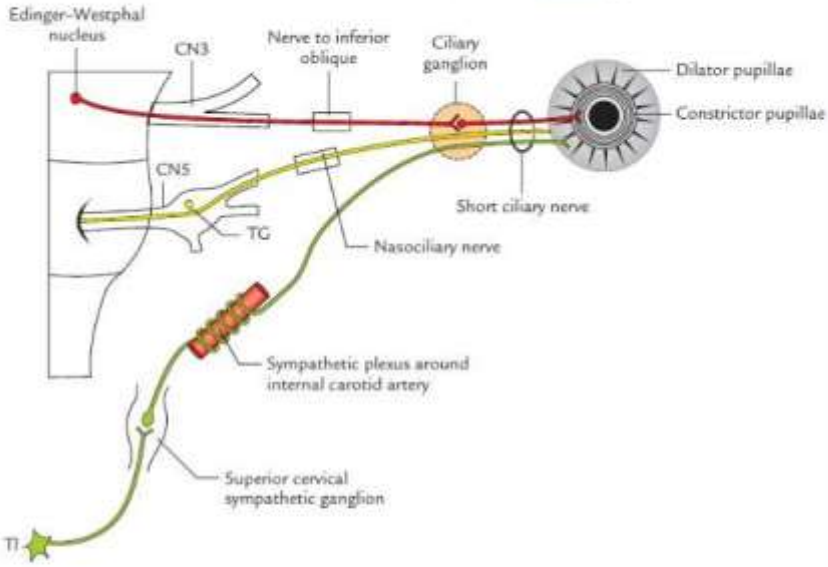
2) Ciliary ganglion [15]

Ans.

Ciliary ganglion is a peripheral parasympathetic ganglion placed in the course of oculomotor nerve near the apex of orbit.

Roots of ciliary ganglion:

- Sensory root comes from the nasociliary nerve.
- Sympathetic root comes from internal carotid plexus. These fibres do not relay here and pass along the short ciliary nerves to supply the blood vessels of the eyeball.
- Parasympathetic root arises from the nerve to inferior oblique muscle and carries the preganglionic fibres from the Edinger-Westphal nucleus. These fibres relay here and postganglionic fibres pass through the short ciliary nerves.
- **Short ciliary nerves**, branches of ciliary ganglion, supply the sphincter pupillae and ciliary muscle



Roots and distribution of the ciliary ganglion.

Importance: The ciliary ganglion is blocked to produce dilatation of pupil before cataract extraction

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Optics and Refraction

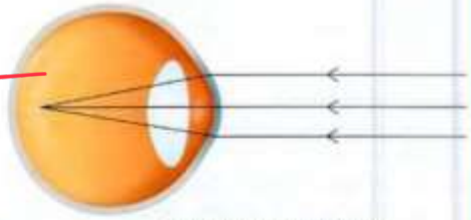
LQs

1) Define Emmetropia. Write about Myopia – etiology, clinical features & management [16, 13, 11]
   a. Surgical treatment of myopia [07]

Ans.

Emmetropia (optically normal eye) can be defined as a state of refraction, where in the parallel rays of light coming from infinity are focused at the sensitive layer of retina with the accommodation being at rest

Myopia or short-sightedness is a type of refractive error in which parallel rays of light coming from infinity are focused in front of the retina when accommodation is at rest.



Refraction in a myopic eye

Etiological classification

1. Axial myopia (MC form) results from ↑ in AP length of the eyeball.
2. Curvatural myopia occurs due to ↑ curvature of the cornea, lens or both.
3. Positional myopia is produced by anterior placement of crystalline lens in the eye.
4. Index myopia results from ↑ in refractive index of lens associated with nuclear sclerosis.
5. Myopia due to ↑ accommodation occurs in patients with spasm of accommodation.

Grading of myopia: American Optometric Association (AOA) has defined 3 grades of myopia:

- Low myopia, when the error is ≤-3D.
- Moderate myopia, when the error is berween-3D to -6D.
- High myopia, when the error is ≥ -6D.

Clinical varieties of myopia

1. Congenital myopia.
2. Simple or developmental myopia – MC variety
3. Pathological or degenerative myopia.
4. Acquired or secondary myopia – occurs secondary to some other factors such as: post-traumatic, post-keratitic, drug-induced, pseudomyopia, space myopia, night myopia, and consecutive myopia.

CONGENITAL MYOPIA

- Present since birth, is usually diagnosed by the age of 2-3 years.
- Most commonly unilateral. Rarely, it may be bilateral.
- High degree of error, about 8 to 10D, is usually present
- Convergent squint may develop in order to preferentially see clear at its far point
- Can be a/w other congenital anomalies such as cataract, aniridia, megalocornea, and congenital separation of retina.

SIMPLE MYOPIA

It is considered as a physiological error not associated with any disease of the eye. Since, the sharpest rise occurs at school going age, i.e. between 8 years to 12 years so, it is also called school myopia.

- Etiology: It results from normal biological variation in the development of eye. It can be :–
  - Axial type of simple myopia.
  - Curvatural type of simple myopia.

» Myopia is aggravated by close work, watching TV, smart phones, computers, limited outdoor activity & not using proper glasses

Clinical features of Simple Myopia	
Symptoms	**Signs**
▪ Short-sightedness ▪ Asthenopic symptoms ▪ Half shutting of the eyes may be complained by parents of the child. ▪ A change in psychological outlook of the children – Due to poor far vision and normal near vision, the children become introvert, studious and develop little interest in the outdoor activities.	➔ Prominent eyeballs: myopic eyes are typically large ➔ Anterior chamber is deeper than normal. ➔ Pupils are slightly large & react sluggishly to light ➔ Fundus is normal; rarely temporal myopic crescent maybe seen. ➔ Magnitude of refractive error: Simple myopia usually occurs in children and it keeps on increasing till about 18- 20 years of age at a rate of about - 0.5 ± 0.30 every year.

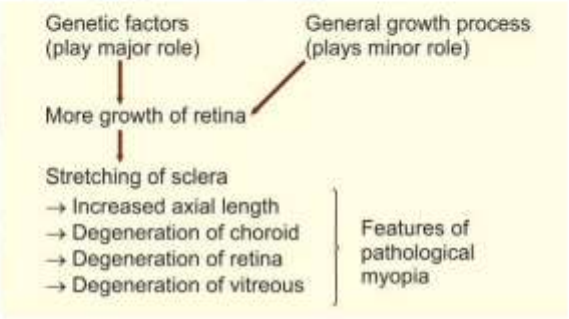
➔ **Diagnosis**: is confirmed by performing clinical refraction.

PATHOLOGICAL MYOPIA

Pathological/ degenerative/ progressive myopia, is a rapidly progressive error which starts in childhood at 5- 10 years of age and results in high myopia (>6D) during early adult life which is usually associated with degenerative changes in the eye. It is less common (about 2% of population).

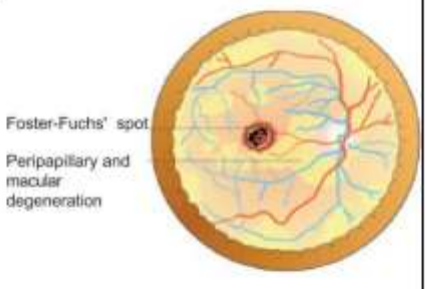
Etiology

1. **Role of heredity**
   - ➔ Familial
   - ➔ Race: More common in certain races like Chinese, Japanese, Arabs and Jews.
   - ➔ Sex: Women > men.
2. **Role of general growth process** – factors such as nutritional deficiency, debilitating diseases & endocrine disturbances also influence the progress of myopia.



Etiological hypothesis for pathological myopia

Clinical features of Pathological Myopia	
Symptoms	**Signs**
1) Defective vision 2) Muscae volitantes, i. e. floating black opacities in front of the eyes. 3) Difficulty in night vision may be complained by very high myopes having marked degenerative changes.	Same as Simple Myopia – except: 1) **Fundus examination reveals**: a. Optic disc appears large and pale and at its temporal edge a characteristic myopic crescent is present. b. Degenerative changes in retina and choroid: ⇒ **Foster-Fuchs' spot** may be present at the macula. ⇒ **Snail track lesions** ⇒ Total retinal atrophy may occur in an advanced case. c. Posterior staphyloma d. Degenerative changes in vitreous: liquefaction, vitreous opacities, and posterior vitreous detachment (PVD) appearing as **Weiss' reflex**. 2) **ERG** may reveal subnormal electroretinogram due to chorioretinal atrophy.

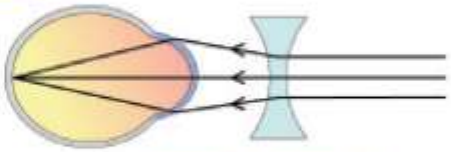


Fundus changes in pathological myopia

Complications: Retinal detachment, Complicated cataract, Vitreous haemorrhage & Choroidal haemorrhage,

Treatment of myopia

1. **Optical treatment of myopia** – prescribe appropriate concave lenses
   - ⇒ Basic rule of correcting myopia is converse of that in hypermetropia, i.e., the minimum acceptance providing maximum vision should be prescribed.
   - ⇒ Contact lenses are justified in cases of high myopia as they avoid peripheral distortion produced by spectacles.



Refraction in a myopic eye corrected with concave lens.

2. **Surgical treatment of myopia:**

Cornea based procedures	Lens based procedures
1. **Radial keratotomy** (RK) – for low to moderate myopia. 2. **Orthokeratology** – can correct upto -5D of myopia 3. **Laser ablation corneal procedures** a. Photorefractive keratectomy (PRK) – can correct upto -6D of myopia. b. Laser in -situ keratomileusis (LASIK) – refractive surgery of choice for myopia of up to -8 D.	1) **Refractive lens exchange (RLE)**: Lens extraction with IOL implantation of appropriate power can be done for myopia of > 12D. 2) **Phakic refractive lens (PRL) or implantable contact lens (ICL)** is also being done for correction of myopia of >8D.

Advantages of LASIK	Disadvantages
a. Minimal post-op pain. b. Early Recovery of vision c. No risk of perforation during surgery. d. No residual haze unlike PRK where sub-epithelial scarring may occur.	1) LASIK is more expensive. 2) It requires greater surgical skill than RK and PHK. 3) There is potential risk of flap related complications

4. **Refractive Lenticule extraction** (ReLEx): aka SMILE (small incision Lenticule extraction) – can correct myopia up to – 10 D.
5. **Intercorneal ring** (ICR) implantation

3. **Low vision aids (LVA)** are indicated in patients with progressive myopia having advanced degenerative changes, where useful vision cannot be obtained with spectacles and contact lenses.

Preventive measures:

1) **General measures**:
   - » Balanced diet rich in vitamins and proteins.
   - » Early management of associated debilitating disease.
   - » Visual hygiene.
   - » Avoidance of excessive near work and excessive use of video display units (VDUs)
   - » ↑ Outdoor activity in childhood may prevent progression of myopia.
2) **Genetic counselling**: the hereditary transfer of disease may be decreased by advising against marriage between two individuals with progressive myopia. However, if they do marry, they should not produce children

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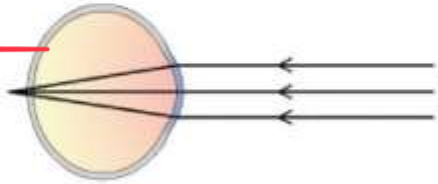
2) Define ametropia. Discuss Hypermetropia – types, clinical features, complications and management [14, 13]

Ans.

Ametropia (a condition of refractive error), is defined as a state of refraction, when the parallel rays of light coming from infinity (with accommodation at rest), are focused either in front or behind the sensitive layer of retina, in one or both the meridians.

The ametropia includes: Myopia, Hypermetropia, and Astigmatism.



Refraction in a hypermetropic eye.

Hypermetropia (hyperopia) or long-sightedness is the refractive state of the eye wherein parallel rays of light coming from infinity are focused behind the retina with accommodation being at rest

Etiology: Hypermetropia may be Axial, Curvatural, index, Positional or due to absence of crystalline lens.

1) Axial hypermetropia (MC): axial shortening of eyeball. 1 mm ↓in AP diameter of the eye = 3D of hypermetropia.
2) Curvatural hypermetropia: ↓ curvature of cornea, lens or both.
3) Index hypermetropia occurs due to ↓ in the refractive index of the lens in old age
4) Positional hypermetropia results from posteriorly placed crystalline lens.
5) Absence of crystalline lens either congenital or acquired (following surgical removal) leads to aphakia- a condition of high hypermetropia.
6) Consecutive hypermetropia may result due to:
   a. Overcorrected myopia following refractive surgery [e.g., LASIK & implantable contact lens].
   b. Underpowered intraocular lens (IOL) implantation during cataract surgery and refractive lens exchange (RLE).

Clinical types – There are 3 clinical types of hypermetropia:

1) **Simple or Developmental or Physiological hypermetropia (MC form):** It can be axial or curvatural
2) **Non-physiological hypermetropia**
   - Congenital causes - Microphthalmos, Microcornea Congenital posterior subluxation of lens etc.
   - Acquired causes – Aphakia, Posterior subluxation of lens, Senile hypermetropia, Consecutive hypermetropia due to surgically overcorrected myopia etc.
3) **Functional hypermetropia:** Occurs due to paralysis of accommodation – 3rd nerve paralysis and internal ophthalmoplegia

Clinical features

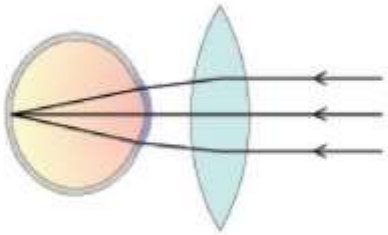
Symptoms	Signs
1) Asymptomatic – in young patients → corrected by mild accommodative effort 2) Asthenopic symptoms develop due to sustained accommodative efforts → tiredness of eyes, frontal or frontotemporal headache, watering and mild photophobia. 3) Defective vision + asthenopic symptoms. 4) Defective vision only – if amount of hypermetropia is very high, the patients usually do not accommodate (especially adults)	1. Small Size of eyeball. 2. Cornea may be slightly smaller than the normal. 3. Anterior chamber is comparatively shallow. 4. Retinoscopy and autorefractometry reveals hypermetropic refractive error. 5. Fundus examination reveals a small optic disc. The retina as a whole may shine due to greater brilliance of light reflections (shot silk appearance). 6. A-scan ultrasonography (biometry) reveal short AP length of the eyeball in axial hypermetropia

Complications

1) Repeated rubbing of the eyes (to get relief from fatigue and tiredness) → Recurrent styes, blepharitis or chalazia.
2) Accommodative convergent squint may develop in children due to excess use of accommodation.
3) Amblyopia may develop.
4) Predisposition to develop primary narrow angle glaucoma.

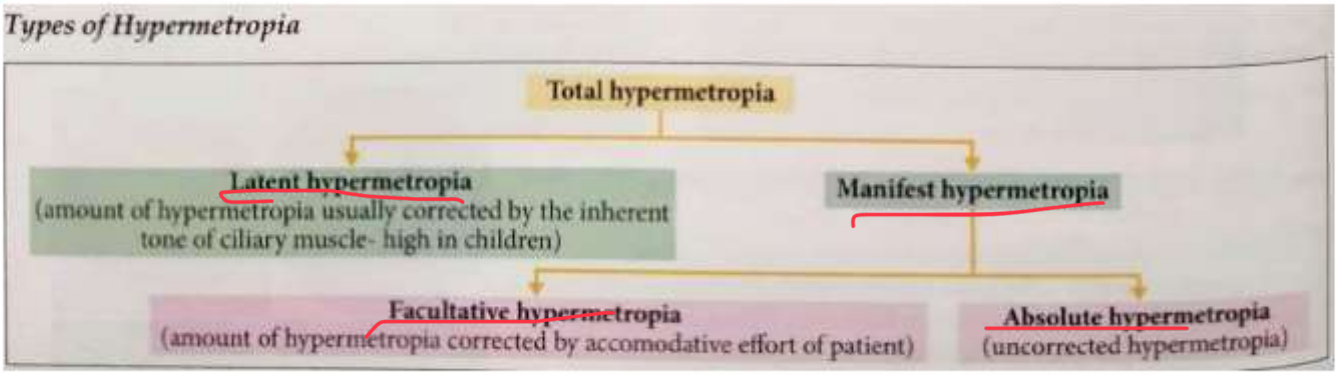
Treatment

Optical treatment – prescribe convex (plus) lenses (either Spectacles or Contact lenses)



Refraction in a hypermetropic eye corrected with convex lens

Types of Hypermetropia



Surgical treatment of Hypermetropia

Cornea based procedures	Lens based procedures
1) Thermal laser keratoplasty (TLK) used for low degree of hyperopia. 2) Conductive keratoplasty (CK) can correct hyperopia of up to 3D. 3) Hyperopic PRK using excimer laser 4) Hyperopic LASIK can correct hypermetropia up to +4D	a) **Phakic refractive lens (PRL) or implantable contact lens (ICL)** is being considered a surgical option for hyperopia of > + 4D. b) **Refractive lens exchange (RLE)** is a good option for high hyperopia especially in presbyopic age

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SQs

1) Astigmatism [16, 14, 12]

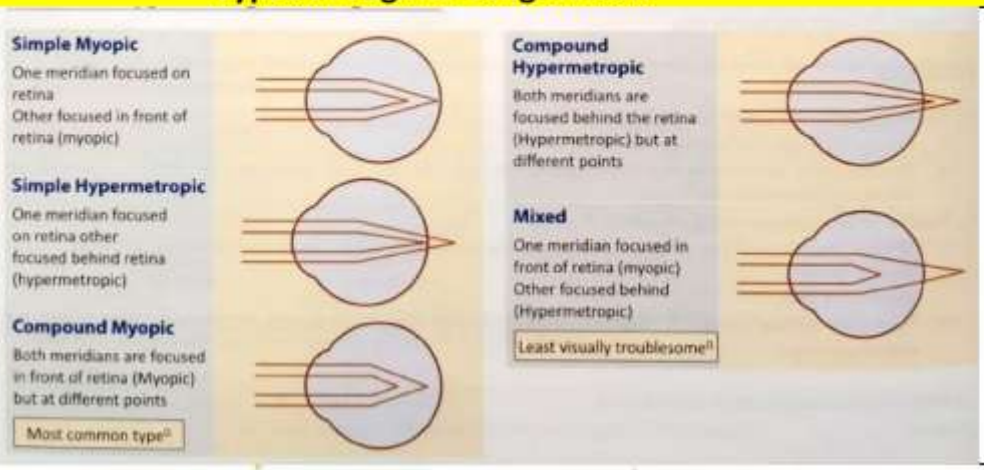
Ans.

Astigmatism is a type of refractive error wherein the refraction varies in different meridians of the eye due to which light rays fail to converge to a point focus

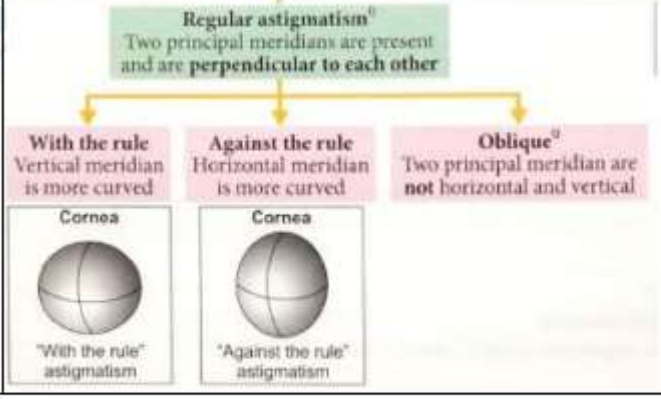
Broadly, there are 2 types of astigmatism: regular and irregular.

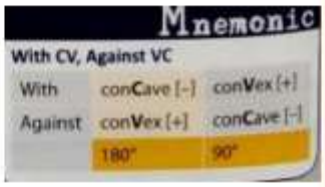
Types of regular astigmatism

Based on position of the two focal lines in relation to retina



Based on the axis and the angle between the two principal meridians



	REGULAR ASTIGMATISM	IRREGULAR ASTIGMATISM
	The astigmatism is regular when the refractive power changes uniformly from one meridian to another (i.e., there are 2 principal meridians).	It is characterized by an irregular change of refractive power in different meridians
Etiology	1. Corneal astigmatism – occur due to abnormal curvature of cornea (MCC). 2. Lenticular astigmatism – occur due to abnormalities of the lens 3. Retinal astigmatism occurs due to oblique placement of macula	2 Types **1)** Curvatural irregular astigmatism – seen in corneal scars or keratoconus. **2)** Index irregular astigmatism – occur due to variable refractive index in different parts of the lens (cataract)
Symptoms	**1)** Asthenopia (tiredness of eyes relieved by closing the eyes) **2)** Blurring of vision – on reading, letters are seen to be "running together". **3)** Elongation of objects may be noticed in high astigmatism.	⇒ Defective vision, ⇒ Distortion of objects, and ⇒ Polyopia (seeing multiple images).
Signs	1. Half closure of the lid (Like myopes) 2. Head tilt – to bring their axes nearer to the horizontal or vertical meridians. 3. Oval optic disc may be seen on ophthalmoscopy. 4. Different power in 2 meridians is revealed on retinoscopy or autorefractometry.	» Retinoscopy reveals irregular pupillary reflex. » Slit-lamp examination reveal corneal irregularity or Keratoconus. » Placido's disc test reveals distorted circles. » Photokeratoscopy and computerized corneal topography give photographic record of irregular corneal curvature
Investigations	1) Retinoscopy. 2) Keratometry reveal corneal astigmatism. 3) Astigmatic fan test 4) Jackson's cross cylinder test useful in confirming the power & axis of cylindrical lenses.	
Optical treatment	1. **With the rule Astigmatism** ➔ **Concave Cylinder at 180 °** or Convex cylinder at 90° 2. **Against the rule Astigmatism** ➔ Convex Cylinder at 180 ° or Concave cylinder at 90° 	Toric Contact lens
Surgical Techniques	1) Astigmatic keratotomy 2) Astigmatic LASIK 3) Limbal Relaxing Incision 4) Ruiz Procedure – for post-keratoplasty astigmatism 5) Phototherapeutic keratectomy (PTK) performed with excimer laser	

Optics of regular astigmatism can be understood from the configuration of a **Sturm's conoid**

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2) Presbyopia [15, 11, 04]

Ans.

Presbyopia (eye sight of old age) is not an error of refraction but a condition of physiological insufficiency of accommodation leading to a progressive fall in near vision.

Pathophysiology: After the age of 40 years, near point of accommodation recedes beyond the normal reading or working range. This condition of failing near vision due to age-related decrease in the amplitude of accommodation is called presbyopia.

Etiology:

1. Hardening of lens with age
2. Weakness of ciliary muscles & suspensory ligaments with age
3. Excessive Close work
4. Causes of premature presbyopia are:
   - Uncorrected hypermetropia.
   - Chronic simple glaucoma.

Symptoms

1) Difficulty in near vision – ex: reading small prints, in threading a needle, etc.
2) Vision improves if held further away
3) Asthenopic symptoms due to fatigue of the ciliary muscle
4) Intermittent diplopia may be experienced by few patients.

Treatment

- **Optical treatment** - prescribe appropriate convex glasses for near work.

Rough guide for providing presbyopic glasses in an emmetrope can be made from the age of the patient.

- 45 years: + 1 to + 1.25D
- 50 years: + 1.5 to 1. 750
- 55 years: +2 to+ 2.25D
- 60 years: +2.5 to + 30

Presbyopic spectacles may be unifocal, bifocal or varifocal.

- **Surgical treatment:**

Refractive surgery for presbyopia, still under trial, includes:

Cornea based procedures	Lens based procedures	Sclera based procedures
1. Monovision conductive keratoplasty (CK) 2. Monovision LASIK. 3. Presbyopic bifocal LASIK 4. Presbyopic multifocal (PML) LASIK	⇒ Multifocal or accommodating IOL implantation after cataract surgery ⇒ Monovision with intraocular lenses.	1) Anterior ciliary sclerotomy 2) Scleral ablation with erbium: YAG laser

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3) Contact lens [11, 08, 03]

Ans.

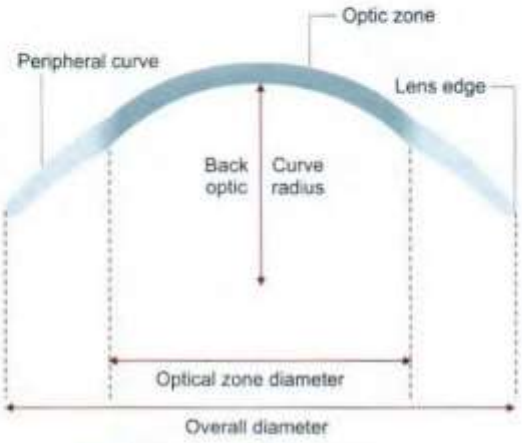
Contact lens is an artificial device whose front surface substitutes the anterior surface of the cornea.

Nomenclature for contact lens :

1. **Diameters of the contact lens**:
   a. *Overall diameter (OD)* of the lens is the linear measurement of the greatest distance across the physical boundaries of the lens.

b. *Optic zone diameter (OZ)* is the central optic zone of lens which is meant to focus rays on the retina.

2. **Curves of the lens** are as follows:
   a) ***Base curve (BC)*** is a curve on the back surface of the lens to fit the front surface of cornea.
   b) ***Peripheral curves*** – These are concentric to base curve and are meant to serve as reservoir of tears
   c) ***Front curve (FC)*** is the curve on the anterior surface – It determines the power of contact lens.
3. **Edge of the lens** – It is the polished and blended union of the peripheral posterior and anterior curves of the lens.
4. **Thickness of the lens** – measured in the centre of the lens
5. **Tint** – It is the colour of the lens.



Types of contact lenses

Depending upon the nature of the material used in their manufacturing, the contact lenses can be divided into following 3 types:

	Hard lenses	Rigid gas permeable lenses {semisoft lenses}	Soft lenses
Made up of	PMMA (polymethylmethacrylate)	Materials which are permeable to oxygen – ***Ex***: Silicone acrylate, Cellulose acetate butyrate	HEMA (hydroxyethylmethacrylate).
Advantages	light in weight, nontoxic, durable and cheap	oxygen permeable	Being soft and oxygen permeable, they are most comfortable and so well tolerated
Disadvantages	1. PMMA is impermeable to O_2 thus restricting the tolerance. 2. Being hard, it can cause corneal abrasions.	these are also hard & hence not popular	Wettability, getting cracked, limited Life, inferior optical quality, more chances of corneal infections

Indications of contact lens use

1. **Optical indications** - for patients with refractive error like anisometropia, unilateral aphakia, high myopia, keratoconus and irregular astigmatism.
2. **Therapeutic indications**:
   a) Corneal diseases, e.g., non-healing corneal ulcers, keratitis & recurrent corneal erosions.
   b) Diseases of iris – aniridia, coloboma and albinism to avoid glare.
   c) In glaucoma as vehicle for drug delivery.
   d) In amblyopia, opaque contact lenses are used for occlusion.
3. **Preventive indications**:
   ⇒ Prevention of symblepharon and restoration of fornices in chemical bums.
   ⇒ Exposure keratitis.
   ⇒ Trichiasis.

4. **Diagnostic indications**: They are used during gonioscopy, electroretinography, Fundus photography, Goldmann's 3 mirror examination etc.
5. **Operative indications**: They are used during goniotomy operation for congenital glaucoma; vitrectomy etc.
6. **Cosmetic indications**:
   (i) Unsightly corneal scars (colour contact lenses);
   (ii) Ptosis (haptic contact lens); and
   (iii) Cosmetic scleral lenses in phthisis bulbi.
7. **Occupational indications** include use by sportsmen; pilots; and actors.

Contraindications for contact lens

1. Mental incompetence, and poor motivation;
2. Chronic dacryocystitis; Chronic blepharitis; Chronic conjunctivitis;
3. Dry eye syndromes;
4. Corneal dystrophies and degenerations; and
5. Recurrent diseases like episcleritis, scleritis and iridocyclitis.

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4) Advantage of Intraocular lenses in treatment of Aphakia [09]
   a. Signs of Aphakia [07]

Ans. **APHAKIA**

Absence of crystalline lens → Converging power of eye decreases → Light rays come to focus behind the retina → high degree of hypermetropia & Accommodation is lost fully

Causes

1) Congenital absence of lens.
2) Surgical aphakia – occur after removal of lens (MC).
3) Aphakia due to absorption of lens matter after trauma in children.
4) Traumatic extrusion of lens from the eye.
5) Posterior dislocation of lens in vitreous.

Clinical features	
Symptoms	**Signs of aphakia**
▪ Defective vision for both far and near vision. ▪ Erythropsia and cyanopsia, i.e., seeing red and blue images {due to excess entry of UV & IR rays in absence of crystalline lens}	⊙ Limbal / corneal scar may be seen in surgical aphakia. ⊙ Anterior chamber is **deeper** than normal. ⊙ Iridodonesis, i.e., tremulousness of iris can be demonstrated. ⊙ Pupil is **jet black** in colour. ⊙ Purkinje's image test shows only **two** images ⊙ Fundus examination shows hypermetropic small disc. ⊙ *Retinoscopy & autorefractometry* reveal high hypermetropia.

Treatment

1) **Spectacles**:
   ✸ +10D with cylindrical lenses for surgically induced astigmatism are commonly used.
   ✸ **Disadvantages of spectacles**
      a. Problem of spherical and chromatic aberrations of thick lenses.
      b. Field of vision is limited.

c. Prismatic effect of thick glasses.
d. Cosmetically, the eyes look enlarged (Frog Eyes) behind the thick spectacles

2) **Contact lenses.**
   - ⊙ **Advantages of contact lenses over spectacles include**: No aberrations and prismatic effect of thick glasses, Wider and better field of vision & cosmetically more acceptable:
   - ⊙ **Disadvantages**: expensive, cumbersome to wear & corneal complications may be associated.

3) **Intraocular lens implantation** is the best available method of correcting aphakia.
4) **Refractive corneal surgery**: Hyperopic LASIK may be tried in cases where IOL cannot be implanted

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5) Pseudophakia [2000]

Ans. The condition of aphakia when corrected with an IOL is referred to as Pseudophakia or artephakia.

Refractive status of a Pseudophakic eye: It depends upon the power of the IOL implanted –

1) Emmetropia – If power of the IOL implanted is exact. It is an ideal situation.
2) Consecutive myopia – if IOL implanted overcorrects the refraction of eye.
3) Consecutive hypermetropia – if under power IOL is implanted.
4) Varying degree of surgically induced astigmatism is also present in pseudophakia.

Signs of pseudophakia (with posterior chamber IOL).

- Surgical scar may be seen near the limbus.
- Anterior chamber is slightly deeper than normal.
- Mild iridodonesis (tremulousness) of iris may be demonstrated.
- Purkinje image test shows **4** images.
- Shimmering light reflex is present.
- Presence of IOL is confirmed on slit-lamp examination after dilating the pupil.
- Visual status and refraction will vary depending upon the power of IOL implanted

Management of pseudophakia:

1. Spectacles
   ⇒ for near vision alone (in pseudophakia with emmetropia) or
   ⇒ Bifocal glasses for both distance and near vision (in pseudophakia with consecutive refractive error).
2. LASIK or Advanced surface ablation (ASA) may be required in moderate consecutive refractive error.
3. Intraocular lens (IOL) exchange or pigiback IOL is required in large consecutive refractive error.

VSQs

1) Uses of convex lenses in Ophthalmology [15]

Ans.

(i) for correction of hypermetropia, aphakia and presbyopia;

(ii) As a magnifying lens – in oblique illumination examination, in indirect ophthalmoscopy & in many other equipments.

CONTENTS

Diseases of Conjunctiva

LQs

1. Ophthalmia neonatorum – aetiology, symptoms, signs & treatment [09, 85]
   a. Purulent conjunctivitis [99]

Ans.

Ophthalmia neonatorum, or neonatal conjunctivitis is a bilateral inflammation of the conjunctiva occurring in a neonate.

Etiology – Infection may occur in 3 ways: **before birth**, **during birth** or **after birth**

1) **Before birth** → through infected liquor amnii in mothers with ruptured membranes.
2) **During birth**: (MC mode) in vaginally delivered infants.
3) **After birth** → Ex: during 1st bath of newborn

Clinical features

1) Pain **&** tenderness in the eyeball → Infant is irritable
2) Hyperaemia **&** chemosis in conjunctiva
3) Periocular vesicles & Corneal involvement (superficial punctate keratitis) – occur in HSV infection.

Causative agent	Incubation period	Conjunctival Discharge	Smear & culture
▪ **Chemical {silver nitrate}**	6 hours	Watery	Negative Culture
▪ **Gonococcal**	2-5 days	Copious purulent discharge	Intracellular Gram-ve diplococci culture positive on blood agar
▪ **Non-Gonococcal bacteria** {Staph, Strep & Haemophilus species}	5-8 days	Mucopurulent	Gram +ve or Gram –ve organisms Positive culture
▪ **Neonatal inclusion conjunctivitis** {serotypes D to K of Chlamydia trachomatis}	5-14 days	Mucopurulent	Positive culture
▪ **Herpes simplex**	6-15 days	Watery	Multinucleated giant cells, cytoplasmic inclusion bodies and **negative** culture

Complications:

- Corneal ulcer, which may perforate → corneal opacification or staphyloma formation.
- Leukoma; Phthisis bulbi

Differential Diagnosis: Congenital dacryocystitis, Congenital glaucoma

Treatment – Culture sensitivity swabs should be taken before starting the treatment.

1) For Chemicals (silver nitrate) → **just wash eye**; it is self-limiting & doesn't require any treatment.
2) For Gonococcus:
   - Topical therapy:
     - Saline lavage hourly till the discharge is eliminated.
     - Bacitracin eye ointment 4 times/ day.
   - Systemic therapy: one of the following regimes can be used for 7 days.
     - Ceftriaxone 75-100 mg/ kg/ day IV or IM, qid **or**
     - Cefotaxime 100-150 mg/ kg/ day IV or IM, 12 hourly **or**

- Ciprofloxacin 10-20 mg/kg/day **or**
- Norfloxacin 10 mg/ kg/ day

3) **For Other Non-gonococcal bacteria** – prescribe broad-spectrum Abx eye drops & Neomycin-bacitracin eye ointments for 2 weeks.
4) **For Neonatal inclusion conjunctivitis** – prescribe topical tetracycline 1% **or** erythromycin 0.5% eye ointment qid for 3 weeks.
   - But, systemic erythromycin (125 mg orally, qid for 3 weeks) should also be given since the presence of chlamydia agents in the conjunctiva implies colonization of upper respiratory tract as well. Both parents should also be treated with systemic erythromycin
5) **For Herpes simplex conjunctivitis** – prescribe topical antivirals (ex: acyclovir 3 % ointment)

Prophylaxis – Antenatal, natal and postnatal care.

a. **Antenatal care**: Prenatal diagnosis and treatment of birth canal infections.
b. **Natal Care:** Aseptic delivery. Newborn baby's closed lids should be thoroughly cleansed & dried.
c. **Postnatal care**: Clean the eyelids with sterile gauze dipped in Povidon-iodine 2.5% solution **or** 1 % tetracycline ointment **or** 0.5% erythromycin ointment.

⊙ Single injection of ceftriaxone 50 mg/kg IM or IV should be given to infants born to mothers with untreated gonococcal infection.

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2. Trachoma – clinical features and management [09, 05, 04]
   a. Pannus [12, 03, 95]
   b. Complications of Trachoma [11]

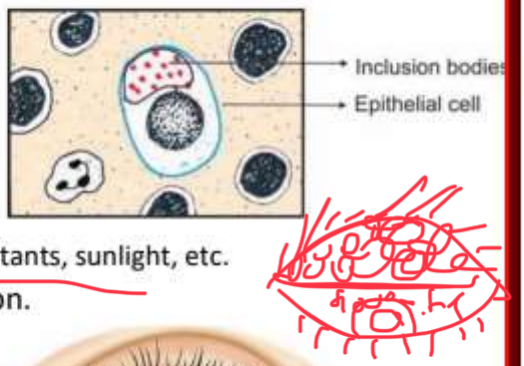
Ans.

Trachoma (aka Egyptian ophthalmia) is a chronic keratoconjunctivitis, affecting the superficial epithelium of conjunctiva and cornea simultaneously. {'Trachoma' in Greek means 'rough'}.

Etiopathogenesis:

Causative organism – Chlamydia trachomatis biovar TRIC. {TRIC = Trachoma and Inclusion Conjunctivitis;}

⊙ The organism is epitheliotropic & produces HP {= Halberstaedter Prowazek} bodies (intracytoplasmic inclusion bodies).
⊙ **A to K** Serotypes (aka **Serovars**) of C. trachomatis are together called **TRIC agents**
⊙ Presently, 12 serovars of Chlamydia trachomatis biovar TRIC have been identified out of which –
- Serovars A, B, B_a and C are a/w hyperendemic (blinding) trachoma.
- Serovars D to K are a/w oculogenital chlamydial disease.

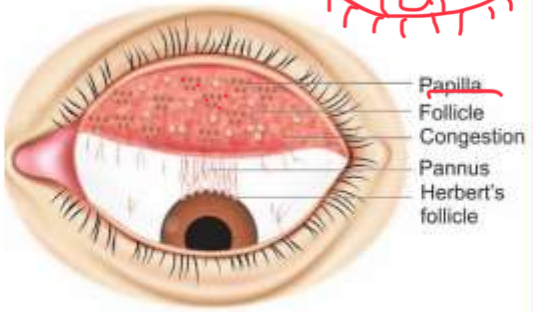


Predisposing factors:
- **Age** – Infancy & early childhood.
- **Sex**: females > males.
- Poor Socioeconomic status; unhygienic and crowded surroundings
- Environmental factors like dry weather, exposure to dust, smoke, irritants, sunlight, etc.

Source of infection: conjunctival discharge of the affected person.

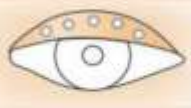
Modes of infection:
1) Direct spread – by airborne or waterborne modes.
2) Vector transmission through flies.
3) Through contaminated fingers of doctors, nurses and contaminated tonometers, common towel, handkerchief, bedding and surma-rods.

Prevalence: Trachoma is responsible for **15- 20%** of the world's blindness, being second only to cataract.



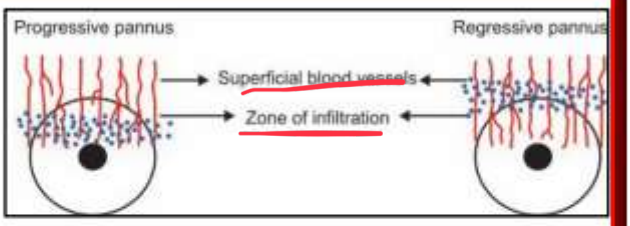
Signs of active trachoma

Clinical features – described in 2 phases:

	Phase of active trachoma	Phase of cicatricial trachoma
Occurs in	Childhood due to active chlamydial infection **Incubation period**: 7 to 14 days	Middle age due to chronic inflammation {Type IV HSN reaction to chlamydial antigens} Here, infection is no longer present, i.e., only trachoma sequelae are present.
Symptoms	➤ In the absence of 2° infection: Mild foreign body sensation, lacrimation, stickiness of the lids & Scanty mucoid discharge. ➤ In the presence of 2° infection: symptoms resemble acute mucopurulent conjunctivitis	
Signs	➤ **Conjunctival signs** 1) **Congestion** of upper tarsal & forniceal conjunctiva. 2) Conjunctival Follicles look like **boiled sago-grains**. Sometimes, follicles may be seen on the bulbar conjunctiva (pathognomonic of trachoma). 3) Papillary hyperplasia – Impart red and velvety appearance to the tarsal conjunctiva. ➤ **Corneal signs** 1) Superficial keratitis in the upper part. 2) **Herbert follicles**– present in the limbal area (similar to conjunctival follicles). 3) Progressive pannus, i.e., infiltration of the cornea is ahead of vascularization. 4) Corneal ulcer may develop	➤ **Conjunctival signs** 1) Conjunctival scarring, which may be irregular, star-shaped or linear. **Linear scar present in the sulcus subtarsalis is called Arlt's line** 2) **Concretions** – whitish deposits formed due to accumulation of dead epithelial cells and mucus in the glands of Henle  3) Others – pseudocyst, **xerosis** & symblepharon ➤ **Lid Signs:** trichiasis, entropion, tylosis, ptosis, madarosis etc. ➤ **Lacrimal Apparatus:** chronic dacryocystitis & dacryoadenitis ➤ **Corneal signs** 1) **Regressive pannus** (pannus siccus) – vessels extend beyond the area of infiltration 2) **Herbert pits** are the pitted scars, left after healing of Herbert follicles 3) **Blinding sequelae:** Corneal opacity, corneal ectasia, corneal xerosis etc.
Treatment	○ **Topical therapy**: - Tetracycline **or** erythromycin 1 % eye ointment BD for 6 weeks **or** - Sulfacetamide (20%) eye drops t.i.d + 1% tetracycline eye oint at bed time for 6 weeks ○ **Systemic antibiotics regimes**: ▪ Azithromycin 20 mg/kg body weight up to maximum 1 g as single oral dose is as effective as 6 weeks of topical therapy and so is the 1st DOC. It is not used in pregnancy and children < 6 years of age. ▪ Tetracycline or erythromycin 250 mg orally, q.i.d. for 3-4 weeks ▪ Doxycycline 100 mg orally BD for 3-4 weeks ○ **Combined topical & systemic therapy**– preferred in severe infections	» Remove **Concretions** with a hypodermic needle » Artificial tears for Conjunctival **xerosis** » Electrolysis, Cryolysis etc. – for Trichiasis » Surgery to correct Cicatricial entropion » Measures to treat Corneal Opacity: • Penetrating keratoplasty (PK) • Keratoprosthesis (KP) – in B/L blind cases • Punctal occlusion & lateral tarsorrhaphy

Grading of trachoma – WHO classification **(FISTO):**

1) **T**F: Trachomatous inflammation-follicular.
2) **T**I: Trachomatous inflammation intense
3) **T**S: Trachomatous scarring in the tarsal conjunctiva.
4) **T**T: Trachomatous trichiasis - eyelash rubs the eyeball.
5) **C**O: Corneal opacity



Complications: Corneal ulcer

Diagnosis

- **Clinical diagnosis** – made from its typical signs. Clinical grading of each case should be done as per WHO classification into TF, TI, TS, TT or CO.
- **Laboratory diagnosis**:
  - Conjunctival cytology: Giemsa-stained smears show PMN reaction with presence of plasma cells and Leber cells.
  - ELISA for chlamydial antigens.
  - Polymerase chain reaction (PCR) is also useful.

Prophylaxis for Trachoma: The WHO's **G**ET 2020 program (**G**lobal **E**limination of **T**rachoma by 2020), has adopted the **SAFE strategy** for prophylaxis against trachoma which includes:

S: Surgery (Tertiary prevention),
A: Antibiotic use (Secondary prevention),
F: Facial hygiene (Primary prevention), &
E: Environmental changes (Primordial prevention).

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3. Describe the aetiology, symptoms, signs and treatment of allergic conjunctivitis [88]
   a. Vernal conjunctivitis / Vernal Catarrh / Spring Catarrh. [13, 11]
   b. Phlyctenular conjunctivitis [03, 95, 91]
   c. Phlycten [08, 05]
   d. Fascicular ulcer [07]

Ans.

Allergic conjunctivitis: It is the inflammation of conjunctiva due to allergic reactions which may be immediate (humoral) or delayed (cellular). The conjunctiva is 10 times more sensitive than the skin to allergens.

Types of Allergic Conjunctivitis:

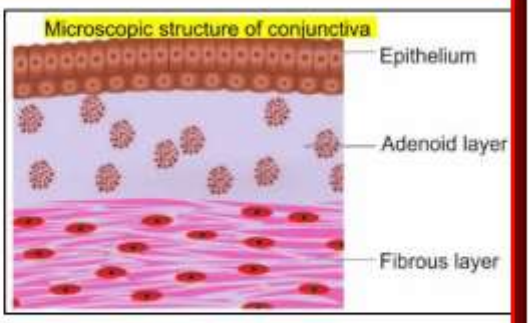
1) Simple allergic conjunctivitis – It can be seasonal or perennial
2) Vernal keratoconjunctivitis (VKC) – it is seasonal
3) Atopic keratoconjunctivitis (AKC) – adult form of VKC
4) Phlyctenular keratoconjunctivitis (PKC)
5) Giant papillary conjunctivitis
6) Contact Dermoconjunctivitis (Drop Conjunctivitis)

VERNAL KERATOCONJUNCTIVITIS OR SPRING CATARRH: VKC is a bilateral, interstitial, self-limiting, allergic inflammation of the conjunctiva having a periodic seasonal incidence.

Etiopathogenesis

- Predisposing factors:
  1) Sex: boys > girls.
  2) Season: More common in summer; hence the name spring catarrh.
  3) Climate: More prevalent in tropics, less in temperate zones
  4) Other atopic manifestations, such as eczema or asthma, are associated in 40- 75% cases.
  5) Family history of atopy is found in 40-60% of patients

- Pathological Changes:
  1) Hyperplasia of Conjunctival epithelium → downward projections into the sub-epithelial tissue.
  2) Adenoid layer shows **infiltration** by mast cells, eosinophils, plasma cells, lymphocytes & histiocytes.
  3) Fibrous layer shows **proliferation** which later on undergoes hyaline changes.
  4) Conjunctival vessels also show proliferation, ↑ permeability and vasodilation.



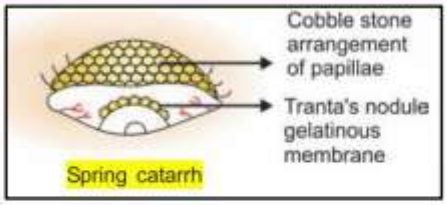
- All these pathological changes lead to formation of multiple papillae in the upper tarsal conjunctiva

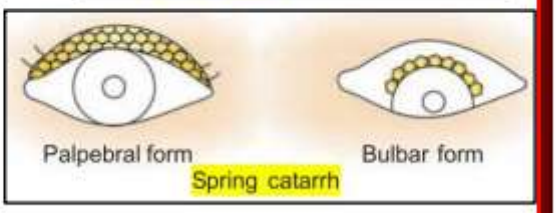
Clinical features

Symptoms	Signs
» Burning and itching sensation which is ↑ when patient comes in a warm humid atmosphere. Itching is more marked with palpebral form of disease. » Other associated symptoms: mild photophobia, lacrimation, stringy *(ropy)* discharge & heaviness of lids.	1) **Palpebral form**: Usually upper tarsal conjunctiva of both eyes is involved. ⇒ Hard, flat topped, papillae are arranged in a 'cobble-stone' or 'pavement stone fashion along with conjunctival hyperemia. ⇒ **In severe cases**, papillae may hypertrophy to produce cauliflower-like 'giant papillae'. 2) **Bulbar limbal form**. It is characterised by: ➔ Dusky red triangular congestion of bulbar conjunctiva in palpebral area, ➔ Limbal papillae occur as gelatinous, thickened confluent accumulation of tissue around limbus ➔ Presence of whitish raised dots along the limbus **(Horner-Tranta's spots)** 3) **Mixed form**: It shows combined features of both palpebral & bulbar forms.

Corneal involvement in VKC includes the following types of lesions:

1. Punctate epithelial keratitis
2. Epithelial erosions – occurs due to coalescence of punctate epithelial lesions.
3. Vernal corneal plaques – occurs due to coating of bare areas of epithelial erosions with a layer of altered exudates
4. Ulcerative vernal keratitis **(shield ulcers)**
5. Subepithelial scarring (ring scar).
6. **Pseudogerontoxon** can develop in recurrent limbal disease and is characterised by a classical 'cupid's bow' outline.





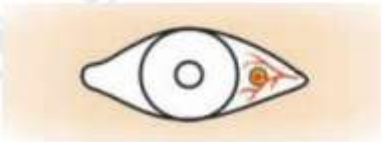
- **Differential diagnosis**: Palpebral form of VKC needs to be differentiated from trachoma
- **Treatment of VKC**
  - » Topical anti-inflammatory therapy with **steroids** (fluorometholone), **mast cell stabilizers** (sodium cromoglycate (2%) drops), **antihistamines** (azelastine, ketotifen), and **NSAIDs** forms the mainstay of treatment of VKC. Tacrolimus (0.03% ointment) is an immune-modulator, which can be useful in refractory cases.
  - » Topical lubricants & mucolytics
    1. Artificial tears, such as carboxymethyl cellulose, provide soothing effect.
    2. Acetyl cysteine (0.5%) – mucolytic; useful in the treatment of early plaque formation
  - » Systemic therapy - Oral antihistaminics & steroids in severe cases.
  - » Treatment of large papillae: Supratarsal injection of long-acting steroid **or** Cryo application, **or** Surgical excision.

» Supportive measures:
  - Dark goggles to prevent photophobia.
  - Cold compresses and ice packs have soothing effects.
  - Maintenance of air-conditioned atmosphere.
  - Change of place from hot to cold area
» Desensitization
» Treatment of vernal keratopathy
  - Punctate epithelial keratitis requires steroids
  - A large vernal plaque requires surgical excision by superficial keratectomy.
  - Surgical treatment for Shield ulcers → debridement, superficial keratectomy, excimer laser therapeutic keratectomy; amniotic membrane transplantation to ↑ re-epithelialization.

PHLYCTENULAR KERATOCONJUNCTIVITIS - aka microbial allergic conjunctivitis **-** It is a characteristic nodular **affection** (phlycten) occurring as an allergic response of the conjunctival and corneal epithelium to some endogenous allergens to which they have become sensitized.

Etiology: It is a delayed hypersensitivity (Type IV-cell mediated) response to endogenous microbial proteins.

- **Causative allergens** – Tuberculous proteins, Staphylococcus proteins, proteins of Moraxella bacillus and certain parasites (worm infestation).
- **Predisposing factors**
  - Sex: Girls > boys.
  - Malnutrition, Overcrowding, unhygienic practices etc.


Phlyctenular conjunctivitis

Pathology

1) **Stage of nodule formation**: exudation & infiltration of WBCs into deep layers of conjunctiva → nodule formation.
2) **Stage of ulceration**: Later on, necrosis occurs at the apex of the nodule and an ulcer is formed.
3) **Stage of granulation**: Eventually, floor of the ulcer covered by granulation tissue.
4) **Stage of healing**.

Clinical features Disease is usually U/L (in contrast to vernal keratoconjunctivitis which is B/L).

- **Symptoms**: mild discomfort in the eye, irritation and reflex watering. However, usually there is associated mucopurulent conjunctivitis due to secondary bacterial infection.
- **Signs**: The phlyctenular conjunctivitis can present in 3 forms: **simple, necrotizing & miliary**.

Corneal involvement in PKC may occur secondarily from extension of conjunctival phlycten; or rarely as a primary disease. 2 forms are seen -

1) **Ulcerative phlyctenular keratitis** may occur in the following 3 forms:
   a. Scrofulous ulcer is a shallow ulcer formed due to breakdown of small limbal phlycten. Such an ulcer usually clears up without leaving any opacity.
   b. Fascicular ulcer has a prominent parallel leash of blood vessels. This ulcer remains superficial but leaves behind a band-shaped superficial opacity after healing.
   c. Miliary ulcer: multiple small ulcers are scattered over a portion of or whole of the cornea.

2) **Diffuse infiltrative phlyctenular keratitis** – this appears in the form of central infiltration of cornea with characteristic rich vascularization from the periphery, all around the limbus.

Differential diagnosis: episcleritis, scleritis, and conjunctival foreign body granuloma.

Management

1. Local therapy
   - Topical steroids (dexamethasone or betamethasone)
   - Antibiotic drops and ointment should be added to cover secondary infection
   - Atropine (1%) eye ointment should be applied once daily when cornea is involved.
2. Specific therapy
   a. Tuberculous infection should be excluded by X-rays chest, Mantoux test, TLC, DLC and ESR. In case, a tubercular focus is discovered, antitubercular treatment should be started
   b. Septic focus, in the form of tonsillitis, adenoiditis, or caries teeth, when present should be adequately treated by systemic antibiotics and necessary surgical measures.
   c. Parasitic infestation should be ruled out by repeated stool examination and when discovered should be adequately treated for complete eradication.
3. General measures – provide high protein diet supplemented with vitamins A, C and D.

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4. Describe the aetiology, symptoms, signs and treatment of membranous conjunctivitis [87]

Ans.

	TRUE MEMBRANE	PSEUDOMEMBRANE
1. Structure	Fibrinous exudate is situated over and within the conjunctival epithelium	Fibrinous exudate is situated over the surface of conjunctival epithelium.
2. On peeling	It cannot be peeled off easily.	It is separated easily.
3. Bleeding	Bleeding occurs when the membrane is removed.	There is no bleeding.

» Streptococcus pyogenes (haemolyticus) causes pseudomembranous conjunctivitis.

» Corynebacterium diphtheriae causes acute membranous conjunctivitis.

Such infections are not known nowadays.

SQs

1) Pterygium [14, 04, 99]

Ans. Pterygium is a wing-shaped fold of conjunctiva encroaching upon the cornea from either side within the interpalpebral fissure.

Etiology: it is a response to prolonged effect of environmental factors like sunlight (UV rays), dry heat, high wind and dust

Pathology: Pterygium is a degenerative and hyperplastic condition of **conjunctiva** which proliferates as vascularised granulation tissue under the corneal epithelium & encroaches the cornea by destroying the corneal epithelium, Bowman's layer & superficial stroma are destroyed.

Clinical features

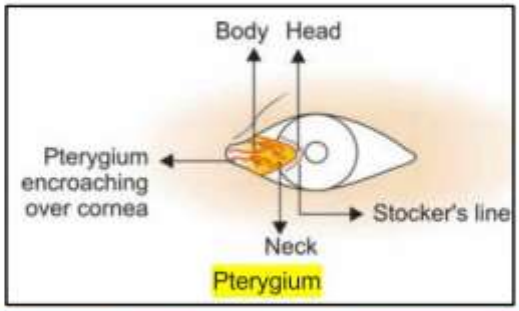
Symptoms

- **Age**: Usually seen in old age.
- **Sex**: More common in males doing outdoor work than females
- Cosmetic intolerance; Foreign body sensation and irritation.
- Defective vision occurs when it encroaches the pupillary area
- Diplopia may occur due to limitation of ocular movements.

Signs

➤ Triangular fold of conjunctiva encroaching on the cornea in the area of palpebral aperture is typical presentation of pterygium.

- Stocker line (deposition of iron) may be seen in corneal epithelium anterior to the advancing head of pterygium.
- **Parts of a fully-developed pterygium are as follows**:
  - **Head**: Apical part present on the cornea,
  - **Neck**: Constricted part present in the limbal area
  - **Body**: Scleral part – extend between limbus & canthus.
  - **Cap**: Semilunar whitish infiltrate present just in front of the head.



Types

1. Type 1 pterygium extends < 2 mm onto the cornea.
2. Type 2 pterygium involves upto 4 mm of the cornea.
3. Type 3 pterygium encroaches onto > 4 mm of the cornea and involves the visual axis.

Complications: Cystic degeneration & infection; neoplastic change to epithelioma, fibrosarcoma or malignant melanoma, may occur rarely.

	Pterygium	Pseudopterygium
1. Etiology	Degenerative process	Inflammatory process
2. Age	Usually occurs in elder persons	Can occur at any age
3. Site	Always situated in the palpebral aperture	Can occur at any site
4. Stages	Either progressive, regressive or stationary	Always stationary
5. Probe test	Probe cannot be passed underneath	A probe can be passed under the neck

Differential diagnosis

- Pterygium must be differentiated from pseudopterygium.
- Pseudopterygium is a fold of bulbar conjunctiva attached to the cornea. It is formed as a response to an acute inflammatory episode such as chemical burn, marginal corneal ulcer, corneal trauma and cicatrizing conjunctivitis.

Treatment

- Medical treatment of not much use.
  - Tear substitutes for dry eye symptom.
  - Topical steroids for associated inflammation.
  - Sunglasses to protect from UV rays (↓ the growth stimulus).
- **Surgical excision is the only satisfactory treatment which may be indicated for**: Cosmetic disfigurement, Visual impairment, Continued progression threatening to encroach onto the pupillary area & Diplopia.
- Recurrence of the pterygium after surgical excision, can be ↓ by any of the following measures:
  - Surgical excision with free conjunctival limbal autograft (CLAU)
  - Surgical excision with amniotic membrane graft and mitomycin-C
  - Surgical excision with lamellar keratectomy and lamellar keratoplasty

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2) Sub-conjunctival Haemorrhage [14, 06, 04]

Ans.

Ecchymosis or subconjunctival haemorrhage may vary in extent from small petechial haemorrhage to an extensive one spreading under the whole of the bulbar conjunctiva and thus making the white sclera of the eye invisible.

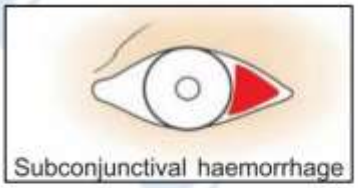
Etiology:

1. Trauma (MCC) → **local trauma** to the conjunctiva (Ex: due to surgery & subconjunctival inj.) or **retrobulbar haemorrhage** (Ex: due to trauma of orbit) which spreads below the bulbar conjunctiva.
2. Inflammations of the conjunctiva: Petechial subconjunctival haemorrhages are usually associated with acute haemorrhagic conjunctivitis caused by picornaviruses, pneumococcus, leptospirosis etc.

3. Venous congestion of head (Ex: in whooping cough, epileptic fits, strangulation or compression of jugular veins) → sudden rise in pressure → rupture of conjunctival capillaries
4. Vascular diseases such as arteriosclerosis, HTN & DM → Spontaneous rupture of fragile capillaries
5. Blood dyscrasias like anaemias, leukaemias and dysproteinaemias.
6. Bleeding disorders like purpura, haemophilia and scurvy.
7. **Acute febrile systemic infections** such as malaria, typhoid, diphtheria, meningococcal septicaemia, measles and scarlet fever.
8. **Vicarious bleeding** associated with menstruation is an extremely rare cause of subconjunctival haemorrhage.

Clinical features.

- **Symptom**: Red eye is the most predominant feature
- **Sign**: Fresh bright red blood is visible under the conjunctiva
- Most of the time it is absorbed completely within 7 to 21 days. During absorption colour changes are noted from bright red to orange and then yellow.
- In severe cases, some pigmentation may be left behind after absorption.
- There may be symptoms of associated causative disease


Subconjunctival haemorrhage

Treatment.

- No Rx needed; resolves spontaneously in 2 weeks
- Treat the cause when discovered.
- Placebo therapy with astringent eye drops.
- Psychotherapy and assurance to the patient
- Cold compresses to check the bleeding in the initial stage and hot compresses may help in absorption of blood in late stages.

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3) Angular Conjunctivitis [05]

Ans.

It is a type of **chronic conjunctivitis** characterised by mild grade inflammation confined to the conjunctiva and lid margins near the angles a/w maceration of the surrounding skin.

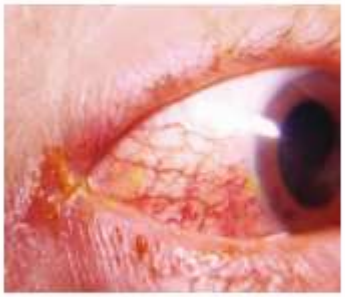
Etiology

- Causative organisms: Moraxella Axenfield (MA, aka diplobacilli) is MCC. Rarely, staphylococci may also cause angular conjunctivitis.
- Source of infection: usually nasal cavity.
- Mode of infection: Infection is transmitted from nasal cavity to the eyes by contaminated fingers or handkerchief.
- Predisposing factors are same as for 'simple chronic conjunctivitis'

Pathology: MA bacillus produces a proteolytic enzyme which collects at the angles by the action of tears and thus macerates the epithelium of the conjunctiva, lid margin and the skin. The maceration is followed by mild grade chronic inflammation. Skin may show eczematous changes.

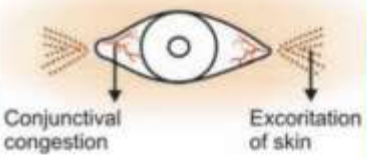
Clinical features

Symptoms	Signs
• Irritation, burning sensation and discomfort in the eyes. • H/o dirty-white foamy discharge at the angles. • Redness in the angles of eyes.	⊙ Hyperaemia of bulbar conjunctiva near the canthi. ⊙ Hyperaemia of lid margins near the angles. ⊙ Excoriation of the skin around the angles. ⊙ Foamy mucopurulent discharge at the angles.



Signs of angular conjunctivitis

Complications: blepharitis & shallow marginal catarrhal corneal ulceration.



Angular conjunctivitis

Treatment

- **Prophylaxis** – treatment of associated nasal infection & good personal hygiene.
- **Curative treatment** consists of:
  1. Oxytetracycline (1 %) eye ointment, 2- 3 times a daily for 9-14 days.
  2. **Zinc lotion instilled in day time** and **zinc oxide ointment at bed time** inhibits the proteolytic ferment and thus helps in reducing the maceration.

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4) Follicular Conjunctivitis [13, 03]

Ans.

It is the inflammation of conjunctiva, characterised by formation of follicles, conjunctival hyperaemia and discharge from the eyes. Follicles are formed due to localised aggregation of lymphocytes in the adenoid layer of conjunctiva.

Follicular conjunctivitis

Etiology

- Exposure to certain chemicals and toxins, e.g., pilocarpine, atropine etc.
- Viruses, e.g., herpes and adenovirus
- Any conjunctivitis of long duration may cause this condition.

Symptoms: Foreign body sensation, slight irritation, redness, watering. mild mucoid discharge, mild photophobia

Signs: Multiple follicles are present in the lower fornix. There is no scarring which differentiates it from trachoma.

Types

1) **Inclusion conjunctivitis**—It is caused by serotypes D to K of Chlamydia trachomatis → produce inclusion bodies similar to those occurring in trachoma. The primary source of infection is contaminated water of swimming pools (hence the name swimming pool conjunctivitis).
2) **Epidemic keratoconjunctivitis**—It is caused by adenovirus. It is treated by adenine arabinoside (Ara-A).
3) **Pharyngoconjunctival fever**— It is also caused by adenovirus. A/w pharyngitis & fever.
4) **Acute herpetic conjunctivitis**—It is common in young children. A/w Corneal dendritic ulcers
5) **New castle conjunctivitis**—It is caused by new castle virus from infected owls.

Complications: Follicles may persist for several years but always resolve without scarring.

Treatment

- Astringent eyedrops are applied frequently; Choose Antibiotic/Antiviral based on the causative agent
- Supportive Treatment: Improve general health and nutrition of the patient.
- Treat associated adenoids, tonsils and upper respiratory tract infection promptly and adequately.

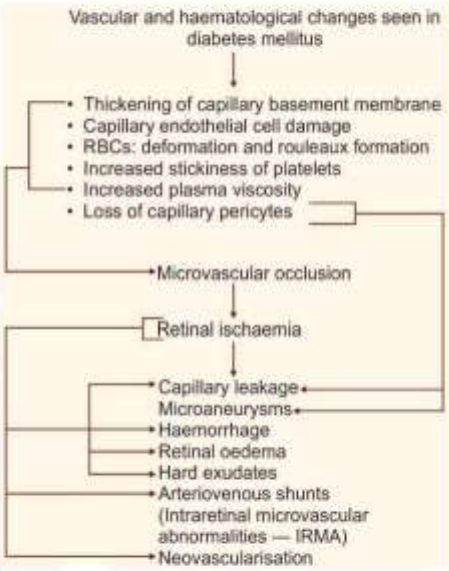
Diseases of Retina

LQs

1) Diabetic retinopathy – classification, ocular fundus signs and treatment [16, 11, 10, 07]
   a. Dot and blot haemorrhages [13]
   b. Retinal hemorrhages [12]

Ans.

Diabetic retinopathy (DR) refers to retinal changes seen in patients with diabetes mellitus.



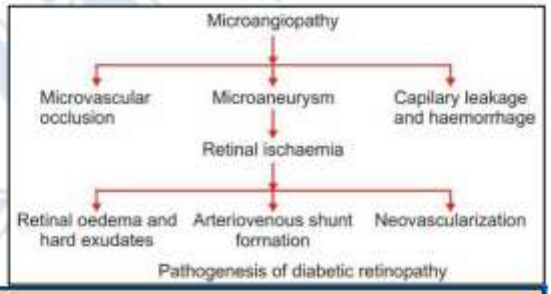
Flowchart depicting pathogenesis of diabetic retinopathy

Risk factors

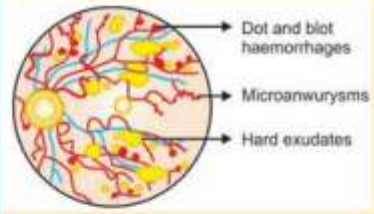
1. Duration of diabetes after the onset of puberty is the most important determining factor
2. Sex: Females > males (4:3).
3. Poor metabolic control
4. Heredity: It is transmitted as autosomal recessive trait.
5. Other risk factors include Pregnancy, HTN, smoking, obesity, anaemia and hyperlipidaemia.

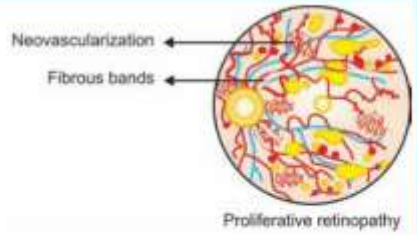
Pathogenesis:

Hyperglycemia, in uncontrolled diabetes mellitus, is the starting point for development of DR.



Pathogenesis of diabetic retinopathy

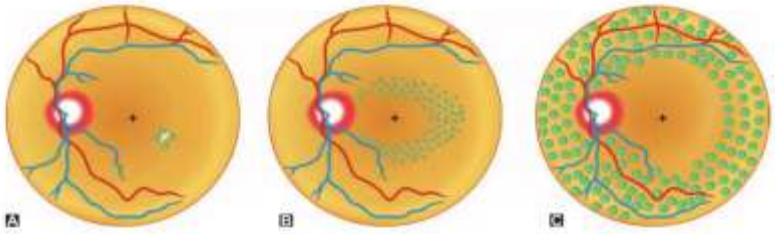
Classification of Diabetic Retinopathy	Ophthalmoscopic features
1. Non-proliferative Diabetic Retinopathy (NPDR) It can be mild, moderate, severe & very severe {**ETDRS** Classification} 	➤ **Microaneurysms** in the macular area (the earliest detectable lesion): These appear as red dots and leak fluid and also fluorescein dye on FFA. They look like cluster of Grapes at end of vascular twigs ➤ **Retinal haemorrhages**: Both deep (**dot & blot haemorrhages – in outer plexiform layer**) and superficial haemorrhages (flame-shaped – in nerve fibre layer), occur from rupture of microaneurysms ➤ Retinal oedema ➤ Hard exudates (lipid deposition) – seen in macular area; ➤ Cotton-wool spots – represent areas of nerve fibre infarcts ➤ Venous abnormalities (beading, looping and dilatation) occur adjacent to area of capillary non-perfusion. ➤ Intraretinal microvascular abnormalities (IRMA) seen as fine irregular red lines connecting arterioles with venules, represent A-V shunts
2. Proliferative diabetic retinopathy (PDR)	**Occurrence of neovascularization** over the changes of very severe NPDR is the hallmark of PDR. It results in the formation of:

	⇒ Fibrovascular epiretinal membrane formed due to condensation of connective tissue around the new vessels ⇒ Vitreous detachment and vitreous haemorrhage 
3. Diabetic maculopathy (Changes in macular region)	Diabetic Macular Oedema (DME) occurs due to ↑ permeability of the retinal capillaries. It can be Clinically significant macular edema (CSME) or clinically non-significant macular edema (non-CSME) **Clinico-angiographic classification of diabetic maculopathy**: 1) Focal exudative maculopathy – FFA reveals focal leakage with adequate macular perfusion 2) Diffuse exudative maculopathy – FFA reveals diffuse leakage 3) Ischaemic maculopathy - occurs due to microvascular blockage. FFA shows areas of non-perfusion in the form of foveal avascular zone (FAZ) 4) Mixed maculopathy: In it combined features of ischaemic & exudative maculopathy are present **OCT (Optical coherence tomography) classification of DME:** 1) Non-tractional DME – Ex: Cystoid macular oedema (CME) 2) Tractional DME – Ex: Vitreo-foveal traction (VFT)
4. Advanced diabetic eye disease (ADED)	It is the end result of uncontrolled PDR & is marked by complications such as: Persistent vitreous haemorrhage, Tractional retinal detachment, and Neovascular glaucoma.

Management of Diabetic Retinopathy:

- **Screening for diabetic retinopathy** – The recommendations for are as follows:
  - First examination, 5 years after diagnosis of type 1 DM **&** at the time of diagnosis in type 2 DM.
  - Every year, till there is no diabetic retinopathy or there is mild NPDR.
  - Every 6 months, in moderate NPDR.
  - Every 3 months, in severe NPDR.
  - Every 2 months, in PDR with no high-risk characteristics.
- **Investigations in a case of DR include**: Urine examination, 24-hour urinary protein, Blood sugar estimation, Renal function tests, Lipid profile, HbA1C, FFA & Optical coherence tomography (OCT)
- **Treatment of diabetic retinopathy**:
  - Metabolic control of diabetes mellitus & associated risk factors:
    - Target blood glucose level: fasting <120 mg%, post-prandial <180 mg%, and HbA_1c <7%.
    - Target lipid profile (fasting): Cholesterol <200 mg%, Triglycerides <150 mg%, HDL >50 mg%, and LDL <150 mg%.
    - Control of associated anaemia: Target hemoglobin >10 mg%.
    - Control of associated hypertension: Target blood pressure levels:130/80 mm Hg.
    - Life style changes - regular exercises, stop smoking and alcohol consumption etc.
  - Intravitreal anti-VEGF {Vascular endothelial growth factor} drugs: e.g., Bevacizumab (1.25 mg) and Ranibizumab (0.5 mg).
    - Effects of the anti-VEGFs last for 4–6 weeks and frequent injections are warranted.
    - There is risk of endophthalmitis
  - Intravitreal steroids – ex: triamcinolone acetonide (IVTA) (20 mg) – restores inner retinal barrier and has some anti-VEGF effects as well. Used along with anti-VEGFs, in recalcitrant cases
    - There is risk of glaucoma, steroid induced cataract, endophthalmitis etc.

- **Laser therapy:**
  - ETDRS had recommended **focal laser for focal DME** and grid laser for diffuse DME.
  - Laser helps by stimulating the RPE pump mechanism and by inhibiting VEGF release.
  - Laser therapy is performed using double frequency YAG laser 532 nm or argon green laser or diode laser.
  - **Ex**: Macular photocoagulation (Focal / Grid) & Panretinal photocoagulation (PRP)
  - PRP causes destruction of hypoxic retina which is responsible for the production of vasoformative factors.
  - Indications for PRP are:
    - PDR with HRCs
    - Neovascularization of iris (NVI)
    - Severe NPDR associated with: Poor compliance for follow-up, Before cataract surgery/YAG capsulotomy, Renal failure, One eyed patient and Pregnancy.



Protocols of laser application in diabetic retinopathy: A, focal treatment; B, grid treatment and; C, panretinal photocoagulation

- **Surgical treatment** {Pars plana vitrectomy (PPV)} is indicated in following cases:
  - ⇒ **Tractional DME with NPDR**: PPV + removal of posterior hyaloid.
  - ⇒ **Advanced PDR with dense vitreous haemorrhage**: PPV + removal of opaque vitreous gel & endophotocoagulation.
  - ⇒ **Advanced PDR with extensive fibrovascular epiretinal membrane**: PPV + removal of fibrovascular epiretinal membrane & endophotocoagulation.
  - ⇒ **Advanced PDR with tractional retinal detachment**: PPV with endophotocoagulation and reattachment of detached retina + scleral buckling and internal tamponade using intravitreal silicone oil or gases like sulphur hexafluoride (SF_6).

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2) Retinoblastoma – aetiology, pathology, C/F & management [09, 05, 90].
   a. Stages of retinoblastoma [15]
   b. Amaurotic cat's eye reflex [96]
   c. Discuss the differential diagnosis of 'Cat's eye' in children [90]

Ans.

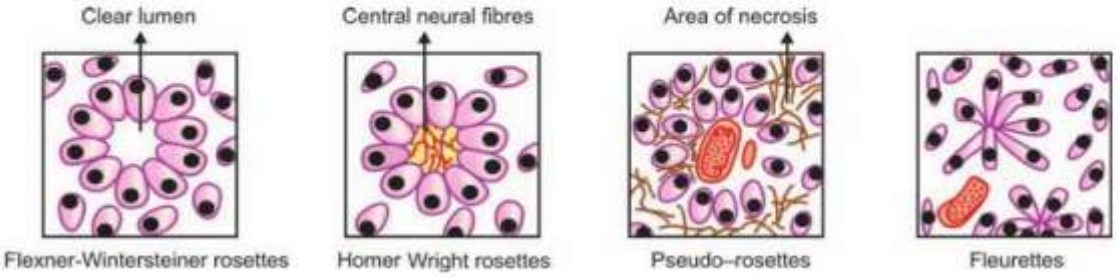
Retinoblastoma is a common congenital malignant tumour of the retina occurring in early childhood.

Etiopathogenesis:

- **Origin**: Retinoblastoma is a tumour derived from neurosensory retina. It occurs due to the proliferation of neural cells which have failed to evolve normally
- Of all cases, only 10% are familial (inherited by **autosomal dominant mode**) and the rest about 90% occur sporadically.
- Retinoblastoma (RB) gene has been identified as 14 band on the long arm of chromosome 13 (**13q 14**) and is a 'cancer suppressor' gene.
- Deletion or inactivation of both the normal alleles of RB gene by 2 mutations (**Knudson's two hit hypothesis**) leads to formation of retinoblastoma.
- **Microscopic examination** shows 2 types of cellular characteristics:
  1) Poorly differentiated cells with large hyperchromatic nuclei and scanty cytoplasm along with necrosis. They resemble the nuclear layer of the retina.
  2) Well-differentiated tumour cells may be arranged in 2 special forms: **a.** Rosettes **b.** Fleurettes.
     - **a. Rosettes**
       - ⇒ Flexner-Wintersteiner rosettes—specific for retinoblastoma.
       - ⇒ Homer Wright rosettes— not specific for retinoblastoma (can also be seen in neuroblastoma & medulloepithelioma).

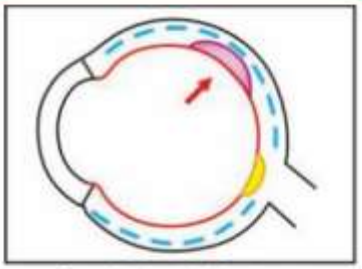
⇒ Pseudorosettes—tumour cells are clustered around blood vessels in necrotic retinoblastoma.

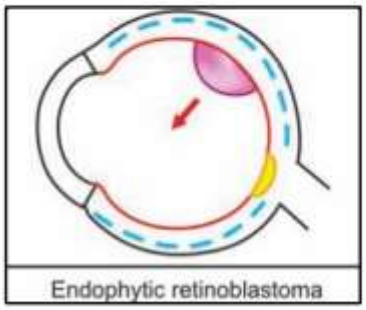
b. Fleurettes: specific for retinoblastoma – flower bouquet type aggregation of tumour cells.



Symptoms	Signs
1. **Leucocoria** (MC)—Peculiar yellow or white pupillary reflex called the "amaurotic cat's eye". It is due to reflection of light from the yellow-white mass in the retrolental area. 2. **Squint** usually convergent is the 2nd MC presenting symptom. 3. **Nystagmus** is seen in bilateral cases. 4. Severe pain may be present due to ↑ intraocular pressure. 5. Enlargement of the globe with protrusion of the eyeball.	1. Multiple polypoid masses are seen in the fundus. There may be haemorrhages on the surface of the tumour. 2. The tumour mass may spread into the vitreous cavity. 3. Pseudohypopyon with esotropia (convergent squint). 4. Acute secondary glaucoma may occur if tumour cells clog the trabecular meshwork → Large Eyeball (Buphthalmos)

- **Growth of RB** - 2 types:
  1. Glioma exophytum—it grows outwards separating the retina from the choroid. It resembles detachment of retina.
  2. Glioma endophytum—it grows inwards towards the vitreous.
- **Stages**- 4 clinical stages:
  1) **The quiescent stage**—It lasts from six months to one year.
  2) **The glaucomatous stage**—enlargement of the globe, proptosis and severe pain a/w ↑ IOP.
  3) **The stage of extraocular extension** – The tumour bursts through the limbus followed by rapid growth.
  4) **The stage of metastasis**—the tumour spreads by the:
     - Direct spread (MC route) – occurs via Optic nerve
     - Lymphatics—Pre-auricular and cervical lymph nodes.
     - Bloodstream—via choroidal vessels. MC sites are bone & liver.


Exophytic retinoblastoma


Endophytic retinoblastoma

Differential Diagnosis

1) **Pseudoglioma** – Various conditions other than retinoblastoma, which present as leukocoria are collectively called as 'Pseudoglioma'.
   - **a.** Congenital cataract
   - **b.** Inflammatory deposits in the vitreous following a plastic cyclitis or choroiditis.
   - **c.** Tuberculosis of the choroid specially the confluent type.
   - **d.** Toxocara infestation.
   - **e.** Persistent hyperplastic Primary vitreous.
   - **f.** Retrolental fibroplasia—it is common in premature babies due to hyperoxygenation.
2) **Other causes** – Coats disease, Choroidal coloboma & Retinal dysplasia.

Diagnosis of Retinoblastoma

1. **Examination under anaesthesia** - Fundus examination of both eyes after full mydriasis with atropine (direct as well as indirect ophthalmoscopy), measurement of IOP & corneal diameter.
2. **LDH levels** are raised in the aqueous humour.
3. **Plain X-ray orbit** —Calcification occurs in 75% cases of retinoblastomas.
4. **Ultrasonography, CT scan and MRI** confirm the diagnosis & also demonstrate extension to optic nerve, orbit and CNS, if any

Treatment

1. Radiation and chemotherapy—Retinoblastoma is a highly radiosensitive tumour
   - Standard dose CVE regimen: consists of 3-weekly, 6 cycles of **C**arboplatin (18.6 mg) on day 1, **V**incristine (0.05 mg) on day 1 & **E**toposide (5 mg) on day 1 and 2
2. Cryotherapy—used for small tumours located anterior to equator
3. Photocoagulation by argon laser or diode laser – used for small tumour located posterior to equator
4. Enucleation (removal of whole eyeball with optic nerve)—treatment of choice if tumour involves more than half of the retina / Optic nerve is involved / Glaucoma is present.
5. Exenteration of the orbit—it is done in stage 3. It is a mutilating surgical procedure → not preferred by many surgeons. Hence only Palliative treatment (CVE regimen, Debulking, External beam radiotherapy) is given in Stage 3 & 4

Prognosis

1) It is always bad if untreated.
2) It is fair if the eye is removed before the onset of extraocular extension.
3) Prognosis is poor if the optic nerve is involved, tumour cells are undifferentiated and in 3rd and 4th clinical stages.
4) Spontaneous regression with massive necrosis and calcification may occur occasionally due to the immunological mechanisms.

SQs

1. Retinal detachment - its classification, predisposing factors and clinical features. [17]
   a. Tractional retinal detachment [13]

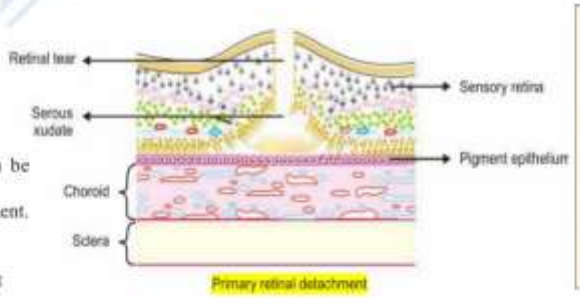
Ans.

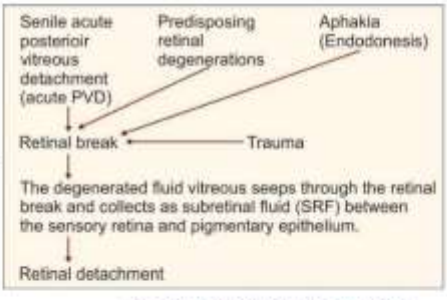
Retinal detachment {RD} is the separation of neurosensory retina proper from the pigment epithelium.

Classification

Clinico-etiologically retinal detachment can be classified into three types:

1. Rhegmatogenous or primary retinal detachment.
2. Tractional retinal detachment — Secondary retinal detachment
3. Exudative retinal detachment — Secondary retinal detachment





Flowchart depicting pathogenesis of rhegmatogenous retinal detachment

	Rhegmatogenous RD	Tractional RD	Exudative (solid) RD
Etiology	It occurs due to a break in the retina in the form of a hole or tear. This allows the fluid from	It occurs due to retina being mechanically pulled away from its bed by the	Exudative RD occurs due to the retina being pushed away by a neoplasm or

	the vitreous to seep through and raise the retina from its bed. Risk Factors are: ↳ **Age**: elderly (40-60 yrs) ↳ **Sex**: M > F. ↳ Myopia. ↳ Aphakia & pseudophakia. ↳ **Retinal degenerations**: Ex: Snail track degeneration, Lattice degeneration etc. ↳ Trauma. ↳ Senile posterior vitreous detachment (PVD).	contraction of fibrous tissue in the vitreous (vitreoretinal tractional bands). Risk Factors are: • Post-traumatic retraction of scar tissue • Proliferative diabetic retinopathy • Retinopathy of prematurity • Plastic cyclitis • Sickle cell retinopathy, • Vitreomacular traction syndrome, • Incontinentia pigmenti, • Retinal dysplasia, and • Toxocariasis.	accumulation of fluid beneath the retina **Causes are**: – ▪ **Systemic diseases:** preecclampsia, renal HTN, blood dyscrasias & polyarteritis nodosa. ▪ **Ocular diseases:** - Congenital: Nanophthalmos, choroidal coloboma & FEVR; - Inflammations: Harada's disease, posterior scleritis, orbital cellulitis etc. - Vascular diseases: Ex: Coats disease; - Neoplasms, e.g., retinoblastoma (exophytic type), haemangioma, etc. - Uveal effusion syndrome. - Choroidal neovascularization
Symptoms	- Dark spots (floaters) in front of the eye - Photopsia – sensation of flashes of light due to irritation of retina - Localised relative loss in the field of vision (of detached retina) - Sudden painless loss of vision occurs when the detachment is large and central		
Signs	✚ **Plane mirror examination**—no red glow seen in the area of detached retina ✚ **Fundus examination**—done by direct & indirect ophthalmoscope **a.** Detached retina looks greyish-white & raised above the surface **b.** **Detached retina is thrown into multiple folds which oscillate with the eye movement** **c.** Retinal vessels are dark with no central light reflex. **d.** In total RD, the retina is funnel-shaped being	↳ Absence of photopsia, floaters & retinal tears ↳ Presence of vitreoretinal bands with lesions of the causative disease ↳ Retinal mobility is severely reduced and shifting fluid is absent	Exudative RD can be differentiated from Rhegmatogenous RD by: ➔ Absence of photopsia, holes, tears & folds ➔ Shifting fluid: changing position of the detached area with gravity is the hallmark of exudative retinal detachment ➔ On transillumination test a simple detachment appears transparent while solid detachment is opaque ➔ FFA may show the source of fluid

	attached to the disc & ora serrata. e. **A/w retinal degeneration, pigmentation & haemorrhage** **Visual field charting**—Scotomas are present corresponding to the area of the detached retina **Electroretinography (ERG)**—it is subnormal or absent **Ultrasonography** confirms the diagnosis of retinal detachment in cases when retina cannot be visualised, e.g., senile mature cataract, corneal opacity, vitreous opacities		
Configuration	Convex	Concave	Convex
Treatment	1) To seal retinal breaks ➔ Photocoagulation or Cryosurgery or Diathermy 2) To approximate the sclera, choroid and detached retina ➔ **Scleral buckling**, Drainage of subretinal fluid (SRF), Pars plana vitrectomy etc.	Pars plana vitrectomy to cut the vitreoretinal tractional bands & Internal tamponade with either a long-acting gas or silicon oil	Treat the cause Ex: Enucleation for intraocular tumours
Complications	Proliferative vitreoretinopathy (PVR), Complicated (posterior cortical) cataract, uveitis and phthisis bulbi		

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2. Retinitis pigmentosa [15, 12, 03, 01]

Ans.

It is a slow degenerative, hereditary disease of the retina predominantly affecting the rods more than the cones.

Etiopathogenesis:

- Occurs due to consanguinity of the parents
- It appears in the childhood and progresses slowly causing blindness in middle age
- Retinitis pigmentosa is inherited as AD > AR > X-linked recessive disease (in the order of occurrence & Prognosis)
- It is Bilateral – both eyes are equally affected
- **Sex**: Males > Female
- Associations of Retinitis pigmentosa:
  - Ocular associations: myopia, primary open-angle glaucoma, microphthalmos, conical cornea (keratoconus) and posterior subcapsular cataract
  - Systemic associations – Ex: Cockayne's syndrome, Refsum's syndrome, Usher's syndrome etc.

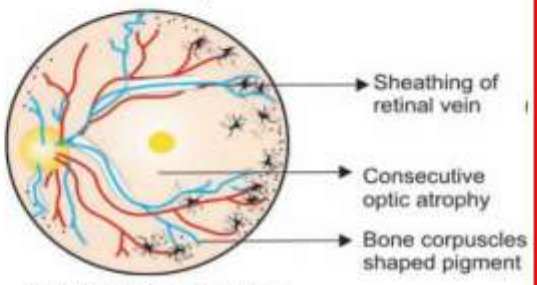
Symptoms:

1) Night blindness is an early complaint (since rods are degenerated early and cones are involved late)

2) **Tubular vision** – Loss of peripheral vision with preservation of central vision.
3) Central vision is also lost ultimately after many years

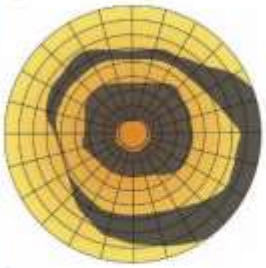
Signs

1. **Fundus examination**
   - Retina is studded with jet black spots (**pigments**) which resemble bone corpuscles with a spidery outline.
   - Retinal arterioles become extremely attenuated and thread-like.
   - Retinal veins may have a sheath of pigment for part of their course.
   - Thinning & atrophy of retinal pigment epithelium (RPE)
   - Optic disc—It shows features of optic atrophy, i.e., pale, wax-like, yellowish appearance.



Visual retinitis pigmentosa

2. **Visual fields**
   - Annular or ring scotoma is present which progress to tubular vision.
   - There is complete blindness in the later stage.
3. **Dark adaptation** is increased due to rods dysfunction.
4. **The electroretinogram** (ERG) and electro-oculogram (EOG) are markedly subnormal or completely extinguished early in the disease



Some Atypical forms of retinitis pigmentosa

- Retinitis pigmentosa sine pigmento: It is characterised by all the clinical features of typical retinitis pigmentosa, except that there are no visible pigmentary changes in the fundus.
- Sectorial retinitis pigmentosa: It is characterized by involvement of only one sector of the retina

Differential Diagnosis – Congenital syphilis & Night blindness

Treatment: till date there is no effective treatment for the disease.

1. **Measures to stop progression**: vasodilators, placental extracts, transplantation of rectus muscles into suprachoroidal space, acupuncture therapy, ultrasonic therapy etc.
2. **Correct any refractive errors** – prescribe glasses
3. **Low vision aids may be useful**. Ex: magnifying glasses' and 'night vision device'
4. **Systemic acetazolamide** (500 mg po) for associated cystoid macular oedema
5. **Stem cell therapy** is still under trial
6. **Rehabilitation** of the patient

Prophylaxis: Genetic counselling is advised. There should be no consanguineous marriages

Prognosis: The central vision steadily becomes very poor in advanced life.

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3. Hypertensive Retinopathy [15, 12, 08, 03]
   a. Keith & Wegner grading of hypertensive retinopathy [13]
   b. Toxaemia of pregnancy retinopathy [95]

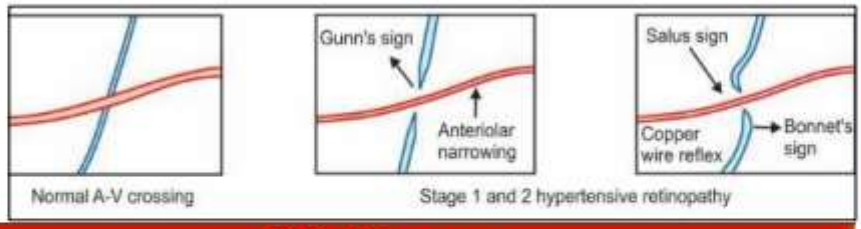
Ans.

Hypertensive retinopathy refers to fundus changes occurring in patients suffering from systemic HTN.

Pathogenesis: 3 factors which play role in the pathogenesis of hypertensive retinopathy are: -

1) Vasoconstriction of retinal arterioles & choroidal vessels ➔ choroidal & RPE ischaemia→ hypertensive choroidopathy
   - Vasoconstriction of peripapillary choroid → optic nerve head ischaemia → hypertensive optic neuropathy
2) Arteriosclerotic changes in the vessels

3) Increased vascular permeability results from hypoxia & is responsible for haemorrhages, exudates, focal retinal oedema, macular oedema etc.

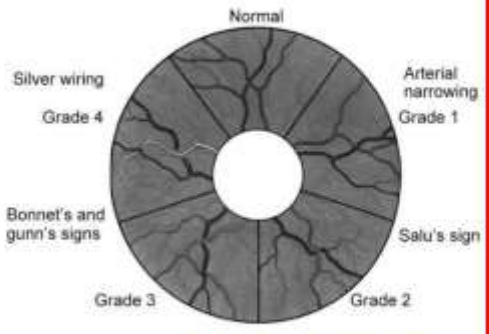


Clinical Types	Clinical Features
1) Simple hypertension without sclerosis	⊙ It is seen in young patients with elastic retinal arterioles ⊙ Generalized constriction of the arterioles occur ⊙ Superficial flame-shaped haemorrhages & cotton wool exudates may be present.
2) Hypertension with involutionary (senile) sclerosis	• Seen in elderly (above 50 yrs); • Changes at arteriovenous crossings are diagnostic (Gunn's sign, Bonnet sign & Salu's sign) • There is deposition of hard exudates • Retinal haemorrhages **without any oedema** may occur
3) Chronic hypertension with compensatory arteriolar sclerosis	✸ It is seen in young patients ✸ a/w chronic glomerulonephritis & the ophthalmic picture is known as '**albuminuric' or 'renal' retinopathy** ✸ The vessels are narrow and tortuous with nicking at AV crossing ✸ Multiple retinal haemorrhages are seen **with oedema**, diffuse cotton wool exudates (early), and hard exudates (later) ✸ Vision is seriously impaired
4) Malignant (acute) hypertensive retinopathy	» Marked arteriolar narrowing with generalized oedema & exudates ➔ papilloedema, Disc pallor macular star along with superficial flame-shaped haemorrhages & Focal intraretinal periarteriolar transudates (FIPTs – due to breakdown of blood-retinal barrier) » Elschnig's spots (focal white spots) & Siegrist streaks (fibrinoid necrosis) are formed » Cotton wool spots are also more marked » a/w renal insufficiency » The visual prognosis is grave unless controlled medically

Keith & Wegner grading of hypertensive retinopathy	
Grade	Fundus Examination
Grade 1	Mild arteriolar attenuation, particularly of small branches, with broadening of the arteriolar light reflex
Grade 2	Marked arteriolar attenuation a/w deflection of veins at arteriovenous crossings (Salus' sign)
Grade 3	Grade 2 + ▪ Copper wiring of arterioles, banking of veins distal to arteriovenous crossings (Bonnet sign), tapering of veins on either side of the crossings (Gunn sign) ▪ Flame-shaped haemorrhages, cotton-wool spots and hard exudates are also present
Grade 4	Grade 3 + silver-wiring of arterioles and papilloedema

Management

- Mild cases require BP control only.
- Moderate cases (with retinal haemorrhages, microaneurysms & cotton-wool spots): BP control + risk reduction therapy (e.g., cholesterol lowering agents).
- Accelerated hypertensive retinopathy (with bilateral disk swelling & severe HTN) patients: stepwise control of BP over a few hours to avoid a sudden ↓ in BP which may ↓ perfusion of optic nerve head and CNS (causing stroke).



Hypertensive retinopathy

Retinopathy in Pregnancy-induced Hypertension (aka 'toxaemia of pregnancy'), is a disease of

- PIH is characterised by ↑ BP, proteinuria and generalised oedema.
- In PIH, Retinal changes are marked when BP rises above $\frac{200}{130}$ mm of Hg.
  - Narrowing of nasal arterioles – Earliest change.
  - Persistent spasm of vessels causes retinal hypoxia - 'cotton wool spots' & superficial haemorrhages.
  - Retinal oedema & exudation is marked & a/w 'macular star' or 'flat macular detachment'
  - Further progression of retinopathy occurs rapidly if pregnancy is allowed to continue.
  - Prognosis for retinal **re**attachment is good, as it occurs spontaneously within a few days of termination of pregnancy.
- **Management**:
- Changes of retinopathy are reversible and disappear after the delivery
- If the patient responds well to conservative Mx, the pregnancy can be continued under close observation.
- Advent of hypoxic retinopathy (cotton wool spots, retinal oedema and haemorrhages), however, should be considered an indication for termination of pregnancy; otherwise, permanent visual loss or even loss of life (of both mother and foetus) may occur.

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4. Central Retinal Vein Occlusion. [10]

Ans. Retinal vein occlusions are more common than the artery occlusions.

- Pressure on the vein by an atherosclerotic retinal artery can cause the occlusion
- **Predisposing factors**: HTN, DM, Hyperviscosity of blood, ↑IOP, orbital cellulitis, orbital tumors etc.
- **Site of Occlusion**: It is just behind the lamina cribrosa
- **Symptom**: There is Painless sudden onset of impaired vision
- **Signs**:

1) **In ischemic CRVO**
   - Retinal veins are dilated, engorged & tortuous.
   - Retina is covered with multiple haemorrhages (splashed tomato appearance) & soft exudates. ☞
   - RAPD present.
   - Retinal edema
   - ↓ amplitude of b-wave of electroretinogram (ERG)
   - In late stages, Neovascularization may be seen at the disc (NVD) or in the periphery (NVE)

Obstruction to the outflow of blood and stagnation
↓
Rise in intravascular pressure
↓
Retinal oedema, abnormal leakage and haemorrhage
↓
Formation of collaterals and neovasularisation

2) **In non-ischemic CRVO**: mild venous congestion, few superficial flame-shaped haemorrhages & other mild lesions but **no RAPD,** relatively normal retina

- **Investigations:**
  - Record Visual acuity, IOP
  - Gonioscopy to rule out neovascularization of angle (NVA).
  - Goldmann perimetry & ERG evaluation: to differentiate ischaemic vs non-ischaemic CRVO.

- FFA should be done (to assess state of retinal perfusion) after resolution of retinal haemorrhage
- Routine investigations: to look for predisposing factors

Complications

1. Neovascular glaucoma occurs at a later stage (usually within 3 months – aka 90 days Glaucoma) due to sclerosis and neovascularisation at the angle of anterior chamber
2. Vitreous haemorrhage and subhyaloid haemorrhage may occur.
3. Complete blindness develops eventually

Differential Diagnosis:

- Diabetic retinopathy is generally bilateral and CRVO is usually unilateral.
- Ocular ischaemic syndrome (OIS) due to carotid occlusive disease has only dilated veins without tortuosity (in CRVO tortuosity is also seen)

Treatment

1) Observation & monitoring in patients with mild to moderate visual loss (since CRVO resolves with almost normal vision.)
2) For patients with marked visual loss: **Intravitreal anti-VEGF drugs**: e.g., Bevacizumab **& Intravitreal steroids** – ex: triamcinolone acetonide: useful for associated CME & Neovascularisation
3) Treatment of Predisposing factors – Ex: HTN, DM
4) Neovascular glaucoma (NVG) can be prevented by PRP.
5) If NVG develops → Pars plana placement of glaucoma drainage device (GDD)

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5. Photoretinitis [05]

Ans. Photoretinitis aka Solar retinopathy or eclipse retinopathy, refers to retinal injury induced by direct or indirect sun viewing.

Etiology:

- Religious sun gazing, solar eclipse observing, telescopic solar viewing, sun bathing and sun watching in psychiatric disorders.
- Causes other than sun exposure are: Welding arc exposure, Lightening & phototoxicity from ophthalmic instruments like operating microscope.

Pathogenesis – Solar radiations damage the retina through:

- Photochemical effects produced by UV rays & IR rays
- Thermal effects may enhance the photochemical effects.

Clinical features

Symptoms	Signs
▪ Persistence of negative after-image of the sun, progressing later into a +ve scotoma & metamorphopsia. ▪ Decreased vision which develops within 1 to 4 hours after solar exposure, usually improves within 6 months	▪ Shortly after exposure a small yellow spot appear in the foveal region. ▪ Later, central burnt-out hole in the pigment epithelium is seen – appears as a **bean- or kidney-shaped pigmented spot** with yellowish white centre in the foveal region. ▪ In worst cases, typical macular hole may appear

Treatment:

- There is no effective treatment for Photoretinitis, so emphasis should be on prevention.
- Eclipse viewing should be discouraged unless there is proper use of protective eye wear filters (to block UV & IR rays).
- Prognosis is guarded, since some scotoma & loss in visual acuity by 1 or 2 lines mostly persists

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CONTENTS

Diseases of Cornea

LQs

1. Discuss symptoms, signs and treatment of herpes simplex keratitis [14]
   a. Herpes Zoster Ophthalmicus [17]
   b. 4 causes of Superficial Punctate Keratitis [15, 01]
   c. Viral keratitis [12, 09]
   d. Dendritic corneal ulcer [03, 95]

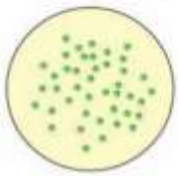
Ans.

Viral Keratitis

- Most of the viruses tend to affect the epithelium of both the conjunctiva and cornea, hence the typical viral lesions constitute the viral keratoconjunctivitis.
- Causative Organisms: Herpes Simplex Virus (most common), herpes zoster & adenovirus keratitis (rare).

Herpes Simplex Keratitis

- Etiology: HSV is a DNA virus.
  - HSV-1 is acquired by kissing or coming in contact with a patient of herpes labialis.
  - HSV-2 is transmitted to eyes of neonates through infected genitalia of the mother.
- Types: Primary Herpes (mainly conjunctival lesions seen) & Recurrent Herpes (mainly corneal lesions seen)
- Symptoms: Vesicles are seen on lips, nose, cornea (HSV-1) & genitals (HSV-2); photophobia, ↓ visual acuity, ocular discomfort.
- Signs:
  - Skin lesions – Vesicles heal without scar formation.
  - Epithelial lesions – occur due to active viral replication
    1) Superficial punctate keratitis: many pin-head size plaques of epithelial cells
    2) Dendritic ulcer with terminal buds
    3) Confluent ulcer—Large geographical pattern type of ulcers
  - Stromal lesions
    - Immune stromal keratitis: occurs due to Type 3 hypersensitivity
    - Necrotising stromal keratitis
  - Endothelial lesions – Disciform keratitis: disc shaped areas of deep stroma/endothelial edema – occurs due to type 4 hypersensitivity.
  - Metaherpetic keratitis – is not an active viral disease, but is a mechanical healing problem due to persistent defects in the basement membrane of corneal epithelium which occurs at the site of a previous herpetic ulcer
  - Corneal Anaesthesia – due to 5th cranial nerve palsy


Superficial punctate keratitis


Confluent ulcer


Dendritic ulcer

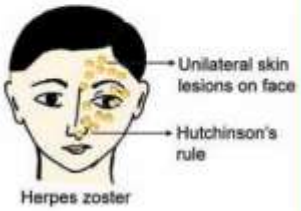

Disciform keratitis

- Diagnosis: By immunofluorescence of epithelial scrapings
- Complications: Corneal opacity, Iritis and iridocyclitis
- Treatment:
  - Topical Antivirals: Acyclovir—3% eye ointment 5 times daily for 10-14 days.
    - Topical Steroids can be given (except in epithelial lesions)
  - Debridement – can be done for dendritic ulcers. This protects healthy epithelium from infection
  - For Metaherpetic keratitis – Promote healing by use of lubricants (artificial tears), bandage soft contact lens and lid closure (tarsorrhaphy)
  - Atropine and warm compresses are useful in controlling iritis
  - Systemic Antivirals - Oral acyclovir 400 mg BD × 7 days. It is the method of choice in recurrent cases
  - Full thickness keratoplasty is done in cases of permanent corneal opacity. The eye must be quiet for a year at least

➢ **Herpes Zoster Opthalmicus** (aka Shingles): It is is an acute infection of Gasserian ganglion of the 5th cranial nerve by the varicella-zoster virus (VZV). It is a/w chickenpox infection in childhood.

➢ Pathogenesis:

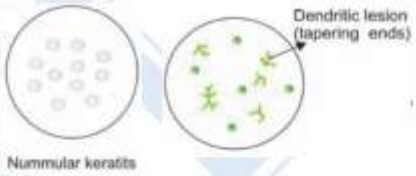
- It often occurs in elderly with ↓ cellular immunity, e.g. as in diabetics, alcoholics, cancer patients etc.
- It is always unilateral; affecting the gasserian ganglion from where the virus travels down the ophthalmic (V_1) branch of 5th nerve affecting the dermatomes supplied by it.



Herpes zoster

➢ Symptoms:

- Fever, malaise & Severe neuralgic pain – a/w Acute phase lesions
- Rows of vesicular eruption along V_1 nerve. Vesicles rupture & cause pitted scar.
- Skin of lid and face becomes red and oedematous



➢ Signs

- Conjunctivitis
- Slit-lamp examination reveals: Superficial punctate keratitis, pseudo-dendritic ulcers (no terminal buds), Nummular keratitis & Disciform keratitis
- Hutchinson's rule: More chances of HZO if the rash affects the tip of the nose (nasociliary nerve)
- Corneal Anaesthesia

➢ Complications: Iridocyclitis, scleritis, Secondary glaucoma, postherpetic neuralgia, cranial nerve palsies (3rd , 6th , 7th cranial nerves)

➢ Treatment:

- Topical Antivirals: Acyclovir—3% eye ointment 5 times daily for 10-14 days
- Systemic Antivirals—Oral acyclovir 800 mg is given 5 times daily for 14 days
- Topical steroids are useful particularly in disciform keratitis, scleritis and iridocyclitis
- Systemic steroids are indicated in cranial nerve palsies
- Analgesics & anti-inflammatory Drugs - mephenamic acid + PCM or even pethidine (in severe cases)
- Antibiotic skin ointment is applied over the skin lesion to prevent secondary infection.
- Topical atropine is applied in cases of keratitis, iridocyclitis, and scleritis
- Surgical treatment:
  - Lateral tarsorrhaphy
  - Full thickness keratoplasty is required for visual rehabilitation of patients with dense scarring. The eye must be quiet for a year at least.

➢ **Causes of Superficial Punctate Keratitis**

1. Viral infections – herpes simplex, herpes zoster, adenovirus, pharyngo-conjunctival fever etc.
2. Chlamydial infections – trachoma and inclusion conjunctivitis.
3. Toxic lesions e.g., due to staphylococcal toxin
4. Trophic lesions e.g., exposure keratitis and neuroparalytic keratitis
5. Allergic lesions e.g., vernal keratoconjunctivitis.
6. Irritative lesions e.g., effect of some drugs such as idoxuridine.
7. Disorders of skin and mucous membrane, such as acne rosacea and pemphigoid.
8. Dry eye syndrome, i.e., keratoconjunctivitis sicca.
9. Specific types of idiopathic SPK e.g., Thygeson's superficial punctate keratitis.
10. Photo-ophthalmitis.

2. Describe aetiology, clinical features, Rx & complications of Bacterial Corneal Ulcer [14, 13, 10]
   a. Nebula [14]

Ans.

Etiology: There are 2 factors in the production of purulent corneal ulcer: Damage to corneal epithelium & Infection of the eroded area.

However, 3 pathogens can ulcerate even the intact corneal epithelium → N. gonorrhoeae, N. meningitidis & C. diphtheriae.

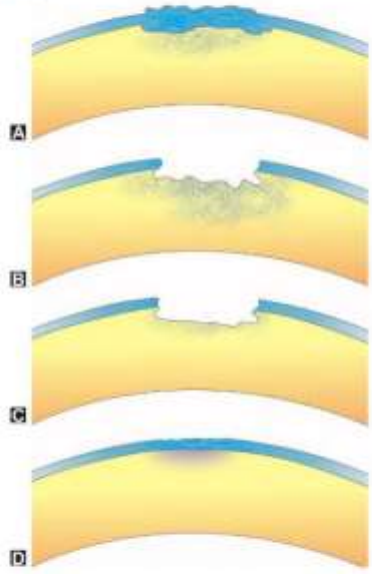
- Corneal epithelial damage: It may occur in following conditions:
  a. Corneal abrasion due to small foreign body, trivial trauma in contact lens wearers.
  b. Epithelial drying as in xerosis and exposure keratitis.
  c. Necrosis of epithelium as in keratomalacia.
  d. Desquamation of epithelial cells as a result of corneal oedema as in bullous keratopathy.
  e. Epithelial damage due to trophic changes as in neuroparalytic keratitis.
- Sources of infection include:
  - Exogenous source like conjunctival sac, lacrimal sac (dacryocystitis), infected foreign bodies and waterborne or airborne infections.
  - From the ocular tissue – Ex: diseases of the conjunctiva readily spread to corneal epithelium, those of sclera to stroma, and of the uveal tract to the endothelium of cornea.
- Causative organisms: Staphylococci, Pseudomonas, Streptococcus pneumonia, Enterobacteriaceae and Neisseria.

Pathogenesis of corneal ulcer: Pathological changes can be described under 4 stages:

1. Stage of progressive infiltration,
2. Stage of active ulceration,
3. Stage of regression, and
4. Stage of cicatrization.

Course of corneal ulcer may take 1 of the 3 forms:

a. Ulcer may become localised and heal,
b. Penetrate deep leading to corneal perforation, and
c. Spread fast to involve whole cornea as sloughing corneal ulcer.



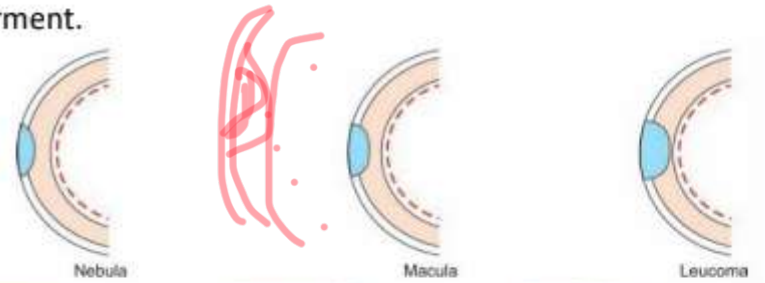
Pathology of corneal ulcer: A, stage of progressive infiltration; B, stage of active ulceration; C, stage of regression; D, stage of cicatrization

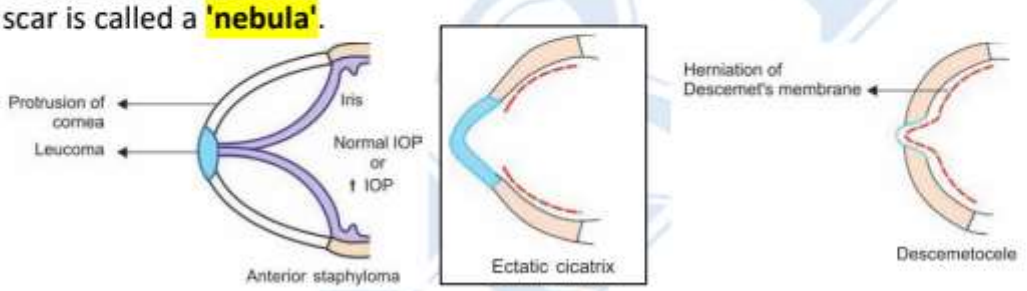
Clinical features: Corneal ulcers may manifest with/without hypopyon

Symptoms	Signs
1. Pain & foreign body sensation – occur due to mechanical effects of lids and toxins on the V_1 nerve endings. 2. Watering from the eye - occurs due to reflex hyperlacrimation. 3. Photophobia – occurs due to stimulation of nerve endings. 4. Blurred vision results from corneal haze. 5. Redness of eyes – occurs due to congestion of circumcorneal vessels	1) Swelling of lids & Blepharospasm. 2) Conjunctiva shows hyperaemia and ciliary congestion. 3) Corneal ulcer ↳ Epithelial defect seen in early stage ↳ Yellowish-white ulcer ↳ Can be oval or irregular in shape ↳ Margins of the ulcer are swollen and over hanging ↳ Floor is covered by necrotic material ↳ Stromal oedema is present surrounding the ulcer area 4) Anterior chamber may show pus (hypopyon). 5) Iris may be slightly muddy in colour. 6) Pupil may be small due to associated toxin induced iritis. 7) IOP may sometimes be raised (inflammatory glaucoma).

Complications of Corneal Ulcer

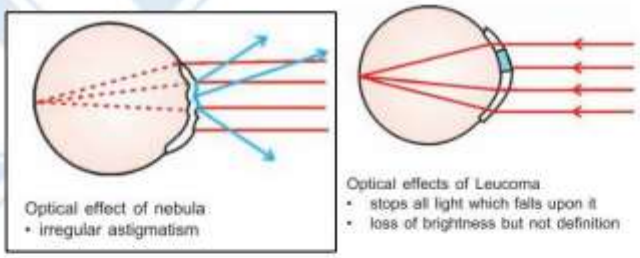
1) Toxic iridocyclitis – occurs due to absorption of toxins in the anterior chamber.
2) Secondary glaucoma – occurs due to fibrinous exudates blocking the angle of anterior chamber (inflammatory glaucoma).
3) Descemetocele – develop if ulcer extend up to Descemet's membrane. This is a sign of impending perforation and is a/w severe pain.
4) Perforation of corneal ulcer: Sudden strain due to cough, sneeze or spasm of orbicularis muscle may convert impending perforation into actual perforation.
5) Corneal scarring → permanent visual impairment.
   - Depending upon the clinical course of ulcer, corneal scar noted may be nebula, macula, leucoma, ectatic cicatrix or anterior staphyloma.
   - When corneal ulcer involves Bowman's membrane and few superficial stromal lamellae, the resultant scar is called a **'nebula'**.





MANAGEMENT OF CORNEAL ULCER

- Clinical evaluation: history taking, physical examination & Ocular examination
- Laboratory Investigations
  1) Routine investigations - CBP, blood sugar, complete urine and stool examination
  2) Microbiological investigations – to identify causative organism and guide the treatment
- **Treatment**



I. Treatment of Uncomplicated corneal ulcer

1. Specific treatment	Topical antibiotics	Initial therapy should be with any of the following 2 drugs: a. Fortified Cefazoline 5% & Fortified tobramycin, 1.3% or b. Fortified vancomycin 5%, and one of FQ (0.3% ciprofloxacin, or 0.3% ofloxacin or 0.5% moxifloxacin). If the response is poor, immediately change Abx as per sensitivity report
	Systemic antibiotics	a cephalosporin & an aminoglycoside or ciprofloxacin (750 mg BD) may be given in fulminating cases with perforation
2. Non-specific Supportive treatment	Cycloplegic drugs – 1% atropine eye drops	• To reduce pain from ciliary spasm and • To prevent the formation of synechiae from iridocyclitis. • Atropine also increases the blood supply to anterior uvea
	Systemic NSAIDs	Paracetamol & ibuprofen relieve the pain & ↓ oedema
	Multi-Vitamins (A, B-complex and C)	Help in early healing of ulcer
3. General measures	• Hot fomentation –gives comfort, reduces pain and causes vasodilatation. • Dark goggles may be used to prevent photophobia. • Rest, good diet and fresh air may have a soothing effect	

II. Treatment of non-healing corneal ulcer:

1) Removal of any known cause of non-healing ulcer.
   - Local causes: ↑ IOP, misdirected cilia, impacted foreign body, dacryocystitis etc.
   - Systemic causes: DM, severe anaemia, malnutrition, patients on systemic steroids etc.
2) Mechanical debridement of ulcer to remove necrosed material may hasten the healing.
3) Cauterisation of the ulcer with pure carbolic acid or 10–20% trichloroacetic acid.
4) Bandage soft contact lens.
5) Peritomy, i.e., severing of perilimbal conjunctival vessels can be performed when excessive corneal vascularization is hindering healing.

III. Treatment of impending perforation:

1. No strain: avoid sneezing, coughing & straining during stool, etc.
2. Pressure bandage should be applied to give some external support.
3. ↓ IOP by simultaneous use of acetazolamide, IV mannitol, oral glycerol & 0.5% timolol eye drops,
4. Tissue adhesive glue such as cyanoacrylate is helpful in preventing perforation.
5. Bandage soft contact lens may also be used.
6. Cover the cornea with Conjunctival flap – to give support to the weak tissue.
7. Amniotic membrane transplantation may also be considered as an option.
8. Penetrating therapeutic keratoplasty (tectonic graft) may be undertaken in suitable cases

IV. Treatment of perforated corneal ulcer:

Depending upon the size of perforation, measures like use of tissue adhesive glues, covering with conjunctival flap, bandage soft contact lens or therapeutic keratoplasty should be undertaken.
Best option is an urgent tectonic keratoplasty.

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3. Describe the aetiology, clinical features, and treatment of interstitial keratitis [15, 02, 96]
   a. Causes of Interstitial Keratitis [17]

Ans.

Interstitial keratitis is inflammation of only Corneal Stroma without primary involvement of the epithelium or endothelium

Causes: Congenital Syphilis, Acquired syphilis, Cogan Syndrome, Trypanosomiasis, Malaria, TB, Leprosy, sarcoidosis etc.

Syphilitic (Luetic) Interstitial Keratitis.

- Pathogenesis: It is a manifestation of local antigen-antibody reaction.
  - Treponema pallidum invades the cornea and sensitizes it in the foetal stage.
  - Later, fresh invasion by Treponema excites the inflammation in the sensitized cornea.
- Clinical features
  - The disease is generally B/L in inherited syphilis and U/L in acquired syphilis
  - In Congenital Syphilis – Hutchinson's triad: interstitial keratitis, Hutchinson's teeth & vestibular deafness.
  - Clinical features of interstitial keratitis can be divided into 3 stages: initial progressive stage, florid stage and stage of regression.
    1. Initial progressive stage – pain, lacrimation, photophobia, blepharospasm and circumcorneal injection followed by a diffuse corneal haze (ground glass appearance). This stage lasts for about 2 weeks.

2. Florid stage: In this stage, eye remains acutely inflamed. Deep vascularization of cornea develops. Since, these vessels are covered by hazy cornea, they look dull reddish pink which is called 'Salmon patch appearance'. Superficial vessels and conjunctiva heap at the limbus in the form of epulit. This stage lasts for about 2 months.
3. Stage of regression: The acute inflammation resolves. This stage may last for about 1 to 2 years.

- **Diagnosis**: +ve VDRL or Treponema pallidum immobilization test confirms the diagnosis.
- **Treatment**
  - Topical treatment for keratitis:
    - Topical corticosteroid drops e.g., dexamethasone 0.1% drops
    - Atropine eye ointment
    - Dark goggles to be used for photophobia.
    - Keratoplasty – done in cases where dense corneal opacities are left.
  - Systemic treatment: Penicillin & steroids.

Tuberculous Interstitial Keratitis

- The features of tubercular interstitial keratitis are similar to syphilitic interstitial keratitis except that it is more frequently unilateral and sectorial (usually involving a lower sector of cornea).
- **Treatment** consists of systemic antitubercular drugs, topical steroids and cycloplegics.

Cogan's Syndrome:

- It comprises the interstitial keratitis of unknown etiology, acute tinnitus, vertigo, and deafness.
- It typically occurs in middle-aged adults and is often bilateral.
- **Treatment**: topical and systemic corticosteroids.

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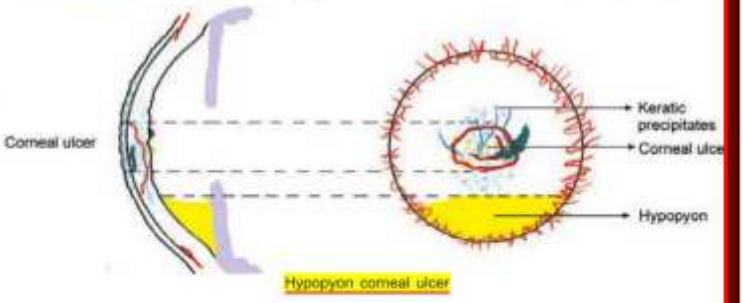
4. Hypopyon Corneal ulcer – Etiology, signs, symptoms, laboratory findings, Dx & treatment [08, 03]
   a. Ulcus serpens [16]

Ans.



Hypopyon corneal ulcer

Etiopathogenesis:

Causative organisms:

- 'Hypopyon corneal ulcer' = Ulcus Serpens ➔ Pneumococcus
- 'Corneal ulcer with hypopyon' ➔ caused by other organisms such as Staphylococci, Streptococci, Gonococci, Moraxella and Pseudomonas.

Factors predisposing to development of hypopyon: chronic dacryocystitis {pneumococcus}, old people, alcoholics etc.

Mechanism of development of hypopyon: Corneal ulcer is associated with some iritis owing to diffusion of bacterial toxins. When the iritis is severe the outpouring of leucocytes from the vessels is so great that these cells gravitate to the bottom of the anterior chamber to form a hypopyon. Once the ulcerative process is controlled, the hypopyon is absorbed.

Clinical features: same as bacterial corneal ulcer.

During initial stage of ulcus Serpens, there is little pain. As a result, the treatment is often unduly delayed.

Characteristic features of ulcus Serpens are:

- It is a greyish white or yellow disc-shaped ulcer occurring near the centre of cornea
- Ulcer has a tendency to creep over the cornea in a serpiginous fashion.
- Violent iridocyclitis is commonly associated with a definite hypopyon.
- Hypopyon increases in size very rapidly and often results in secondary glaucoma.
- Ulcer spreads rapidly and has a great tendency for early perforation.

Management of hypopyon corneal ulcer: same as for other bacterial corneal ulcer.

Special points which need to be considered are:

- Secondary glaucoma should be anticipated and treated with Anti-glaucoma drugs
- Source of infection, i.e., chronic dacryocystitis if detected, should be treated by dacryocystectomy

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SQs

1) Mention various causes of loss of Corneal Transparency. Write a note on Vascularization of Cornea. [17]
   a. Adherent leucoma [13, 03]
   b. Leucoma grade corneal opacity [06]
   c. Corneal opacity [05]

Ans.

The word 'corneal opacification' literally means loss of normal transparency of cornea.

Cornea is an avascular structure. Small loops derived from the anterior ciliary vessels invade its periphery for about 1 mm. Actually, these loops are not in the cornea but in the subconjunctival tissue which overlaps the cornea.

Causes of Corneal Opacity

Tears in the endothelium and Descemet's membrane (STUMPED) corneal ulcers and inflammation (STUMPED).

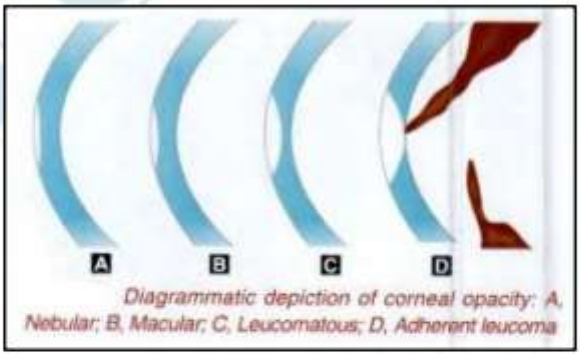
Congenital

- **S**: Sclerocornea
- **T**: Tears in Descemet's membrane
- **U**: Ulcer
- **M**: Metabolic conditions such as mucolipidosis, mucopolysaccharidosis
- **P**: Posterior corneal defect such as Peter's anomaly
- **E**: Endothelial dystrophy such as congenital hereditary endothelial dystrophy
- **D**: Dermoid.

Acquired

- Post-trauma following chemical injuries, mechanical injuries
- Corneal degenerations
- Postinfection or inflammation of cornea following infective or non-infective keratitis.

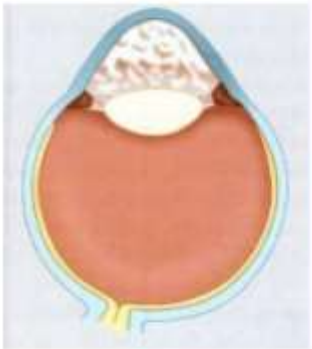
Clinical features: Loss of vision (when dense opacity covers the pupillary area) or blurred vision (due to astigmatic effect).



Diagrammatic depiction of corneal opacity: A, Nebular; B, Macular; C, Leucomatous; D, Adherent leucoma

Types of corneal opacity:

1. **Nebular corneal opacity**: It is a faint opacity – occurs due to superficial scars
2. **Macular corneal opacity**: It is a semi-dense opacity produced when scarring involves about half the corneal stroma.
3. **Leucomatous corneal opacity** (leucoma simplex): It is a dense white opacity which results due to scarring of more than half of the stroma.
4. **Adherent leucoma**: It results when healing occurs after perforation of cornea with incarceration of iris
5. **Corneal facet**: Sometimes, the corneal surface is depressed at the site of healing (due to less fibrous tissue); such a scar is called facet.
6. **Kerectasia**: In this condition, corneal curvature is ↑ at the site of opacity (bulge due to weak scar).
7. **Anterior staphyloma**: {refer 6th SQ}


Anterior staphyloma

Secondary changes seen in long-standing corneal opacity: hyaline degeneration, calcareous degeneration, pigmentation and atheromatous ulceration.

Treatment:

1) Optical iridectomy – done in cases with central macular or Leucomatous corneal opacities, provided vision improves with pupillary dilatation.

2) Phototherapeutic keratectomy (PTK) performed with excimer laser is useful in superficial (nebular) corneal opacities.
3) Keratoplasty can be done in uncomplicated cases where optical iridectomy is not of much use.
4) Cosmetic-coloured contact lens – BEST option for an eye with ugly scar having no potential for vision
5) Tattooing of scar – suitable only for firm scars in a quiet eye without useful vision

---

2) Corneal edema [16]

Ans. The water content of normal cornea is 78%. It is kept constant by a balance of factors which draw water in the cornea (e.g., IOP and swelling pressure of the stromal matrix) and the factors which draw water out of cornea (viz. the active pumping action of corneal endothelium, and the mechanical barrier action of epithelium and endothelium).

- Disturbance of any of the above factors leads to corneal oedema, wherein its hydration becomes above 78%, central thickness increases and transparency reduces.

Causes of corneal oedema

1) ↑IOP
2) Endothelial damage
   - Due to injuries, such as birth trauma (forceps delivery), surgical trauma during intraocular operation, contusion injuries and penetrating injuries.
   - Due to corneal dystrophies such as, Fuchs dystrophy, congenital hereditary endothelial dystrophy and posterior polymorphous dystrophy.
   - Secondary to inflam-mations such as uveitis, endophthalmitis and corneal graft infection.
3) Epithelial damage due to – mechanical injuries, chemical burns, radiational injuries, thermal injuries, inflammation and infections.

Clinical features

- Initially there occurs stromal haze with reduced vision.
- Permanent oedema with epithelial vesicles and bullae formation (bullous keratopathy) – In long-standing cases with chronic endothelial failure (e.g., in Fuch's dystrophy) there occurs.
- This is associated with marked loss of vision, pain, discomfort and photophobia, due to periodic rupture of bullae.

Treatment

1) Treat the cause wherever possible, e.g., raised IOP and ocular inflammations.
2) Dehydrate the cornea using:
   - Hypertonic agents e.g., 5% sodium chloride drops.
   - Hot forced air from hair dryer.
3) Therapeutic soft contact lenses may be used to get relief from discomfort of bullous keratopathy.
4) Penetrating keratoplasty is required for longstanding cases of corneal oedema, non-responsive to conservative therapy.

---

3) Indications of Keratoplasty [11]
   a. Indications for Lamellar Keratoplasty [11]
   b. Types of Keratoplasty [10]

Ans.

Keratoplasty, also called corneal grafting or corneal transplantation, is a surgery in which the patient's diseased cornea is replaced by the healthy clear cornea.

Types of Keratoplasty

A. Autokeratoplasty:

1) Rotational keratoplasty, in which patient's own cornea is trephined and rotated to transfer the pupillary area having a small corneal opacity to the periphery.

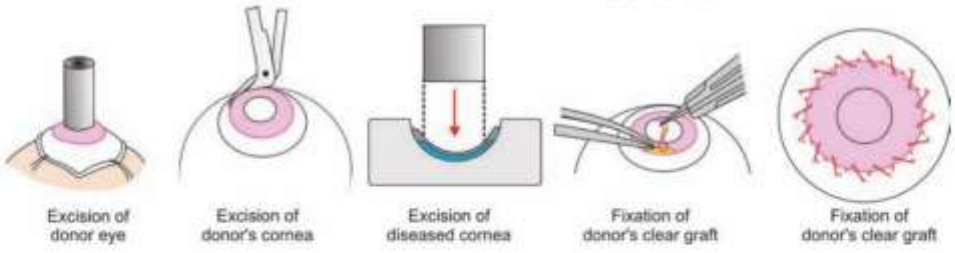
2) Contralateral keratoplasty: It is indicated when cornea of one eye of the patient is opaque and the other eye is blind due to posterior segment disease (e.g., optic atrophy & retinal detachment, etc.) with clear cornea

B. Allografting or Allo-keratoplasty: In it, patient's diseased cornea is replaced by the donor's healthy cornea. It can be of following types:

1) Penetrating Keratoplasty (PK) (full-thickness grafting) – indicated in pseudophakic bullous keratopathy

2) Lamellar Keratoplasty (partial-thickness grafting) which may be:

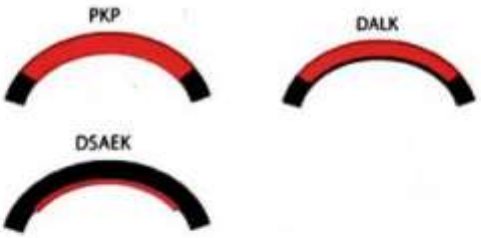
- DALK = Deep anterior lamellar keratoplasty – corneal tissue is removed upto Descemet's membrane – indicated in keratoconus.
- DSEK = Descemet's stripping endothelial keratoplasty – Removal of only endothelium & Descemet's membrane – indicated after the surgical trauma (Ex: phacoemulsification).



Schematic diagram of technique of penetrating keratoplasty

3) Small patch graft (for small defects), which may be full thickness or partial thickness.

Indications of Keratoplasty



1) **Optical**, i.e., to improve vision in – corneal opacity, bullous keratopathy, corneal dystrophies, advanced keratoconus.

2) **Therapeutic**, i.e., to replace inflamed cornea not responding to conventional therapy.

3) **Tectonic graft**, i.e., to restore integrity of eyeball e.g., after corneal perforation and in marked corneal thinning.

4) **Cosmetic**, i.e., to improve the appearance of the eye

Donor tissue: The donor eye should be removed as early as possible (within 6 hours of death). It should be stored under sterile conditions.

Complications of Keratoplasty

- Early complications: flat anterior chamber, iris prolapse, infection, secondary glaucoma, etc.
- Late complications – graft rejection, recurrence of disease and astigmatism.

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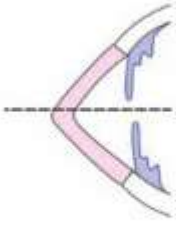
4) Keratoconus [09, 01]

Ans.

Keratoconus (conical cornea) is a non-inflammatory ectatic condition of cornea.

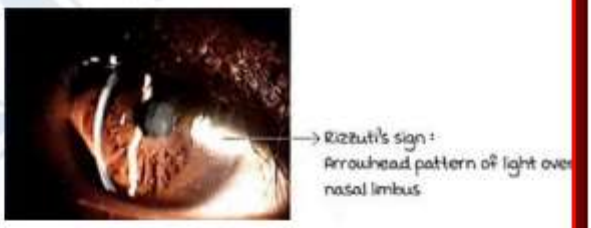


Etiopathogenesis: It is still not clear. It usually starts at puberty and progresses slowly with thinning and ectasia which occur as a result of defective synthesis of mucopolysaccharide and collagen tissue.

Clinical features	
Symptoms	**Signs**
Patient presents with a defective vision due to progressive myopia and irregular astigmatism, which does not improve fully despite full correction with glasses	**1)** Window reflex is distorted. **2)** Placido disc examination shows irregularity of the circles **3)** Slit-lamp examination ➔ thinning and ectasia of central cornea, opacity at the apex and Fleischer's ring at the base of cone, folds in Descemet's and Bowman's membranes. ↳ Very fine, vertical, deep stromal striae (Vogt lines) which disappear with external pressure on the globe are peculiar feature. **4)** Retinoscopy reveals a yawning reflex (scissor reflex) and irregular astigmatism. **5)** On distant direct ophthalmoscopy an annular dark shadow (due to total internal reflection of light) is seen which separates the central and peripheral areas of cornea (oil droplet reflex). **6)** Munson's sign – bulging of lower lid when patient looks down. **7)** **Keratometric values are increased** {Normal value is 45 D} and based on it the severity of keratoconus is graded as: **mild** (< 48 D), **moderate** (48–54 D), and **severe** (>54 D). **8)** Corneal topography, i.e., study of shape of corneal surface, is the most sensitive method for detecting early keratoconus, Forme fruste refers to the earliest subclinical form of keratoconus detected on topography.

Morphological classification: Depending upon the size & shape of the cone, the keratoconus is of 3 types:

1) Nipple cone has a small size (<5 mm) and steep curvature.
2) Oval cone is larger (5–6 mm) and ellipsoid in shape.
3) Globus cone is very large (>6 mm) and globe like.



Complications: Rupture of Descemet's membrane → Acute hydrops ➔ sudden corneal oedema, marked defective vision, pain, photophobia and lacrimation

Associations of Keratoconus:

- Ocular conditions e.g., ectopia lentis, congenital cataract, aniridia, retinitis pigmentosa, and vernal keratoconjunctivitis (VKC).
- Systemic conditions e.g., Marfan's syndrome, atopy, Down's syndrome, Ehlers-Danlos syndrome, osteogenesis imperfecta and mitral valve prolapse.

Treatment options:

1. Spectacle correction may improve vision in early cases.
2. Contact lenses (rigid gas permeable) also improve the vision in early cases.
   ↳ In early to moderate cases a specially designed scleral contact lens (Rose-K) may be useful.
3. Intacs, the intracorneal ring segments, are also useful in early to moderate cases.
4. C3R – Corneal collagen cross linking with riboflavin and UV-A rays – to slow the progression of disease.
5. Keratoplasty is required in later stages.

---

5) Staphyloma [07]

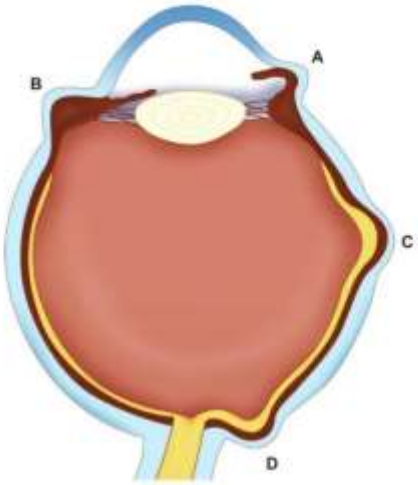
a. Types of Staphylomas [03]

Ans.

Staphyloma refers to a localised bulging of weak and thin outer tunic of the eyeball (cornea or sclera), lined by uveal tissue which shines through the thinned out fibrous coat.

Types: Anatomically, it can be divided into anterior, intercalary, ciliary, equatorial and posterior staphyloma.

1) **Anterior staphyloma**: An ectasia of pseudocornea (the scar formed from organised exudates and fibrous tissue covered with epithelium) which results after total sloughing of cornea, with iris plastered behind it is called anterior staphyloma.
2) **Intercalary staphyloma**: It refers to the localised bulge in limbal area lined internally by the root of iris and the anterior most part of ciliary body. It results due to ectasia of weak scar tissue formed at the limbus, following healing of a perforating injury or a peripheral corneal ulcer.
3) **Ciliary staphyloma**: As the name implies, it is the bulge of weak sclera lined by the ciliary body.
   - Its common causes are thinning of sclera after a perforating injury, scleritis, developmental glaucoma and end stage primary or secondary glaucoma.



Staphylomas (diagrammatic depiction) A, Intercalary; B, Ciliary; C, Equatorial; D, Posterior

4) **Equatorial staphyloma**: it results due to bulge of sclera lined by the choroid in the equatorial region.
   - Its causes are scleritis and degeneration of sclera in pathological myopia and chronic uncontrolled glaucoma.
5) **Posterior staphyloma**. It refers to bulge of weak sclera lined by the choroid behind the equator.
   - Here again the common causes are pathological myopia (most common cause), posterior scleritis and perforating injuries. It is diagnosed on ophthalmoscopy.

---

6) Fungal Keratitis [05]

Ans.

Etiology

- **Causative Organisms**:
  - Filamentous fungi: e.g.
    - Septate fungi: Aspergillus, Fusarium, Alternaria, Penicillium etc.
    - Non-septate fungi: Mucor and Rhizopus.
  - Yeasts e.g., Candida and Cryptococcus.
  - Dimorphic fungi – histoplasma, coccidioides and Blastomyces
  - Fungi responsible for mycotic corneal ulcers: Aspergillus (most common), Candida & Fusarium.
- **Modes of infection**
  - Injury by vegetative material such as crop leaf, branch of a tree etc. – seen in field workers especially during harvesting season.
  - Injury by animal tail
  - Secondary fungal ulcers are common in patients who are immunosuppressed systemically or locally such as patients suffering from dry eye, herpetic keratitis, bullous keratopathy or postoperative cases of keratoplasty.
- **Role of antibiotics and steroids**: Antibiotics disturb the symbiosis between bacteria and fungi; and the steroids make the fungi facultative pathogens which are otherwise symbiotic saprophytes. Hence, excessive use of these drugs predisposes the patients to fungal infections.

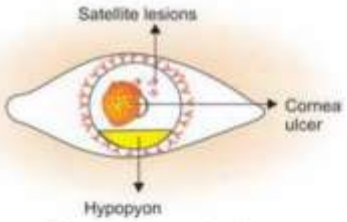
Clinical features

Symptoms are similar to the central bacterial corneal ulcer (refer 2nd LQ), but in general they are less marked than the equal-sized bacterial ulcer and the overall course is slow.

Signs.

- Corneal ulcer is dry-looking, greyish white, with elevated rolled out margins.
- Pigmented ulcer (brownish) can be seen in some species of fungi, e.g., dermatiaceous fungi.
- Feathery finger-like extensions are present into the surrounding stroma under the intact epithelium.
- Sterile immune ring (yellow line of demarcation) present where fungal antigen & host antibodies meet.
- Multiple, small satellite lesions may be present around the ulcer.
- Usually, a big hypopyon is present even if the ulcer is very small. Unlike bacterial ulcer, the hypopyon may not be sterile as the fungi can penetrate into the anterior chamber without perforation.
- Endothelial plaque, composed of fibrin and leucocytes, may be located under the stromal lesion.
- Perforation in mycotic ulcer is rare but can occur.
- Corneal vascularization is conspicuously absent in pure mycotic ulcer.

Diagnosis



Fungal corneal ulcer

1. Typical clinical manifestations associated with history of injury by vegetative material are highly suspicious of a mycotic corneal ulcer.
2. Chronic ulcer worsening inspite of most efficient treatment should arouse suspicion of mycotic involvement.
3. Lab investigations: include examination of wet KOH, Gram's and Giemsa-stained films for fungal hyphae and culture on Sabouraud's agar medium.
4. PCR for rapid results.

Treatment

- **Specific treatment with Antifungals**
  - Topical antifungal eyedrops should be used for a long period (6 to 8 weeks).
    - **Ex**: Natamycin (5%), Amphotericin B (0.1 to 0.3%), Fluconazole (0.2%) etc.
    - **Nystatin** (3.5%) eye ointment, five times a day is effective against Candida.
  - Intracorneal administration of voriconazole in cases with intraocular extension or anterior chamber involvement.
  - Systemic antifungal drugs – in severe cases of deeper fungal keratitis➔ Tablet fluconazole or ketoconazole or voriconazole may be given for 2–3 weeks.
- **Non-specific treatment** – similar to that of bacterial corneal ulcer (refer 2nd LQ).
- **Therapeutic penetrating keratoplasty** is done non-responsive cases.

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7) Exposure keratitis [05, 98]

Ans.

It occurs when eyes are covered insufficiently by the lids (Lagophthalmos).

Causes:

1. Extreme proptosis due to any cause will allow inadequate closure of lids.
2. Symblepharon causing lagophthalmos
3. Bell's palsy or any other cause of facial palsy.
4. Ectropion of severe degree.
5. Deep coma a/w inadequate closure of lids.
6. Physiological lagophthalmos: Lagophthalmos during sleep may occur in healthy individuals

Pathogenesis: Due to exposure the corneal epithelium dries up ➔ desiccation. After the epithelium is cast off, invasion by infective organisms may occur.

Clinical features

- Symptoms and signs of the causative disease are evident.
- Ocular irritation, burning, foreign body sensation and redness. Symptoms are gets worse in the morning.

❖ **Signs of exposure keratopathy are**:

- Drying of cornea
- There is absence of reflex blinking and defective closure of lids during sleep
- Punctate epithelial defects develop in the cornea
- Corneal ulceration may develop soon due to necrosis followed by bacterial superinfection.

Treatment

1) Prophylaxis: Once lagophthalmos is diagnosed following measures should be taken to prevent exposure keratitis.
   - Artificial tears should be instilled frequently.
   - Instillation of ointment and closure of lids by a tape during sleep.
   - Soft bandage contact lens with frequent instillation of artificial tears
   - Treatment of cause of exposure: e.g., proptosis, ectropion, symblepharon should be taken up.
2) Treatment of the corneal ulcer once developed.
3) Tarsorrhaphy is required when it is not possible to treat the cause.

---

8) Neuroparalytic keratitis [2000]

Ans.

There is loss of corneal sensation which results in the formation of corneal ulcer.

- **Etiology**: There is 5th nerve (trigeminal nerve) paralysis → It occurs typically as a result of injecting alcohol in gasserian ganglion in cases of trigeminal neuralgia.
- **Symptom**: It is a painless condition due to corneal anaesthesia.
- **Signs**: The characteristic feature is desquamation of corneal epithelium.
  - Large corneal ulcers are seen due to peeling of the epithelium.
  - There is corneal anaesthesia
  - The stroma is cloudy and yellow often associated with hypopyon.
  - Ciliary congestion is marked.
- **Treatment options**
  - Treat the corneal ulcer on usual line of treatment. Special care is taken to protect the eye with an eye shield.
  - Artificial tears and eye ointment are applied to lubricate the cornea.
  - Closure of the lacrimal puncta may be done to conserve moisture.
  - Paramedian or lateral tarsorrhaphy is indicated in these cases.

---

9) Keratomalacia [94]

Ans.

Keratomalacia refers to corneal necrosis due to vitamin A deficiency➔ non-healing corneal ulcer

In this condition, there is no inflammatory reaction

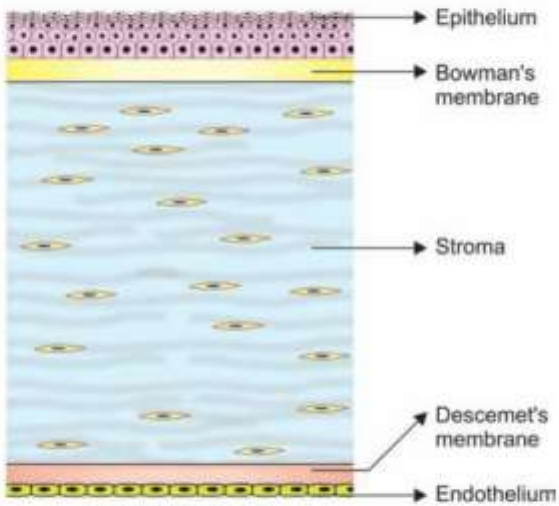
VSQs

1. Corneal endothelium [10]

Ans.

It is the innermost layer of cornea

- It consists of single layer of flattened hexagonal cells.
- It helps to maintain corneal transparency by
  - Active Sodium-Potassium-ATPase pump to maintain corneal dehydration
  - Tight junctions between adjacent endothelial cells – to prevent aqueous humour from entering cornea
- **Investigation done for endothelial cell count ➔** Specular microscopy.
- Normal endothelial cell count: 2400-3000 cells/ mm^2
- If endothelial cells are between 500-2400 per mm^2, cornea adapts by Polymegathism and Polymorphism.
- If endothelial cells are < 500 per mm^2, it leads to corneal decompensation (hazy, edematous cornea)



Structure of cornea

Diseases of Vitreous

SQs

1. Vitreous hemorrhage – causes, C/F & fate [16, 12, 06]

Ans.

Vitreous haemorrhage occurs from the retinal vessels and may present as preretinal (subhyaloid) or an intragel haemorrhage.

Causes:

1. Retinal tear, PVD (posterior vitreous detachment) and RD.
2. Trauma to eye.
3. Inflammatory diseases – Acute chorioretinitis, Eales' disease, or secondary to uveitis.
4. Vascular disorders, e.g., hypertensive retinopathy, and central retinal vein occlusion.
5. Metabolic diseases – ex: diabetic retinopathy.
6. Exudative age-related macular degeneration.
7. Blood dyscrasias, e.g., retinopathy of anaemia, leukaemias, polycythemia and sickle-cell.
8. Bleeding disorders, e.g., purpura, haemophilia and scurvy.
9. Neoplasms – rupture of vessels due to acute necrosis in tumours like retinoblastoma and malignant melanoma of choroid.
10. Other causes – Coat's diseases, radiation retinopathy, retinal capillary aneurysm.

Clinical features

Symptoms	Signs
◈ Floaters of sudden onset (if vitreous haemorrhage is small). ◈ Sudden painless loss of vision occurs in massive vitreous haemorrhage	1) Distant direct ophthalmoscopy reveals black shadows against the red glow in small haemorrhages and no red glow in a large haemorrhage. 2) Direct & indirect ophthalmoscopy – show blood in the vitreous cavity in small vitreous haemorrhage and non-visualization of fundus in large vitreous haemorrhage. 3) Slit-lamp examination shows a reddish mass in the vitreous. 4) Ultrasonography with B-scan is helpful in diagnosing vitreous haemorrhage

Fate of vitreous haemorrhage

1. Complete absorption may occur without organization
2. Organization of haemorrhage with formation of a yellowish-white debris occurs in persistent or recurrent bleeding.
3. Complications like vitreous liquefaction, degeneration & khaki cell glaucoma (in aphakia) may occur.
4. Retinitis proliferans may occur which may be complicated by tractional retinal detachment.

Treatment

1) **Conservative treatment**: bed rest & elevation of patient's head – (to allow blood to settle down).
2) **Treatment of the cause**: Once the blood settles down, indirect ophthalmoscopy should be performed to locate and manage the causative lesion.
3) **Vitrectomy by pars plana route** should be considered to clear the vitreous, if the haemorrhage is not absorbed after 3 months.

VSQs

1. At macula [05]

Ans.

The macula is situated at the posterior pole with its centre (foveola) being lateral to temporal margin of disc. Normal macula is slightly darker than the surrounding retina. Its centre imparts a bright reflex (foveal reflex).

Following abnormalities may be seen at the macula:

1) **Macular hole** – It looks red in colour with punched-out margins.
2) **Macular haemorrhage** is red and round.
3) **Cherry red spot** is seen in central retinal artery occlusion, Tay-Sach's disease, Niemann-Pick's disease, Gaucher's disease and Berlin's oedema.
4) **Macular oedema** may occur due to trauma, intraocular operations, uveitis & diabetic maculopathy.
5) **Pigmentary disturbances** may be seen after trauma, solar burn, age-related macular degeneration (ARMD), central chorioretinitis and chloroquine toxicity.
6) **Bull's eye macular lesions** are seen in ARMD, Stargardt disease, chloroquine retinopathy and cone dystrophy.
7) **Hard exudates –** may be seen in hypertensive retinopathy, exudative diabetic maculopathy, Coat's disease, CNVM.
8) **Macular scarring –** It may occur following trauma and disciform macular degeneration.

---

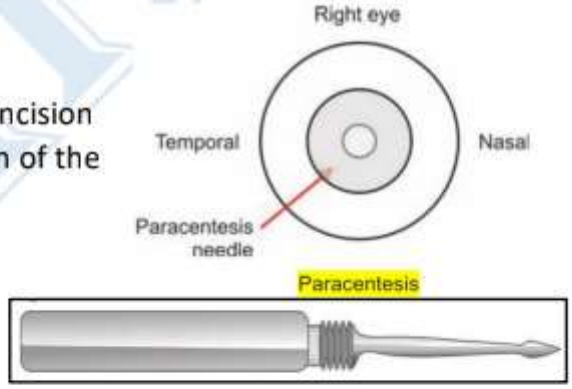
2. Paracentesis {incl it's indications} [05, 03]

Ans.

Aqueous is slowly released from the anterior chamber by an incision on cornea via paracentesis needle. This improves the nutrition of the cornea

Indications

- Hypopyon ulcer or hyphaema with raised tension.
- Impending perforation of corneal ulcer (descemetocele).



CONTENTS

Diseases of Sclera

SQs

1) Episcleritis [14, 04]

a. Clinical features and treatment of Episcleritis [17]

Ans.

Episcleritis is benign, recurrent, inflammation of the episclera, involving the overlying Tenon's capsule but not the underlying sclera.

Etiology

1. Idiopathic.
2. Can be a/w –
   - → Systemic diseases like gout, rosacea, psoriasis and connective tissue diseases.
   - → Infections like herpes zoster virus, syphilis, Lyme disease and TB

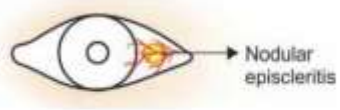
Pathology: Lymphocytic infiltration of episcleral tissue → oedema & congestion of Tenon's capsule and conjunctiva.

Clinical features

Symptoms	Signs
Redness, mild ocular discomfort (foreign body sensation). Rarely, photophobia & lacrimation may occur	On examination 2 clinical types of episcleritis may be recognised. 1. **Simple episcleritis** is sectorial inflammation of episclera. 2. **Nodular episcleritis** is characterised by a pink or purple nodule which is firm, tender, can be moved separately from the sclera and the overlying conjunctiva also moves freely

Differential diagnosis

- Simple episcleritis may be confused rarely with conjunctivitis.
- Nodular episcleritis may be confused with inflamed pinguecula, foreign body lodged in bulbar conjunctiva and, very rarely with scleritis.



Treatment

1. Topical NSAIDs, e.g., ketorolac 0.3%.
2. Topical mild corticosteroid eye drops, e.g., fluorometholone instilled 2- 3 hourly
3. Topical artificial tears, e.g., 0.5% carboxy methyl cellulose have soothing effect.
4. Cold compresses applied to the closed lids may offer symptomatic relief.
5. Systemic NSAIDs e.g., indomethacin (25 mg t.i.d.) – for recurrent cases.

---

2) Scleritis [99, 97]

Ans.

Scleritis refers to inflammation of the sclera proper.

Etiology

1) Idiopathic

2) Almost 50% cases of scleritis are associated with systemic diseases like: –

a. *Autoimmune collagen disorders* - rheumatoid arthritis (MC), Wegener's granulomatosis, SLE etc.

b. *Metabolic disorders* – gout & thyrotoxicosis

c. *Infections* → herpes zoster ophthalmicus, staphylococcal and streptococcal infection.

d. *Granulomatous diseases* → TB, syphilis, sarcoidosis, leprosy.

e. Miscellaneous conditions like irradiation, chemical burns, Bechet's disease and rosacea.

f. Surgically induced scleritis (SIS) is a rare complication of ocular surgery.

Pathology : It is a chronic granulomatous disorder characterised by fibrinoid necrosis, destruction of collagen with infiltration by PMNs, lymphocytes, plasma cells and macrophages. The granuloma is surrounded by multinucleated epithelioid giant cells & some of them may show evidence of vasculitis.

Classification of Scleritis		
A. Non-infectious scleritis	**I. Anterior scleritis (98%)**	a. Non-necrotizing scleritis (85%) – can be diffuse or nodular
		b. Necrotizing scleritis (13%) ± Inflammation
	II. Posterior scleritis (2%)	

B. Infectious scleritis – 5–10% of all cases

Clinical features

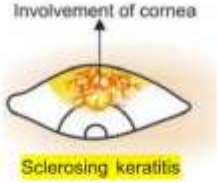

Necrotizing scleritis

Symptoms

- **Ocular Pain**: moderate to severe and often wakes the patient early in the morning. Radiates to the jaw and temple.
- Redness may be localized or diffuse.
- Photophobia and lacrimation.
- Diminution of vision may occur occasionally.

Signs of Infectious scleritis:		
Purulent exudates, painful nodules, Formation of fistulae, conjunctival & scleral ulcers		
Signs of Non-infectious scleritis		
Anterior scleritis	**Non–necrotizing anterior scleritis**	1) Non-necrotizing anterior diffuse scleritis – widespread inflammation of the anterior sclera. 2) Non-necrotizing anterior nodular scleritis – 1 or 2 hard, purplish, immovable scleral nodules, usually situated near the limbus.
	Necrotizing anterior scleritis	1. Necrotizing Anterior scleritis with inflammation – intense localised inflammation a/w areas of infarction ➔ sclera becomes transparent & uveal tissue shine through it. 2. Necrotizing Anterior scleritis without inflammation (scleromalacia perforans) –typically occurs in elderly females with longstanding rheumatoid arthritis.
Posterior scleritis		It is an inflammation of the sclera behind the equator. a/w inflammation of adjacent structures like exudative retinal detachment, macular oedema etc.

Complications: These are common with necrotizing scleritis and include sclerosing keratitis, keratolysis, complicated cataract and secondary glaucoma.



Sclerosing keratitis

Investigations – Following laboratory studies may be helpful in identifying associated systemic diseases:

1. TLC, DLC and ESR.
2. Serum levels of complement (C3), immune complexes, rheumatoid factor, antinuclear antibodies and L.E cells for an immunological survey.
3. FTA–ABS, VDRL for syphilis.
4. Serum uric acid for gout.
5. Urine analysis.

6. Mantoux test.
7. X-rays of chest, paranasal sinuses, sacroiliac joint and orbit (to rule out foreign body especially in patients with nodular scleritis).

Treatment

A. Non-infectious scleritis	1. Non-necrotizing scleritis	a. **Topical steroid eyedrops** b. **Systemic indomethacin 75 mg BD**
	2. Necrotizing scleritis	1) Topical steroids 2) Oral steroids on heavy doses, tapered slowly. 3) Immunosuppressive agents like methotrexate or cyclophosphamide may be required in nonresponsive cases. 4) Surgical treatment, in the form of scleral patch graft may be required to preserve integrity of the globe in extensive scleral melt and thinning.
B. Infectious scleritis		↳ Most of the time diagnosis is delayed and patients are put on topical and oral steroids which worsen the infective scleritis. ↳ Topical + oral Abx is required in an aggressive manner. ↳ Surgical debridement – by debulking the infected scleral tissue. It also facilitates the effect of antibiotics.

Diseases of Uveal Tract

LQs

1. Anterior uveitis – Etiology, clinical features, complications & management [15, 05]
   a. Seclusio Pupillae [15]; Festooned pupil [14]
   b. Keratic precipitates [14, 11, 09]
   c. Aqueous flare [14]; Flare [07, 04]
   d. Acute iridocyclitis – etiology, clinical picture, Management & complications [13, 10]
   e. Granulomatous uveitis – etiology, signs, symptoms and management [02]
   f. Iris nodules [02]

Ans.

Anterior uveitis: It is inflammation of the uveal tissue from iris up to pars plicata of ciliary body. It may be subdivided into:

- Iritis – Inflammation predominantly affects the iris.
- Iridocyclitis – Iris & pars plicata part of ciliary body are *equally* involved, and
- Anterior cyclitis – pars plicata part of ciliary body is predominantly affected.

Etiology – [proposed by Duke Elder]

1) **Infective uveitis**: Uveal infections may be exogenous, secondary or endogenous.
   - Exogenous infection – ex: from penetrating injuries, perforation of corneal ulcer etc.
   - Secondary infection –from neighbouring structures, e.g., acute conjunctivitis, keratitis, scleritis, etc.
   - Endogenous infections are caused by the organisms situated elsewhere in the body.

   ➢ Types of infectious uveitis:
   a. Bacterial – These may be granulomatous, e.g., TB, leprosy, syphilitic, brucellosis or pyogenic such as streptococci, staphylococci, pneumococci and gonococcus.
   b. Viral – herpes simplex, herpes zoster and CMV.
   c. Fungal – aspergillosis, candidiasis, blastomycosis.
   d. Parasitic – toxoplasmosis, toxocariasis, onchocerciasis and amoebiasis.
   e. Rickettsial – scrub typhus & epidemic typhus.
2) **Immune related uveitis**: It may be caused by Microbial allergy (TB, streptococci etc.), anaphylactic uveitis, Atopic uveitis, autoimmune uveitis & HLA-associated uveitis.
3) **Toxic uveitis:**
   - Endotoxins – produced inside the body as in pneumococcal or gonococcal infections
   - Endocular toxins – Ex: in patients with blind eyes, intraocular haemorrhage
   - Exogenous toxins – Ex: chemical substances of inorganic, animal or vegetative origin.
4) **Traumatic uveitis** – seen in accidental or operative injuries to the uveal tissue.
5) **Uveitis associated with systemic diseases** like DM, gout, Sarocoidosis, PAN, rheumatoid arthritis etc.
6) **Idiopathic uveitis** – Ex: Fuchs' heterochromic iridocyclitis.

Clinical Features

Symptoms

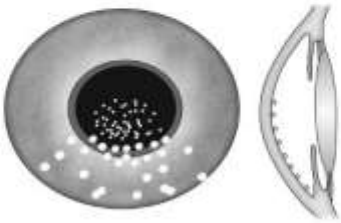
1) Pain – dull aching throbbing sensation which is typically worse at night; referred along the distribution of 5th nerve, especially towards forehead and scalp.
2) Redness – occurs due to circumcorneal congestion
3) Photophobia and blepharospasm – occurs due to a reflex between sensory fibres of 5th nerve (which are irritated) and motor fibres of the 7th nerve, supplying the orbicularis oculi muscle.

4) Lacrimation occurs due to lacrimatory reflex mediated by 5th nerve (afferent) and secretomotor fibres of the 7th nerve (efferent).

5) Defective vision – it can be because of induced myopia due to ciliary spasm, corneal haze (due to oedema & KPs), pupillary block due to exudates, complicated cataract, vitreous haze, etc.

Signs - Slit-lamp examination reveals:

1. **Lid** oedema
2. **Circumcorneal congestion** is marked in acute cases and minimal in chronic cases.
3. **Corneal signs**: Corneal oedema, KPs & posterior corneal opacities.

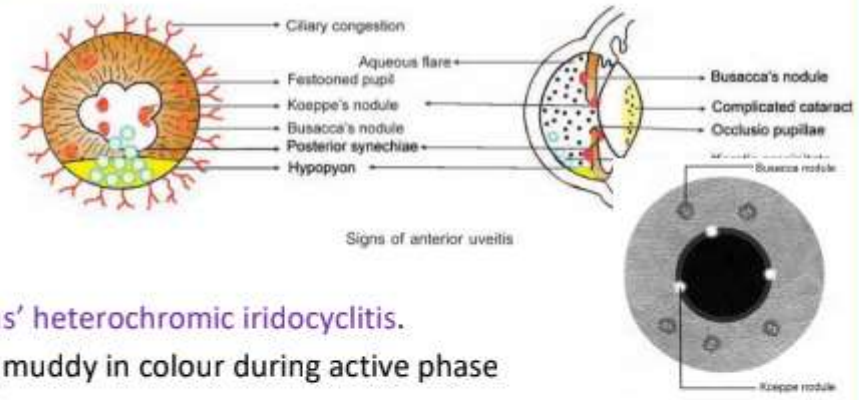


Keratic precipitates. Mutton fat KP's at the bottom, small and medium KP's above

- Keratic precipitates (KPs) are proteinaceous cellular deposits occurring at the back of cornea. The composition and morphology of KPs varies with the type of uveitis. Types of KPs:

Types of KPs	Seen in	Composed of	Description
1) **Mutton fat KPs**	Granulomatous uveitis	Epithelioid cells and macrophages	They are large, thick, fluffy, lardaceous KPs, having a greasy or waxy appearance
2) **Small & medium KPs**	Non-granulomatous uveitis	Lymphocytes	These small, discrete, dirty white KPs are arranged irregularly
3) **Old KPs**	Sign of healed uveitis		
4) **Fine KPs, aka 'stellate' KPs**	Fuch's heterochromic iridocyclitis, Herpetic iritis and CMV retinitis	They cover entire corneal endothelium (Endothelial dusting)	

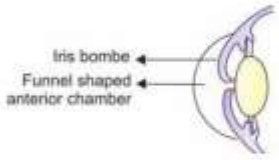
4. **Anterior chamber signs**
   - Aqueous cells – should be counted in an oblique slit-lamp beam
   - Aqueous flare – earliest sign of acute anterior uveitis – occurs due to leakage of protein into the aqueous humour from damaged blood vessels.
   - Hypopyon – When exudates are heavy and thick, they settle down in lower part of the anterior chamber as hypopyon (sterile pus in the anterior chamber)
   - Haemorrhagic hypopyon is a feature of uveitis a/w herpetic infection, trauma & rubeosis iridis.
   - Changes in depth & shape of anterior chamber – occur due to synechiae formation. Ex: Funnel-shaped in annular synechiae with iris bombe.
   - Changes in the angle of anterior chamber are observed with gonioscopic examination. In active stage, cellular deposits and in chronic stage peripheral anterior synechiae may be seen.



Signs of anterior uveitis

5. **Iris signs**
   - Loss of normal pattern –occurs due to oedema of iris in active phase and due to atrophic changes in chronic phase. Iris atrophy is typically observed in Fuchs' heterochromic iridocyclitis.
   - Changes in iris colour - Iris becomes muddy in colour during active phase
   - Iris nodules - seen typically in granulomatous uveitis.
     - Koeppe's nodules are situated at the pupillary border and may initiate posterior synechia.
     - Busacca's nodules are larger and located away from the pupil.

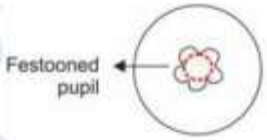
- Posterior synechiae - between the posterior surface of iris and anterior capsule of crystalline lens
  - » Annular (ring) posterior synechiae are 360° adhesions of pupillary margin to anterior capsule of lens → funnel-shaped Anterior chamber → impair circulation of aqueous humour from posterior chamber to anterior chamber (seclusio pupillae/ Festooned pupil). Thus, aqueous collects behind the iris and pushes it anteriorly (leading to 'iris-bombe' formation) → ↑ IOP



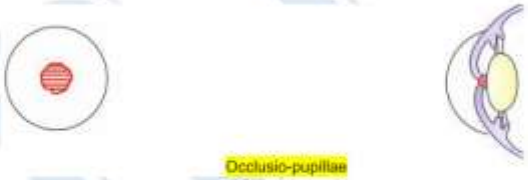
- Neovascularisation of iris (rubeosis iridis) - seen in chronic cases & in Fuch's heterochromic iridocyclitis.

6. Pupillary signs

- Narrow pupil - occurs due to Iris oedema & irritation of sphincter pupillae by toxins.
- Irregular pupil shape – (Festooned pupil) – occurs due to posterior synechiae. Dilatation of pupil with mydriatics (e.g., atropine) looks like festive paper decoration



- Ectropion pupillae (eversion of pupillary margin).
- Sluggish Pupillary reaction – due to oedema and hyperaemia of iris which hamper pupil movements.
- Occlusio pupillae – occurs if the pupil is completely occluded due to organisation of exudates across the entire pupillary area.



7. Changes in the lens

- Pigment dispersal on the anterior capsule of lens
- Exudates deposit on the lens in cases with acute plastic iridocyclitis → cyclitic membrane

8. Changes in the vitreous and retina

- Exudates & inflammatory cells seen in the anterior vitreous
- Cystoid macular oedema (CME) may occur in chronic cases.

Complications

1. Complicated cataract – 'polychromatic lustre' & 'bread-crumb' appearance
2. Secondary glaucoma.
   - » Early glaucoma (hypertensive uveitis)– occurs due to clogging of trabecular meshwork because of exudates and inflammatory cells in the anterior chamber
   - » Late glaucoma (post-inflammatory glaucoma) is the result of pupil block (seclusio pupillae due to ring synechiae formation, or occlusio pupillae due to organised exudates) not allowing the aqueous to flow from posterior to anterior chamber.
3. Cyclitic membrane – occurs due to deposition of exudates on the lens.

4. Choroiditis.
5. Retinal complications: CME, macular scar, macular hole, epiretinal membrane, exudative retinal detachment, retinal scars and sub-retinal fibrosis.
6. Papillitis (inflammation of the optic disc) – seen in severe cases
7. Band-shaped keratopathy – seen in chronic cases, especially in children with Still's disease.
8. Phthisis bulbi – end result of any form of chronic uveitis in which the eye becomes soft, shrink and atrophic.

Investigations

- Haematological investigations: TLC, DLC, ESR, Blood sugar levels, Blood uric acid. Serological tests for syphilis, toxoplasmosis, antinuclear antibodies, Rh factor, LE cells, C-reactive proteins, ACE (for sarcoidosis).
- Urine examination for WBCs, pus cells, RBCs and culture to rule out UTI.

- Stool examination for cyst and ova to rule out parasitic infestations.
- Radiological investigations:
  - X-rays of chest, paranasal sinuses, sacroiliac joints and lumbar spine.
  - CT scan of thorax should be considered for suspected sarcoidosis cases.
  - MRI scan of head for suspected sarcoidosis, demyelination and lymphomas.
- Skin tests: tuberculin test, Kveim's test for sarcoidosis, lepromin test and pathergy test for Behcet's disease.

Treatment

- **Specific treatment of the cause** – for Ex: antitubercular drugs for TB etc.
- **Nonspecific treatment**

Local therapy	1. Cycloplegic drugs – Ex: 1% atropine sulfate eye ointment or drops – it relieves the spasm of iris sphincter, prevents the formation of synechiae & increases the blood supply to anterior uvea 2. Corticosteroids – Ex: dexamethasone, betamethasone etc. – anti-inflammatory effect 3. Broad spectrum Abx drops - are usually prescribed with topical steroid preparations to provide an umbrella cover for them.
Systemic therapy	1) Corticosteroids – esp. in non-granulomatous uveitis & other cases resistant to topical therapy. Ex: prednisolone (60–100 mg) Drawback: Steroids (both topical and systemic) may cause many ocular (e.g., steroid-induced glaucoma and cataract) and systemic side-effects 2) NSAIDs ↳ Aspirin can be used where steroids are contraindicated ↳ Phenylbutazone – used if uveitis is a/w rheumatoid disease 3) Immunosuppressive drugs – used in severe cases with imminent danger of blindness – Ex: cyclophosphamide, chlorambucil, azathioprine and methotrexate 4) Azithromycin or tetracycline or erythromycin – used to treat chlamydial infection in patients and their sexual partners with Reiter's syndrome
Physical measures	→ Hot fomentation - It is very soothing, diminishes pain and increases circulation. As a result, more antibodies are brought and toxins are drained. → Dark goggles - used in sunlight to ↓ photophobia, lacrimation and blepharospasm

- **Treatment of complications**
  - Inflammatory glaucoma (hypertensive uveitis): 0.5% timolol maleate eyedrops + usual treatment of iridocyclitis. Pilocarpine and latanoprost eye drops are contraindicated in inflammatory glaucoma.
  - Post-inflammatory glaucoma due to ring synechiae is treated by laser iridotomy.
  - Complicated cataract requires lens extraction
  - Exudative Retinal detachment usually settles itself if uveitis is treated aggressively. A tractional detachment requires vitrectomy
  - Phthisis bulbi especially when painful, requires removal by enucleation operation.

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2. Describe the aetiology, clinical features and management of red eye [11, 07]
   a. Differential diagnosis and treatment for Red Eye [05]

Ans.

	Feature	Acute conjunctivitis	Acute iridocyclitis	Acute congestive glaucoma
1.	Onset	Gradual	Usually gradual	Sudden
2.	Pain	Mild discomfort	Moderate in eye and along the first division of trigeminal nerve	Severe in eye and the entire trigeminal area
3.	Discharge	Mucopurulent	Watery	Watery
4.	Coloured halos	May be present	Absent	Present
5.	Vision	Good	Slightly impaired	Markedly impaired
6.	Congestion	Superficial conjunctival	Deep ciliary	Deep ciliary
7.	Tenderness	Absent	Marked	Marked
8.	Pupil	Normal	Small and irregular	Large and vertically oval
9.	Media	Clear	Hazy due to KPs, aqueous flare and pupillary exudates	Hazy due to oedematous cornea
10.	Anterior chamber	Normal	May be deep	Very shallow
11.	Iris	Normal	Muddy	Oedematous
12.	Intraocular pressure	Normal	Usually normal	Raised
13.	Constitutional symptoms	Absent	Little	Prostration and vomiting

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SQs

1) Endophthalmitis [13]

Ans.

Endophthalmitis is defined as an inflammation of the inner structures of the eyeball, i.e., uveal tissue and retina a/w pouring of exudates in the vitreous cavity, anterior chamber & posterior chamber.

Etiology

- **Infective endophthalmitis**
  - *Modes of infection*
    1) Exogenous infections – from perforating injuries, perforation of corneal ulcers etc.
    2) Secondary infection –from neighbouring structures – Ex: orbital cellulitis, infected corneal ulcers etc.
    3) Endogenous infections are caused by the organisms situated elsewhere in the body
  - *Causative organisms*
    1. Bacterial – MC is Gram +ve cocci, i.e., Staphylococcus epidermidis and Staphylococcus aureus.
       - Others: Streptococci, Pseudomonas, Pneumococci, Corynebacterium, Propionibacterium acnes and Actinomyces
    2. Fungal endophthalmitis (rare) – is caused by Aspergillus, Fusarium, Candida, etc.
- **Non-infective (sterile) endophthalmitis** – Results from toxins released in following situations:
  1) Postoperative – Ex: Chemicals adherent to intraocular lens (IOL) or instruments.
  2) Post-traumatic – Ex: retained intraocular foreign body.
  3) Phacoanaphylactic – induced by lens proteins in patients with Morgagnian cataract.
  4) Intraocular tumour necrosis may present as sterile endophthalmitis (masquerade syndrome).

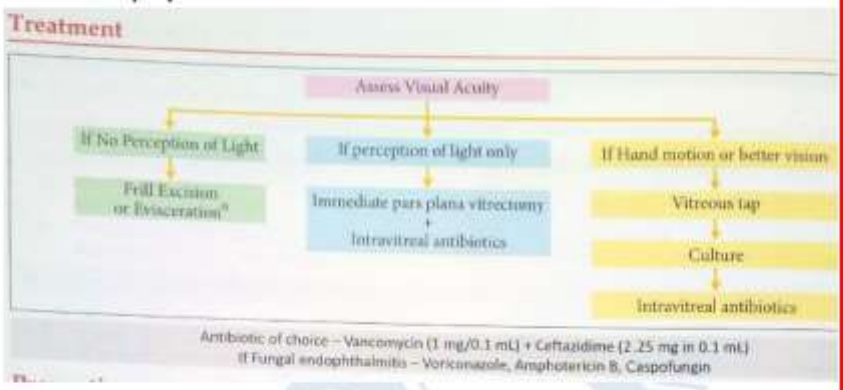
Clinical feature of bacterial endophthalmitis – Onset may be acute or delayed

- *Acute onset* - between 1–7 days of operation.
- *Delayed onset* - a week to month after surgery. Fungi are the most common cause

➤ **Symptoms:** severe ocular pain, redness, lacrimation, photophobia and loss of vision.

➤ **Signs**:

1. **Lids** become red and swollen.
2. **Conjunctiva** shows chemosis and marked circumcorneal congestion.
3. **Cornea** is oedematous, cloudy and ring infiltration may be formed.
4. In exogenous form, Edges of wound become yellow and necrotic.
5. **Anterior chamber** shows hypopyon→ iris and pupil details are not seen.
6. **Iris**, when visible, is oedematous and muddy.
7. **Pupil shows yellow reflex** due to purulent exudation in vitreous.
8. **Vitreous exudation:** vitreous cavity is filled with exudation and pus. Soon a yellowish white mass is seen through fixed dilated pupil. This sign is called **amaurotic cat's-eye reflex**.



9. **IOP** is raised in early stages, but in severe cases, the ciliary processes are destroyed, and a fall in IOP may ultimately result in shrinkage of the globe.

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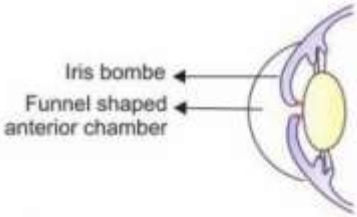
2) Iris bombe [12, 11, 91]

Ans. The iris bow forwards due to collection of aqueous in the posterior chamber.

Physiological iris bombe—on dilatation of the pupil there is crowding of the iris in the angle of anterior chamber causing obstruction to the flow of aqueous from the posterior to the anterior chamber



Pathological causes: as a complication of Anterior Uveitis

Complications: Post-inflammatory glaucoma

DDx: Cyst of posterior epithelium

Treatment: YAG laser iridotomy is the treatment of choice to prevent secondary glaucoma

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3) Panophthalmitis [04]

Ans. It is an intense purulent inflammation of the whole eyeball including the Tenon's capsule.

Etiology

- Panophthalmitis is an acute bacterial infection.
- Mode of infection and causative organisms are same as described for bacterial endophthalmitis

Clinical features

Symptoms	Signs
▪ Severe ocular pain and headache, ▪ Complete loss of vision, ▪ Profuse watering, ▪ Purulent discharge, ▪ Marked redness and swelling of the eyes, and	1) *Lids* show a marked oedema and hyperaemia. 2) *Eyeball* is slightly proptosed, ocular movements are limited and painful. 3) *Conjunctiva* shows marked chemosis and ciliary as well as conjunctival congestion. 4) *Cornea* is cloudy and oedematous. 5) *Anterior chamber* is full of pus. 6) *Vision* is completely lost and perception of light is absent. 7) *IOP* is markedly raised.

Panophthalmitis

▪ Constitutional symptoms like malaise and fever.	8) *Globe perforation* may occur at limbus, pus comes out and intraocular pressure falls.

Complications: Orbital cellulitis, cavernous sinus thrombosis, and Meningitis or encephalitis

Treatment

1) *Anti-inflammatory* and analgesics should be started immediately to relieve pain.
2) *Broad spectrum* antibiotics should be administered to prevent further spread of infection in the surrounding structures.
3) *Evisceration* operation should be performed to avoid intracranial dissemination of infection.

VSQs

1. Hyphema [14]

Ans.

Collection of blood in the anterior chamber is called Hyphema.

Causes of Hyphema – HOTS
H erpetic uveitis
O phthalmia nodosa
T rauma
S yphilis

Treatment:

- Most hyphaemas absorb spontaneously and thus need no treatment.
- Acetazolamide and hyperosmotic agents can be given to ↓ IOP.
- If the blood does not get absorbed in a week's time, then a paracentesis should be done to drain the blood.

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2. Circulus arteriosus iridis major& minor [13]

Ans.

- Anterior ciliary arteries pierce the sclera near the limbus to enter the ciliary muscle; where they anastomose with the two long posterior ciliary arteries to form the **circulus arteriosus major**, near the root of iris.
- Branches arise from the **circulus arteriosus major** to supply the ciliary processes (1 branch for each process) & **form circulus arteriosus minor** which supply the pupillary margins & some central part of iris

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3. Hard Exudates [05]

Ans. Hard exudates are lipid deposits in the outer plexiform layer of retina which occur following leaky capillaries in severe hypertensive retinopathy.

- They appear as yellowish waxy spots with sharp margins. They are generally seen in posterior pole and may be arranged as macular-fan or macular-star.
- They are temporary and may disappear in 3-6 weeks

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4. Nodule at the Limbus [04]

Ans. A limbal nodule is any nodular lesion at the limbus (junction of the cornea & sclera) of the eye.

DDx for a limbal nodule can include:

- Early Pterygium
- Foreign body / foreign body granuloma
- Phlycten, an inflamed nodule of lymphoid tissue
- Episcleritis & Scleritis
- Granuloma
- Limbal dermoid, Malignant melanoma, naevus etc.

CONTENTS

Diseases of Lens

LQs

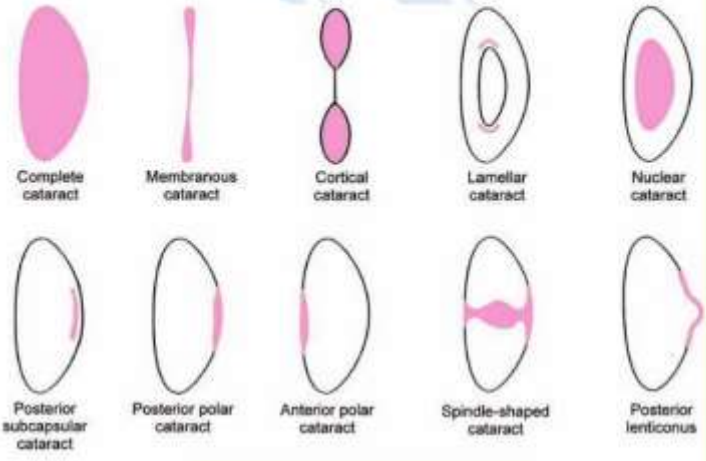
1) Congenital cataract – Classification, clinical features, Dx and management [07, 03]
   a. Zonular cataract [16]
   b. Lamellar cataract [15, 02]

Ans.

Congenital cataracts occur due to some disturbance in the normal growth of the lens before birth.

ETIOLOGY

1. **Idiopathic** - About one-third cases
2. **Hereditary** - About one-third cases
   - E.g., of familial cataracts: Zonular cataract, Coronary cataract, Cataracta pulverulenta etc.
   - Can occur with/without systemic disorders like cerebro-oculo-facial syndrome, Lowe's syndrome, Stickler syndrome etc.
3. **Maternal factors**
   - Malnutrition during pregnancy ➔ zonular cataract
   - Maternal infections like rubella are associated with cataract in 50% of cases
   - Ingestion of thalidomide, corticosteroids during pregnancy
   - Exposure to radiation during pregnancy
4. **Foetal factors**
   1) Anoxia due to placental haemorrhage.
   2) Birth trauma, may cause cataract.
   3) Metabolic disorders – galactosemia, galactokinase deficiency and neonatal hypoglycemia.
   4) Congenital anomalies e.g., as seen in Lowe's syndrome etc.



The various presentations of congenital cataract

Classification of Congenital Cataract	
A. Congenital capsular cataracts	1. Anterior capsular cataract 2. Posterior capsular cataract
B. Polar Cataracts	1) Anterior polar cataract 2) Posterior polar cataract
C. Congenital Nuclear cataracts	1. Cataracta pulverulenta 2. Lamellar cataract 3. Sutural and axial cataracts: Floriform cataract, Coralliform cataract, Anterior axial embryonic cataract & Dendritic suture cataract 4. Total nuclear cataract
D. Generalized cataracts	1) Coronary cataract 2) Blue dot cataract 3) Total congenital cataract 4) Congenital membranous cataract

CLINICAL FEATURES

A. Congenital capsular cataracts

- Anterior capsular cataracts are nonaxial, stationary and visually insignificant.
- Posterior capsular cataracts are rare and can be a/w persistent hyaloid artery remnants

B. Polar Cataracts – involve the poles of the lens

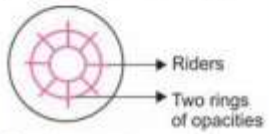
1. Anterior polar cataract: It arise either due to delayed development of anterior chamber or due to corneal perforation.
2. Posterior polar cataract: It is a very common lens anomaly & maybe a/w Persistent hyaloid artery remnants, Posterior lenticonus, and Persistent hyperplastic primary vitreous (PHPV).

C. Congenital Nuclear cataracts

a) **Cataracta centralis pulverulenta** – it is B/L & is characterised by a small rounded opacity lying in the centre of the lens. The opacity has a powdery appearance (pulverulenta) and usually does not affect the vision.

b) **Lamellar (Zonular) cataract** – it refers to the developmental cataract in which the opacity occupies a discrete zone in the lens. It is the MC type of congenital cataract presenting with visual impairment

- Etiology: It may be either genetic (autosomal dominant) or environmental (vit D deficiency, hypocalcaemia, maternal rubella infection).
- Characteristic features: 👉
  - Typically, this cataract occurs in a zone of foetal nucleus surrounding the embryonic nucleus
  - Small linear opacities like spokes of a wheel (riders) may be seen towards the equator.
  - It is usually B/L and causes severe visual defects



Zonular cataract

c) **Sutural and axial cataracts**:

- They are punctate opacities scattered around the Y-sutures and do not have much effect on the vision
- They vary in size and shape and have different pattern and thus are named accordingly as –
  1. Floriform cataract - the opacities are arranged like the petals of a flower
  2. Coralliform cataract – spindle- shaped opacity with off shoots resembling a coral
  3. Anterior axial embryonic cataract occurs as fine dot near the anterior Y-suture
  4. Dendritic sutural cataract occurs as fine dots along the dendritic sutures.

d) **Total nuclear cataract**: It involves the embryonic and fetal nucleus and sometimes infantile nucleus as well. It is characterized by B/L, dense chalky white central opacity seriously impairing vision.

D. Generalized cataracts

1. Coronary cataract
   - occur around puberty 👉
   - The opacities are many hundreds in number and have a regular radial distribution in the periphery of lens


Coronary cataract

2. Blue dot cataract: this is the MC type of congenital cataract. It usually forms in the first two decades of life. The opacities are in the form of rounded bluish dots situated in the periphery
3. Total congenital cataract – it can be due to heredity or rubella. In rubella, the child is born with a progressive 'pearly white' nuclear cataract
4. Congenital membranous cataract: Sometimes there may occur total or partial absorption of congenital cataract, leaving behind thin membranous cataract. This is a/w **Hallermann-Streiff-Francois Syndrome**

DIFFERENTIAL DIAGNOSIS: Congenital cataracts presenting with leukocoria need to be differentiated from other conditions presenting with leukocoria such as retinoblastoma, retinopathy of prematurity, persistent hyperplastic primary vitreous (PHPV), etc.

MANAGEMENT:

1) Clinico-investigative work up:

- Ocular examination – is done to know Density and morphology of cataract, assess visual function & note any associated ocular defects like microphthalmos, glaucoma, PHPV, optic nerve hypoplasia etc.
- Laboratory investigations – is done to detect:
  - Intrauterine infections by TORCH test
  - Galactosemia by urine test
  - Lowe's syndrome by urine chromatography for amino acids.
  - Hyperglycemia by blood sugar level
  - Hypocalcemia by serum calcium and phosphate levels and X-ray skull

2) **Prognostic factors** - Density of cataract, U/L or B/L, time of presentation & associated ocular or systemic defects

3) **Indications and timing of paediatric cataract surgery**
   - Partial cataracts & small central cataracts which are visually **insignificant** can safely be ignored
   - Bilateral dense cataracts should be removed within 6 weeks of birth to prevent amblyopia
   - Unilateral dense cataract should be removed as early as possible (within days) after birth

4) **Surgical Technique** – extra capsular cataract extraction involving anterior capsulorrhexis and lens aspiration or lensectomy is used.

5) **Correction of paediatric aphakia**
   - ⊙ Children > 2 years – Implant PCIOL during surgery.
   - ⊙ Children below 2 yrs – treated by extended wear contact lens. Later on, secondary IOL implantation may be done.

6) **Correction of amblyopia** – It can be done via occlusion therapy, Penalization, Pleoptic exercises etc.

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2) Senile Cortical Cataract – C/F & management [04, 03]
   - **a.** Local anaesthesia for cataract surgery [13]
   - **b.** Morgagnian hypermature cataract [13]
   - **c.** Four complications of cataract surgery [12]
   - **d.** Different etiology for cataract [07]

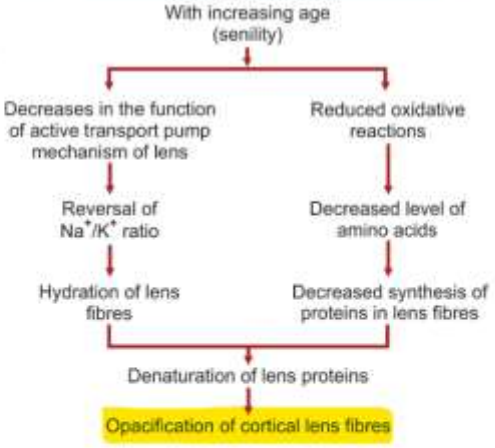
Ans.

❖ 'Age-related cataract' also called as senile cataract is the commonest type of acquired cataract affecting equally persons of either sex usually above the age of 50 years.

❖ Morphologically, the senile cataract occurs in 2 forms: the cortical (soft cataract) and the nuclear (hard cataract).

Risk Factors:

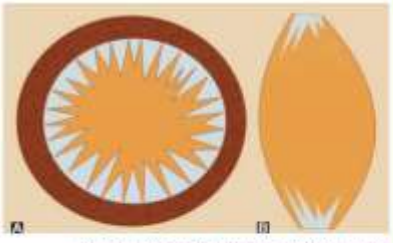
1. Age - usually occurs after the age of 50 years.
2. Heredity – can predispose to pre-senile cataract
3. Exposure to UV radiations from sunlight.
4. Diet deficient in proteins, amino acids, vitamins (riboflavin, Vit E, Vit C)
5. Dehydrational crisis - (due to diarrhoea, cholera, etc.)
6. Smoking.
7. Causes of pre-senile cataract: (before 50 years of age) ➔ Heredity, DM, Myotonic dystrophy & Atopic dermatitis



Mechanism of loss of transparency of Lens: 👉

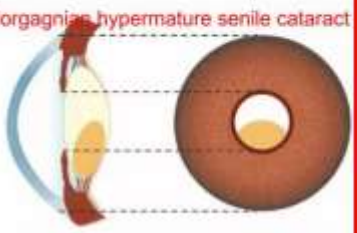
Stages of Maturation of Senile Cortical Cataract:

1. **Stage of lamellar separation** – earliest change is separation of cortical fibres by fluid.
2. **Stage of incipient cataract** – 2 types of senile cortical cataracts can be recognised at this stage:
   - **a)** Cuneiform senile cortical cataract – It is characterised by wedge-shaped opacities which starts at periphery and extends centrally → radial spoke-like pattern of greyish white opacities. 👉
   - **b)** Cupuliform senile cortical cataract – it is a saucer-shaped opacity develops just below the capsule in the posterior cortex (posterior subcapsular cataract), which gradually extends outwards.
3. **Immature senile cataract (ISC)**: opacification becomes more diffuse and irregular. Iris shadow is visible. In some patients, at this stage, lens may become swollen due to continued hydration. This condition is called '**intumescent cataract**'. Due to swollen lens anterior chamber becomes shallow. 👉



Diagrammatic depiction of immature senile cataract (cuneiform type): A, as seen by oblique illumination; B, in optical section with the beam of the slit-lamp

4. **Mature senile cataract (MSC):** In this stage, opacification becomes complete, i.e., whole of the cortex is involved. Lens becomes pearly white in colour. It is aka **'ripe cataract'**.
5. **Hypermature senile cataract (HMSC):** it may occur in any of the two forms:
   a. Morgagnian hypermature cataract: here, whole cortex liquefies & the lens is converted into a bag of milky fluid. The small brownish nucleus settles at the bottom.
   b. Sclerotic type hypermature cataract: here, cortex becomes disintegrated and the lens shrink due to leakage of water → anterior chamber becomes deep & iris becomes tremulous (iridodonesis).



Clinical features

Symptoms

1) Glare – intolerance of bright light; such as direct sunlight or the headlights of an oncoming motor vehicle.
2) Uniocular polyopia: It occurs due to irregular refraction by the lens
3) Coloured halos: they form because of breaking of white light into coloured spectrum due to presence of water droplets in the lens.
4) Black spots in front of eyes.
5) Image blur & distortion of images
6) **Deterioration of vision**: It is painless and gradually progressive in nature.

↳ Patients with central opacities (e.g., cupuliform cataract i.e., posterior subcapsular cataract) have early loss of vision. These patients see better when pupil is dilated due to dim light in the evening (Day blindness).

↳ Patients with peripheral opacities (e.g., cuneiform cataract) visual loss is delayed and the vision improves in bright light when pupil is contracted.

Signs of senile cataract

Examination	Nuclear cataract	ISC	MSC	HMSC (M)	HMSC (S)
1. Visual acuity	6/9 to PL+	6/9 to CF+	HM+ to PL+	PL+	PL+
2. Colour of lens	Grey, amber, brown, black or red	Greyish white	Pearly white with sinking brownish nucleus	Milky white	Dirty white with hyper white spots
3. Iris shadow	Not seen	Seen	Not seen	Not seen	Not seen
4. Distant direct ophthalmoscopy with dilated pupil	Central dark area against red fundal glow	Multiple dark areas against red fundal glow	No red glow but white pupil due to complete cataract	No red glow milky white pupil	No red glow dirty white pupil
5. Slit-lamp examination	Nuclear opacity clear cortex	Areas of normal with cataractous cortex	Complete cortex is cataractous	Milky white cortex with sunken brown-ish nucleus	Shrunken cataractous lens with thickened anterior capsule

ISC: Immature senile cataract, MSC: Mature senile cataract, HMSC (M) Hypermature senile cataract (Morgagnian), HMSC (S): Hypermature senile cataract (Sclerotic), PL: Perception of light, HM: Hand movements, CF: Counting finger

Differential diagnosis

1. Immature senile cataract (ISC) can be differentiated from the nuclear sclerosis
2. Mature senile cataract can be differentiated from retrolental causes of white pupillary reflex (leukocoria)

ISC	Nuclear sclerosis
1. Painless progressive loss of vision	1. Painless progressive loss of vision
2. Greyish colour of lens on oblique illumination examination	2. Greyish colour of lens
3. Iris shadow is present	3. Iris shadow is absent
4. Black spots against red glow are observed on distant direct ophthalmoscopy	4. No black spots are seen against red glow
5. Slit-lamp examination reveals area of cataractous cortex	5. Slit-lamp examination reveals clear lens with nuclear sclerosis
6. Visual acuity does not improve on pin-hole testing	6. Visual acuity usually improves on pin-hole testing

Complications

1) Phacoanaphylactic uveitis – Lens proteins leak into the anterior chamber in hypermature cataract ➔ may act as antigen and induce antigen-antibody reaction➔ Phacoanaphylactic uveitis.
2) Lens-induced glaucoma:
   a. Phacomorphic glaucoma (MC)– it is caused by intumescent (swollen) lens. It is a type of 2° angle closure glaucoma.
   b. Phacolytic glaucoma: Lens proteins are leaked into the anterior chamber in hypermature cataract→ proteins are engulfed by the macrophages → The swollen macrophages clog the trabecular meshwork → Phacolytic Glaucoma. It is a type of 2° open angle glaucoma.
   c. Phacotopic glaucoma: Hypermature cataractous lens may subluxate/dislocate and cause glaucoma by blocking the pupil or angle of anterior chamber.
3) Subluxation or dislocation of lens – occur due to degeneration of zonules in hypermature stage

MANAGEMENT OF CATARACT IN ADULTS:

Non-Surgical measures

- Treatment of cause of cataract – Ex: control of DM, stop cataractogenic drugs (corticosteroids, miotics etc.)
- Measures to improve vision – Ex: Prescription of glasses

Types of Cataract Surgeries:

1) Intra-Capsular Cataract Extraction (ICCE) - Complete lens with the capsule is removed
   - ⇢ Not done now
   - ⇢ Only current indication: Subluxation of lens due to >180° zonular dehiscence
2) Extra-Capsular Cataract Extraction (ECCE) – Lens is removed but not the capsule.
   - ↳ Techniques:
   - ↳ *Latest Technique* - Femtosecond Laser Assisted Cataract Surgery (FLACS)

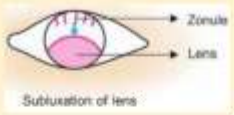
	Site of Incision	Size of Incision
a. Conventional ECCE	Limbus	8 – 10 mm
b. Small Incision Cataract Surgery (SICS)	Sclera	5 – 7 mm
c. Phacoemulsification	Cornea	2.2 – 3.2 mm

Local anaesthesia for cataract surgery:

- **Peribulbar injection**: Bupivacaine + Lignocaine + 1:200000 Adrenaline + Hyaluronidase.

Complications of Cataract Surgery:

Intra-op Complications	Acute Post-op Complications	Late Post-op Complications
1) Posterior Capsular rupture 2) UGH Syndrome (Uveitis, Glaucoma, Hyphema) 3) Expulsive Choroidal hemorrhage 4) Descemet's membrane detachment	• Shallow anterior chamber • Endophthalmitis	» PCO – Posterior Capsular Opacification: aka After Cataract/secondary cataract » Displacement of IOL 1) Superior IOL Dislocation - Sunrise Syndrome 2) Inferior IOL Dislocation - Sunset Syndrome 3) Complete Subluxation of Lens - Lost Lens Syndrome » Irvin Gas Syndrome » Anterior Capsular Phimosis



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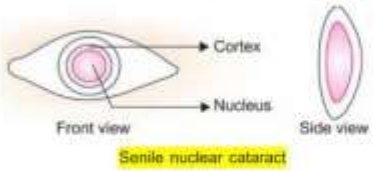
3) Discuss the signs, symptoms, diagnosis and management of nuclear cataract [02]

Ans. It is a type of Senile Cataract – (Refer 2nd LQ)

it occurs due to sclerosis, it causes hard cataract
↓
Increase in refractive index of lens
↓
Increase in power (converging of lens)
↓
Index myopia – loss of far vision or near sightedness leads to second sight

- Aka central cataract
- **Pathogenesis:**
- **Main Clinical Features:**
  - » Loss of vision in daytime: (pupils constrict in daytime=> light is blocked by the cataractous nucleus => LOV)
  - » Improvement of vision in dim light /night
  - » Second sight – Improvement of near vision due to nuclear cataract in a patient who is already presbyopic
- **Grading of Nuclear Cataract**: 4 Grades
  1. Grade 1 – yellow due to deposition of urochrome pigment ➔ C/F yellow coloured vision (xanthopsia)
  2. Grade 2 – Amber
  3. Grade 3 – Brown called as cataracta brunescens
  4. Grade 4 - Black called as cataracta nigra
- **Management of Nuclear Cataract** – (Refer 2nd LQ)



SQs

1) Aetiology and clinical features of complicated cataract [14]

Ans.

Complicated cataract refers to opacification of the lens 2° to some other intraocular disease

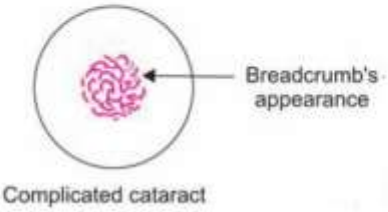
Aetiology – UMAR

» Chronic Anterior Uveitis (MCC)

» High Myopia

» Angle Closure Glaucoma

» Fundus dystrophies like Retinitis Pigmentosa



Complicated cataract

Clinical features

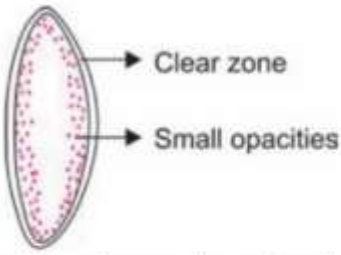
- Typically, the complicated cataract starts as posterior subcapsular cortical cataract (PSC).
- The opacity is irregular in outline and variable in density.
- In the beam of slit-lamp the opacities have:
  - **'Breadcrumb' appearance**.
  - **'Polychromatic lustre'** i.e., appearance of iridescent coloured particles of reds, greens and blue is a very characteristic sign (Rainbow cataract).
  - Diffuse yellow-haze is seen in the adjoining cortex.
  - Slowly the opacity spreads in the rest of the cortex, and finally the entire lens becomes opaque, giving dirty white or chalky white appearance.
  - Deposition of calcium is common in the later stages.

---

2) Traumatic Cataract [08]

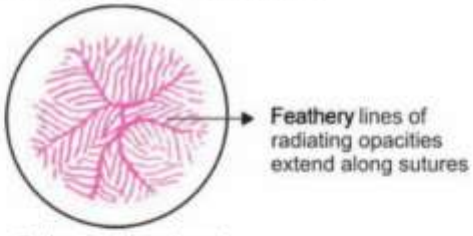
Ans.

- Aka Concussion cataract: It occurs mainly due to imbibition of aqueous and partly due to direct mechanical effects of the injury on lens fibres.
- It may assume any of the following shapes:
  - Discrete subepithelial opacities (MC).
  - Blunt Trauma causes Rosette Shaped Cataract
    - Early rosette cataract (punctate) – It appears as feathery lines of opacities along the star-shaped suture lines; usually in the posterior cortex
    - Late rosette cataract: It develops in the posterior cortex 1 to 2 years after the injury. Its sutural extensions are shorter and more compact than the early rosette cataract.



Punctate subcapsular cataract

  - Infrared Rays cause – glassblower's cataract – occurs due to true exfoliation of lens capsule.
  - Lightning/electric shock causes anterior capsular opacities
  - Traumatic zonular cataract.
  - Diffuse (total) concussion cataract.
  - Early maturation of senile cataract may follow blunt trauma.



Early rosette cataract

- **Treatment of traumatic cataract**: (Refer 2nd LQ)

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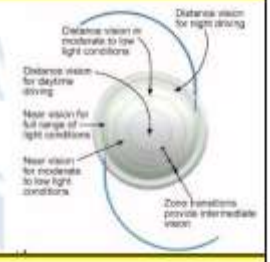
3) Intraocular lens [03, 98]

a. Types of Intraocular lenses [16]

Ans.

Types of IOL:

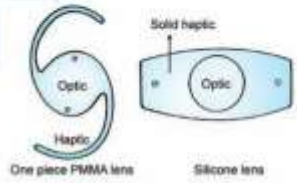
Based on the method of fixation in the eye	1) ACIOL (Angle supported IOLs) - made of PMMA (poly-methyl meth acrylate): Drawback: bullous keratopathy 2) Iris-supported lenses: These lenses are fixed on the iris with the help of sutures. These lenses are also not used due to high incidence of postoperative complications. Example of iris supported lens is Singh & Worst's iris claw lens 3) PCIOL – rest entirely behind the iris: made of Silicone or Acrylic – Ex: C-Shaped Haptic
Based on the material of manufacturing	▪ Rigid IOLs – made of PMMA ▪ Foldable IOLs - made of Silicone or Acrylic ▪ Rollable IOL - made of hydrogel
Based on the focussing ability	➤ Unifocal IOLs - Depending upon the power of IOLs implanted, these can make the patient emmetropic, myopic or hypermetropic. ➤ Multifocal IOLs - These are of 2 types, either refractive or diffractive optics types. These are also called pseudoaccommodative IOLs. ➤ Accommodative IOLs exhibit some anterior movement of optic to improve the near vision. Ex: Crystalens 
Special function IOLs	1) Toric IOL – in pre-existing astigmatism 2) Square edged IOL – To ↓ occurrence of Posterior capsular opacification (PCO). 3) Aniridia IOLs: These are devised to cosmetically cover the defects of aniridia

Calculation of IOL Power is known as Biometry

Formulae to Calculate IOL power: SRK-T, Holladay, Hoffer Q & Haigis-L

Indications of IOL implantation: All cases being operated for cataract

Best Site for IOL implantation – in the Capsular Bag



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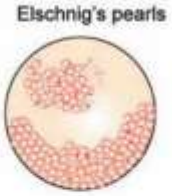
4) After cataract [2000]

Ans.

- **PCO** = Posterior Capsular Opacification: aka After Cataract/secondary cataract
- It is the MC late complication of Cataract Surgery;
- Occur 6-12 months after surgery;
- **Types of PCO**:
  - a. Elsching Pearls (90% cases): forms due to posterior migration of residual epithelial cell
  - b. Sommering's Pearls (10% cases)
- **Treatment of PCO**: Nd: YAG laser posterior capsulotomy;
  Nd: YAG - 1064 nm; MOA – Photodisruption



VSQs

1. Lenticonus [14]

Ans.

- Lenticonus is a bulging of the lens capsule and the underlying cortex.
- It results in Myopia
- It is typically seen in Alport's syndrome
- Diagnosis of lenticonus is made by biomicroscopic examination

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2. Snow Blindness [09]

Ans.

Snow blindness occur due to exposure to reflected UV rays of sun from snow surface. It can lead to Photophthalmia.

Symptoms: extreme burning pain, photophobia, lacrimation and blepharospasm due to desquamation of corneal epithelium

Signs: multiple epithelial erosions a/w blepharospasm & swelling of the palpebral conjunctiva

Treatment

- Cold compresses, astringent lotions and atropine ointment are effective.
- Bandage both eyes for 24 hours. This helps in regeneration of the epithelium.

Prophylaxis: Wearing of dark glasses (Crooke's glasses) to block UV rays when such exposure is anticipated.

Glaucoma

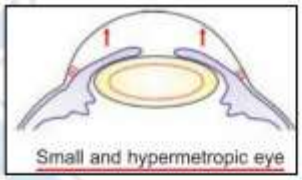
LQs

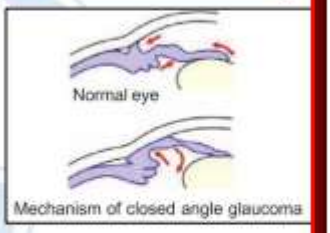
1. Discuss the aetiology, clinical features and management of acute congestive glaucoma [15, 14, 12]
   a. Mention stages of primary narrow angle glaucoma [14]
   b. Describe aetiology, signs, symptoms & treatment of various stages of congestive glaucoma [97]

Ans.

PRIMARY ANGLE-CLOSURE GLAUCOMA: It is characterised by apposition of iris against the trabecular meshwork (TM) → obstruction of aqueous outflow by closure of an already narrow angle of the anterior chamber.

Etiopathogenesis:





❖ **Risk Factors**:

➢ Demographic risk factors
- Age: it is more common in elderly: – 50-70 years
- Gender: Female > Male (3:1)
- Race: it is more common in South-East Asians, Chinese & Eskimos

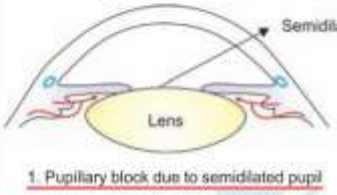
➢ Anatomical and ocular risk factors
- Hypermetropic eyes with shallow anterior chamber & short axial length
- Eyes with narrow angle of anterior chamber – Ex: small eyeball
- Plateau iris configuration
- Heredity – some anatomical factors like shallow anterior chamber & narrow angles are common in 1st degree relatives of patients

❖ **Pathomechanisms of ↑ IOP**: 3 mechanisms

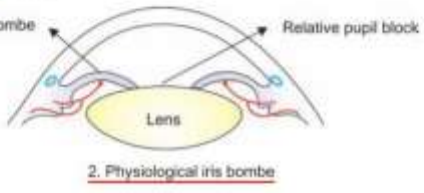
1) **Pupillary block mechanism** – Evident in almost 70% cases
   - Precipitating Factors: mydriasis (ex: atropine), miosis (pilocarpine), Valsalva manoeuvre etc.
   - Precipitating factors → mid dilatation of the pupil → ↑ apposition between iris and lens → relative pupil block → aqueous collects in the posterior chamber & pushes the iris anteriorly (Iris bombe) → iridocorneal contact → peripheral anterior synechiae → angle closure → ↑IOP

2) **Plateau iris configuration**– Evident in 10 % Cases = Angle Closure Glaucoma without pupillary block.
   - » **Anterior positioned ciliary processes** push the peripheral iris forward → closure of anterior chamber angle
   - » Iridotomy is sufficient to control IOP in such patients.

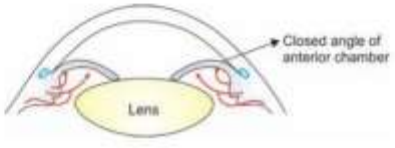
3) **Phacomorphic mechanism**: Abnormal lens may either cause pupillary block or push the peripheral iris forward into the angle structures.



1. Pupillary block due to semidilated pupil



2. Physiological iris bombe



Classification: based on natural history (IOP, gonioscopy, disc and visual field evaluation):

1. Primary Angle Closure Suspect (PACS),
2. Primary Angle Closure (PAC), and
3. Primary Angle Closure Glaucoma (PACG).

<table>
<tr><th colspan="2"></th><th>PACS
(Primary Angle Closure Suspect)</th><th colspan="2">PAC (Primary Angle Closure)</th><th>PACG
(Primary Angle Closure Glaucoma)</th></tr>
<tr><th colspan="2"></th><th></th><th>Subacute PAC</th><th>Acute PAC</th><th></th></tr>
<tr><td colspan="2">Symptoms</td><td>Absent</td><td>• U/L transient blurring of vision
• Coloured halos around light, headache & eyeache on the affected side
• Self-termination of the attack occurs due to physiological miosis induced by bright light, sleep</td><td>» Pain – sudden onset of very severe pain in the eye; radiates along the branches of 5th nerve
» Nausea, vomiting are a/w the pain
» Rapidly progressive impairment of vision, redness, photophobia and lacrimation
» Past history of intermittent attacks of subacute PAC</td><td rowspan="2">• It occurs due to gradual synechial closure of the angle of anterior chamber.
• PACG may clinically manifest as subacute, acute or chronic PACG
• Subacute and acute PACG clinically present as similar to subacute and acute PAC except that glaucomatous optic disc changes and visual field defects are always present in PACG.
• Chronic PACG - Clinical features are similar to POAG except that angle closure is present (refer 2nd LQ)</td></tr>
<tr><td colspan="2">Signs</td><td>1. Eclipse sign – Indicates ↓ axial anterior chamber depth.
2. Slit-lamp signs
↳ ↓ axial anterior chamber depth
↳ Convex shaped iris lens diaphragm
↳ Close proximity of the iris to cornea in periphery
3. Van Herick slit-lamp grading of the angle
<table><tr><th>Grade</th><th>Angle</th><th>PACD</th></tr><tr><td>4</td><td>Wide open angle</td><td>= 0.75 to 1 CT</td></tr><tr><td>3</td><td>Mild narrow angle</td><td>= 0.25 to 0.5 CT</td></tr><tr><td>2</td><td>Moderate narrow angle</td><td>= 0.25 CT</td></tr><tr><td>1</td><td>Extremely narrow angle</td><td>< 0.25 CT</td></tr><tr><td>0</td><td>Closed angle</td><td>Nil</td></tr><tr><td colspan="3">PACD = peripheral anterior chamber depth; CT = corneal thickness</td></tr></table></td><td>→ Same as PACS
→ Usually, during examination the eye is white and not congested</td><td>• Lid Edema
• Conjunctiva is chemosed & congested
• Cornea is edematous and insensitive
• Anterior chamber is shallow; + Aqueous flare & Cells
• Iris – discoloured
• Pupil is semi dilated & fixed. It is non-reactive to both light and accommodation</td></tr>
<tr><td rowspan="4">Diagnostic criteria</td><td>Gonioscopy</td><td>Irido-trabecular contact (ITC) angle > 270°
No PAS (peripheral anterior synechiae)</td><td colspan="2">ITC angle > 270°
PAS present</td><td rowspan="4"></td></tr>
<tr><td>IOP</td><td>Normal</td><td colspan="2">elevated</td></tr>
<tr><td>Optic disc</td><td>Normal – No glaucomatous change</td><td>Normal in Subacute PAC;</td><td>Edematous & hyperaemic in Acute PAC</td></tr>
<tr><td>Visual fields</td><td>Normal</td><td colspan="2">Normal</td><td>Visual fields show glaucomatous defects</td></tr>
<tr><td colspan="2">Impression</td><td>The angle is at risk</td><td colspan="2">Angle is abnormal
either in function (↑ IOP) and/or structure (PAS +ve).</td><td>Angle is abnormal in function (↑ IOP) and structure (PAS +ve).</td></tr>
<tr><td rowspan="2">Management</td><td>Tests</td><td>Provocative tests to precipitate closure of the angle:
➔ Ex: Prone-darkroom test & Mydriatic provocative test
➔ Positive provocative test indicates that angle is capable of spontaneous closure
➔ Negative provocative test does not rule out a possibility of spontaneous closure. So, patient should be warned of possible symptoms of an acute attack of PAC.</td><td colspan="2" rowspan="2">Acute PAC is a serious ocular emergency and needs to be managed aggressively
1) Immediate Medical therapy to lower IOP
↳ Systemic hyperosmotic agents – Ex: i.v. Mannitol
↳ Systemic CA inhibitors: Ex: i.v. acetazolamide 500 mg stat
↳ Topical antiglaucoma drugs –
- Beta-blockers – Ex: 0.5% timolol
- Alpha adrenergic agonists, e.g., 0.1–0.2% brimonidine
- Prostaglandin analogue, e.g., 0.005% latanoprost
↳ Analgesics and antiemetics
↳ Topical steroid, e.g., dexamethasone eye drops
↳ Compressive gonioscopy
2) Definitive therapy
➤ Laser Peripheral Iridotomy – LPI (with Nd: YAG laser or Argon Laser)
➤ Filtration surgery, i.e., trabeculectomy – done if LPI is not effective
➤ Clear lens extraction by phacoemulsification – done in Phacomorphic etiology
3) Prophylaxis of the fellow eye – Prophylactic laser iridotomy on the fellow asymptomatic eye
4) Long-term glaucoma surveillance and IOP management in both eyes of a patient with acute PAC is must to ultimately prevent glaucomatous blindness</td><td rowspan="2">Laser iridotomy ± medical therapy ➔ if it fails, then → Trabeculectomy (filtration surgery)

Prophylactic laser iridotomy in fellow eye must also be performed.</td></tr>
<tr><td>Treatment</td><td>• Prophylactic laser iridotomy
• Periodic follow up</td></tr>
</table>

Absolute PACG: PACG, if untreated, gradually passes into the final phase of absolute glaucoma.

- **Clinical features**: Painful blind eye, Perilimbal reddish blue zone, Caput medusae, bullous keratopathy, Shallow Anterior chamber; Iris becomes atrophic; Pupil becomes fixed and dilated and gives a greenish hue;
  - Optic disc shows glaucomatous optic atrophy.
  - IOP is high; eyeball becomes stony hard.
- **Management of absolute glaucoma**
  1. Retrobulbar alcohol injection – to relieve pain. It destroys the ciliary ganglion.
  2. Cyclocryotherapy or Cyclodiathermy or Cyclophotocoagulation – to destroy secretory ciliary epithelium → ↓ IOP
  3. Enucleation of eyeball – if pain is not relieved by conservative methods.
- **Complications of absolute glaucoma** (due to prolonged high IOP):
  - Corneal ulceration
  - Staphyloma formation: As a result of continued high IOP, sclera becomes very thin and atrophic and ultimately bulges out either in the ciliary region (ciliary staphyloma) or equatorial region (equatorial staphyloma).
  - Atrophic bulbi.

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2. Describe the clinical features, pathology, Dx and Mx of primary open angle glaucoma [08, 2000]
   - **a.** Optic nerve head changes in Open Angle Glaucoma [17]
   - **b.** Field changes in Primary Open Angle Glaucoma [10, 03, 02]
   - **c.** Field defects in Chronic Simple Glaucoma [07]
   - **d.** Describe the aetiology, clinical features, and treatment of chronic simple glaucoma [95, 91, 90]

Ans.

Primary open-angle glaucoma (POAG), also known as chronic simple glaucoma of adult onset, is characterised by slowly progressive ↑ IOP (>21 mm Hg) associated with:

- Open normal appearing anterior chamber angle,
- Characteristic optic disc cupping, and
- Specific visual field defects.

ETIOPATHOGENESIS:

A. Predisposing factors:

1) **Age**: elders between 5th and 7th decades.
2) **Race**: black > whites
3) **Family history**: POAG has a polygenic inheritance – Myocilin C, Optineurin & WD repeat domain 36 genes
4) **Ocular Factors**: Myopes; Low Central corneal thickness (CCT) & ↑ IOP
5) DM, smoking
6) Blood Pressure: Diastolic perfusion pressure (DBP – IOP) of <55 mm Hg.
7) Graves' ophthalmic disease
8) Corticosteroid responsiveness.

B. Pathogenesis of rise in IOP: Trabecular meshwork stiffening & apposition of Schlemm's canal ➔ Failure of aqueous outflow pump mechanism ➔ ↓ in the aqueous outflow ➔ ↑ IOP.

⇒ Such changes are caused by age-related:

- Sclerosis of trabecular meshwork with faulty collagen tissue.
- Narrowing of intertrabecular spaces.
- Deposition of amorphous material in the juxtacanalicular space.
- Collapse of Schlemm's canal

C. Pathogenesis of glaucomatous optic neuropathy: All glaucomas are characterized by a progressive optic neuropathy which occurs due to death of retinal ganglion cells (RGCs) which results in characteristic optic disc appearance and specific visual field defects.

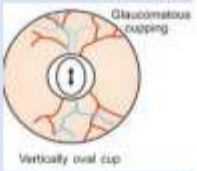
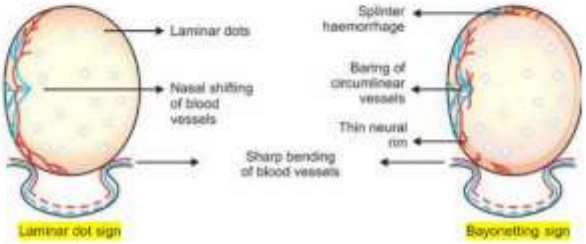
- Mechanical effect of ↑ IOP push the lamina cribrosa and squeezes the nerve fibres within it's meshes to disturb axoplasmic flow.

CLINICAL FEATURES:

Symptoms

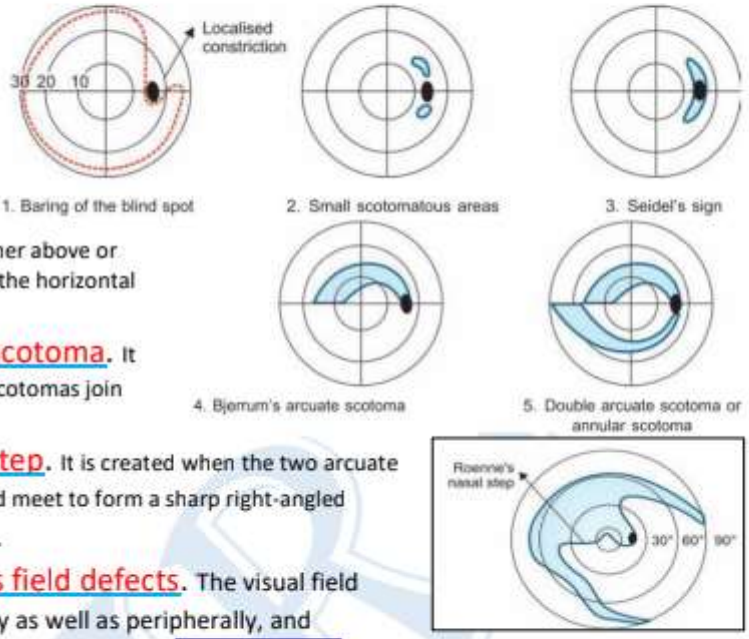
1) Asymptomatic – POAG is insidious. Hence, periodic eye examination is required after middle age.
2) Headache and eye ache.
3) Scotoma (defect in the visual field)
4) Increasing difficulty in reading and close work – occurs due to accommodative failure as a result of constant pressure on the ciliary muscle and its nerve supply. Patients complain of, frequent changes in presbyopic glasses.
5) Delayed dark adaptation
6) Blindness is the end result of untreated cases of POAG.

Signs of POAG

1. Anterior segment signs		a) Slit- lamp examination may reveal normal anterior segment. b) In late stages, pupil reflex becomes sluggish and cornea may show slight haze c) A low (<555 mm) central corneal thickness is a risk factor for POAG.
2. IOP changes		↳ In the initial stages, the IOP may not be raised permanently ↳ Diurnal variation in IOP of > 5 mm Hg is suspicious and > 8 mm Hg is diagnostic of glaucoma ↳ In later stages, IOP is permanently raised above 21 mm of Hg
3. Optic disc changes (Detected on fundus examination)	**Early Changes**	1) Vertically oval cup due to selective loss of neural rim tissue in the inferior and superior poles. 2) Asymmetry of the cups of two eyes. 3) Large cup size ≥ 0.6 (normal cup size is 0.3 to 0.4). 4) Splinter haemorrhages on or near the optic disc margin. 5) Pallor areas on the disc 
	Advanced changes	a) Marked cupping (cup size 0.7 to 0.9) b) Thinning of neuroretinal rim is seen as a crescentric shadow adjacent to the disc margin. c) Notching of the rim specially up to disc margin is pathognomic. d) Nasal shifting of retinal vessels which have the appearance of being broken off at the margin is Bayonetting sign. e) Pulsations of the retinal arterioles may be seen at the disc margin (a pathognomic sign of glaucoma if IOP is very high. f) Laminar dot sign: the pores in the lamina cribrosa are slit-shaped and are visible up to the margin of the disc. 
	Glaucomatous optic atrophy	As the damage progresses, all the neural tissue of the disc is destroyed and the optic nerve head appears white and deeply excavated

4. Visual field defects (in sequence)

1) Isopter contraction –mild constriction of visual field.
2) Baring of blind spot – It means exclusion of the blind spot from the central field due to inward curve of the outer boundary of 30° central field
3) Small wing-shaped paracentral scotoma – It is the earliest clinically significant field defect.
4) Seidel's scotoma – paracentral scotoma joins the blind spot to form a sickle-shaped scotoma known as Seidel's scotoma
5) Arcuate or Bjerrum's scotoma – formed by the extension of Seidel's scotoma either above or below the fixation point to reach the horizontal line.
6) Ring or double arcuate scotoma. It develops when the two arcuate scotomas join together.
7) Roenne's central nasal step. It is created when the two arcuate scotomas run in different arcs and meet to form a sharp right-angled defect at the horizontal meridian.
8) Advanced glaucomatous field defects. The visual field loss gradually spreads centrally as well as peripherally, and eventually only a small island of central vision (tubular vision) and an accompanying temporal island are left.



5. Ocular associations

POAG may be associated with high myopia, Fuchs' endothelial dystrophy, retinitis pigmentosa, central retinal vein occlusion and primary retinal detachment.

INVESTIGATIONS

1. **Tonometry** – Applanation tonometry should be preferred over Schiotz tonometry – to measure IOP
2. **Central corneal thickness (CCT) measurement**
3. **Diurnal IOP variation test** is useful in detection of early cases
4. **Gonioscopy** – It reveals a wide-open angle of anterior chamber – rule out other forms of glaucoma.
5. **Documentation of optic disc changes**.
6. **Slit-lamp examination** of anterior segment to rule out causes of secondary open-angle glaucoma.
7. **Perimetry** to detect the visual field defects.
8. **Nerve fibre layer analyzer (NFLA)** is a device which helps in detecting the glaucomatous damage to the retinal nerve fibres before the appearance of actual visual field changes
9. **Provocative tests** are required in border-line cases. The test commonly performed is water drinking test.
   - Water drinking test. It is based on the theory that glaucomatous eyes have a greater response to water drinking. In it after 8 hours fast, baseline IOP is noted and the patient is asked to drink one litre of water, following which IOP is noted every 15 minutes for 1 hour. A rise of 8 mm of Hg or more is said to be diagnostic of POAG.

DIAGNOSIS: Depending upon the level of IOP, glaucomatous cupping of the optic disc and the visual field changes, the patients are assigned to one of the following diagnostic entities:

A. Primary open-angle glaucoma: POAG is labelled when raised IOP (>21 mm of Hg) is a/w definite glaucomatous optic disc cupping and visual field changes.
B. Ocular hypertension - patient has an IOP constantly > 21 mm of Hg but no optic disc and visual field changes.
C. Normal tension glaucoma (NTG) or low-tension glaucoma (LTG): Here, typical glaucomatous disc cupping ± visual field changes is associated with an IOP < 21 mm of Hg

MANAGEMENT

- **Baseline evaluation** – done to monitor future progress.
  - **It includes**: visual acuity, slit-lamp examination of anterior segment, tonometry; measurement of CCT, optic disc evaluation (preferably with fundus photography), gonioscopy and visual field charting.
  - **Grading:** American Academy of Ophthalmology (AAO) grades glaucoma damage into mild, moderate & severe.

Degree	Description
Mild	Characteristic optic-nerve abnormalities are consistent with glaucoma but with normal visual field.
Moderate	Visual-field abnormalities in one hemi-field and not within 5 degrees of fixation.
Severe	Visual-field abnormalities in both hemi-fields and within 5 degrees of fixation.

- **Therapeutic choices.**
  - **Medical therapy with Antiglaucoma drugs**
    - » Identify a target pressure from the baseline evaluation data
    - » Based on your target, prescribe a Single/Combination of antiglaucoma drugs
    - » Monitor the patient for disc changes and field changes – tonometry is most essential on regular follow-up.

Anti-Glaucoma Drugs (↓ IOP)	
Drugs to ↑ trabecular outflow	Pilocarpine (miotics) Dipivefrine Bimatoprost
Drugs to ↑ uveoscleral outflow	→ Dipivefrine → Latanoprost
Drugs to ↓ aqueous production	• CA inhibitors (ex: acetazolamide) • α agonist (ex: dipivefrine, epinephrine) • β blockers (ex: timolol)
Hyperosmotic agents	Ex: Mannitol

  - **Laser trabeculoplasty** can be done using argon laser (ALT), or diode laser (DLT) and selective laser trabeculoplasty (SLT). It should be considered in patients where IOP is uncontrolled despite maximal tolerated medical therapy.
  - **Surgical therapy** - Filtration surgery – Ex: Trabeculectomy

    Indications of Surgery
    a) Uncontrolled glaucoma despite maximal medical therapy and laser trabeculoplasty.
    b) Noncompliance of medical therapy and nonavailability of ALT/SLT.
    c) Eyes with advanced disease, i.e., having very high IOP, advanced cupping and advanced field loss.

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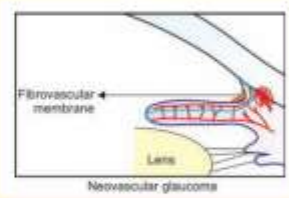
3. Enumerate the causes of Secondary Glaucoma. Add a note on its treatment [98, 94]
   a. Phacomorphic Glaucoma [15]

Ans.

Depending upon the causative primary disease, secondary glaucomas are named as follows:

Secondary Glaucoma	Treatment
1) Lens-induced (phacogenic) glaucomas	▪ Medical therapy to lower the IOP ▪ Cataract extraction with implantation of PCIOL (in quiet eyes) ▪ Laser iridotomy – for Phacomorphic Glaucoma ▪ Irrigation-aspiration of the lens particles from the anterior chamber – in Lens Particle Glaucoma & Phacoantigenic Glaucoma
2) Inflammatory glaucoma	Treat iridocyclitis + Medical therapy to lower IOP Trabeculectomy – can be done if medical treatment fails
3) Pigmentary glaucoma.	behaves like POAG and is thus managed on the same lines.
4) Pseudoexfoliative glaucoma.	behaves like POAG and is thus managed on the same lines.

5) Neovascular glaucoma.  Neovascular glaucoma	» Panretinal photocoagulation – to prevent further neovascularization » Glaucoma drainage device, i.e., artificial filtration shunt (Seton operation) may control the IOP. » Medical therapy and conventional filtration surgery are usually ***not effective*** in controlling the IOP.
6) Glaucomas a/w iridocorneal endothelial syndromes.	» Glaucoma drainage device, i.e., artificial filtration shunt (Seton operation) may control the IOP. » Medical therapy and conventional filtration surgery are usually ***not effective*** in controlling the IOP.
7) Glaucomas a/w intraocular haemorrhage.	Examples are: Red cell glaucoma, Haemolytic glaucoma, Ghost cell glaucoma & Hemosiderotic glaucoma
8) Glaucoma-in-aphakia.	Examples are: ▪ Steroid-induced glaucoma: Discontinue steroids + Medical therapy to ↓ IOP ▪ Traumatic glaucoma: Treat the cause + Medical therapy to ↓ IOP + Surgery
9) Glaucoma a/w intraocular tumours	Enucleation of the eyeball should be carried out as early as possible

Lens induced glaucoma can be classified as below:

- Lens-induced secondary angle closure glaucoma:
  - Phacomorphic glaucoma (due to swollen lens)
  - Phacotopic glaucoma (due to anterior lens displacement).
- Lens-induced secondary open angle glaucoma: Phacolytic glaucoma, Lens particle glaucoma & Phacoanaphylactic glaucoma.

Phacomorphic Glaucoma

- **Causes**: Phacomorphic glaucoma is an acute secondary **angle-closure** glaucoma caused by:
  - Intumescent lens i.e., swollen cataractous lens due to rapid maturation of cataract
  - Anterior subluxation or dislocation of the lens and spherophakia (congenital small spherical lens) are causes of Phacotopic (a variant of Phacomorphic) glaucoma.
- **Pathogenesis**: The swollen lens pushes the iris forward and obliterates the angle → secondary acute angle closure glaucoma.
- **Clinical presentation**: Phacomorphic glaucoma presents as acute congestive glaucoma with features almost similar to Acute PAC (refer 1st LQ) except that the lens is always cataractous and swollen.
- **Treatment** should be immediate and consists of:
  - Medical treatment to control IOP by IV mannitol, systemic acetazolamide & topical β-blockers.
  - Laser iridotomy may be effective in breaking the angle-closure attack.
  - Cataract extraction with implantation of PCIOL should be performed once the eye becomes quiet.

SQs

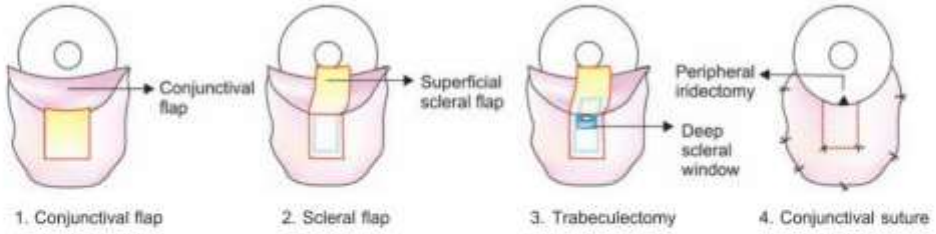
1) Trabeculectomy [15, 02]

Ans.

- It is the most frequently performed external Filtration Surgery till date
- In this Surgery, a new channel (fistula) is created around the margin of sclera, through which aqueous flows from anterior chamber into the subconjunctival space

➤ **Surgical technique** 👉

- A fistula is created between anterior chamber and subtenon's space,
- MCC of failure – fibrosis of fistula
- To prevent fibrosis, use antimetabolites (mitomycin, 5-FU)



➤ **Indications**

1. Primary angle-closure glaucoma with peripheral anterior synechiae involving > 270° angle.
2. Primary open-angle glaucoma not controlled with medical treatment.
3. Congenital and developmental glaucomas where trabeculotomy and goniotomy fail.
4. Secondary glaucomas where medical therapy is not effective.

➤ **Complications**: postoperative shallow anterior chamber, hyphaema, iritis, cataract due to accidental injury to the lens, and endophthalmitis.

➤ Sutureless trabeculectomy is also available nowadays

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2) Buphthalmos [10, 05, 02]

Ans.

It is defined as Congenital Glaucoma occurring prior to age of 3 years.

❖ As it results due to retention of aqueous humour (watery solution) it is also termed as 'hydrophthalmos'

❖ **Etiology**: It occurs due to congenital abnormality at the angle of anterior chamber (Ex: Absence of canal of Schlemm, incomplete separation of iris from cornea). It is transmitted as an autosomal recessive trait.

❖ **Symptoms**: Lacrimation, Photophobia, defective vision & enlargement of cornea and the eye as a whole.

❖ **Signs**:

- Corneal enlargement occurs along with the enlargement of the globe—Buphthalmos (Bull-like eyes)
- There is corneal oedema & opacities due to endothelium damage and rupture of Descemet's membrane (Haabs' striae)
- Corneal Diameter > 13 mm
- Lens is flattened and displaced backwards due to stretching of the zonule of Zinn
- Deep anterior chamber - due to backward displacement of the lens.
- Sclera becomes thin and bluish as the uveal tissue shines through it
- IOP is Raised (due to retention of Aqueous humour)

❖ **Treatment**: it is primarily surgical

➤ **Medical treatment**: not very effective; ↓IOP by anti-glaucoma drugs {α_2- agonist (brimonidine) causes CNS depression in children and is contraindicated}

➤ **Surgical procedures**:

- Incisional angle surgery, which can be performed by the internal approach (goniotomy) or by external approach (trabeculotomy).
- Filteration surgery – Ex: Trabeculectomy
- Glaucoma drainage devices (GDD) are required in incalcitrant cases

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VSQs

1. Classify Glaucoma [04, 03]

Ans.

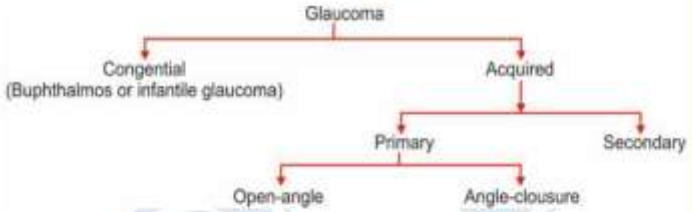
Glaucoma is not a single disease process but a group of disorders characterized by a progressive optic neuropathy resulting in a characteristic appearance of the optic disc and a specific pattern of irreversible visual field defects that are associated frequently but not invariably with ↑ IOP.

Classification: Clinico-etiologically glaucoma may be classified as follows:

A. Congenital/developmental glaucomas
   1. *Primary congenital glaucoma (without associated anomalies).*
   2. *Developmental glaucoma (with associated anomalies).*

B. Primary adult glaucomas
   1) Primary open-angle glaucomas (POAG)
   2) Primary angle-closure glaucoma (PACG)
   3) Primary mixed mechanism glaucoma

C. Secondary glaucomas



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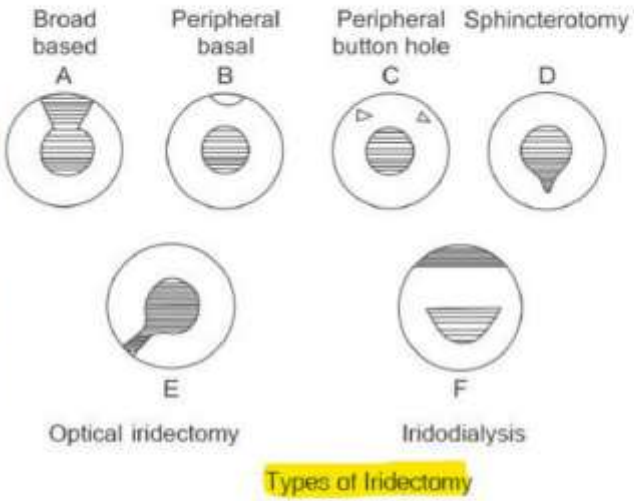
2. Indications & types of iridectomy [06, 05]

Ans.

Iridectomy consists of the abscission or cutting of a portion of the iris

Indications:

1) Prolapsed iris
2) Treatment of all stages of primary angle-closure glaucoma.
3) Prophylaxis in the fellow eye
4) Foreign body in the iris
5) Optical iridectomy—In central leucomatous corneal opacity, temporal (for distant vision, e.g. farmers) or nasal (for near work, e.g. clerk, goldsmith) iridectomy is done according to the requirement and job of the patient



Types of Iridectomy

Neuro-ophthalmology

LQs

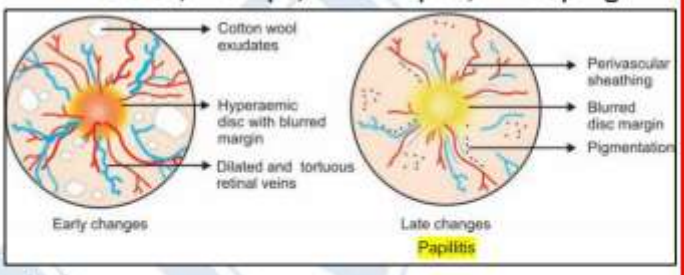
1) Discuss Optic Neuritis – C/F, DDx and treatment [11]
   a. Acute Retrobulbar neuritis [16]
   b. Papillitis [05, 03]

Ans.

Optic neuritis refers to inflammatory & demyelinating disorders of the optic nerve.

Etiology

1. **Demyelinating disorders** (MCC) – Ex: MS, neuromyelitis optica (Devic's disease) etc.
2. **Hereditary optic neuritis** (Leber's disease)
3. **Parainfectious optic neuritis** – a/w viral infections – Ex: measles, mumps, chickenpox, whooping cough and glandular fever.
4. **Infectious optic neuritis** may be sinus related (with acute ethmoiditis) or a/w cat scratch fever, syphilis, TB, Lyme disease and cryptococcal meningitis in patients with AIDS.
5. **Autoimmune disorders** a/w optic neuritis include sarcoidosis, SLE, PAN, GBS & Wegener's granulomatosis
6. **Idiopathic**
7. **Toxic optic neuritis** – (refer 3rd SQ)

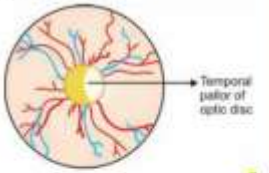


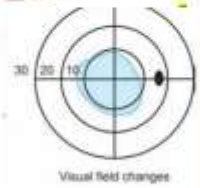
Anatomical types: Optic neuritis can be classified into 3 anatomical types:

1. **Papillitis** – It refers to involvement of the optic disc. This is usually U/L but sometimes may be B/L.
2. Neuroretinitis refers to combined involvement of optic disc & surrounding retina in the macular area.
3. **Retrobulbar neuritis** refers to the involvement of optic nerve behind the eyeball. Clinical features of acute retrobulbar neuritis are similar to that of acute papillitis except for the fundus changes and ocular changes

Clinical features

Symptoms	Signs
➔ Visual loss – Monocular, sudden, progressive ➔ ↓Dark adaptation ➔ Visual obscuration in bright light. ➔ Impairment of colour vision. ➔ Movement phosphenes and sound induced phosphenes may be perceived by patients ➔ Depth perception, particularly for the moving object may be impaired (Pulfrich's phenomenon). ➔ **Pain** – aggravated by ocular movements.	1) ↓ Visual acuity. 2) Impairment of colour vision & Contrast sensitivity. 3) Marcus Gunn pupil – it indicates RAPD 4) **Ophthalmoscopic features**: • Papillitis is characterised by hyperaemia & edema of the disc and blurring of the margins. Physiological cup is obliterated. Retinal veins are congested and tortuous. Splinter haemorrhages & fine exudates are seen on the disc • Neuroretinitis = Papillitis + macular star formation • In retrobulbar neuritis fundus appears normal and the condition is typically defined as a disease where neither the ophthalmologist nor the patient sees anything. Occasionally, temporal pallor of the disc may be seen 5) Visual field– central scotoma (MC defect) 6) Visually evoked response (VER) shows ↓ amplitude & delay in the transmission time. 7) Fundus fluorescein angiography reveals mild to moderate leak.





Differential diagnosis

» Papillitis should be differentiated from papilloedema, ischaemic optic neuropathy, anterior orbital compressive neuropathy and pseudopapilloedema

» Acute retrobulbar neuritis must be differentiated from malingering, hysterical blindness, cortical blindness and indirect optic neuropathy.

Treatment

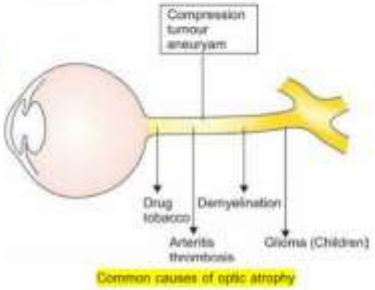
1) ***Identify & treat the cause.***
2) ***Corticosteroid therapy***:
   - If brain MRI scan shows lesions of MS → i.v. methylprednisolone (1 gm daily) for 3 days followed by oral prednisolone (1 mg/kg/day) for 11 days. Then taper prednisolone over 4 days.
   - This therapy will delay conversion to clinical MS over the next 2 years.
3) ***Interferon therapy***:
   - It reduces the recurrences in patients with MS.
   - But this is very expensive and with unknown long-term benefits.

SQs

1. Optic atrophy – Classification, Aetiology, pathology & C/F [16, 14, 12, 08]
   a. Primary optic atrophy [07, 02]
   b. Consecutive Optic Atrophy [05, 02]

Ans.

Optic atrophy refers to degeneration of the optic nerve, which occurs as an end result of any process that damages axons in the anterior visual system, i.e., from retinal ganglion cells to lateral geniculate body



Common causes of optic atrophy

Classification & Etiopathogenesis– based on its ophthalmoscopic appearance:

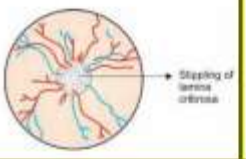
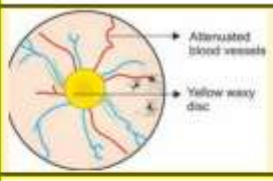
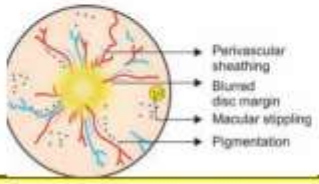
Type of optic atrophy	Occurs due to	Seen in
1) ***Primary (simple) optic atrophy:***	Lesions proximal to the optic disc	MS, retrobulbar neuritis, toxic amblyopias, tabes dorsalis, intracranial tumours pressing directly on the anterior visual pathway (e.g., pituitary tumour) etc.
2) ***Consecutive optic atrophy:***	Destruction of ganglion cells	Diffuse chorioretinitis, retinitis pigmentosa, pathological myopia and CRAO
3) ***Post-neuritic optic atrophy***	Occurs due to longstanding papilloedema or papillitis	
4) ***Glaucomatous optic atrophy***	Occurs due to the effect of long-standing ↑IOP	
5) ***Vascular (ischaemic) optic atrophy***	Disc ischaemia	Giant cell arteritis, severe haemorrhage, severe anaemia and quinine poisoning

Pathological features

1. Degeneration → gliosis - astrocytes arrange themselves in longitudinal columns replacing the nerve fibres (columnar gliosis) ➔ seen in primary optic atrophy
2. Degeneration of the nerve fibres → **excessive** gliosis (regeneration) ➔ seen in consecutive and post-neuritic optic atrophy.
3. Degeneration of the nerve fibres with negligible gliosis ➔ seen in glaucomatous & ischaemic (vascular) optic atrophy.

Clinical features of optic atrophy

1. Loss of vision, may be of sudden or gradual onset (depending upon the cause of optic atrophy)
2. Pupil is semi-dilated and direct light reflex is very sluggish or absent.
3. Swinging flash light test depicts Marcus Gunn pupil (RAPD).
4. Visual field loss.
5. Ophthalmoscopic appearance:

Type of optic atrophy	Ophthalmoscopic appearance
1) Primary (simple) optic atrophy: 	▪ Disc appear chalky white. ▪ Disc Edges are sharply outlined ▪ Lamina cribrosa is clearly seen at the bottom of the physiological cup. ▪ Retinal vessels and surrounding retina are normal
2) Consecutive optic atrophy: 	Disc appears yellow waxy. Retinal vessels are attenuated
3) Post-neuritic optic atrophy 	» Disc appears dirty white in colour » Due to gliosis, its edges are blurred » Physiological cup is obliterated and lamina cribrosa is not visible » Retinal vessels are attenuated + Perivascular sheathing
4) Glaucomatous optic atrophy	Wide cupping of the optic disc & nasal shift of blood vessels
5) Vascular (ischaemic) optic atrophy	Pallor of the optic disc + marked attenuation of the vessels

Treatment of underlying cause may help in preserving some vision in patients with partial optic atrophy. But, once complete atrophy has set in, the vision cannot be recovered

---

2. Papilledema {15, 12, 08, 05}

Ans.

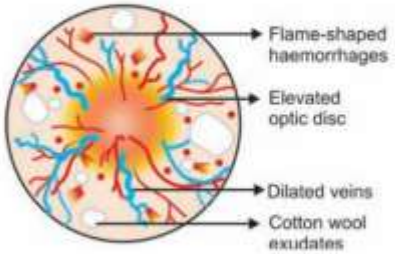
Papilloedema is a non-inflammatory oedema of optic disc which occurs 2° to ↑ intracranial pressure.

Causes of papilloedema

1) Congenital conditions: aqueductal stenosis and craniosynostosis.
2) Intracranial infections such as meningitis and encephalitis.
3) Diffuse cerebral oedema from blunt head trauma
4) Intracranial haemorrhages & Cerebral venous sinus thrombosis
5) Obstruction of CSF absorption via arachnoid villi which have been damaged previously.
6) Intracranial space-occupying lesions (ICSOLs) & Tumours of spinal cord.
7) Idiopathic intracranial hypertension (IIH) also known as pseudotumour cerebri
8) Systemic conditions: malignant hypertension, PIH, cardiopulmonary insufficiency, blood dyscrasias and nephritis.

Clinical features

- **General features** of ↑ ICP ➔ headache, nausea, projectile vomiting and diplopia.
- **Ocular features**

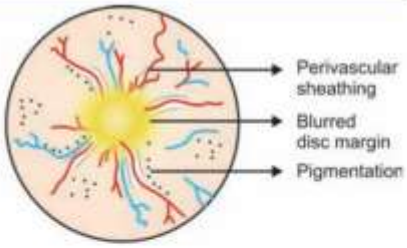


Early changes

Stage of Papilledema	Symptoms	Signs		
		Pupillary Reaction	Ophthalmoscopic Features	Visual Field
1) Early (incipient)	Absent	Normal	» Obscuration of the disc margins » Absence of spontaneous venous pulsation at the disc » Blurring of peripapillary nerve fibre layer » Hyperaemia of the disc	Normal
2) Established (fully developed)	Transient visual obscurations	Normal	▪ Blurring of the disc margin ▪ Engorged & tortuous veins ▪ Physiological cup is obliterated ▪ Disc becomes markedly hyperaemic ▪ Paton's lines i.e., circumferential greyish white folds may develop due to separation of nerve fibres by the oedema.	Enlargement of Blind Spot
3) Chronic or long-standing (vintage)	↓Visual acuity	Normal	Optic disc appears as dome of a champagne cork. The central cup remains obliterated 	Enlargement of Blind Spot + Visual fields begin to constrict
4) Atrophic	Severely impaired visual acuity	Light reflex is impaired	⇉ Greyish white discolouration of the disc ⇉ ↓Prominence of the disc ⇉ Retinal arterioles are narrowed	Concentric contraction of peripheral fields

Treatment and prognosis

- Urgent neuroimaging (CT scan or MRI with a gadolinium enhancement) to reveal the cause.
- As a rule, unless the causative disease is treatable or cerebral decompression is done, the course of papilloedema is chronic and visual prognosis is bad



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3. Toxic Amblyopia [09]
   a. Amblyopia [10]
   b. Tobacco Amblyopia [90]

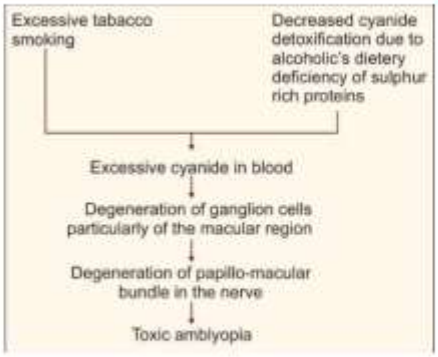
Ans.

Amblyopia means partial loss of sight in one or both eyes, in the absence of obvious defect in the eye.

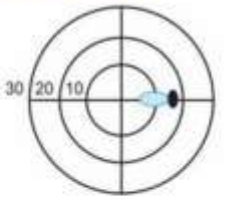
- ❖ It may be either congenital or acquired.
- ❖ Acquired amblyopia may be organic (toxic amblyopia) or Functional.

- ❖ Functional amblyopia occurs due to psychical suppression of the retinal image. It may be anisometropic, strabismic or due to stimulus deprivation
- ❖ **Toxic amblyopia** aka **Toxic/nutritional optic neuropathy,** is basically chronic retrobulbar neuritis.
  - ⊙ Poisons (exo/endogenous) → damage the optic nerve → visual loss
  - ⊙ It is frequently bilateral and has a chronic course with permanent visual deterioration.
  - ⊙ Some Varieties of toxic amblyopia are: Tobacco amblyopia, Ethyl alcohol amblyopia, Methyl alcohol amblyopia, Quinine amblyopia & Ethambutol amblyopia.

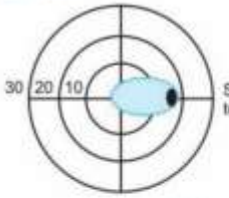
⊙ Tobacco amblyopia



- Seen in in men (40-60 years) who are pipe smokers, heavy drinkers and have a diet deficient in proteins and vitamin B complex; and hence also labelled as 'tobacco-alcohol-amblyopia'.
- **Pathogenesis**
- **Symptoms**: B/L, progressive Impairment of central vision. Patients complain of fogginess and difficulty in doing near work.
- **Signs**
  - Visual field: bilateral centrocaecal scotomas with diffuse margins
  - Fundus examination is almost normal



Central caecal scotoma



Scotoma extending to fixation point

- **Treatment.**
  - Complete cessation of tobacco and alcohol consumption
  - Hydroxocobalamin 1000 mg IM injections weekly for 10 weeks
  - Care of general health and nutrition.
- **Prognosis:** It is good, if complete abstinence from tobacco and alcohol is maintained. Visual recovery is slow and may take several weeks to months

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4. Colour Vision [09]
   a. Mention any four commonly employed colour vision tests [16]
   b. Trichromatic theory of color vision [15]

Ans.

- Colour Vision is the ability of the eye to discriminate between different colours excited by light of different wavelengths.
- Colour vision is a function of the cones and thus better appreciated in photopic vision.
- **Theories of colour vision**:

  1) Young-Helmholtz's trichromatic theory:
     - It postulates the existence of 3 kinds of cones, each containing a different photopigment which is maximally sensitive to one of the three primary colours viz. red, green and blue.
     - The sensation of any given colour is determined by the relative frequency of the impulse from each of the three cone systems.
  2) Hering's Opponent colour theory – it points out that some colours appear to be 'mutually exclusive' – colour opponency occurs at ganglion cell onwards
- **Commonly employed colour vision tests are**:
  1. Pseudoisochromatic charts (MC) – Ex: Ishihara's chart – for red-green defects
  2. City university colour vision test
  3. Edridge-Green lantern test
  4. Farnsworth-Munsell 100 hue test – It is the most sensitive test for colour vision defects
  5. Farnsworth D15 hue discrimination test
  6. Nagel's anomaloscope
  7. Holmgren's wools test

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5. Snellen's chart for vision testing [90]

Ans.

- Snellen's chart is used to record the visual acuity (Normal distant visual acuity is 6/6)
- Snellen's chart consists of a series of letters arranged in lines each diminishing in size.
- The lines from above downwards should be read at 60, 36, 24, 18, 12, 9, 6, 5 m, respectively.
- At these distances the letters subtend a visual angle of 5' at the nodal point.
- It is kept at a distance of 6 m so that the rays of light are parallel for practical purpose.

CONTENTS

Disorders of Ocular Motility

SQs

1) Heterophoria [10, 97]

Ans. Heterophoria also known as **'latent strabismus'**, is a condition in which the tendency of the eyes to deviate is kept latent by fusion. Therefore, when the influence of fusion is removed the visual axis of one eye deviates away.

Types of heterophoria

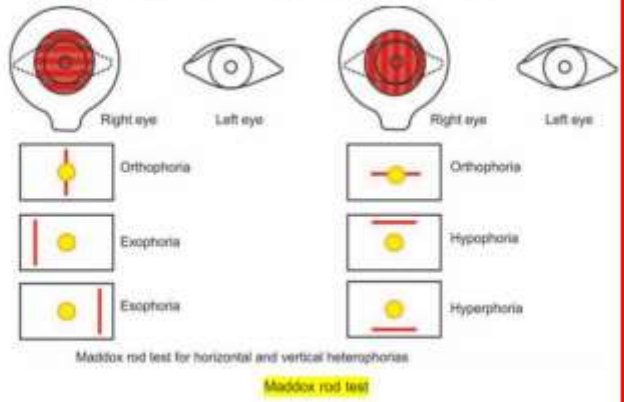
1. **Esophoria** or latent convergent squint refers to tendency of eyeballs to deviate inward.
2. **Exophoria** or latent divergent squint refers to tendency of the eyeballs to deviate outwards.
3. **Hyperphoria** is a tendency of the eyeball to deviate upwards, while **hypophoria** is a tendency to deviate downwards.
4. **Cyclophoria** or torsional deviation is a tendency of the eyeball to rotate around the anteroposterior axis. When the 12 O'clock meridian of cornea rotates nasally, it is called incyclophoria and when it rotates temporally it is called encyclophoria.

Etiology

Anatomical factors	Physiological factors
1. Orbital asymmetry. 2. Abnormal interpupillary distance (IPD): A wide IPD is a/w exophoria and small with esophoria. 3. Weakness or Faulty insertion of extraocular muscle. 4. Anomalous central distribution of the tonic innervation of the two eyes. 5. Variation in the position of the macula in relation to the optical axis of the eye.	1) **Age**: **Esophoria** is more common in **younger** age group as compared to **exophoria** which is often seen in **elderly**. 2) **Role of accommodation**: ↑ **accommodation** is associated with **esophoria** (as seen in hypermetropes and individuals doing excessive near work) and ↓ **accommodation** with **exophoria** (as seen in simple myopes). 3) **Dissociation factor** such as prolonged constant use of one eye may result in **exophoria** (seen in individuals using uniocular microscope and watch makers using uniocular magnifying glass).

Symptoms: Depending upon the symptoms heterophoria can be divided into compensated & decompensated types.

- *Compensated heterophoria*: It is a/w **no** subjective symptoms. Compensation of heterophoria depends upon the reserve neuromuscular power to overcome the muscular imbalance.
- *Decompensated heterophoria*: It is a/w multiple symptoms which may be grouped as under:
  1. Symptoms of muscular fatigue: Headache and eyeache, Difficulty in changing the focus from near to distant objects, Photophobia due to muscular fatigue is not relieved by using dark glasses, but relieved by closing one eye.
  2. Symptoms of failure to maintain binocular single vision: Blurring of words while reading, Intermittent diplopia & Intermittent squint
  3. Symptoms of defective postural sensations cause problems in judging distances and positions especially of the moving objects. This difficulty may be experienced by cricketers, tennis players and pilots during landing.



Maddox rod test

Examination of a case of heterophoria

1. Testing for vision and refractive error
2. **Special Tests**: Cover-uncover test; Prism cover test; Maddox rod test & Maddox wing test

Treatment

1. ***Correction of refractive error***
2. ***Orthoptic Exercises*** – will improve the convergence insufficiency. They can be done with synoptophore.
3. ***Prescription of prism in glasses*** – Prism is prescribed with apex towards the direction of phoria to correct only $\frac{1}{2}$ or at the most $\frac{2}{3}$ of heterophoria.
4. ***Surgical treatment***: It is opted if symptoms are not relieved by other measures. Aim of the surgical management is to strengthen the weak muscle or weaken the strong muscle.

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2) Diplopia [96, 91]

Ans.

Diplopia refers to simultaneous perception of two images of a single object. Diplopia may be binocular or uniocular.

Binocular diplopia: Occurs due to formation of image on dissimilar points of the two retinae.

✪ **Causes of binocular diplopia**:

- Paralysis or paresis of the extraocular muscles (commonest cause)
- Displacement of one eyeball – seen in space occupying lesion in the orbit, & fractures of the orbital wall
- Mechanical restriction of ocular movements – Ex: by thick pterygium, symblepharon and thyroid ophthalmopathy
- Deviation of ray of light in one eye as caused by decentred spectacles
- Anisometropia i.e., disparity of image size between two eyes (e.g., uniocular aphakia with spectacle correction).

✪ **2 Types of Binocular diplopia:**

	Definition	Seen in
1. **Uncrossed (harmonious) diplopia:**	False image is on the same side as deviation	Convergent squint as in lateral rectus paralysis
2. **Crossed (unharmonious) diplopia:**	False image is seen on the opposite side	Divergent squint as in medial rectus paralysis

Uniocular diplopia: Here, object appears double from the affected eye even when the normal eye is closed. Causes of uniocular diplopia are:

- Subluxated lens (pupillary area is partially phakic and partially aphakic).
- Subluxated intraocular lens (pupillary area is partially aphakic and partially pseudophakic).
- Double pupil due to congenital anomaly, or large peripheral iridectomy or iridodialysis.
- Incipient cataract.
- Keratoconus.

Treatment of diplopia: Treat the causative disease.

Temporary relief from annoying diplopia can be obtained by occluding the affected eye.

VSQs

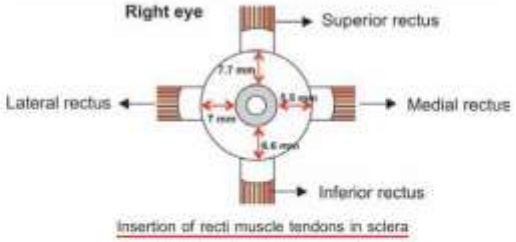
1. Insertion of extra ocular muscles [16]

Ans.

The recti muscles are inserted into the sclera by flat tendons at various distances from the limbus.

Superior oblique ➔ gets inserted into the upper and outer part of the sclera behind the equator.

Inferior oblique ➔ It is inserted into the outer part of the sclera behind the equator.



Insertion of recti muscle tendons in sclera

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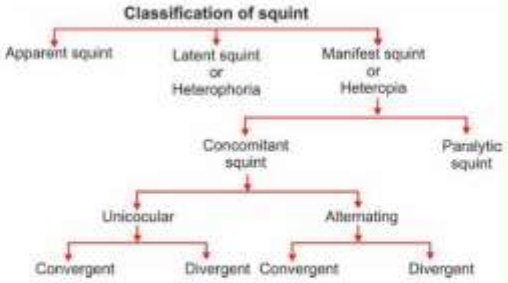
2. Clinical features of concomitant squint [15]

Ans.



1. **Ocular deviation**
   - » Unilateral (monocular squint) or alternating (alternate squint).
   - » Inward deviation (esotropia) or outward deviation (exotropia) or vertical deviation (hypertropia).
   - » Primary deviation (of squinting eye) is equal to secondary deviation (deviation of normal eye under cover when patient fixes with squinting eye).
   - » Ocular deviation is equal in all the directions of gaze.
2. **Ocular movements** are not limited in any direction.
3. **Refractive error** may or may not be associated.
4. **Suppression and amblyopia** may develop as sensory adaptation to strabismus.
5. **A-V patterns** may be observed in horizontal strabismus.

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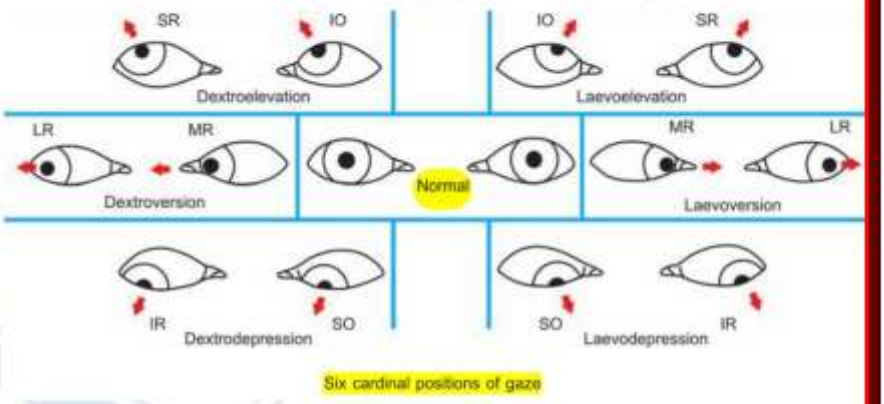
3. Cardinal positions of gaze [15]

Ans.

These are the positions which allow examination of each of the 12 extraocular muscles in their main field of action. There are 6 cardinal positions of gaze, viz, dextroversion, levoversion, dextroelevation, levoelevation, dextrodepression and levodepression



Six cardinal positions of gaze

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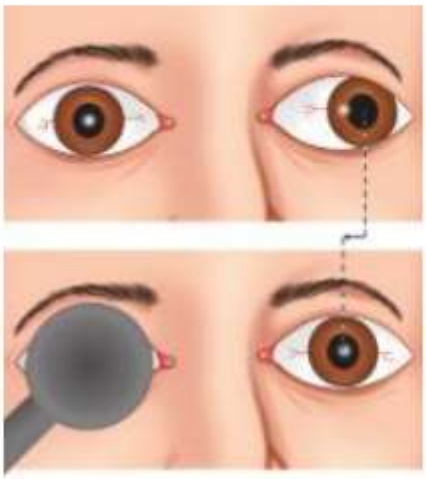
4. Cover test [09]

Ans.



- **Direct cover test** – It confirms the presence of manifest squint. To perform it, the patient is asked to fixate on a point light. Then, the normal looking eye is covered while observing the movement of the uncovered eye. In the presence of squint, the uncovered eye will move in **opposite** direction to take fixation, while in apparent squint there will be no movement.
- **Alternate cover test:** It reveals whether the squint is unilateral or alternate and also differentiates concomitant squint from paralytic squint (where secondary deviation is greater than primary).

g. 14.17 *Direct cover test depicting left exotropia*

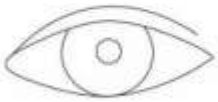
Disorders of Eyelids

SQs

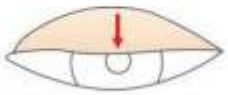
1. Ptosis – types, etiology, C/F & Mx [17, 12, 10]
   a. Myogenic Ptosis [16]

Ans.

Abnormal drooping of the upper eyelid is called ptosis



Normal eye



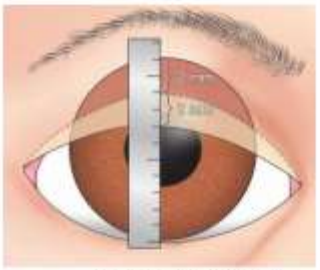
Narrow palpebral aperture

Ptosis

Types of Ptosis		Etiology & C/F
1. Congenital ptosis		• It is due to congenital weakness of the LPS.
2. Acquired ptosis	a. Neurogenic ptosis	Occurs due to innervational defects like: Third nerve palsy, Horner's syndrome & Multiple sclerosis
	b. Myogenic ptosis	Occurs due to acquired disorders of the LPS muscle or of the myoneural junction as seen in myasthenia gravis, dystrophia myotonica, ocular myopathy, muscular dystrophy, following trauma to LPS, thyrotoxicosis, and Lambert-Eaton myasthenia syndrome
	c. Aponeurotic ptosis	It develops due to defects of the levator aponeurosis in the presence of a normal functioning muscle. It includes: → Involutional (senile) ptosis, → Postoperative ptosis (after cataract & retinal detachment surgery), → Traumatic dehiscence of the aponeurosis.
	d. Mechanical ptosis	↪ It may result due to excessive weight on the upper lid as seen in patients with lid tumours, multiple chalazia and lid oedema. ↪ It may also occur due to scarring (cicatricial ptosis) as seen in patients with ocular pemphigoid and trachoma

Clinical Evaluation:

1. **History**: age of onset, family history, history of trauma, eye surgery & variability in degree of ptosis.
2. **Examination**
   1) Exclude pseudoptosis on inspection.
   2) Observe the following points in each case:
      ⇒ Whether ptosis is unilateral or bilateral. **Causes of bilateral ptosis** ➔ congenital ptosis, myasthenia gravis, myotonic dystrophy, Lambert-Eaton myasthenic syndrome etc.
      ⇒ Function of orbicularis oculi muscle.
      ⇒ Eyelid crease is present or absent.
      ⇒ Jaw-winking phenomenon is present or not.
      ⇒ Associated weakness of any extraocular muscle.
      ⇒ Bell's phenomenon (up and outrolling of eyeball during forceful closure) is present or absent.



Measurement of degree of ptosis

   3) Measurement of amount (degree) of ptosis: Mild (2 mm) or Moderate (3 mm) or Severe ptosis (4 mm)
   4) Margin reflex distance (MRD): It is the distance between the upper lid margins and corneal light reflex. Normal value of MRD is 4–5 mm.
   5) Assessment of levator function (Burke's method)
   6) Special investigations:
      a. **Tensilon test**: ptosis improve with i.v. injection of edrophonium (Tensilon) in myasthenia.

b. Phenylephrine test is done if Horner's syndrome is suspected.
c. Neurological investigations done to find out the cause in patient with neurogenic ptosis.

7) Photographic record of the patient should be maintained for comparison.

Treatment

Treatment of Congenital ptosis:

- In severe ptosis, surgery should be performed at the earliest to prevent amblyopia.
- In mild and moderate ptosis, surgery should be delayed until the age of 34 years, when accurate measurements are possible.
- **Surgical Techniques**:

1. Tarso-conjunctivo-Mullerectomy (Fasanella-Servat operation): for mild ptosis (1.5–2 mm)
2. Levator resection – for moderate and severe grades of ptosis. Levator muscle can be resected by either Conjunctival (Blaskowics' operation) or Skin approach (Everbusch's operation)
3. Frontalis sling operation (Brow suspension) – for severe ptosis with no levator function.

Treatment of acquired ptosis

- Treat the underlying cause wherever possible.
- Conservative treatment should be done & surgery deferred at least for 6 months in neurogenic ptosis.
- Surgical procedures for acquired ptosis are same as described for congenital ptosis.

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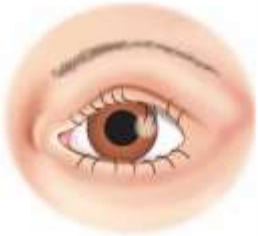
2. Aetiology and complications of Trichiasis [17, 14]

Ans.



It refers to inward misdirection of cilia (which rub against the eyeball) with normal position of the lid margin.

Etiology: cicatrising trachoma, ulcerative blepharitis, healed membranous conjunctivitis, hordeolum externum, mechanical injuries, burns, and operative scar on the lid margin.

CLINICAL FEATURES

Symptoms	Signs
Foreign body sensation & photophobia. Patient may feel irritation, pain and lacrimation	❖ ≥ 1 Misdirected cilia touching the cornea. ❖ Reflex blepharospasm and photophobia occur. ❖ Conjunctiva may be congested. ❖ Signs of causative disease viz. trachoma, blepharitis, etc. may be present

Complications: recurrent corneal abrasions, superficial corneal opacities, corneal vascularisation and non-healing corneal ulcer.

Treatment:

1. Epilation (mechanical removal with forceps) – recurrence occurs within 3–4 weeks.
2. Electrolysis –lash follicles are destroyed by electric current → loosened cilia are then removed with epilation forceps.
3. Cryoepilation: Its main disadvantage is depigmentation of the skin.
4. Surgical correction – if many cilia are misdirected, surgical correction similar to cicatricial entropion should be used.

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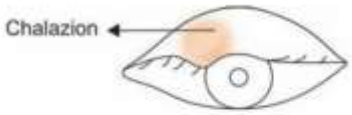
3. Chalazion [15, 13, 11, 10, 07, 06]

Ans.



Chalazion, also called a tarsal or meibomian cyst, is a chronic non-infective (non-suppurative) lipogranulomatous inflammation of the meibomian gland. This is the commonest of all lid lumps. **Etiopathogenesis**

» Predisposing factors are similar to hordeolum externum.

» Mild infection of the meibomian gland by very low virulent organisms→ proliferation of the epithelium and infiltration of the duct walls, which are blocked → retention of secretions (sebum) in the gland → enlargement of the blocked meibomian glands and surrounding tissue.

Clinical features

Symptoms:

- Painless swelling in the eyelid, gradually increasing in size.
- Mild heaviness in the lid may be felt.
- Large chalazion → press on the cornea → induce astigmatism → Blurred vision.
- Large chalazion of lower eyelid → eversion of lower punctum → Watering (epiphora)

Signs:

- Nodule – present slightly away from lid margin, firm to hard and non-tender on palpation.
- Upper lid is involved more commonly than the lower lid (upper lid contains more meibomian glands than the lower lid).
- Reddish purple area is seen on the palpebral conjunctiva after eversion of the lid.
- Marginal chalazion, occurring occasionally, may present as small reddish grey nodule on lid margin.

Clinical course and complications

- Slow increase in size is often seen.
- If the lesion bursts on the conjunctival side → Fungating mass of granulation tissue may be formed.
- Secondary infection may lead to formation of hordeolum internum.
- Calcification may occur.
- Malignant change into meibomian gland adenocarcinoma (sebaceous cell carcinoma) may be seen occasionally in elderly people. In recurrent chalazion malignancy should be ruled out
- Complete spontaneous resolution may occur rarely

Treatment

1. Conservative treatment – In small, soft and recent chalazion, self-resolution may be helped by conservative treatment in the form of hot fomentation, topical antibiotic eyedrops and oral anti-inflammatory drugs.
2. Intralesional injection of long-acting steroid (triamcinolone) – resolution in about 50% cases.
3. Incision and curettage: It is the conventional and effective treatment for chalazion.
4. Diathermy. – Better option for marginal chalazion.
5. Oral tetracycline should be given as prophylaxis in recurrent chalazia, especially if associated with acne rosacea or seborrhoeic dermatitis.

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4. Lagophthalmos [12, 99]

Ans.

It is characterised by inability to close the eyelids voluntarily.

Etiology:

⇒ It occurs in patients with paralysis of orbicularis oculi muscle, cicatricial contraction of the lids, symblepharon, severe ectropion, proptosis, following over-resection of the levator muscle for ptosis, and in comatosed patients.

⇒ Physiologically some people sleep with their eyes open (nocturnal lagophthalmos).

Clinical features: incomplete closure of the palpebral aperture a/w features of the causative disease.

Complications: conjunctival and corneal xerosis and exposure keratitis.

Treatment:

1) Measures to prevent exposure keratitis
   - Artificial tear drops & antibiotic eye ointment (esp. during sleep and in comatosed patients).
   - Soft bandage contact lens.
   - Tarsorrhaphy may be performed to cover the exposed cornea when indicated.
2) Measures to treat the cause of lagophthalmos, wherever possible should be taken.

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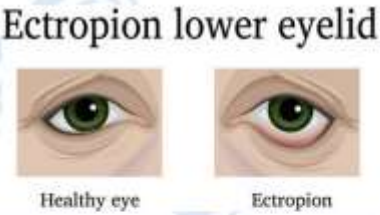
5. Ectropion [12]

Ans.



Out rolling or outward turning of the lid margin is called ectropion

Types of Ectropions	Etiopathogenesis
1. Congenital ectropion	Seen in Down syndrome and blepharophimosis syndrome & is due to congenital shortage of the skin
2. Cicatricial ectropion	It occurs due to scarring of the skin as seen in thermal burns, chemical burns, lacerating injuries and skin ulcers
3. Senile (involutional) ectropion	Affects only the lower lid in elder people. Factors which contribute for its development are: • Horizontal laxity of the lid due to weakening of orbicularis muscle. • Medial canthal tendon laxity • Lateral canthal tendon laxity • Disinsertion of lower lid retractors.
4. Mechanical ectropion	It occurs in conditions where either the lower lid is pulled down (as in tumours) or pushed out and down (as in proptosis and chemosis of conjunctiva).
5. Paralytic ectropion	It mainly occurs in the lower lids & is due to paralysis of the seventh nerve as seen in Bell's palsy, head injury, infections of the middle ear and operations on parotid gland

Clinical features

Symptoms

- Epiphora is the main symptom in ectropion of the lower lid.
- Symptoms due to associated chronic conjunctivitis: irritation, discomfort and mild photophobia.

Signs

- Lid margin is outrolled – Depending upon degree of outrolling, ectropion can be divided into 3 grades:
  - Grade I: In it only punctum is everted.
  - Grade II: Lid margin is everted and palpebral conjunctiva is visible.
  - Grade III: The fornix is also visible.
- Signs of the etiological condition such as:
  - Skin scars in cicatricial ectropion and
  - 7th nerve palsy in paralytic ectropion may also be seen.

❖ In involutional ectropion, the following signs can be elicited:

- Horizontal lid laxity ➔ positive snap test, i.e., lid can be easily pulled away from the globe but fails to snap back to the normal position on release.
- Medial canthal tendon laxity: Normally on pulling the lid laterally the inferior punctum moves by 1–2 mm only; while it can the moved upto limbus in medial tendon laxity.
- Lateral canthal tendon laxity is evidenced by its > 2 mm movement on pulling the lid medially.

Complications

- Dryness and thickening of conjunctiva and corneal ulceration (exposure keratitis) may occur due to prolonged exposure.
- Eczema and dermatitis of the lower lid skin may occur due to prolonged epiphora.

Treatment

1. **Congenital ectropion**: Mild ectropion need no treatment. Moderate or severe ectropion is treated with horizontal lid tightening & full thickness skin graft to vertically lengthen anterior lamella.
2. **Cicatricial ectropion**: Depending upon the degree it can be corrected by any of the following plastic operations:
   a. V-Y operation – for mild degree ectropion
   b. Z-plasty (Elschnig's operation) – for mild to moderate degree of ectropion.
   c. Excision of scar tissue and full thickness skin grafting – It is performed in severe cases.
3. **Involutional ectropion**. Depending upon the severity, following 3 operations are done:
   a) Medial conjunctivoplasty – for mild degree of ectropion.
   b) Horizontal lid shortening – for moderate degree of ectropion.
   c) Byron Smith's modified Kuhnt-Szymanowski operation – done in severe degree of ectropion.
   d) Lateral tarsal strip technique – useful for generalized ectropion a/w horizontal lid laxity.
4. **Paralytic ectropion**:

- Often resolves spontaneously within 6 months. Therefore, temporary measures are taken initially which include:
  a) Topical lubricants,
  b) Taping temporal side of eyelid, and
  c) Suture tarsorrhaphy
- Permanent surgical treatment is required only in non-resolving cases. This includes:
  - Horizontal lid tightening or
  - Palpebral sling operation.

5. **Mechanical ectropion**. It is corrected by treating underlying mechanical force causing ectropion.

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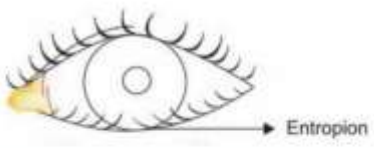
6. Entropion [10]

Ans.

Entropion refers to inward rolling and rotation of the lid margin toward globe.



Types of Entropions	Etiopathogenesis
1. **Congenital entropion**	- Lower eyelid congenital entropion is caused by improper development of the lower lid retractors. - Upper eyelid congenital entropion is secondary to mechanical effects of microphthalmos

2. Cicatricial entropion	It is caused by cicatricial contraction of the palpebral conjunctiva as seen in trachoma, membranous conjunctivitis, chemical burns, pemphigus and Stevens-Johnson syndrome
3. Senile (involutional) entropion	Affects only the lower lid in elder people. Factors which contribute for its development are: ↳ Horizontal laxity of the lid due to weakening of orbicularis muscle. ↳ Vertical lid instability due to weakening of lower lid retractor. ↳ Over-riding of pretarsal orbicularis. ↳ Laxity of orbital septum along with prolapse of orbital fat into the lower lid
4. Mechanical entropion	It occurs due to lack of support provided by the globe to the lids as seen in phthisis bulbi, enophthalmos and after enucleation or evisceration operation

Clinical features

- **Symptoms** occur due to rubbing of cilia against the cornea and conjunctiva and are thus similar to trichiasis. These include foreign body sensation, irritation, lacrimation and photophobia.
- **Signs are as follows:**
  - **In-turning of lid margins**: Depending upon the degree of inturning, it can be divided into 3 grades:
    - Grade I entropion – only the posterior lid border is inrolled,
    - Grade II entropion – Inturning up to the inter-marginal strip, and
    - Grade III entropion – whole lid margin including the anterior border is inturned.
  - **Signs of causative disease**, e.g., scarring of palpebral conjunctiva in cicatricial entropion, and horizontal lid laxity in involutional entropion may be seen.
  - **Signs of complications**: recurrent corneal abrasions, superficial corneal opacities, corneal vascularization and even corneal ulceration.

Treatment

1. Congenital entropion may resolve with time without need of any intervention or may require excision of a strip of skin and muscle with plastic reconstruction of the lid crease (Hotz procedure).
2. Cicatricial entropion:
   - It is treated by a plastic operation, which is based on any of the following principles: Altering the direction of lashes, or transplanting the lashes, or straightening the distorted tarsus.
   - Surgical techniques employed for correcting cicatricial entropion are as follows:
     a) Anterior lamellar resection – done in mild entropion
     b) Tarsal wedge resection.
     c) Transposition of tarsoconjunctival wedge. (Modified Ketssey's operation)
     d) Posterior lamellar graft – done in severe entropion.
3. Senile entropion: Surgical techniques are as below:
   a. Transverse everting suture – for temporary cure in very old patients
   b. Wies operation (Transverse lid split and everting sutures) – for long term cure in patients with little horizontal laxity.
   c. Plication of lower lid retractors **(Jones operation)**: It is performed in severe cases or when recurrence occurs after the above-described operations.
   d. **Quickert procedure**: This is indicated in patients having associated marked horizontal lid laxity. Quickert procedure basically combines horizontal lid shortening with Weis procedure.
4. Mechanical entropion – It is corrected by treating underlying mechanical force causing entropion

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7. Hordeolum Internum [09, 04, 02]

Ans.

It is a suppurative inflammation of the meibomian gland associated with blockage of the duct.

Etiology

» Hordeolum internum may occur as: Primary Staphylococcal infection of the meibomian gland or due to Secondary infection in a chalazion (infected chalazion)

» Predisposing factors are similar to hordeolum externum.

Clinical features

- Symptoms: similar to hordeolum externum, except that pain is more intense, due to the swelling being embedded deeply in the dense fibrous tissue.
- Signs: a localized, firm, red, tender swelling of the lid associated with marked oedema
  - Point of maximum tenderness and swelling is away from the lid margin and that pus usually points on the tarsal conjunctiva (seen as yellowish area on everting the lid)

Treatment: It is similar to hordeolum externum, except that, when pus is formed, it should be drained by a vertical incision from the tarsal conjunctiva.

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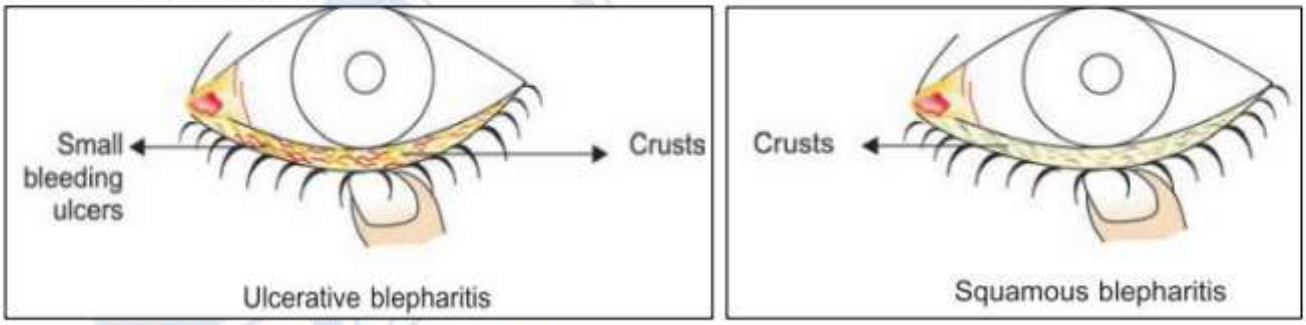
8. Blepharitis [08, 05, 01]
   a. Squamous blepharitis – etiology, signs, symptoms & Rx [16]
   b. Ulcerative Blepheritis [02]

Ans.

Blepharitis is a subacute or chronic inflammation of the lid margins. It can be clinically divided into:

1. Bacterial blepharitis,
2. Seborrhoeic or squamous blepharitis,
3. Mixed Bacterial & seborrhoeic blepharitis,
4. Posterior blepharitis or meibomitis, and
5. Parasitic blepharitis



Parasitic Blepharitis (Lash Infestation)

Etiology: It is seen in persons living in poor hygienic conditions → infestations of lashes with lice

Clinical features:

- Symptoms: chronic irritation, itching, burning, and mild lacrimation.
- Signs:
  - Lid margins are red and inflamed.
  - Lice anchoring the lashes with their claws may be seen on slit-lamp examination.
  - Nits (eggs) may be seen as opalescent pearls adherent to the base of cilia.

Treatment – Mechanical removal of the lices and nits with forceps.

» Abx eye ointment + Delousing of the patient & family members is done to prevent recurrences.

ANTERIOR BLEPHARITIS

	BACTERIAL BLEPHARITIS – aka chronic anterior blepharitis, or staphylococcal blepharitis or ulcerative blepharitis	SEBORRHOEIC or SQUAMOUS BLEPHARITIS:
Etiology	**Causative organisms** are ➔ Coagulase +ve Staphylococci (MC). ➔ Rarely, Streptococci, Propionibacterium acnes & Moraxella.	It is a/w seborrhoea of scalp (dandruff). → Glands of Zeis secrete abnormal excess lipids which are split by Corynebacterium acne into irritating free fatty acids.
Symptoms	Chronic irritation, itching, mild lacrimation, gluing of cilia, and mild photophobia. The symptoms are characteristically worse in the morning	whitish material (soft scales) deposit at the lid margin → irritation, occasional watering H/o falling of eyelashes
Signs	1. **Yellow crusts** are seen at the root of cilia which glue them together. 2. Small ulcers, which bleed easily, are seen on removing the crusts. 3. Red, thickened lid margins are seen with dilated blood vessels (rosettes). 4. Mild papillary conjunctivitis and conjunctival hyperemia.	⇒ Accumulation of white dandruff-like scales is seen on the lid margin, among the lashes. ⇒ The lashes fall out easily but are usually replaced quickly without distortion. ⇒ Lid margin is thickened and the sharp posterior border tends to be rounded leading to epiphora, in long standing cases.
Complications	a) Lash abnormalities: madarosis (sparseness or absence of cilia), trichiasis (misdirected cilia), and poliosis (greying of lashes). b) **Tylosis** – thickening and scarring of lid margin. c) Eversion of punctum leading to epiphora. d) Eczema of skin and ectropion may develop due to prolonged watering. e) Recurrent styes (external hordeola). f) Marginal keratitis & phlyctenulosis g) Tear film instability, and dry eye. h) Secondary inflammatory & mechanical changes in the conjunctiva and cornea.	
Treatment	1) **Lid hygiene**: » Warm compresses for 5–10 minutes to soften the crusts, » Remove Crust & clean lid margin with 3% $NaHCO_3$ » Avoid rubbing of the eyes 2) **Antibiotics**: ↳ Abx eye drops: 3–4 times/day. ↳ Oral antibiotics - used in unresponsive patients 3) **Topical steroids** (weak) such as fluorometholone – for patients with papillary conjunctivitis, marginal keratitis & phlyctenulosis. 4) **Artificial tear drops** - for dry eyes	1. Associated seborrhoea of the scalp should be adequately treated. 2. Local measures: removal of scales from the lid margin with the help of lukewarm solution of 3% $NaHCO_3$ 3. Antibiotics, as described in bacterial blepharitis, may be required in patients with mixed seborrhoeic and bacterial blepharitis.

Posterior Blepharitis (Meibomitis): inflammation of Meibomian glands occurs in chronic & acute forms.

- **Chronic meibomitis:**
  - Seen in middle aged persons, especially those with acne rosacea and/or seborrhoeic dermatitis.
  - Bacterial lipases play main role in the pathogenesis of chronic meibomitis.
  - **Clinical features**
    - Symptoms: chronic irritation, burning, itching, grittiness, mild lacrimation with remissions and exacerbations intermittently. Symptoms are characteristically worse in the morning.
    - Signs:
      1. White frothy (foam like) secretions are seen on the eyelid margins and canthi (meibomian seborrhoea).
      2. Opening of meibomian glands become prominent with thick secretions which can be expressed out by pressure on the lids giving toothpaste appearance.
      3. Vertical yellowish streaks shining through conjunctiva can be seen on eversion of the lids. These represent the meibomian ducts filled with thick secretion.
      4. Oily and foamy tear film with accumulation of froth on the lid margins or inner canthus.
      5. **Secondary changes:** papillary conjunctivitis, and inferior corneal punctate epithelial erosions.
- **Acute meibomitis:** it occurs due to staphylococcal infection. It is characterized by painful swelling. Pressure on it results in expression of pus followed by serosanguinous discharge.

Treatment of meibomitis

1. Lid hygiene: Warm compresses & Expression of accumulated secretions by repeated vertical massage of lids in the form of milking.
2. **Topical antibiotics** in the form of eye ointment should be rubbed at the lid margin immediately after massage, and Eye drops may be used 3–4 times a day.
3. **Systemic tetracyclines**, e.g., doxycycline 100 mg b.d. for 1 week and then o.d. for 6–12 weeks (to block staphylococcal lipase production).
4. **Erythromycin** may be used where tetracyclines are contraindicated.
5. **Ocular lubricants** – artificial tear drops.
6. **Topical steroids** (weak) such as fluorometholone – required in patients with papillary conjunctivitis.

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9. Symblepharon [03]

Ans.

In this condition, lids become adherent with the eyeball as a result of adhesions between the palpebral and bulbar conjunctiva.

Etiology: It results from healing of the kissing raw surfaces upon the palpebral and bulbar conjunctiva.

- Common causes are thermal or chemical burns, membranous conjunctivitis, injuries, conjunctival ulcerations, ocular pemphigus and Stevens-Johnson syndrome.

Clinical features:

1. Ocular movements become restricted,
2. Diplopia may be experienced due to restricted ocular motility,
3. Lagophthalmos, i.e., inability to close the lids may occur due to adhesions.
4. Cosmetic disfigurement is a common complaint.
5. Types of symblepharon, depending upon the extent of adhesions, are as below:

Types of symblepharon

- Anterior symblepharon—adhesions present only in the anterior part
- Posterior symblepharon—adhesions present in the fornices.
- Total symblepharon—adhesions involving whole of the lid.

Complications: Dryness, thickening and keratinisation of conjunctiva due to prolonged exposure and corneal ulceration (exposure keratitis).

Treatment

1. Prophylaxis: During the stage of raw surfaces, the adhesions may be prevented by:
   - Sweeping a glass rod coated with lubricant around the fornices several times a day
   - Therapeutic soft contact lens of large size, also helps in preventing the adhesions.
2. Curative treatment consists of symblepharectomy. The raw area created may be covered by:
   - Mobilising the surrounding conjunctiva in mild cases.
   - Conjunctival or buccal mucosal graft is required in severe cases.
   - Amniotic membrane transplantation (AMT), also gives good results

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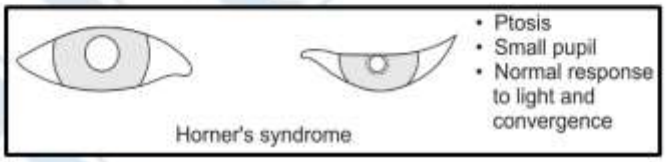
VSQs

1) Homer's Syndrome [11]

Ans.

Horner's syndrome, occurring due to oculo-sympathetic paresis, is characterised by classic triad of:

1. Mild ptosis (due to paralysis of Muller's muscles),
2. Miosis (due to paralysis of dilator pupillae), and
3. Reduced ipsilateral sweating (anhydrosis),



- Other features include mild enophthalmos, loss of cilio-spinal reflex, heterochromia, i.e., ipsilateral iris is lighter in color, pupil is slow to dilate, and there occurs slight elevation of the lower eyelid

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2) Indications of Tarsorrhaphy [09]

Ans.

In this operation, adhesions are created between a part of the lid margins with the aim to narrow down or almost close the palpebral aperture

Indications of TEMPORARY Tarsorrhaphy	Indications of PERMANENT Tarsorrhaphy
1) To protect the cornea when 7th nerve palsy is expected to recover.	1. Cases of VII nerve palsy where there is no chance of recovery; and
2) To assist healing of an indolent corneal ulcer.	2. Established cases of neuroparalytic keratitis with severe loss of corneal sensations
3) To assist in healing of skin-grafts of the lids in the correct position.	

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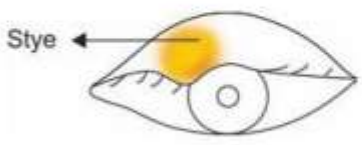
3) Hordeolum Externum (stye) [08]

Ans.

It is an acute suppurative inflammation of lash follicle and its associated glands of Zeis or Moll.



Etiology:

Causative organism: Staphylococcus aureus (MC).

Predisposing factors:

- Age – more common in children and young adults.
- Habitual rubbing of the eyes or fingering of the lids and nose, chronic blepharitis and DM.
- Chronic debility, excessive intake of carbohydrates and alcohol.

Clinical features

Symptoms: acute pain a/w swelling of lid, mild watering and photophobia.

Signs:

- Stage of cellulitis – localised, firm, red, tender swelling at the lid margin.
- Stage of abscess formation – visible pus point on the lid margin in relation to the affected cilia.

Treatment

1) Hot compresses 2–3 times a day – useful especially in cellulitis stage.
2) Clear the pus by epilating the involved cilia.
3) Surgical incision is required for a large abscess.
4) Antibiotic eye drops, eye ointment & Systemic Abx should be used for early control of infection
5) Systemic anti-inflammatory and analgesics relieve pain and reduce oedema.
6) In recurrent styes, try to find out and treat the associated predisposing condition.

---

4) List the swellings in the lid [07]

Ans.

Oedema (swelling) of the lids is classified as inflammatory, solid and passive oedema:

- **Inflammatory oedema**: It is seen in the following conditions.
  1) Inflammations of the lid itself – dermatitis, stye, hordeolum internum, insect bites, cellulitis and lid abscess.
  2) Inflammations of the conjunctiva – acute conjunctivitis.
  3) Inflammations of the lacrimal sac, i.e., lacrimal abscess.
  4) Inflammations of the lacrimal gland, i.e., acute dacryoadenitis.
  5) Inflammations of the eyeball – acute iridocyclitis, endophthalmitis and panophthalmitis.
  6) Inflammations of the orbit – orbital cellulitis, orbital abscess and pseudotumour.
  7) Inflammations of the paranasal sinuses, e.g., maxillary sinusitis.
- **Solid oedema of the lids**. It is chronic thickening of the lids, which follows recurrent attacks of erysipelas.
- **Passive oedema of the lids**. It may occur due to local or general causes.
  - **Local causes**: cavernous sinus thrombosis, head injury and angioneurotic oedema.
  - **General causes**: congestive heart failure, renal failure, hypoproteinaemia and severe anaemia

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Diseases of Lacrimal Apparatus

LQs

1) Chronic Dacryocystitis – etiology, C/F, complications & Mx [17, 10, 03]
   a. Indications for Dacryocystorhinostomy (DCR) [08]
   b. Indications of dacryocystectomy [07]
   c. D.C.R. [96]

Ans.

Chronic Dacryocystitis is a chronic suppurative inflammation of the lacrimal sac due to the obstruction of the nasolacrimal duct.

Etiology: It occurs due to a vicious cycle of stasis and mild infection of long duration.

✪ **Predisposing factors**

1. Age - more common in elderly (40-60 yrs).
2. Sex: predominantly seen in females (80%) (due to narrow lumen of the bony canal).
3. Heredity.
4. Low Socio-economic status & Poor personal hygiene.

✪ **Factors responsible for stasis of tears in lacrimal sac**

1. Anatomical factors → retard drainage of tears – Ex: narrow bony canal of NLD etc.
2. Foreign bodies in the sac → block opening of NLD.
3. Excessive lacrimation causes stagnation of tears in the sac.
4. Mild grade inflammation of lacrimal sac due to recurrent conjunctivitis may block the NLD.
5. Obstruction of lower end of the NLD by nasal polyps, DNS, tumours, atrophic rhinitis etc.

✪ **Source of infection**: Lacrimal sac may get infected from the conjunctiva, nasal cavity (retrograde spread), or paranasal sinuses.

✪ **Causative organisms**: Staphylococci, Streptococci, Pneumococci and Pseudomonas pyocyanea. Rarely chronic granulomatous infections like TB, syphilis, leprosy and occasionally Rhinosporidiosis.

Clinical features: They can be divided into 4 stages:

1. Stage of chronic catarrhal dacryocystitis: Mild inflammation of lacrimal sac → block NLD.
   - ➢ Watering eye is the only symptom in this stage
   - ➢ On syringing the lacrimal sac, either clear fluid or few fibrinous mucoid flakes regurgitate.
   - ➢ Dacryocystography reveals block in NLD.
2. Stage of lacrimal mucocele. It follows chronic stagnation causing distension of lacrimal sac.
   - ▪ Here constant epiphora & a swelling is seen just below the inner canthus
   - ▪ Regurgitation test: Milky or gelatinous mucoid fluid regurgitates from the lower punctum on pressing the swelling.
   - ▪ Dacryocystography at this stage reveals a distended sac with blockage somewhere in the NLD.
   - ▪ *Encysted mucocele*: Sometimes due to chronic infection, opening of both the canaliculi into the sac are blocked and a large fluctuant swelling is seen at the inner canthus with a negative regurgitation test. This is called encysted mucocele.
3. Stage of chronic suppurative dacryocystitis: Due to pyogenic infection, the mucoid discharge becomes purulent, converting the mucocele into 'pyocoele'.
   - ♦ Here epiphora, recurrent conjunctivitis & swelling at the inner canthus with mild erythema of the overlying skin is seen.
   - ♦ Regurgitation test: a frank purulent discharge flows from the lower punctum.
   - ♦ If openings of canaliculi are blocked at this stage the so called encysted pyocoele results.

4. **Stage of chronic fibrotic sac**: repeated infections → thickening of mucosa → small fibrotic sac due to persistent epiphora and discharge. Dacryocystography, at this stage reveals a very small sac with irregular folds in the mucosa.

Complications

- Chronic intractable conjunctivitis; Acute on chronic dacryocystitis.
- Ectropion of lower lid; maceration and eczema of lower lid skin due to prolonged watering.
- Corneal ulceration.
- High risk of developing endophthalmitis if an intraocular surgery is performed in the presence of dacryocystitis.

Treatment

a. **Conservative treatment** by probing & lacrimal syringing.

b. **Balloon catheter dilation** (aka balloon dacryocystoplasty) – done in patients with partial NLD obstruction.

c. **Dacryocystorhinostomy (DCR)**: It is the operation of choice as it re-establishes the lacrimal drainage. DCR can be done by external or endoscopic approach.

d. **Dacryocystectomy (DCT)**: It should be performed only when **DCR** is contraindicated.

   Indications of DCT include:
   - Too old patient.
   - Markedly shrunken and fibrosed sac.
   - TB, syphilis, leprosy or mycotic infections of sac.
   - Tumours of sac.
   - Gross nasal diseases like atrophic rhinitis

e. **Conjunctivodacryocystorhinostomy (CDCR)**: It is performed in the presence of blocked canaliculi.

Endonasal DCR	*External DCR*
Advantages	*Disadvantages*
• No external scar	• Cutaneous scar
• Relatively blood less surgery	• Relatively more bleeding during surgery
• Better visualisation of nasal pathology	
• Less chances of injury to ethmoidal vessels and *cribriform* plate	• Potential injury to adjacent medial canthus structures
• Less time consuming (15–30 minutes) since nasal mucosal flaps and sac wall flaps are not made	• More operating time (45–60 minutes)
• No postoperative morbidity	• Significant postoperative morbidity
Disadvantages	*Advantages*
• Less success rate (70–90%)	• More success rate (95%)
• Requires skilled ophthalmologist and/or rhinologist • Expensive equipment	• Easily performed by ophthalmologists • Cheap (expensive equipment not required)
• Requires reasonable access to middle meatus and familiarity with endoscopic anatomy	• Does not require familiarity with endoscopic anatomy

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2) Acute Dacryocystitis – etiology, C/F, complications & Mx [14, 90]
   a. 4 complications of acute dacryocystitis [15]
   b. Lacrimal Abscess [01]

Ans.

Acute Dacryocystitis is the acute suppurative inflammation of the lacrimal sac.

Etiology

- Causative organisms: Streptococcus haemolyticus, Pneumococcus and Staphylococcus
- Acute dacryocystitis may develop in 2 ways:
  1. As an acute exacerbation of chronic dacryocystitis.
  2. As an acute peridacryocystitis due to neighbouring infected structures such as: paranasal sinuses etc.

Clinical features – can be divided into 3 stages:

1) **Stage of cellulitis**: It is characterised by a painful swelling in the region of lacrimal sac a/w epiphora, fever and malaise. The swelling is red, hot, firm and tender. Redness and oedema also spread to the lids & cheek.
2) **Stage of lacrimal abscess**: Continued inflammation → occlusion of the canaliculi due to oedema. Lacrimal sac is filled with pus, distends and its anterior wall ruptures → Lacrimal abscess.
3) **Stage of fistula formation**: When the lacrimal abscess is left un-treated, it discharges, leaving an external fistula below the medial palpebral ligament. Rarely, the abscess may open up into the nasal cavity forming an internal fistula.

Complications: Acute conjunctivitis, Lid abscess, Osteomyelitis of lacrimal bone, Orbital cellulitis, Conversion of corneal abrasion into corneal ulceration, Facial cellulitis etc.

Treatment

1. **During cellulitis stage** – systemic & topical Abx to control infection; Analgesics & hot fomentation to relieve pain and swelling.
2. **During stage of lacrimal abscess**, in addition to the above treatment, pus should be should be gently squeezed out and dressing should be done with betadine-soaked gauze. Later on, either DCT or DCR operation should be carried out, otherwise recurrence will occur.
3. **Treatment of external lacrimal fistula**: After controlling the acute infection with systemic Abx, fistulectomy along with DCT or DCR operation should be performed.

SQs

1. Epiphora [97]
   a. Causes of Epiphora [12]

Ans.

Epiphora is the watering of eyes due to inadequate drainage (outflow) of normally secreted tears.

Causes of epiphora:

1) **Physiological cause** is ' lacrimal pump' failure due to lower lid laxity or weakness of orbicularis muscle.
2) **Mechanical obstruction** in lacrimal passages may lie at the level of punctum, canaliculus, lacrimal sac or NLD.

a. **Punctal causes**:
   - Eversion of lower punctum: seen in old age (due to laxity of the lids), chronic conjunctivitis etc.
   - Punctal obstruction:
     - Congenital absence of puncta
     - a small foreign body can also block the punctum.
     - Prolonged use of drugs like idoxuridine and pilocarpine is also a/w punctal stenosis.

b. **Canalicular obstruction**: It may be congenital or acquired due to FB, trauma, canaliculitis due to actinomyces etc.

c. **Causes in the lacrimal sac**: congenital mucous membrane folds, traumatic strictures, dacryocystitis, infections like TB and syphilis, dacryolithiasis and tumours.

d. **Causes in the NLD**:
   - Congenital causes: non-canalization, partial canalization or imperforated membranous valves.
   - Acquired causes: traumatic strictures, inflammatory strictures, idiopathic stenosis, tumours etc.

Diagnosis: via Dacryocystography. It tells the exact site, nature and extent of block.

Radionucleotide Dacryocystography (lacrimal scintillography): It is a non-invasive technique to assess the functional efficiency of lacrimal drainage apparatus.

VSQs

1. Causes of dry eye [16]

Ans.

"Dry eye is a multifactorial disease of the ocular surface characterized by loss of homoeostasis of the tear film, and accompanied by ocular symptoms, in which tear film instability and hyperosmolarity, ocular surface inflammation and damage, and neurosensory abnormalities play etiological roles".

Holly and Lemp classified dry eye in 1977. It can be classified as follows

Aqueous deficiency	Lipid deficiency	Mucin deficiency
Keratoconjunctivitis sicca (KCS)	Evaporative dry eye	
Causes		
Sjögren's syndrome	Meibomian gland disease (MGD)	Vitamin A deficiency
Non-Sjögren's	Posterior blepharitis	Pemphigoid
Age-related hyposecretion	Rosacea	Stevens–Johnson syndrome
Congenital alacrima	Atopic keratoconjunctivitis	Chemical burns
Lacrimal gland disease	Low blink rate (Bell's palsy)	Trachoma
Riley day syndrome	Contact lens wear	

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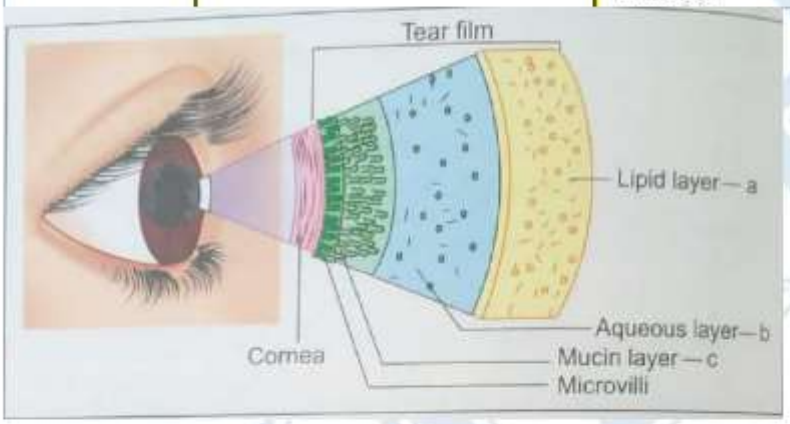
2. Tear film [10]

Ans.

Tear Film consist of 3 layers, which from *anterior to posterior* are Lipid layer, Aqueous layer & Mucus layer

	Lipid Layer	Aqueous Layer	Mucous layer
Secreted by	Meibomian, zeis & moll glands	Lacrimal Glands	Goblet cells & Gland of Manz
Functions	↓ evaporation & lubricates the eyelids	Supplies atmospheric O_2 to cornea & has antibacterial function	Converts hydrophobic corneal surface to hydrophilic



CONTENTS

Disease of Orbit

SQs

1. Orbital Cellulitis – C/F & Complications [15, 07, 05]

Ans.

Orbital cellulitis refers to an acute infection of the soft tissues of the orbit behind the orbital septum

Etiology

1. **Exogenous infection** – from penetrating injury, retention of intraorbital foreign body, and following operations like evisceration, enucleation, dacryocystectomy and orbitotomy.
2. **Extension of infection** from neighbouring structures (MC) like paranasal sinuses, teeth, face, lids, intracranial & intraorbital structures.
3. **Endogenous infection**: It may rarely develop as metastatic infection from breast abscess, puerperal sepsis, thrombophlebitis of legs and septicaemia.

Causative organisms: Staphylococcus aureus, Streptococcus pneumoniae, Streptococcus pyogenes & Haemophilus influenzae.

Pathology – similar to suppurative inflammations in general, except for the following special features:

⇒ Infection establishes early due to absence of lymphatics in the orbit.

⇒ Damage produced is extensive as orbital infection is associated with raised intraorbital pressure due to the tight compartment.

Clinical features of Orbital Cellulitis	
Symptoms	**Signs**
» Swelling & severe pain (↑ by movements of the eyeball) » Associated general symptoms → fever, nausea, vomiting and prostrations. » Vision loss ± diplopia may also occur	1. Swelling of lids – woody hardness & redness. 2. Restriction of ocular movements 3. Chemosis of conjunctiva 4. Axial proptosis of varying degree is present. 5. RAPD may occur. 6. Fundus examination may show congestion of retinal veins and signs of papillitis or papilloedema.

Complications:

1. **Ocular complications** → vision loss due to exposure keratopathy, optic neuritis & CRAO.
2. **Orbital complications** → subperiosteal abscess & orbital abscess – diagnosed by CT scan
   ⇒ Subperiosteal abscess is a collection of pus between the orbital bony wall and periosteum.
   ⇒ Orbital abscess is collection of pus within the orbital soft tissue.
3. **Temporal or parotid abscesses** may occur due to spread of infection around the orbit.
4. **Intracranial complications** → cavernous sinus thrombosis, meningitis & brain abscesses.
5. **General** septicemia or pyaemia

Investigations

1) Bacterial cultures from nasal swabs, conjunctival swabs & blood samples.
2) CBP may reveal leukocytosis.
3) X-ray PNS to identify associated sinusitis.
4) Orbital ultrasonography to detect intraorbital abscess.
5) CT scan and MRI are useful:
   - In differentiating preseptal and postseptal cellulitis;
   - In detecting subperiosteal & orbital abscesses,

- In detecting intracranial extension; and
- In deciding when and from where to drain an orbital abscess.

Treatment

Orbital cellulitis is an emergency and so the patient should be hospitalised for aggressive Mx.

1) **Intensive Abx therapy**: based on susceptibility
   ⇒ For Staphylococcal infections – oxacillin + ampicillin.
   Cefotaxime, ciprofloxacin or vancomycin may be used alternatively.
   ⇒ To cover H. influenzae esp. in children, chloramphenicol or clavulanic acid should also be added.
   ⇒ To cover anaerobes oral metronidazole 500 mg every 8 hours should be added.
2) **NSAIDs** are helpful in controlling pain and fever.
3) **Topical antibiotic eye ointment** – if there is severe proptosis.
4) **Nasal decongestants**
5) **Revaluation**, at least 2-3 times daily in the hospital, is required to monitor the response and modify the treatment accordingly.
6) **Surgical intervention**: Its indications include unresponsiveness to antibiotics, decrease in vision and presence of an orbital or subperiosteal abscess.
   - *Immediate canthotomy/cantholysis* – If the orbit is tight, optic neuropathy is present or ↑ IOP.
   - Subperiosteal abscess is drained by a 2- 3 cm curved incision in the upper medial aspect.
   - In most cases, it is necessary to drain both the orbit as well as the infected paranasal sinuses.

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2. Cavernous sinus thrombosis [13]

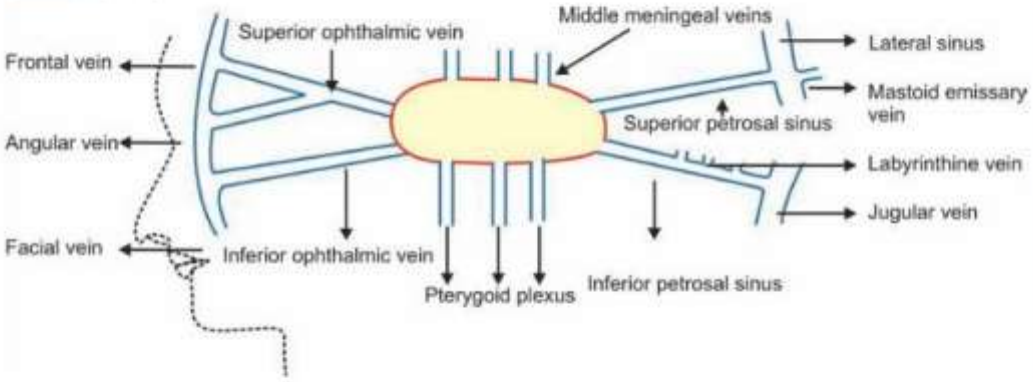
Ans.

Cavernous sinus thrombosis, refers to infected blood clot.

Etiology: It occurs due to the extension of thrombosis from various sources which communicate with the cavernous sinus

Communications of cavernous sinus and sources of Infection

1) **Anteriorly**, the superior & inferior ophthalmic veins drain in the sinus. These veins receive blood from face, nose, para nasal sinuses and orbits.
2) **Posteriorly**, the superior and inferior petrosal sinuses leave it to join the lateral sinus. Labyrinthine veins opening into the inferior petrosal sinuses bring infections from the middle ear. Mastoid emissary veins may spread infection from the mastoid air cells.
3) **Superiorly**, the veins of the cerebrum may be infected from meningitis and cerebral abscesses.
4) **Inferiorly**, the sinus communicates with pterygoid venous plexus.
5) **Medially**, the two cavernous sinuses are connected with each other by transverse sinuses which account for transfer of infection from one side to the other.



Communications of cavernous sinus—lateral view

Clinical features: CST starts initially as a unilateral condition, which soon becomes bilateral due to intercavernous communication.

Hence, appearance of signs and symptoms in the opposite eye is diagnostic of CST.

The condition is characterised by general and ocular features.

- **General features** - abrupt onset of high-grade fever with chills and rigors, vomiting and headache.
- **Ocular features**:
  - Severe pain in the eye and forehead on the affected side.
  - Conjunctiva is swollen and congested.
  - Proptosis develops rapidly.
  - Oedema in mastoid region is a pathognomic sign (occurs due to back pressure in mastoid emissary vein)
  - Ophthalmoplegia is sequential – 6th nerve palsy occurs first as it passes through the cavernous sinus; 3rd and 4th nerves are involved later as they are related to the lateral wall of cavernous sinus.
  - Ipsilateral ptosis, dilated pupil, and absence of direct & consensual pupillary light reflex are signs of 3rd nerve palsy.
  - Corneal anaesthesia, i.e., loss of corneal reflex – due to paralysis of V_1
  - In advanced cases, retinal veins show congestion and there may appear papilloedema.
  - Vision loss may occur in later stages

Investigations

- **CT scan** head and orbit may show involvement of cavernous sinuses and proptosis.
- **Magnetic resonance venography** (angiography) is the investigation of choice which shows an absence of flow void in thrombosed sinuses.
- **Blood culture** is recommended for sepsis.

Complications: At any stage, hyperpyrexia & signs of meningitis or pulmonary infarction may precede death.

Differential diagnosis: orbital cellulitis and panophthalmitis

Treatment

a) **IV Antibiotics** – Massive doses of modern potent broad-spectrum Abx
b) **Analgesics and anti-inflammatory drugs** control pain and fever.
c) **Anticoagulants'** role is controversial

Clinical features	*Cavernous sinus thrombosis*	*Orbital cellulitis*
1. Laterality	Initially unilateral, but soon becomes bilateral	Unilateral
2. Degree of proptosis	Moderate	Marked
3. Vision	Not affected in early stage	Not affected in early stage
4. Cornea and anterior chamber	Clear in early stages	Clear in early stages
5. Ocular movements	Complete limitation to palsy	Marked limitation
6. Oedema in mastoid region	Present	Absent
7. General symptoms with fever, and prostrations	Marked	Mild

VSQs

1. Causes of Enophthalmos [07]

Ans.

Enophthalmos is the inward displacement of the eyeball.

Causes of Enophthalmos:

1. **Congenital** – Microphthalmos and maxillary hypoplasia
2. **Traumatic** – Blow out fractures of floor of the orbit.
3. **Post-inflammatory** – Cicatrization of extraocular muscles as in the pseudotumour syndromes.
4. **Paralytic enophthalmos**: It is seen in Horner's syndrome (due to paralysis of cervical sympathetics).
5. **Atrophy of orbital contents** due to age & irradiation of malignant tumours following cicatrizing metastatic carcinoma and due to scleroderma

---

Ocular Injuries

SQs

1) Closed globe injuries [13]
   a. Vossius Ring [16]
   b. Berlin's Oedema [04]
   c. Effect of blunt injury on the eye [96]

Ans.

Closed-globe injury is the one in which **eyewall** (sclera and cornea) **does not** have a full thickness wound but there is intraocular damage. It includes ***contusion & lamellar laceration***.

- **Contusion** refers to the closed-globe injury due to blunt trauma.
- **Lamellar laceration** refers to the closed-globe injury by a sharp object or blunt trauma.
- **CAUSES**

→ Direct blow to the eyeball by fist, a tennis or cricket ball or blunt instruments like sticks & big stones.

→ Can also occur in roadside accidents, injuries by agricultural and industrial machines etc.

LESIONS OF CLOSED-GLOBE INJURY

1. Cornea.

a) **Simple corneal abrasions** – These are very painful and diagnosed by fluorescein staining.
b) **Recurrent corneal erosions** (recurrent keratalgia) – occur due to finger nail trauma.
c) **Partial corneal tears** (lamellar corneal laceration)
d) **Tears in Descemet's membrane** are known to occur in birth trauma.
e) **Acute corneal oedema**.
f) **Blood staining of cornea**

2. Sclera: Partial thickness scleral wounds (lamellar scleral lacerations) may occur

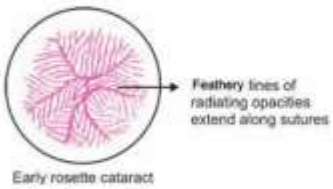
3. Anterior chamber: Traumatic hyphaema (blood in the anterior chamber) & Exudates

4. Iris, pupil and ciliary body

a. Traumatic miosis & Traumatic mydriasis (Iridoplegia)
b. Rupture of the pupillary margin.
c. Radiating tears in the iris stroma.
d. Iridodialysis, i.e., detachment of iris from its root at the ciliary body → D-shaped pupil
e. Anti-flexion & Retroflexion of the iris
f. Traumatic aniridia or iridemia – iris sinks to the bottom of anterior chamber
g. Inflammatory changes – traumatic iridocyclitis, haemophthalmitis, post-traumatic iris atrophy and pigmentary changes.

5. Lens:

1) Vossius ring: It is a circular ring of brown pigment seen on the anterior capsule. It occurs due to striking of the contracted pupillary margin against the crystalline lens. It is always smaller than the size of the pupil.
2) Concussion (traumatic) cataract
3) Traumatic absorption of the lens → aphakia (esp. in young children)
4) Subluxation of the lens – occur due to partial tear of zonules.
5) Dislocation of the lens – occurs due to complete rupture of the zonules.



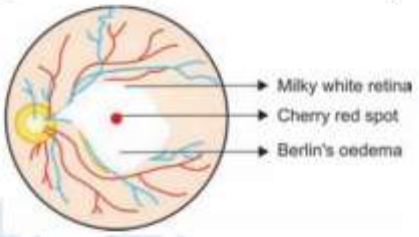
6. Vitreous

- Detachment of the vitreous
- Vitreous haemorrhage.
- Vitreous herniation in the anterior chamber may occur with subluxation or dislocation of the lens.

7. Choroid – rupture, haemorrhage & detachment

8. Retina

a) Commotio retinae (Berlin's oedema) – milky white cloudiness in the posterior pole with a 'cherry-red spot' in the foveal region. It may disappear after some days or may be followed by pigmentary changes.



b) Retinal haemorrhages & Retinal tears.
c) Traumatic proliferative retinopathy (Retinitis proliferans).
d) Retinal detachment
e) Traumatic macular oedema → pigmentary degeneration.

9. IOP changes in closed-globe injury → Traumatic glaucoma & Traumatic hypotony.

10. Traumatic changes in the refraction

- Myopia may follow ciliary spasm or rupture of zonules or anterior shift of the lens.
- Hypermetropia & loss of accommodation may occur from damage to the ciliary body (cyclоplegia).

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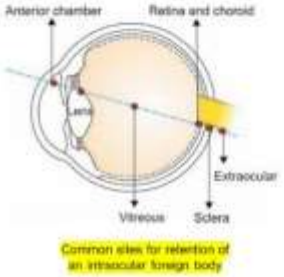
2) Open globe injury – C/F & complications [10]

Ans.



Common sites for retention of an intraocular foreign body

Open-globe injury refers to a full-thickness wound of **eyewall** (sclera & cornea). It includes rupture and laceration of eyewall.

⇒ Globe Rupture (inside to outside break) – caused by a blunt object.

⇒ Globe Laceration (outside to inside break) – caused by a sharp object. It includes penetrating injuries (has entry wound), perforating injuries (has entry and exit wound) & Intraocular FB.

Open globe injury	Clinical Features	Treatment
Globe Laceration 	Wounds of the conjunctiva	Suture if it is > 3 mm
	Uncomplicated corneal wounds	▪ Pad & bandage with atropine & Abx ointments for Small Wounds ▪ Suture for Large wounds
	Complicated corneal wounds (a/w prolapse of iris)	Suture the wound after abscising the iris
	Corneo-scleral tear & Scleral wounds	1st suture should be applied at limbus.
	Wounds of the lens (Ex: lens ruptures)	Lensectomy - Small wounds in the anterior capsule may seal and lead on to traumatic cataract which should managed accordingly
	Severely wounded eye	Excision
Globe rupture	» It is a/w: Prolapse of uveal tissue, vitreous loss, intraocular haemorrhage & dislocation of the lens » Accompanying signs include irregular pupil, hyphaema, commotio retinae, choroidal rupture, and retinal tears	▪ Repair of tear in the eyewall should be done meticulously under general anaesthesia to save the eyeball whenever possible ▪ Post-op Abx, steroids & Atropine ▪ Enucleation in badly damaged eye

---

3) Blow out fracture [09]

Ans.

Blow-out fractures are isolated comminuted fractures which occur when the orbital walls are pressed indirectly. These mainly involve orbital floor and medial wall.

Etiology – Trauma to the orbit by tennis ball, cricket ball, human fist or part of an automobile.

Clinical features

1. Periorbital oedema and blood extravasation in and around the orbit (such as subconjunctival ecchymosis) occur immediately.
2. Emphysema of the eyelids
3. Paraesthesia and anaesthesia in the distribution of infraorbital nerve (lower lid, cheek, side of nose, upper lip and upper teeth)
4. Ipsilateral epistaxis as a result of bleeding from maxillary sinus into the nose
5. Proptosis – occurs because of associated orbital oedema and haemorrhage. 6
6. Enophthalmos (inward displacement of eyeball) and mechanical ptosis.
7. Diplopia.

Orbital imaging

- **Plain X-rays** taken in Water's view reveal ➔ fragmentation of the orbital floor and herniation of contents into maxillary antrum (hanging drop or tear drop sign).
- **CT scan & MRI** – for detailed visualisation of soft tissues & evaluating the extent of the fracture

Management

- **General measures and medical treatment**
  a) Cold compresses, immediately after trauma may ↓ swelling by causing vasoconstriction
  b) Avoid nose blowing, as it may contribute to surgical emphysema and herniation of soft tissue.
  c) Systemic antibiotics should be given to prevent secondary infection from the maxillary sinus.
  d) Analgesics and anti-inflammatory drugs to decrease pain and swelling.
- **Surgical repair** – to restore continuity of the orbital floor.
  Indications of surgical intervention include:
  - Large herniation of tissues into the antrum
  - Diplopia not resolving significantly.
  - Enophthalmos > 3 mm.

---

4) Sympathetic ophthalmitis [09, 99]

Ans.

Sympathetic ophthalmitis is a serious bilateral granulomatous panuveitis which follows a penetrating ocular trauma. The injured eye is called exciting eye and the fellow eye which also develops uveitis is called sympathizing eye. Very rarely, sympathetic ophthalmitis can also occur following an intraocular surgery.

Predisposing factors: child, penetrating injury, Wounds in the ciliary region, incarceration of the iris, lens capsule etc.

Pathogenesis: Allergic theory – it states that the uveal pigment acts as an allergen and excites plastic uveitis in the sound eye.

Pathology: It is characteristic of granulomatous uveitis, i.e., there is:

- Aggregation of lymphocytes, plasma cells, epithelioid cells & giant cells throughout the uveal tract.
- Dalen-Fuchs' nodules are formed due to proliferation of the pigment epithelium (of the iris, ciliary body and choroid) associated with invasion by the lymphocytes and epithelioid cells.
- Sympathetic perivasculitis: Retina shows perivascular cellular infiltration.

Clinical features	
In Exciting (injured) eye	In Sympathizing (sound) eye
↳ Features of persistent low grade plastic uveitis: ciliary congestion, lacrimation and tenderness ↳ Keratic precipitates may be present at the back of cornea (dangerous sign)	Here, Clinical feature can be divided into 2 stages which occur after 4–8 weeks of injury in the other eye: ➢ Prodromal stage: ▪ Symptoms: photophobia & transient indistinctness of near objects (due to weakening of accommodation) ▪ Signs: - Presence of retrolental flare and cells - Presence of KPs on back of cornea. - Mild ciliary congestion, slight tenderness of the globe, - Vitreous haze and disc oedema. ➢ Fully-developed stage – typical signs & symptoms of acute plastic iridocyclitis are seen

Treatment

- Early excision (enucleation) should be done when the injured eye has no useful vision
- Conservative treatment:
  - ⇢ Corticosteroids should be administered by all routes, i.e., systemic, periocular injections & frequent instillation of topical drops.
  - ⇢ Immunosuppressant drugs should be started in severe cases, without delay.
  - ⇢ Atropine should be instilled three times a day in all cases.

Prognosis

» It is good if steroid therapy is commenced early.

» It is bad if the uvea is heavily infiltrated and inflammation has taken firm hold

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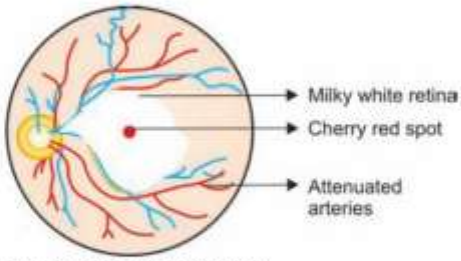
VSQs

1. Cherry - red spot {incl Causes} [16, 15, 09, 05]

Ans.

Differential diagnosis of cherry red spot

1) Tay-Sachs disease
2) Niemann-Pick disease
3) Myoclonus
4) Berlin's oedema
5) Macular hole or haemorrhage.



Central retinal artery occlusion

Ocular Pharmacology

SQs

1) Pilocarpine [16, 11, 08, 02]

Ans.

It is a direct-acting parasympathomimetic drug.

Ocular Indications:

(i) Primary open-angle glaucoma;
(ii) Acute angle closure glaucoma;
(iii) Chronic synechial angle-closure glaucoma.

Contraindications: inflammatory glaucoma, malignant glaucoma and known allergy.

Available preparations and dosage are:

a. **Eye drops** are available in 1 %, 2% and 4% strengths → Effect last upto 4-6 hours. Hence, it is given every 6^{th} or 8^{th} hiurly
b. **Ocusert** are available as pilo-20 and pilo-40. These are changed once in a week.
c. **Pilocarpine gel (4%)** is a bedtime adjunct to the daytime medication.

---

2) Timolol maleate [14]

a. Action and adverse effects of Beta blockers [10]

Ans.

MOA: Timolol is a nonselective β-blocker ➔ block β_2 receptors in the ciliary processes ➔ ↓ aqueous production ➔ ↓ IOP.

Indications:

⊙ β blockers are useful in all types of glaucomas, viz., developmental, primary and secondary; narrow as well as open angle.
⊙ β-blockers are the 1^{st} choice in POAG & all secondary glaucomas.

Contraindications: bronchial asthma, emphysema, COPD, heart blocks, congestive heart failure or cardiomyopathy; Known drug allergies etc.

Preparations: Timolol is available as 0.25% and 0.5% eye drops → effects last up to 24 hours. Hence, it is used once or twice daily.

- The drug is very effective, however the phenomenon of **'short-term escape'** and **'long-term drift'** are well known.

Adverse-effects:

1. **Ocular side-effects**: burning, conjunctival hyperaemia, superficial punctate keratopathy & corneal anaesthesia.
2. **Systemic side-effects**:
   - ⇉ **CVS effects** (due to β_1 block): bradycardia, arrhythmias, heart failure and syncope.
   - ⇉ **Respiratory effects** (due to β_2 block): bronchospasm & airway obstruction, esp in asthmatics.
   - ⇉ **CNS effects**: depression, anxiety, confusion, drowsiness, disorientation, hallucinations etc.
   - ⇉ **Miscellaneous effects**: nausea, diarrhoea, decreased libido, skin rashes, alopecia & exacerbation of myasthenia gravis

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3) Atropine {incl it's uses in Ophthalmology} [12, 11, 09, 07]

a. Homatropine [03]

Ans.

Atropine is a parasympatholytic Drug.

MOA: It competitively block the Muscarinic effects of Ach (competitive antagonism) ➔ {refer pharma}

Uses in Ophthalmology: Atropine, homatropine, cyclopentolate or tropicamide are used topically as mydriatic & cycloplegic for refraction testing

- For fundoscopic examination, short acting agent like Tropicamide is used.
- In iridocyclitis - atropinic mydriatics are used alternatively with miotics to break adhesions between iris & lens.

---

4) Anti-Glaucoma drugs [09, 05]
   a. Topical drugs for glaucoma [05, 99]

Ans.

Anti-Glaucoma Drugs (↓ IOP)	
Drugs to ↑ trabecular outflow	Pilocarpine (miotics) Dipivefrine Bimatoprost
Drugs to ↑ uveoscleral outflow	⇢ Dipivefrine ⇢ Latanoprost
Drugs to ↓ aqueous production	• CA inhibitors (ex: acetazolamide) • α agonist (ex: dipivefrine, epinephrine) • β blockers (ex: timolol)
Hyperosmotic agents	Ex: Mannitol

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5) Uses of steroids in Ophthalmology [04, 03]
   a. Uses of cortisones in Ophthalmology [97]

Ans.

1. Topical preparations are used in scleritis, uveitis, allergic conjunctivitis, allergic keratitis, cystoid macular oedema and after intraocular surgery.
2. Systemic preparations are used in scleritis, posterior uveitis, Vogt-Koyanagi-Harada (VKH) syndrome, papillitis, retrobulbar neuritis, malignant exophthalmos, orbital lymphangioma and corneal graft rejections.

VSQs

1. Cycloplegic drugs [16]

Ans.

Cycloplegics are the drugs which cause paralysis of accommodation and dilate the pupil (cycloplegia).

Ex: {Pa} Atropine, Homatropine, Cyclopentolate, Tropicamide & Phenylephrine.

Indications: These are used for wet retinoscopy

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2. Uses of Acetazolamide in Ophthalmology [13]

Ans.

All types of acute & chronic Glaucomas: to ⇩ IOP by ⇩ formation of aqueous humour.

Used either orally or topically.

Oral preparations have many side effects → Paresthesias, Urinary Frequency, electrolyte imbalance, GI symptoms etc.

Lasers and Cryotherapy in Ophthalmology

SQs

1. Uses of laser in Ophthalmology [12, 08]
   a. Nd YAG laser [14, 02]

Ans.

LASER is an acronym for 'Light Amplification by Stimulated Emission of Radiation'.

LASER EFFECTS	THERAPEUTIC APPLICATIONS
1) Photoradiation (Photochemical effect)	Corneal collagen linking with riboflavin
2) Photocoagulation	1. Eyelid lesions ➔ haemangioma. 2. Cornea ➔ Treatment of corneal neovascularisation. 3. In glaucoma – Ex: Laser iridotomy for narrow-angle glaucoma etc. 4. Lesions of iris: ➢ Photomydriasis for pathologic miotic pupil, ➢ Laser sphincterotomy. 5. Lesions of retina and choroid: ⇒ Diabetic retinopathy – Pan Retinal Photocoagulation (PRP) ⇒ Intraocular tumours – retinoblastoma, malignant melanoma and choroidal haemangioma. ⇒ Macular diseases ➔ Age-related macular degeneration (ARMD). ⇒ For sealing of holes in retinal detachment.
3) Photodisruption	**1) Nd: YAG laser:** {wavelength – 1064 nm} ▪ Capsulotomy for thickened posterior capsule ▪ Membranectomy for pupillary membranes. ▪ Iridotomy & Vitreolysis. ▪ Phacolysis in phacoemulsification technique of cataract extraction 2) **Femtosecond laser**: ✪ Creation of corneal flap for LASIK ✪ Creation of tunnel for intracorneal rings, ✪ Arcuate incisions to correct corneal astigmatism, ✪ Keratoplasty incisions, ✪ Small incision lenticule extraction (SMILE) for correction of myopia ✪ Femtosecond laser assisted cataract surgery (FLACS).
4) Photoablation	**Excimer (Argon fluoride laser)** ❖ Photorefractive keratectomy (PRK) ❖ Laser assisted in situ keratomileusis (LASIK) for correction of refractive errors and ❖ Phototherapeutic keratectomy (PTK) for corneal diseases such as band-shaped keratopathy

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VSQs

1) Uses of cryo in ophthalmology [11]

Ans.

1. **Lids**: Cryosurgery may be used for following lesions:
   (i) Cryolysis for trichiasis
   (ii) Cryotherapy for warts and Molluscum contagiosum,
   (iii) Cryotherapy for basal cell carcinoma and haemangioma.
2. **Conjunctiva**: Cryotherapy is used for hypertrophied papillae of vernal catarrh and ocular surface squamous neoplasia (OSSN).
3. **Cornea**: Herpes simplex keratitis may be treated by cryotherapy.
4. **Lens**: Cryoextraction of the lens used to be the best intracapsular technique. However, nowadays intracapsular cataract extraction (ICCE) is **no more performed**.
5. **Ciliary body:** Cyclocryopexy for absolute glaucoma and neovascular glaucoma.
6. **Retina**:
   a. Cryopexy for sealing retinal holes in retinal detachment.
   b. Prophylactic cryopexy to prevent retinal detachment in predisposed cases.
   c. Anterior retinal cryopexy (ARC) in retinal ischaemic disease, e.g., retinopathy of prematurity to prevent neovascularization.
   d. Cryo treatment of retinoblastoma.

Systemic Ophthalmology

LQs

1. Describe the various viral infections of the eye and its complications [02]

Ans.

Viral Infections	Ocular Lesions & Complications
Measles	Catarrhal conjunctivitis, Koplik's spots on conjunctiva, corneal ulceration, optic neuritis and retinitis
Mumps	Conjunctivitis, keratitis, acute dacryoadenitis and uveitis
Rubella	Congenital microphthalmos, cataract, glaucoma, chorioretinitis and optic atrophy
AIDS	1. Retinal microvasculopathy – develops from vaso-occlusive process. It is characterised by nonspecific lesions: a) Multiple 'cotton wool spots' occur in 50% cases, b) Superficial and deep retinal haemorrhages occur in 15- 40% cases, and c) Microaneurysms and telangiectasia may also be seen rarely. 2. Usual ocular Infections – becomes severe in patients with AIDS. These include: Herpes zoster ophthalmicus, Herpes simplex infections, Toxoplasmosis (chorioretinitis), and Ocular tuberculosis, syphilis and fungal corneal ulcers. 3. Opportunistic infections of the eye: These are caused by micro-organisms which do not affect normal patients. These include: Cytomegalovirus (CMV) retinitis, Candida endophthalmitis, Cryptococcal infections, Pneumocystis carinii, and Choroiditis. 4. Unusual neoplasms: ▪ Kaposi's sarcoma – on the eyelid or conjunctiva ▪ Burkitt 's lymphoma of the orbit. 5. Neuro-ophthalmic lesions: Cranial nerve palsies ➔ paralysis of eyelids, extraocular muscles, Loss of sensory supply to the eye, loss of vision etc.

Management:

- CMV infections can be treated by zidovudine, ganciclovir and foscarnet.
- Kaposi's sarcoma responds to radiotherapy.
- Herpes zoster ophthalmicus is treated by acyclovir.

SQs

1) Ocular features of Vitamin A deficiency [16]
   a. Vitamin A deficiency [03]
   b. Bitot Spot [02]
   c. Avitaminosis Vitamin A (Ocular) [2000]

Ans.

Ocular manifestations of vitamin A deficiency arc referred to as xerophthalmia.

Etiology: It occurs either due to dietary deficiency of vitamin A or its defective absorption.

Clinical features

1) Night blindness: It is the earliest symptom of xerophthalmia in children.
2) Conjunctival Xerosis: It consists of one or more patches of dry, lustreless, non-wettable conjunctiva.

3) **Bitot' s spot:** It is an extension of the xerotic process. The Bitot' s spot is a raised, silvery white, foamy, triangular patch of keratinised epithelium, situated on the bulbar conjunctiva
4) **Corneal xerosis:** cornea lacks lustre.
5) **Corneal ulceration/keratomalacia** ➔ stromal defects ➔ blindness.
6) **Corneal scars:** Healing of stromal defects results in corneal scars of different densities and sizes.
7) **Xerophthalmic Fundus:** It is characterized by typical seed-like, raised, whitish lesions scattered uniformly over the pan of the fundus at the level of optic disc

Treatment:

1. **Local ocular therapy**:
   - **For conjunctival xerosis**: artificial tears (0.7% hydroxypropyl methyl cellulose) should be instilled every 3-4 hours.
   - **For keratomalacia:** full-fledged treatment of bacterial corneal ulcer should be instituted.
2. **Vitamin A therapy**: The WHO recommended schedule is as given below:
   (i) **All patients above the age of 1 year** (except women of reproductive age): 200,000 IU of vitamin A orally or 100,000 IU by IM injection should be given immediately on diagnosis and repeated the following day and 4 weeks later
   (ii) **Children under the age of 1 year –** should be treated with half the doses for patients of more than 1 year of age.
   (iii) **Women of reproductive age, pregnant or not**:
   - **For night blindness, conjunctival xerosis and Bitot' s spots** ➔ Daily dose of 10,000 IU of vitamin A orally (1 sugar coated tablet) for 2 weeks.
   - **For corneal xerophthalmia** ➔ same as described for patients above 1 year of age
3. **Treatment of underlying conditions** such as PEM and other nutritional disorders, diarrhoea, dehydration and electrolyte imbalance, infections and parasitic conditions should be considered simultaneously

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2) Night blindness [12, 04]

Ans.

Night Blindness (Nyctalopia) occur in patients with rod dysfunction, media opacities & advanced POAG.

Causes of Night Blindness		
Rod dysfunction	**Media opacities:**	**Advanced POAG**
It can be due to – ⇒ Vitamin A deficiency, ⇒ Tapetoretinal degenerations (e.g., retinitis pigmentosa), ⇒ Congenital high myopia, ⇒ Congenital night blindness, and ⇒ Oguchi's disease	Here ocular media interfere with the light rays in dim light (i.e., with dilated pupils). Seen in: ▪ Peripheral Cortical cataract & ▪ Corneal opacities	In advanced cases of primary open angle glaucoma, dark adaptation may be so much delayed that patient gives history of night blindness

Treatment of Night Blindness: it depends on the cause; hence proper evaluation is the key.

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3) Common causes of Blindness in India [08]

Ans.

Blindness is defined as, "Visual acuity < 3/60 (Snellen) or counting finger at a distance of 3 meters or central visual field < 10 degrees".

Rapid assessment of avoidable blindness (RAAB) survey has been carried out in India to estimate the magnitude & causes of blindness: 👉

RAAB Survey (2006-07)[10]	
Disease condition	*Percent blindness*
Cataract	72.2
Refractive errors (0.7%) + aphakia (5.6%)	6.3
Glaucoma	4.4
Complications of cataract surgery	3.0
Corneal opacity including trachoma	6.5
Posterior segment disorders (DR, 0.1% + ARMD 0.7% + others 2.2%)	3.0

VSQs

1. Foods rich in Vitamin-A [14]

Ans.

Animal sources: milk, butter, cream, cheese, egg yolk and liver. Fish liver oils (cod liver oil and shark liver oil)

Vegetable sources: **Carrot, Papaya**, **mango**, pumpkins, green leafy vegetables (spinach, amaranth) and drumsticks

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2. WHO classification of Blindness [10]

Ans.

As per WHO, 10th Revision of ICD, the vision impairment (maximum vision less than 6/ 18 Snellen in the better eye) has been divided into 4 categories: 👉

Category of visual impairment		*Level of visual acuity (Snellen)*
Normal vision:	0	6/6 to 6/18
Moderate vision impairment:	1	Less than 6/18 to 6/60
Severe vision impairment:	2	Less than 6/60 to 3/60
Blindness:	3	Less than 3/60 (FC at 3 m) or visual field <10°

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3. Preventable blindness [06]

Ans.

Preventable blindness is that which can be easily prevented by attacking the causative factor at an appropriate time.

Ex: corneal blindness due to vitamin A deficiency and trachoma can be prevented by timely measures.

Community Ophthalmology

SQs

1. NPCB (National Programme for Control of Blindness) [04, 02]
   a. Role of Primary Health Center in NPCB. [11]
   b. Mobile Eye Camp [11]
   c. Primary Eye Care System [08]
   d. Eye Bank [05]

Ans.

National Programme for Control of Blindness and Visual Impairment (NPCB & VI) in India:

- Previously called as NPCB – launched in 1976
- **Objectives**:
  a. Reduction in the backlog of blindness through identification and treatment of blinds.
  b. Development and strengthening of eyecare facilities in every district.
  c. Development of human resources for providing eye care services.
  d. Improvement in quality-of-service delivery.
  e. Securing participation of voluntary organizations and private practitioners in eye care.
  f. Enhancement in community awareness on eye care.
  g. Setting up of mechanism for referral, coordination and feedback between organizations dedicated to prevention, treatment and rehabilitation.
- **Definition of Blindness** (same as definition by WHO):
  - Presenting visual acuity <3/60 in better eye
    or
  - Visual Field limitation to < 10 degrees
- **Prevalence of Blindness**: 1.05%
- **Main Causes**: Cataract (MC) > Refractive errors > Glaucoma
- **Adoption of vision 2020: Right to Sight** in 2001 is the most prestigious major flip for NPCB
  - Vision 2020 was Launched in 1999 in Geneva but India adopted in 2001
  - Aim:
    a) To reduce blind people to as million by 2020
    b) To eliminate avoidable blindness by 2020
  - 7 diseases covered by Vision 2020 in India: Cataract, Refractive Errors, Glaucoma, Corneal Blindness, Childhood Blindness, Trachoma & Diabetic Retinopathy
- Since 2001, NPCB is being implemented as per guidelines of the initiative 'Vision 2020'.
- During 12th five-year plan (2012-2017), from it has been decided to continue the NPCB under the NCD pool of the recently approved 'National Health Mission'.
- The contribution of the central government will be 75% and that of state/UT government will be 25% of the total fund requirement for NPCB.

Primary eye care System: This is defined as the provision of promotive, preventive and therapeutic measures for eye health to individuals and the community at peripheral level (PHCs & subcentres) under NPCB.

- Promotive and preventive activities focus on health education.
- Education is aimed at making the people aware that the majority of blinding diseases are preventable or curable.

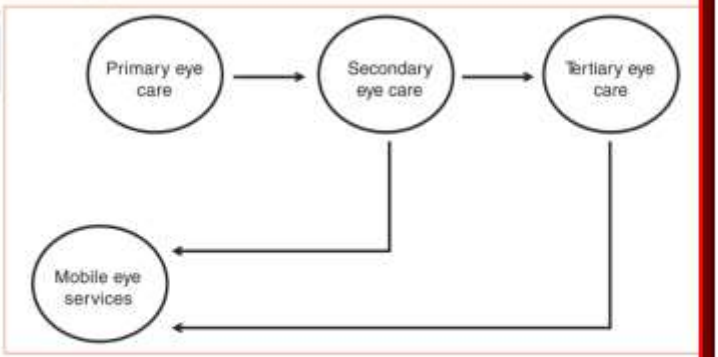
- A locally available, willing person is selected and trained as a primary care worker.
- The health worker is trained to recognize common eye conditions and take appropriate action.
- The 'kit' includes a hand torch, vision measuring chart, antibiotics (usually tetracycline eye ointment), vitamin A capsules, zinc sulphate eye drops, bandages, sticking plaster, epilation forceps and eye shields

Community ophthalmology practice at primary level

Promotive	Preventive	Curative	Rehabilitative
• Nutrition education • Improved maternal and child nutrition • Health education • Face washing • Good antenatal care • Safe water • Improved environmental sanitation	• Ocular prophylaxis at birth • Vitamin A doses • Measles vaccine • Perinatal care • Avoid medication in pregnancy • Avoid hypoxia at birth • Examine neonate's eyes • Nutrition supplementation	• Vision screening • Treatment for vitamin A deficiency • Referral for surgery • Emergency management • Treatment for trachoma • Treatment for other common eye diseases	• Provision of low vision services • Community based rehabilitation • Counselling of the incurable blind • Certification of blind by eye surgeon • Sensitise about concessions

Mobile Eye Camp:

- a team of specialized staff (ophthalmologists, nurses, optometrists and technicians) form a mobile ophthalmic unit which conducts 'eye camps' in the rural areas, with the assistance of several NGOs.
- **Facilities**: screening for common eye diseases, refraction and prescription of glasses, cataract surgery, surgery for angle-closure glaucoma, optical iridectomies and referral of complicated cases. They also provide health education.
- These units are supported by the government to deliver basic eye health facilities to communities who cannot otherwise avail of them
- A central body organizes health education, training of staff, evaluates and monitors all activities related to the National programmes for the prevention of blindness.



Model for an effective eye care delivery system.

Eye bank is an organization which deals with the collection, storage and distribution of cornea for the purpose of corneal grafting, research and supply of the eye tissue for other ophthalmic purposes.

Functions of an eye bank

1) Promotion of eye donation by ↑ awareness about eye donation to the general public.
2) Registration of the pledger for eye donation.
3) Collection of the donated eyes from the deceased.
4) Receiving and processing the donor eyes.
5) Preservation of the tissue.
6) Distribution of the donor tissues to the corneal surgeons.
7) Research activities for improvement of the preservation methodology, corneal substitute and utilisation of the other components of eye.

Eye Bank Personnel:

1) Eye bank in-charge – should be an ophthalmologist

2) Eye bank technician – record data pertaining to donor material and waiting list of patients
3) Clerk-cum-storekeeper – coordinate with other eye banks.
4) Medical social worker – promote voluntary eye donation
5) Driver – maintain vehicle of the eye bank

- Legal aspect: The collection and use of donated eyes come under the preview of 'The Transplantation of Human Organs Act, 1994'.
- Facts about eye donation
  a. The eyes have to be removed within six hours of death.
  b. The eyes cannot be removed from a living human being in spite of his/ her consent and wish

REHABILITATION OF THE BLIND: A blind person needs the following types of rehabilitation:

1) **Medical rehabilitation –** By low vision aids (LVA)
2) **Training and psychosocial rehabilitation**:
   - blinds should be assured and made to feel that they are equally useful and not inferior to the sighted persons.
   - Mobility training with the help of a stick.
   - Training in daily living skills such as bathing, washing, putting on clothes, shaving, cooking etc.
3) **Educational rehabilitation** – 'Blind Schools' with the facility of Braille system of education.
4) **Vocational rehabilitation** – It will help them to earn their livelihood and live as useful citizens. Blinds can be trained in making handicrafts, book binding, candle and chalk making, cottage industries and as telephone operators.

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2. Legal Blindness [2000, 98]

Ans.

Vision in better eye <1/60 to perception light is called Legal Blindness.

"Legal blindness" is a definition used by the US government to determine eligibility for vocational training, rehabilitation, schooling, disability benefits, low vision devices, and tax exemption programs

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Clinical Methods in Ophthalmology

LQs

1) A 45-year-old man, tailor by profession reports to eye O.P.D. with complaints of painless gradual diminution of vision. Describe DDx and principles of treatment [10]
   a. Enumerate causes for painful diminution of vision [15]
   b. Enumerate the causes for sudden loss of vision [11, 03]
   c. Enumerate the causes for gradual loss of vision [08]
   d. Enumerate the causes of Gradual dimension of vision in a person above the age of 50 years and how will you investigate such a case [97]
   e. Sudden loss of vision in one eye – etiology & DDx [90]

Ans.

Sudden Loss of Vision		Gradual Loss of vision	
Painless	**Painful**	Painless	**Painful**
1. CRAO 2. CRVO 3. Massive Vitreous Hemorrhage 4. Retinal Detachment	1) Mechanical injuries to eyeball 2) Chemical injuries to eyeball 3) Acute Congestive Glaucoma 4) Acute iridocyclitis	1. Corneal degenerations 2. Refractive errors 3. Senile cataract 4. Developmental cataract 5. Optic atrophy 6. Diabetic retinopathy	1) Corneal ulcerations 2) Chronic iridocyclitis

Refer to the respective topics for principles.

SQs

1. Fundus Fluorescein Angiography (FFA) – indications, technique and complications [16, 11]

Ans.

FFA is a tool which helps to diagnose various fundus disorders by using fluorescein dye along the vasculature of the retina and choroid.

Indication – Disorders of ocular fundus, viz.,

1. Diabetic retinopathy
2. Vascular occlusions;
3. Eales' disease.
4. Central serous retinopathy,
5. Cystoid macular oedema.

Contraindications: renal impairment and known allergy to fluorescein.

Technique: Rapidly inject 5 mL of 10% solution of sterile sodium fluorescein dye in the antecubital vein and take serial photographs (with fundus camera) of the fundus of the patient who is seated with pupils fully dilated.

Complications: Minor side effects include: discoloration of skin and urine, mild nausea and rarely vomiting. Anaphylaxis or cardiorespiratory problems are extremely rare.

Phases of Angiogram:

1. Pre-arterial phase
2. Arterial phase
3. Arteriovenous phase
4. Venous phase

Abnormalities detected by FFA:

Causes of hyperfluorescence	CAUSES OF HYPO FLUORESCENCE
1. LEAKAGE OF DYE FROM THE VESSELS – SEEN IN CASES OF PROLIFERATIVE DIABETIC RETINOPATHY & IN AGE-RELATED MACULAR DEGENERATION 2. LEAKAGE OF DYE FROM OPTIC NERVE HEAD AS SEEN IN PAPILLEDEMA 3. WINDOW DEFECT IN RPE DUE TO ATROPHY 4. POOLING OF DYE UNDER DETACHED RPE, E.G., ARMD 5. POOLING OF DYE UNDER SENSORY RETINA AFTER BREAKDOWN OF THE OUTER BLOOD-RETINAL BARRIER AS OCCUR IN CENTRAL SEROUS RETINOPATHY (CSR) 6. STAINING, I.E., LONG RETENTION OF DYE BY SOME TISSUES, E.G. AS SEEN IN THE PRESENCE OF DRUSEN AND NORMAL OPTIC DISC	1. Blockage of background fluorescence due to abnormal deposits on retina, e.g., as seen due to the presence of retinal haemorrhage, hard exudates and pigmented clumps 2. Occlusion of retinal or choroidal vasculature, e.g., as seen in centra l retinal artery occlusion and occlusion of capillaries in diabetic retinopathy. 3. Loss of vasculature as occurs in patients with choroideremia and myopic degeneration.

VSQs

1) Relative Afferent Pupillary Defect (RAPD) [17]
   a. Marcus Gunn pupil [13, 12, 09, 05]

Ans.
Marcus Gunn pupil is the paradoxical response of a pupil to light in the presence of optic nerve lesions and severe retinal diseases.
Swinging flash light test: a bright flash light is shone on to one pupil & then quickly moved to the contralateral pupil. This swinging to-and-fro of flash light is repeated several times while observing the pupillary response. Normally, both pupils constrict equally and the pupil to which light is transferred remains tightly constricted. In the presence of RAPD in one eye, the affected pupil will *dilate when the flash light is moved* from the normal eye to the abnormal eye. This response is called **'Marcus Gunn pupil'** or a relative afferent pupillary defect (RAPD).
Significance: Marcus Gunn pupil is the earliest indication of optic nerve disease even in the presence of normal visual acuity.

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2) Applanation Tonometry [16]
Ans. It is based on **Timbert-Fick law** which states that the pressure inside a sphere (P) is equal to the force (W) required to flatten its surface divided by the area of flattening (A); i.e., P = W/A.
Commonly used applanation tonometers are:
1. Goldmann tonometer: It is the most popular and accurate tonometer. It requires slit-lamp.
2. Perkin's applanation tonometer: it is small, easy to carry and does not require slit-lamp.
3. Pneumatic tonometer: In this tonometer, there is a pneumatic-to-electronic transducer, which converts the air pressure to a recording on a paper strip, from where IOP is read.
4. Pulse air tonometer
5. Tono-Pen is a computerised pocket tonometer.

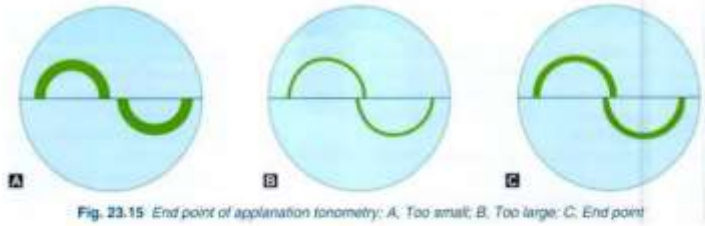

Fig. 23.15 *End point of applanation tonometry: A, Too small; B, Too large; C, End point*

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3) Madarosis [13]

Ans. It refers to partial or complete loss of eyelashes.

Causes:

•Local causes ➔ chronic blepharitis, cicatrizing conjunctivitis, & complication of cryotherapy, radiotherapy or surgery done for any eyelid lesion.

• Systemic causes ➔ alopecia, psoriasis, hypothyroidism and leprosy.

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4) Ciliary Vessels [09]

Ans.

BLOOD SUPPLY OF UVEAL TRACT

Arterial supply: 3

1. Short posterior ciliary arteries – (br. of ophthalmic artery).
2. Long posterior ciliary arteries
3. Anterior ciliary arteries (arise from muscular branches of ophthalmic artery).

Venous drainage: A series of small veins ➔ form the vortex veins (4 in number – Superior temporal, inferior temporal, superior nasal and inferior nasal) ➔ pierce the sclera behind the equator and drain into superior and inferior ophthalmic veins, which in turn, drain into the cavernous sinus

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