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Self Assessment and Review of

OPHTHALMOLOGY

Concepts based on Kanski, Yanoff-Duker, Parson, Khurana
Updated information from latest editions of standard textbooks

Sudha Seetharam

Answers with Precise Explanations

All Recent Questions 2015

All India (2012–2000)

AIIMS (Nov 2015–2000)

PGI (2015-2000)

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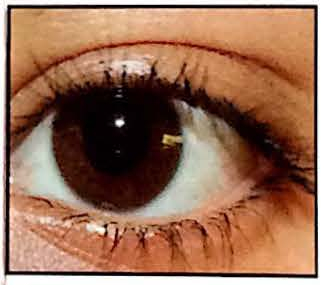

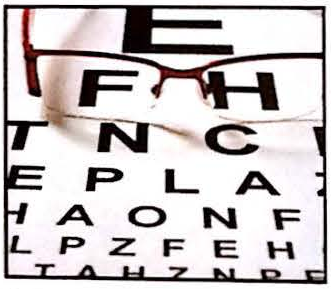

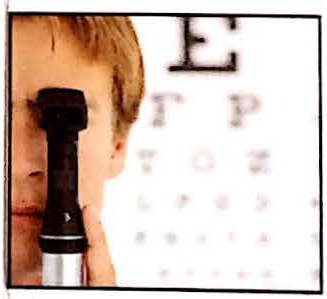

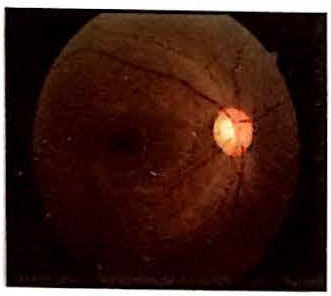

A must-buy book for All India, AIIMS, PGI, JIPMER, DNB, FMGE & State entrance exams

295 -> 200

Self Assessment and Review of Ophthalmology

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Dedicated to

My lucky charm;
Our son, Sreejit

Preface

Dear Students,

Postgraduate medical entrance preparation is undoubtedly one of the most challenging phases in the life of a medico. There are over nineteen subjects to be covered, time is limited and the competition is tremendous. A PG seat in a subject and institution of choice is a dream for every MBBS graduate. But trust me; **this dream can become a reality for you.** What you need is organized preparation in the right direction; motivation to keep up the grueling task and most importantly, the belief that you will succeed.

Ophthalmology has always been a very scoring subject in entrance examinations. Over the past 4–5 years, the trend of questions in Ophthalmology has changed tremendously. The reason is that Ophthalmology is a rapidly evolving subject with new developments taking place at a very fast pace. Thus, diagnostic and treatment modalities keep changing too.

Keeping all this in mind, I have made an attempt to write this book. It is primarily intended for students preparing for PG medical entrance examinations. But it can be useful for MBBS students for a quick revision before their examination. It can also be used by Ophthalmology residents and practitioners as a ready reference.

In this book, I have attempted to include the theoretical discussions relevant for entrance examinations, with special emphasis on recent trends after referencing from standard textbooks like *Clinical Ophthalmology: Kanski, Ophthalmology: Yanoff and Duker* and *Comprehensive Ophthalmology: A K Khurana.* I have also provided MCQs from important examinations like AIIMS, PGI, COMEDK, State PG entrances, DNB. I have carefully chosen the representative questions from each topic so that after going through this book, the students should be able to answer not only the repeat questions but also any new questions that may be asked.

One common mistake that students make in this regard is trying to go through all questions of a particular subject that have been asked in the past 15–20 years. But many of these questions have actually no relevance today because that particular diagnostic or treatment modality may have changed. So those questions will never be asked again. Memorizing these only adds to the confusion and leads to waste of time. With this in mind, I have prepared a concise collection of representative questions mainly from recent examinations. I have also added a picture quiz as there is a rising trend of questions based on photographs. However, despite my best efforts there may be some inadvertent errors which I sincerely regret.

I sincerely hope that you will enjoy reading this book as much as I enjoyed writing it.
Best of luck for your exams and for life!

Sudha Seetharam

Acknowledgements

Writing this book was a big challenge, considering the amount of time, research and reading that it entailed. I was always skeptical as to whether I would be able to dedicate so much time for this endeavor within my busy schedule as a clinical practitioner. I would like to express my heartfelt gratitude to everyone who has helped me in my journey so far.

First and foremost, The Almighty God whose blessings have always been with me in whatever I have done.

My parents: It is because of them that I am what I am today.

My dear husband: He has been a constant source of encouragement at every step in writing this book, never letting me give up. He has also helped me in typing out and arranging the text. Had it not been for him, I would not have seriously considered writing this book.

My parents-in-law: Their blessings and encouragement keep me going.

My brother: My childhood companion and perhaps, my greatest critic, whose honest advice is priceless.

My son: The greatest joy of my life whose smile is enough to make my day. I must acknowledge the many precious personal moments between us that were lost to this difficult task.

My grandparents and grandparents-in-law who, from somewhere in heaven, continue to shower their blessings on me even today.

My teachers at Guru Nanak Eye Centre, Maulana Azad Medical College, especially Dr Jawaharlal Goyal and Dr Ritu Arora who have taught me almost all the ophthalmology that I know today. My teachers at Medical College, Kolkata and South Point High School who have been instrumental in shaping me. My English teacher, Mrs Leena Guha Roy deserves special mention.

Dr Suhas Haldipurkar, Medical Director, Laxmi Eye Institute who initiated me into private practice and opened up a new arena of knowledge.

Mr Ganesh LN, Ms Janaki, Dr Suyog Sahoo, Mr Niraj Salunkhe, Dr Debdatta Mazumdar, Dr Rituparna Mukherjee, Dr Pritesh Singh and all the staff at PGEI whose valuable inputs have helped me during my journey.

I acknowledge Shri Jitendar P Vij (Group Chairman) M/s Jaypee Brothers Medical Publishers (P) Ltd., and his team for the keen interest in publishing this book.

And last but not the least, my students who are the inspiration for this book.

Thank you very, very much!

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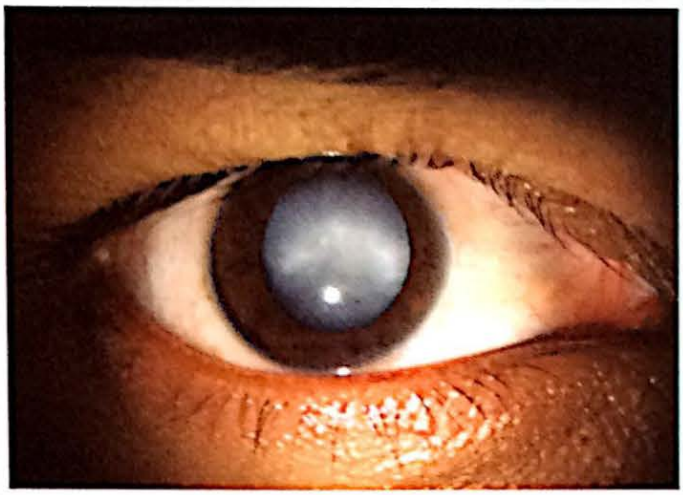
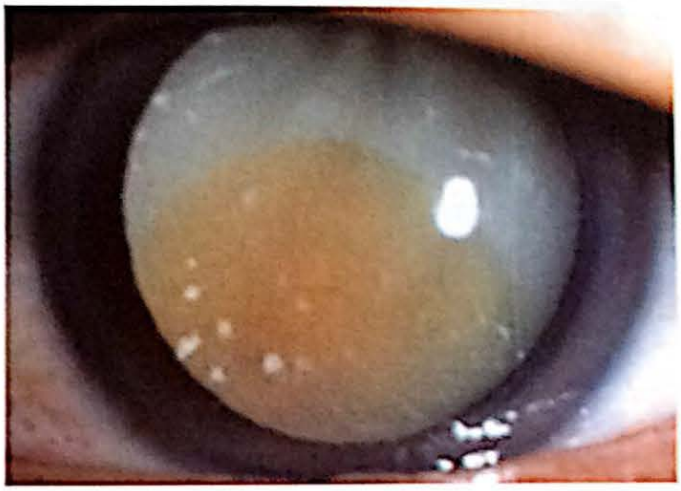
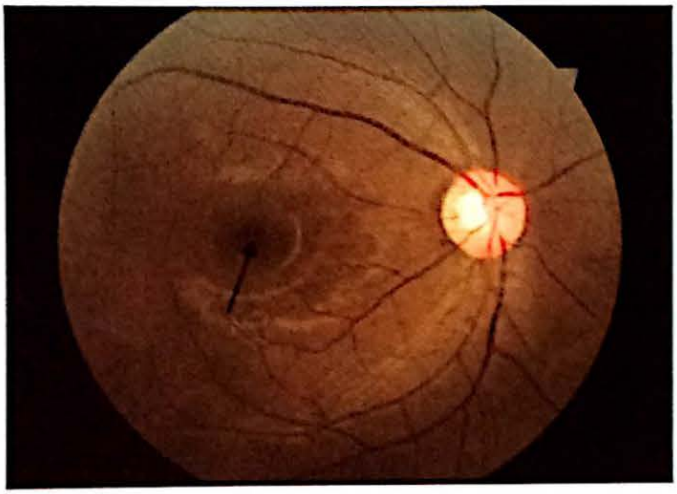


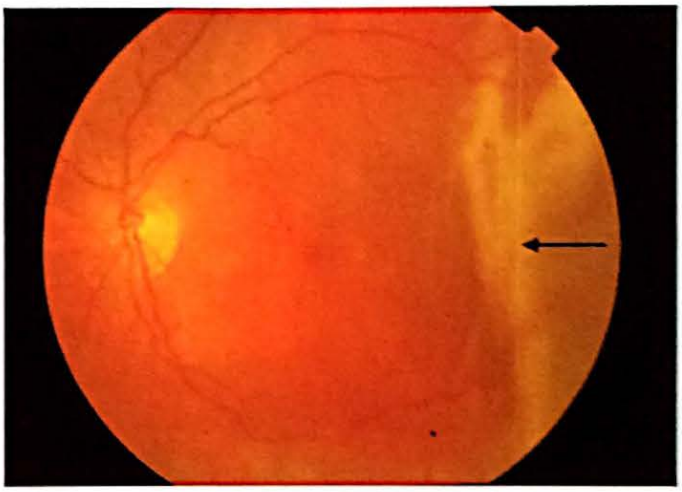
Mature cataract



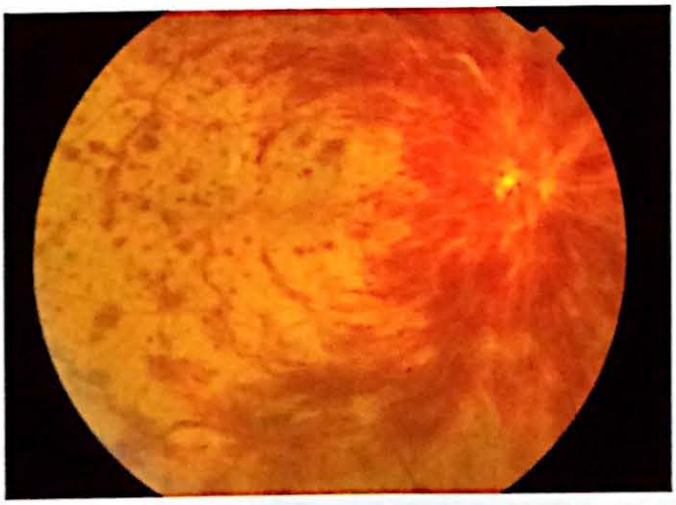
Morgagnian cataract



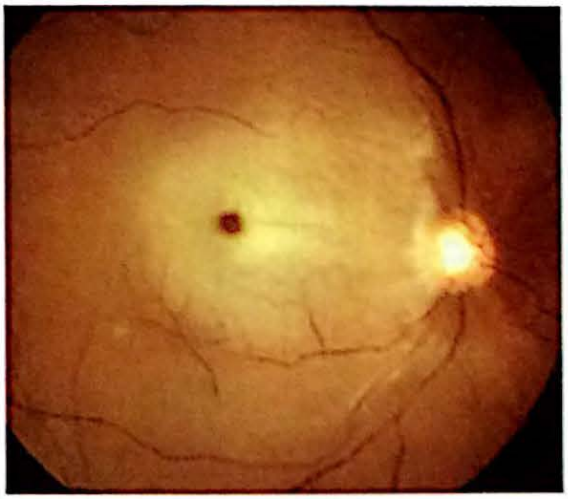
Normal Fundus (arrow points to the fovea)



Retinal detachment

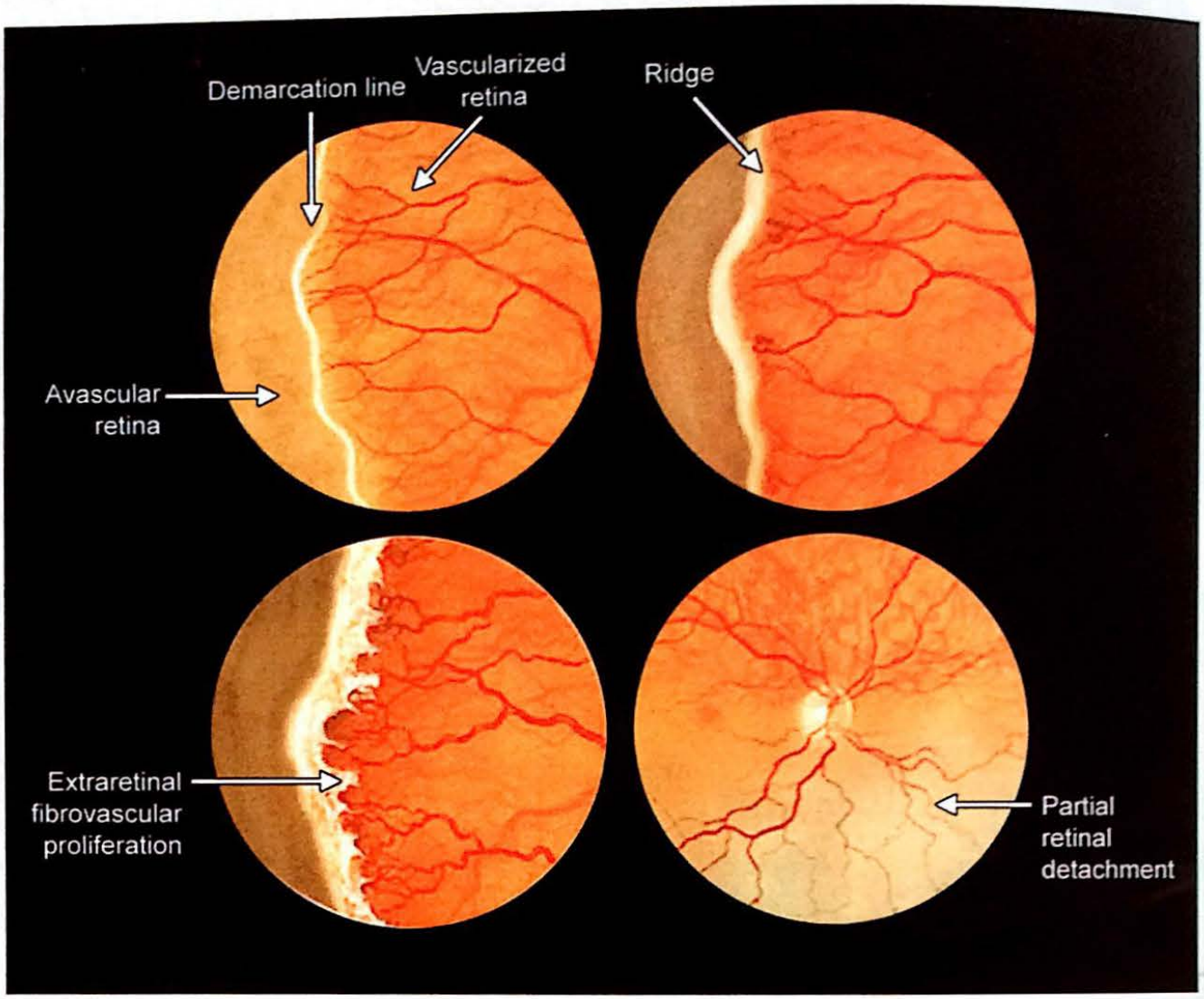


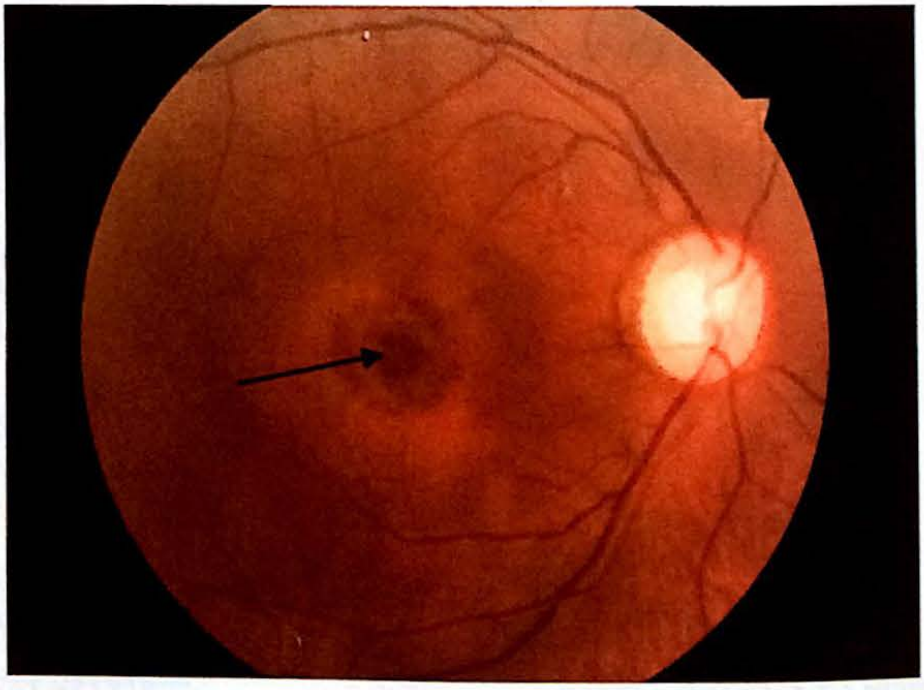
Splashed tomato appearance (CRVO)



Cherry red spot

Plate 2





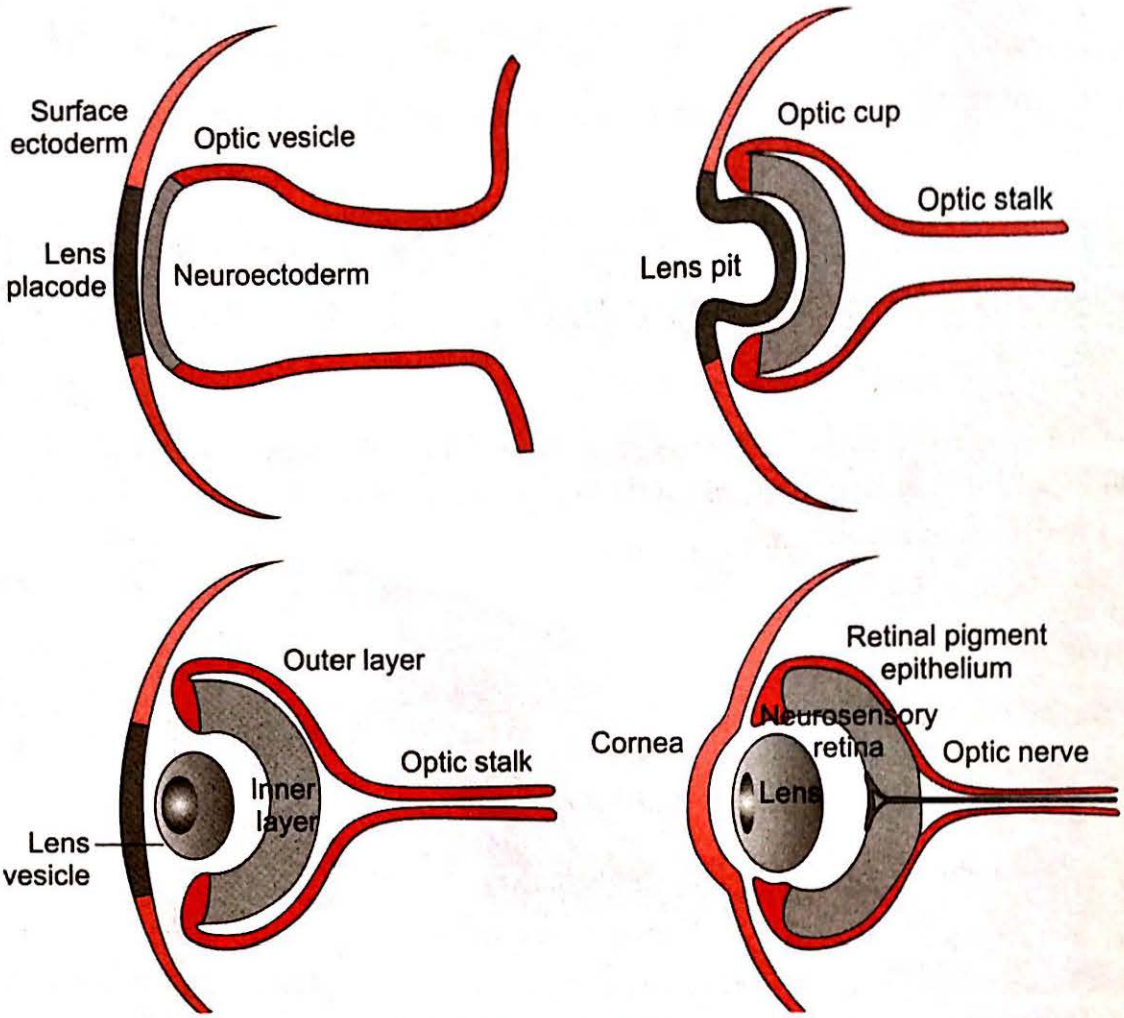
Exudative ARMD/CNVM (indicated by black arrow)

Chapter 1

Ocular Embryology

The eye is formed from three different germ layers, namely neuroectoderm, surface ectoderm and mesoderm with contribution from the neural crest cells. The structures originating from the different layers are:

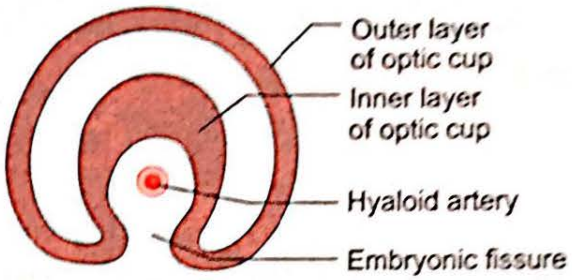
Neuroectoderm
Optic nerve[Q]
Retina including the retinal pigment epithelium[Q]
Epithelium of ciliary body
Epithelium of the iris
Sphincter and dilator pupillae muscles[Q]
Ciliary zonules
Secondary and tertiary vitreous
Surface ectoderm
Epithelium of conjunctiva
Epithelium of cornea
Lens[Q]
Lacrimal glands
Skin of eyelids
Neural crest cells
Stroma, Descemet's membrane and endothelium of the cornea
Angle of anterior chamber[Q]
Stroma of the iris[Q]
Ciliary body and choroid
Primary vitreous
Mesoderm
Sclera
Walls of the orbit
Extraocular muscles
Connective tissue of the orbit
Eyelids



Development of the eye

- The development of the eye starts at about **the third week of gestation.** The neural tube which forms the forebrain gives rise to one diverticulum on either side known as the **optic vesicle**[Q].
- The optic vesicle (neuroectoderm) meets the **surface ectoderm** which shows an area of thickening called the **lens placode.**
- The optic vesicle invaginates to form the two layered **optic cup**[Q]**.** Eventually, **the inner layer** of the cup forms the **neurosensory retina**[Q] whereas the **outer layer** forms the **retinal pigment epithelium**[Q]**.** It then continues backward as the **optic nerve** with its meninges to the brain. The anterior end of the cup later differentiates into the **ciliary epithelium, iris epithelium and muscles of the iris**[Q].
- The invagination of the optic cup however remains incomplete inferonasally in the form of a fissure known as the **embryonic fissure**[Q]. Through this fissure, the hyaloid artery passes to provide nutrition to the developing ocular structures. Eventually, the hyaloid artery disappears and the embryonic fissure closes. The space between the lens and optic cup becomes filled by a clear jelly called the vitreous which is mainly secreted by the neuroectoderm.
- The lens placode invaginates into the optic cup and ultimately gets detached from the surface ectoderm to form **the lens vesicle**[Q]**.** This eventually forms **the crystalline lens.**

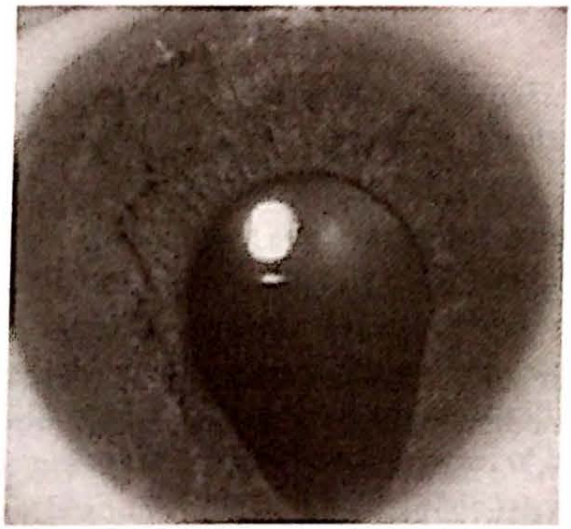
- After formation of the lens vesicle, there is migration of the waves of **neural crest cells**[Q]. These cells eventually differentiate into the **cornea, angle structures and stroma of the iris and ciliary body**[Q].
- While the ectodermal events are taking place, **the mesoderm** surrounding the optic cup differentiates to form the **sclera, extraocular muscles and orbital structures**[Q].



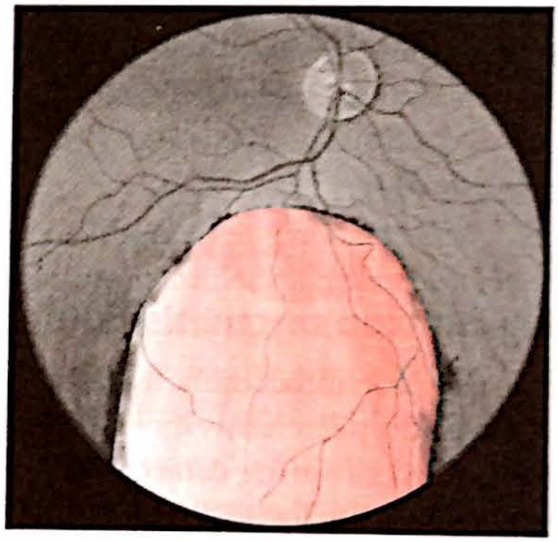
Embryonic fissure

Embryonic Remnants in the Eye

- **Mittendorf's dot**[Q]: It is the remnant of the anterior end of the hyaloid artery and remains attached to the posterior pole of the lens.
- **Bergmeister papilla**[Q]: It is the remnant of the posterior end of the hyaloid artery. It remains attached to the optic disc associated with some glial tissue.
- **Persistent hyperplastic primary vitreous (PHPV):** Failure of the foetal vasculature to regress is called PHPV (explained in detail in the chapter on Retina).
- **Coloboma:** Failure of the embryonic fissure to close gives rise to ocular coloboma.



Iris coloboma



Chorioretinal coloboma

Chapter 2

Conjunctiva, Sclera and Cornea

CONJUNCTIVA

Conjunctiva is a translucent mucous membrane lining the posterior surface of the eyelids and anterior surface of the sclera. The parts of the conjunctiva are:

- Palpebral: It lines the posterior surface of the eyelid and is firmly attached to the tarsus
- Forniceal: It is the loose fold of conjunctiva at the fornix
- Bulbar: It covers the sclera.

Structure of Conjunctiva

It has the following layers:

- Epithelium: It is **stratified squamous nonkeratinized epithelium**Q
- Adenoid layer: It is also called the lymphoid layer and contains the lymphocytes.
- Fibrous layer: Consists of collagenous and elastic fibers, vessels and nerves.

Glands of Conjunctiva

The glands present here are:

- Mucin producing: Goblet cells , Crypts of Henle and Glands of Manz
- Accessory lacrimal glands: **Glands of Krause**Q (in the fornices) and **Wolfring**Q (along tarsal borders).

ALLERGIC CONJUNCTIVITIS

Vernal Keratoconjunctivitis (VKC)

- It is an allergic keratoconjunctivitis seen in boys between 5–15 years of age
- It is a **Type I hypersensitivity**Q reaction mediated by **IgE and mast cells**Q
- It is also called as **spring catarrh**Q
- Symptoms: **Itching**Q associated with ropy discharge
- Signs
  - Conjunctival features:
    - Flat topped papillae are seen on the upper tarsal conjunctiva. The typical appearance is called **cobblestone appearance**Q.
    - In the limbal variety, there is hypertrophy of the superior limbal conjunctiva. This gives rise to raised white nodules close to the upper limbus called **Horner-Trantas spots**Q

  - Corneal features
    - Superficial punctate keratitis
    - **Shield ulcer**[Q]
    - Curved white line close to the upper limbus called **pseudogerontoxon**[Q]
    - **Keratoconus**[Q] is an association
- Treatment
  - Acute episode: The following drugs are given topically:
    - Antihistaminics
    - Steroids
    - Cyclosporine
- Prophylaxis: The following drugs are given topically:
  - **Sodium cromoglycate**[Q]
  - Ketotifen
  - Olopatadine
  - Epinastine

Phlyctenular Conjunctivitis

- It is a **Type IV hypersensitivity**[Q] reaction, mainly to **tuberculous antigen**[Q]. However in western countries, it is said to be mainly associated with **Staphylococcus**[Q].
- It is seen in children (8–15 years)
- It begins as a well-circumscribed nodule at the limbus but it may encroach upon the cornea. This is known as **fascicular ulcer**[Q]
- Treatment is topical steroids. Systemic evaluation and ATT is usually considered in our country.

INFECTIVE CONJUNCTIVITIS

Acute Bacterial Conjunctivitis

This is a very common self-limiting condition seen mainly in children. The symptoms are redness, grittiness, discharge, sticking of lashes. On examination, there is conjunctival congestion, more in fornices, associated with purulent or mucopurulent discharge. **Follicles**[Q] **may be seen (Follicles are collections of lymphocytes in the adenoid layer surrounded by blood vessels)**.The condition is usually self-limiting. Local antibiotics and lubricants may be prescribed. The different types of bacterial conjunctivitis are:

- **Membranous conjunctivitis:** This is caused by organisms of very high virulence like Corynebacterium diphtheria, **Streptococcus haemolyticus**. A **thick yellowish-grey membrane** is formed in the palpebral conjunctiva which **bleeds on peeling. The raw area** left after sloughing of the membrane may lead to complications like symblepharon and entropion due to cicatrisation. Systemic antibiotics and anti-diphtheric serum are also given in addition to local therapy.
- **Pseudomembranous conjunctivitis:** This is the common variety where a pseudomembrane is seen on the palpebral conjunctiva due to organisation of the exudates. It is

adhered loosely to the underlying conjunctiva and may bleed slightly on peeling but no raw area is seen beneath it. It is seen in conjunctivitis caused by **Staphylococcus aureus, Staphylococcus epidermidis,** Streptococcus of low virulence etc.

- **Angular conjunctivitis:** This is a condition where the redness is limited to the inner and outer canthi and excoriation is seen at the lateral eye margins. It is caused by **Moraxella axenfeld**Q. It is treated with zinc and oxytetracycline topically.

Acute Viral Conjunctivitis

This is a common self-limiting condition seen in both children and adults. It presents with redness, watering and foreign body sensation. It is a **follicular conjunctivitis**Q and may be associated with subconjunctival haemorrhage and pseudomembrane. **Pre-auricular lymphadenopathy is seen**Q. It is self limiting and lubricants are prescribed for relief. The different types are:

- **Haemorrhagic conjunctivitis:** It is associated with petechial haemorrhages in the conjunctiva. The causative organisms are **Adenovirus**Q, **Enterovirus**Q, **Echovirus**Q, **Coxsakie**Q **virus**
- **Keratoconjunctivitis:** This is commonly seen with **adenovirus** where nummular lesions are seen on the cornea associated with conjunctivitis. **It is associated with photophobia**Q **and blurring of vision.** Nummulae resolve spontaneously over a period of time but topical steroids are prescribed for quick resolution.

Adult Inclusion Conjunctivitis

- The causative organism is **Chlamydia trachomatis serotypes D-K**Q.
- The primary source of infection is urethritis in males and cervicitis in females because these serotypes are sexually transmitted
- It may also be transferred through contaminated water of swimming pools. Hence, it is also called **swimming pool conjunctivitis**Q
- It is a type of **follicular conjunctivitis**Q associated with preauricular lymphadenopathy.

Trachoma

It is a specific type of keratoconjunctivitis which is characterized by formation of follicles and pannus followed by resolution by cicatrisation. The causative organism is **Chlamydia trachomatis serotypes A, B, Ba and C**Q. It is seen in children < 5 years of age especially in areas of poverty, overcrowding and poor hygiene.

Pathology

- Chlamydia is an epitheliotropic microorganism which affects the conjunctival and corneal epithelium**Q**.
- It can be seen in the epithelial cells as **HP inclusion bodies**Q
- Trachoma is characterized by intense infiltration of lymphocytes in the adenoid layer of the conjunctiva. Aggregation of lymphocytes results in the formation of follicles. **Sago grain follicles are seen on the upper tarsal conjunctiva**Q.

- Follicles seen close to the upper limbus are called as **Herbert's follicles**Q
- On cicatrisation, a white line is seen on the upper tarsal conjunctiva known as **Arlt's line**Q
- Herbert's follicles on scarring form **Herbert's pits**Q.

Prevalence

High endemic (50–70%): Punjab, Rajasthan, UP
Moderately endemic (20–50%): Gujarat, MP Bihar, Assam, Karnataka
Low endemic (<20%): JK, AP, Tamil Nadu, Maharashtra
Very low endemic (0–5%): West Bengal, Odisha

WHO Classification (1987)

- **F (Follicles):** Presence of more than **5 follicles**Q , larger than **0.5 mm diameter**Q on the **upper tarsal conjunctiva**Q
- **I (Inflammation):** Intense inflammation with thickening of the conjunctiva obscuring more than 50% of the deep tarsal vessels
- **S (Scarring):** Evidence of conjunctival cicatrisation with white lines, bands of fibrosis in the tarsal conjunctiva
- **T (Trichiasis):** At least one trichiatic lash
- **O (Opacity):** Corneal opacity obscuring the visual axis and causing a visual acuity < 6/18

Sequelae

- Trichiasis, entropion
- Ptosis, madarosis, tylosis
- Conjunctival and corneal xerosis
- Corneal ulcer and opacity
- Chronic dacryocystitis

Prevalence (in population 0–9 yrs)	Prophylaxis
>10%	**Mass prophylaxis**Q
5–10%	Family prophylaxis
<5%-	Only individual treatment

Treatment

*SAFE Strategy*Q

S: Surgery for trichiasis/entropion/corneal opacity.
A: Antibiotics for active infection (**Azithromycin 500 mg single dose**)Q
F: Facial cleanliness
E: Environmental hygiene

Ophthalmia Neonatorum

It is an acute inflammation of the conjunctiva seen in a newborn child within one month of birth. It is associated with catarrhal discharge (no tears are formed up to 4 weeks of life) The causes may be **chemical, Chlamydia, herpes simplex**, gonococcus, other bacteria.

CONJUNCTIVAL DEGENERATIONS

The different types of degenerations seen in conjunctiva are:

- **Pinguecula (most common)**[Q]
- Pterygium
- Concretions
- Retention cyst

Pterygium

- It is an **elastotic degeneration**[Q] of the conjunctiva
- A degenerated fold of conjunctiva grows on the surface of the cornea to **involve** the **superficial corneal layers up to the stroma**[Q]
- It is associated with UV ray exposure
- A pterygium has three parts: Head, neck and body. Close to the head of the pterygium is a pigmented line due to iron deposition called as **Stocker's line**[Q]
- Pterygium may lead to:
  - Visual disturbance when it encroaches the pupillary axis
  - Astigmatism: Usually, **with the rule**[Q]
  - Diplopia due to restriction of ocular movements
  - Disturbance of tear film
  - Cosmetic blemish
- Treatment
  - Excision with bare sclera: Simple excision is associated with a high level of **recurrence**[Q]
  - Excision with application of **Mitomycin C**[Q] to the bed of pterygium
  - Excision with **conjunctival autograft**[Q]
  - Excision with **amniotic membrane graft**[Q].

SCLERA

Episcleritis

- It is a common self-limiting disorder affecting young adults
- It is associated with mild redness and discomfort
- It may be nodular or diffuse
- It is treated with topical anti-inflammatory drugs.

Scleritis

- It is a painful condition associated with severe redness and watering of the eye
- It is associated with connective tissue disorders like **Rheumatoid arthritis**[Q], Wegener granulomatosis, Polyarteritis nodosa and SLE.
- It may be associated with uveitis also
- Treatment is topical and systemic steroids. It may also need systemic immunosuppressants in severe cases.

Classification

- Anterior
  - Non-necrotizing: It may be nodular or diffuse
  - Necrotizing: It may be with or without inflammation. **Scleromalacia perforans**[Q] is a type of **necrotizing scleritis without inflammation,** associated with long-standing **rheumatoid arthritis**[Q]
- Posterior:
  - Non-necrotizing
  - Necrotizing: Surgically induced necrotizing scleritis (SINS)

Blue Sclera[Q]

- Osteogenesis imperfecta
- Paget's disease
- Ehlers-Danlos syndrome
- Marfan syndrome
- Staphyloma
- Healed scleritis

Staphyloma

It is an ectasia of the outer coats of the eyeball with incarceration of uveal tissue. It is of the following types:

- **Anterior**: It is due to **perforated corneal ulcer**[Q]
- **Intercalary** (within 3 mm of the limbus): It is seen in **peripheral ulcerative keratitis**[Q]
- **Ciliary** (Posterior to 3 mm from the limbus) It is seen in **healed scleritis**[Q]
- **Equatorial**: It is seen in **high myopia, healed scleritis**[Q]
- **Posterior** (behind the equator): It is seen in **pathological myopia**[Q]

CORNEA

Anatomy

The cornea is the transparent outermost coat which covers the anterior one third of the eyeball

- Refractive Index 1.376[Q]

- Refractive Power +43. Dioptres[Q]
- Thickness: Central 450–550 microns
  Peripheral 700-900 microns
- Anterior surface of cornea is **elliptical**. Vertical diameter 11mm
  Horizontal diameter 11.7 mm

Posterior surface is circular. Diameter 11.7 mm

It is composed of five layers. From outside inwards, they are:

- Epithelium: Stratified squamous non-keratinized epithelium
- Bowman's layer
- Stroma: **Thickest layer**[Q]
- Descemet's membrane: **Toughest layer**[Q]
- Endothelium: **Most metabolically active layer.**[Q] It is composed of a single layer of hexagonal cells.

Maintenance of Corneal Transparency

The factors responsible for the maintenance of corneal transparency are:

- The smooth texture of the epithelium and the tear film
- **Arrangement of stromal lamellae**[Q]: The regular arrangement of the stromal lamellae ensure that all the scattered light is lost by mutual interference
- **Avascularity**[Q]
- Unmyelinated nerve fibres
- **Endothelial pump mechanism:** The endothelial pump (Na^+/K^+ ATPase) ensures an optimum level of corneal hydration by pumping out excess water from the cornea **This relative dehydration of the cornea is responsible for its transparency.**[Q] Optimum hydration of cornea is 80%
- Intraocular pressure: Increase in intraocular pressure affects the endothelial pump activity leading to increased hydration of cornea.

Important Investigations Related to Cornea

- Pachymetry: To measure corneal thickness[Q]
- Keratometry: To measure the corneal curvature[Q]
- Corneal topography: Measures corneal curvature, shape and thickness
- **Specular microscopy: To assess the corneal endothelium**[Q]
- Confocal microscope: To evaluate all the layers of the cornea
- Vital stains:
  - Fluorescein stain: To identify epithelial defect
  - Rose Bengal stain: It stains the degenerated epithelial cells. It causes severe stinging, hence rarely used.

INFECTIVE KERATITIS

The different types of infectious keratitis are bacterial, fungal, acanthamoeba and viral.

Bacterial Keratitis and Fungal Keratitis

The corneal epithelium is usually resistant to infection but if the integrity of the epithelium is disturbed, keratitis may result. Hence, the predisposing factors for development of ulcer are:

- Corneal abrasion
- Trauma and foreign body
- Dry eyes
- Contact lens misuse
- Misuse of anaesthetic drops
- Prolonged steroid use
- Neurotrophic/Exposure keratopathy
- Bullous keratopathy
- Chronic blepharitis or dacryocystitis
- Immunosuppression due to any cause

But there are certain bacteria capable of invading an intact epithelium. These are:

- N. gonorrhea, N. meningitis[Q]
- Corynebacterium diphtheriae[Q]
- Listeria[Q]

Features

	Bacterial ulcer	**Fungal ulcer**
History	Presence of predisposing factors	Predisposing factors especially trauma with vegetable matter[Q]
Symptoms	Pain, redness, photophobia and lacrimation	Same symptoms but less pronounced[Q]
Ulcer margins	Well-defined	Indistinct and feathery[Q]
Base of the ulcer	Clean	Necrotic
Number of ulcers	Usually single	Multiple small ulcers surround the main ulcer. Known as satellite ulcers[Q]
Associated features	None	Surrounded by a ring called immune ring
Hypopyon	Sterile and mobile[Q]	Non-sterile[Q], contains fungal hyphae Fixed[Q]
Investigation	Corneal scraping and staining with Gram stain. Innoculation in blood agar	Corneal scraping and KOH mount. Inoculation in Sabouraud's dextrose agar
Treatment(Medical)	Fortified antibiotics • Cefazoline (50 mg/ml) • Tobramycin (14 mg/ml Atropine[Q]	Anti- fungal drops • Natamycin (50 mg/ml)[Q] • Amphotericin B (1.5 mg/ml) • Voriconazole (10 mg/ml) Oral Ketoconazole Atropine[Q]
Treatment (Surgical)	Debridement Therapeutic keratoplasty	Debridement Therapeutic keratoplasty

Things to remember
• Hypopyon corneal ulcer with rapid progression and perforation: **Pseudomonas**[Q]
• **Ulcer serpens: Pneumococcus**[Q]
• Most common causative organism causing fungal ulcer in India: Aspergillus
• **Drug of choice for fungal corneal ulcer (filamentous): Natamycin**[Q]
• Drug of choice for fungal corneal ulcer (non-filamentous): Amphotericin B
• **Steroids are absolutely contraindicated in both bacterial and fungal ulcers**
• Atropine must be given in all cases to relieve ciliary spasm[Q]

Complications of Corneal Ulcer

- Thinning and bulging of cornea
- Bulging of Descemet's membrane called **Descemetocele**[Q]
- Corneal opacity which may be nebular, macular or leucomatous
- Perforation of cornea may lead to:
  - Small perforation is usually plugged by iris resulting in adherent leucoma
  - Large perforation leads to iris prolapse. Exudation forms a pseudocornea over it
  - Anterior staphyloma: Protrusion of the cornea with incarcerated iris tissue.

Acanthamoeba Keratitis

- Acanthamoeba is a **fresh water protozoan**[Q] which is capable of causing corneal infection. It is **seen in contact lens users**[Q] who use outdated solutions, tap water to clean their lenses
- However, the **most common infectious keratitis in contact lens user is caused by Pseudomonas**[Q].

Clinical Features

- Severe pain, out of proportion to the degree of inflammation is the distinctive feature.

Most painful type of keratitis[Q]

- Redness, photophobia and lacrimation
- Starts as a **dendritic ulcer**[Q] but soon becomes **a ring ulcer**[Q]
- Involves the adjoining limbus causing **limbitis**
- Inflammation of the corneal nerves called **radial keratoneuritis**[Q].

Investigation

- Acanthamoeba is basically a diagnosis of exclusion
- Corneal scraping and staining with **Calcoflour white stain**[Q]
- Inoculation of scraped material in **Non-nutrient agar enriched with E.coli**[Q].

Treatment

The drugs used are:

- **Polyhexamethylene Biguanide (0.02%)**Q
- Chlorhexidine (0.02%)
- **Propamidine**Q
- Neomycin

Viral keratitis

Viral keratitis may be by either **Herpes simplex keratitis (HSV)** or **Herpes zoster ophthalmicus (HZO).**

Herpes simplex keratitis

It may be either primary or secondary.

A. Primary infection: This is the first infection by the virus seen in children less than 5 years of age. The features are:
   - Vesicles are seen on the lids and periorbital area which heal without scarring
   - Acute follicular conjunctivitis
   - Mild superficial punctate keratitis (SPK)
   - It is treated with Acyclovir ointment (3%) 5 times/day for 2 weeks.

B. Secondary infection/Reactivation stage: This may have varied presentation
   - **Epithelial keratitis:** This is due to the **direct invasion of the epithelium by the virus**Q. It starts as a **dendritic ulcer with marked decrease in corneal sensitivity**Q. Later, the multiple dendrites may join together to form a **geographical ulcer.** Due to decrease in corneal sensitivity, the ulcer is relatively **painless**Q. Treatment is **Acyclovir (3%) ointment**Q for 7–10 days. **Steroids are absolutely contraindicated**Q.
   - **Stromal keratitis**: This is an **immune response to the viral antigen**Q. Characterized by stromal oedema which may progress to necrosis of the underlying bed.
   - **Disciform keratitis**: This is an **immune response to the viral antigen**Q. It presents with localized stromal oedema, Descemet's folds and keratic precipitates.
   - **Endothelitis**: This is also an **immune response to the viral antigen**Q. It presents with stromal oedema, Descemet's folds and endothelial exudates. Keratic precipitates with anterior uveitis may be present.
   - **Treatment of stromal, disciform keratitis and endothelitis is topical steroids**Q.

Herpes Zoster Ophthalmicus

This is a reactivation of the chickenpox or Varicella zoster virus in conditions where the immune system is suppressed like in elderly, chemotherapy, corticosteroid or immunosuppressive therapy. After the initial exposure, the virus remains latent in the sensory ganglion. When reactivated, it migrates down the sensory nerve and causes skin vesicles in that particular dermatome. HZO is seen when the virus involves the **nasociliary division**Q of the **ophthalmic branch**Q of the **Trigeminal nerve**Q. Thus the distribution of the

skin lesions helps to fairly predict the occurrence of HZO. This is known as **Hutchison's sign**Q **which states that if the lesions involve the tip and sides of the nose, the chance of ophthalmic involvement is high.**

The features of HZO are

- Vesicles on the lids
- Blepharitis
- Acute follicular conjunctivitis
- Episcleritis and Scleritis
- Keratitis: This may be of different types
  - Punctate epithelial keratitis
  - **Pseudodendritic keratitis**Q
  - Stromal keratitis
  - Disciform keratitis
  - Endothelitis
- Iridocyclitis, choroiditis
- Acute retinal necrosis
- Neuroretinitis

In the chronic cases, due to decrease in corneal sensation the patient may develop **neurotrophic keratitis**Q.

Treatment of HZO is Oral Acyclovir (800 mg) five times a day for 2 weeks. Topical therapy in the form of steroids, cycloplegics or topical acyclovir is given according to the type of presentation.

Interstitial keratitis

- This is a very distinctive entity characterized by stromal keratitis with no involvement of epithelium or endothelium
- It is associated with **tuberculosis**Q**, leprosy**Q **and syphilis**Q
- The basis of the disease is essentially an immune reaction to the foreign antigen
- It is a dense keratitis associated with deep corneal vascularisation
- The pinkish discoloration of the cornea associated with the condition is called **Salmon patch of Hutchinson**Q
- Treatment is topical steroids. Therapy for the associated systemic disease has to be initiated.

NON-INFECTIOUS KERATOPATHIES

Peripheral Ulcerative Keratitis (PUK)

- This is an immunological ulcer associated with disorders like **rheumatoid arthritis, SLE, PAN, Wegener's granulomatosis etc**Q
- Ulcer involves the periphery of the cornea, extending up to the limbus. It may lead to thinning and perforation. Healed ulcer gives rise to irregular astigmatismQ
- Treatment is **topical steroids. Corneal patch grafting** may be done in cases of severe thinning

Mooren's Ulcer

- Mooren's ulcer is, by definition, an idiopathic PUK where no systemic association can be identified[Q].
- May be unilateral or bilateral and is **severely painful**
- Response to topical steroids is not very good
- Treatment
  - Systemic steroids and immunosuppressants
  - **Conjunctival resection and cautery[Q]:** It is assumed that the antibodies responsible for corneal destruction and ulceration are brought by the conjunctival vessels. Hence, conjunctival resection is done
  - Corneal patch graft.

Neurotrophic Keratopathy

- This type of keratopathy is seen in patients with **decreased corneal sensation**
- **Causes may be Herpes, Diabetes, Leprosy, Trigeminal nerve palsy[Q], brain tumours, cerebrovascular accidents etc.**
- Decreased sensation leads to decrease in reflex tearing. This leads to epithelial breakdown and poor healing
- It is an indolent, **painless ulcer** which does not respond to conventional management
- Treatment is **artificial tears and tarsorrrhaphy.**

Exposure Keratopathy

- This type of keratopathy is seen in patients of **lagophthalmos[Q] or proptosis**
- Evaporation of tears is increased, leading to excessive drying of the cornea. This leads to epithelial breakdown and poor healing
- Treatment is **artificial tears and tarsorrhaphy.**

Drug-induced Keratopathy

- This is also called **Vortex Keratopathy[Q]** or **Cornea Verticillata[Q]**
- It consists of whorl shaped deposits in the cornea
- The important drugs causing this type of keratopathy are:
  - **Chloroquine[Q]**
  - Chlorpromazine
  - **Amiodarone[Q]**
  - Indomethacin
  - Tamoxifen

CORNEAL ECTASIA

Corneal ectasia refers to a group of disorders where protrusion of the cornea is associated with thinning. The different disorders are:

- Keratoconus

- Keratoglobus
- Pellucid marginal degeneration
- Inflammatory ectasias due to trauma, corneal ulcer etc.

KERATOCONUS

- It is a non-inflammatory corneal ectasia where **conical protrusion of a part of the cornea is seen, associated with thinning.** It is bilateral but asymmetrical where one eye is more affected than the other
- It presents in the adolescent age group. The common complaint is frequent change of refraction and lack of clarity with the prescribed glasses
- **Irregular myopic astigmatism is seen**Q
- **Scissoring Reflex**Q is seen on retinoscopy
- Slit lamp examination shows a conical protrusion of the cornea. **Fine vertical folds (Vogt's striae)**Q **are seen in the deep stroma or Descemet's membrane.** Prominent corneal nerves are seen due to associated thinning of the cornea. At the base of the cone, a pigmented line (**Fleischer's ring**Q) is seen due to iron deposition
- **Munson's sign**Q**:** In advanced cone , the protruded cornea indents the lower lid when the patient is asked to look down
- **Corneal topography** is used for confirmation of the diagnosis
- Complication: Tears in the Descemet's membrane may occur due to excessive stretching. This leads to inflow of aqueous into the cornea. Over hydration leads to corneal edema and loss of transparency. This is called **acute hydrops**Q
- Treatment:
  - Spectacles followed by **rigid gas permeable contact lenses**Q
  - **Collagen cross linking (C3R)**Q —Application of **riboflavin followed by U-V ray** exposure causes cross-linkage of the corneal collagen which leads to flattening of the cornea
  - **Keratoplasty** is done in advanced cases.

Associations

- Down's syndrome
- Vernal Catarrh
- Turner's syndrome
- Blue sclera
- Marfan's syndrome
- Aniridia
- Ehler-Danlos syndrome
- Retinitis pigmentosa
- Osteogenesis imperfecta

KERATOGLOBUS

- It is a rare condition where the entire cornea is abnormally protruded and thin. High myopia is seen
- May be associated with **Leber's congenital amaurosis**Q

- **Scleral contact lenses**[Q] and Keratoplasty are treatment options.

PELLUCID MARGINAL DEGENERATION

- It is a bilateral progressive thinning disorder **involving the inferior peripheral cornea**[Q]
- A crescent shaped band of corneal thinning is present from 4 o'clock to 8 o'clock position. It is separated from the limbus by a zone of normal cornea
- **Irregular myopic astigmatism** is seen
- **Rigid gas permeable contact lenses** are Keratoplasty are the treatment options.

CORNEAL DYSTROPHIES

This is a group of disorders where some layer of the cornea is affected from birth. The presentation of the disease however may be later in life. Depending upon the layer of the cornea which is affected corneal dystrophies are classified as

Anterior dystrophies: These are subdivided into

A. Epithelial dystrophies
   - Cogan microcystic dystrophy
   - Meesmann and Stocker dystrophy

B. Bowman's membrane dystrophies
   - Reis-Buckler dystrophy
   - Thiel-Behnke dystrophy

Anterior dystrophies do not have much effect on vision. In these conditions, the adhesion of the epithelium to the underlying basement membrane is weak. Hence, the epithelium gets eroded from time to time. Thus, presentation of anterior dystrophies is **recurrent corneal erosions**[Q].

Treatment is **Phototherapeutic Keratectomy (PTK)**[Q] or superficial keratectomy.

Stromal dystrophies: In these dystrophies there is deposition of a foreign substance within the corneal stroma. The different types are:

Name of dystrophy	Substance deposited	Test
Lattice dystrophy	Amyloid[Q]	Congo-red[Q]
Granular dystrophy	Hyaline[Q]	Masson's trichrome[Q]
Avellino (Combination of Lattice and Granular dystrophy)	Amyloid and Hyaline	Congo-red and Masson's trichrome
Macular dystrophy	Mucopolysaccharide[Q]	Alcian blue[Q]
Schnyder's Crystalline dystrophy	Phospholipids[Q]	Oil-red O
Gelatinous drop like dystrophy	Amyloid	Congo-red

Stromal dystrophies usually present with **diminution of vision.** Treatment is **keratoplasty.**

Posterior dystrophies: They are the endothelial dystrophies namely

- Congenital hereditary endothelial dystrophy
- Fuchs' endothelial dystrophy
- Posterior polymorphous dystrophy

These conditions present with diminution of vision and bullous keratopathy due to corneal oedema. **Treatment is keratoplasty.**

CORNEAL DEGENERATIONS

This is a group of disorders where degenerative changes take place in a corneal tissue that was normal at the time of birth. The important degenerations are

Arcus Senilis

- It is the **most common corneal degeneration**Q. It is present in about 50% individuals above 60 years of age
- If present in children or young adults, it is called **arcus juvenilis** and it may be a feature of **systemic hyperlipidemias**Q
- Arcus is a bilateral lipid deposition which starts in the superior and inferior perilimbal cornea. It progresses circumferentially to form a band which is about 1 mm wide
- The peripheral border is separated from limbus by a clear zone of cornea.
- Lipid is first deposited in anterior part of the Descemet's membrane and the stroma.

Climatic Droplet Keratopathy

- It is also known as **spheroidal degeneration**Q **or Labrador keratopathy**Q
- Prolonged exposure to UV rays is the cause
- Golden yellow spherules are seen in the interpalpebral area. It may cause visual impairment in advanced cases
- Treatment is phototherapeutic keratectomy (PTK)Q or superficial keratectomy

Band Shaped KeratopathyQ

- This type of degeneration is due to deposition of **Ca++ salts**Q **in the Bowman's layer**Q **of the cornea**
- It is seen as a white band in the interpalpebral area with clear space separating it from the limbus
- Causes
  - Local—Chronic uveitis, Silicon oil in the anterior chamber
  - Systemic causes—Hyperparathyroidism, Hypervitaminosis D, Multiple Myeloma
  - Treatment is **chelation with of calcium with EDTA after scraping the epithelium**Q. Phototherapeutic Keratectomy (PTK) and superficial keratectomy are further options.

KERATOPLASTY

Keratoplasty is a procedure where a part or entire diseased cornea is replaced by cadaveric donor cornea.

Corneal Preservation and Eve Banking

- Corneal donor tissue collection and preservation technique is of prime importance in order to maintain endothelial viability
- Ideal death enucleation time is **6 hours**[Q].

There are different types of storage media:

- **Refrigerated moist chamber:** Eyes are stored in a special bottle with sterile solution placed at the bottom of a jar to produce a moist chamber. The jar is kept in a refrigerator at 4°C. The tissue can be stored for 24–48 hours. This is **short-term preservation.**
- **Modified M.K. medium**[Q] (McCarey-Kaufman medium) – (i) 5% Dextran 40 (ii) HEPES buffer, (iii)Phenol Red as pH indicator (iv) Gentamycin (v) Sodium bicarbonate. The tissue remains viable for a period of 3-4 days. This is **intermediate preservation**
- **Long-term preservation:** Cryopreservation or tissue culture.

Contraindications for Use of Donor Tissue[Q]

- Infections like HIV, Hepatitis B, Hepatitis C
- Intraocular malignancies like retinoblastoma, choroidal melanoma
- Head and neck malignancies
- Rabies[Q]
- Septicemia[Q]
- Prion diseases
- Death from unexplained cause[Q].

Types of Keratoplasty

Keratoplasty may be either full thickness or partial thickness. The different types are:

- **Penetrating keratoplasty**[Q] (Full- thickness keratoplasty): The entire tissue from epithelium to endothelium is transplanted. Indications are
  - Tectonic graft is done to restore globe integrity in perforated corneal ulcer
  - Optical graft is done to improve vision in full-thickness corneal opacities, adherent leucoma etc
- **Lamellar keratoplasty**[Q]**:** In this procedure, transplantation of epithelium and stroma is done over the host endothelium and Descemet's membrane. Possible indications are **keratoconus, stromal dystrophies, corneal opacities**[Q] etc
- **Descemet's stripping endothelial keratoplasty (DSEK)**[Q]**:** In this procedure, only the Descemet's membrane and endothelium is transplanted. **Indications are endothelial dystrophies and bullous keratopathy**[Q].

Complications of Keratoplasty

- Graft rejection[Q]: The donor tissue suffers immunological rejection from the host
- Graft infection
- Secondary glaucoma
- Graft failure: Primary failure is due to poor viability of the host tissue. Secondary failure may be due to infection, rejection etc.

XEROPHTHALMIA

It is the term used to cover all the ocular manifestations of vitamin A deficiency. Night-blindness occurs because of disturbance in the visual cycle involving rhodopsin. The ocular surface features are due to metaplasia of the non-keratinized epithelium to keratinized epithelium[Q].

WHO classification (1982)

- XN—Night blindness
- XIA—Conjunctival xerosis
- XIB—**Bitot's spots**[Q]– triangular, foamy, gray, sharply demarcated patch
- X2—Corneal xerosis
- X3A—Corneal ulceration/keratomalacia< 1/3 corneal surface
- X3B—Corneal ulceration/keratomalacia> 1/3 corneal surface
- XS—Corneal scar or opacity
- XF—Xerophthalmia fundus

Treatment

- Children < 1 year of age or weighing< 8 kg: 1 lakh IU orally on days 0, 1 and 14[Q]
- Children equal to or >1 year of age or weighing > 8 kg: 2 lakh IU orally on days 0, 1 and 14[Q]
- IM dose is half of the oral dose.

QUESTIONS

1. **Which of the following is not true of acute viral conjunctivitis?** *(AIIMS 2013)*
   a. Vision is not affected
   b. Corneal infiltration is seen
   c. Antibiotics are the mainstay of treatment
   d. Pupil remains unaffected
2. **Which of the following causes acute haemorrhagic conjunctivitis?** *(DPG)*
   a. Adenovirus
   b. Staphylococcus
   c. Herpes simplex
   d. Haemophilus
3. **Which of the following does not cause haemorrhagic conjunctivitis?** *(AIPG)*
   a. Adenovirus
   b. Coxsackie virus
   c. Enterovirus
   d. Papilloma virus
4. **Angular conjunctivitis is caused by:** *(PGI)*
   a. Haemophilus b. Adenovirus
   c. Moraxella d. Bacteroides
5. **Angular conjunctivitis is caused by Moraxella which is typically:** *(APPG)*
   a. Gram positive diplococcus
   b. Gram negative diplococcus
   c. Gram positive diplobacillus
   d. Gram negative diplobacillus
6. **Inclusion conjunctivitis is caused by:** *(PGI)*
   a. Chlamydia trachomatis
   b. Chlamydia psitacci
   c. Herpes simplex
   d. Gonococcus
7. **In the grading of trachoma, follicular stage is defined as the presence of:** *(AIIMS)*
   a. Five or more follicles in the lower tarsal conjunctiva
   b. Three or more follicles in the lower tarsal conjunctiva
   c. Five or more follicles in the upper tarsal conjunctiva
   d. Three or more follicles in the upper tarsal conjunctiva
8. **Herbert's pits are seen in:** *(Maharashtra PG)*
   a. Spring catarrh
   b. Trachoma
   c. Phlyctenular conjunctivitis
   d. Sarcoidosis
9. **Arlt's line is seen in:** *(AIPG/PGI)*
   a. Vernal keratoconjunctivitis
   b. Pterygium
   c. Trachoma
   d. Ocular pemphigoid
10. **SAFE strategy is used for:** *(AIIMS)*
    a. Trachoma
    b. Diabetic retinopathy
    c. Onchocerciasis
    d. Glaucoma
11. **Which of the drugs is not effective against trachoma?** *(APPG)*
    a. Azithromycin b. Erythromycin
    c. Ivermectin d. Rifampicin
12. **A recurrent bilateral conjunctivitis occurring with the onset of hot weather in young boys with symptoms of burning, itching and lacrimation with polygonal raised areas on the palpebral conjunctiva is:** *(AIPG)*
    a. Trachoma
    b. Phlyctenular conjunctivitis
    c. Mucopurulent conjunctivitis
    d. Vernal keratoconjunctivitis
13. **Features of vernal keratoconjunctivitis is/are:** *(PGI)*
    a. Papillary hypertrophy
    b. Follicular hypertrophy
    c. Herbert's pits

d. Trantas' spots
e. Ciliary congestion

14. Cobblestone appearance of the conjunctiva is seen in: *(APPG)*
a. Trachoma
b. Spring catarrh
c. Ophthalmia nodosum
d. Long-term use of miotics

15. Topical sodium chromoglycate is used in the treatment of: *(COMEDK)*
a. Phlyctenular conjunctivitis
b. Vernal keratoconjunctivitis
c. Trachoma
d. Subconjunctival haemorrhage

16. Phlyctenular conjunctivitis, false is: *(Manipal)*
a. It is most commonly associated with tuberculosis
b. The lesions are typically found near the limbus
c. It predominantly affects children
d. It is a Type IV hypersensitivity reaction

17. Giant papillary conjunctivitis is caused by: *(COMEDK)*
a. Contact lens
b. Ocular prosthesis
c. Protruding corneal sutures
d. All of the above

18. Changes seen in the conjunctiva in vitamin A deficiency: *(AIIMS 2013)*
a. Actinic degeneration
b. Hyperplasia of goblet cells
c. Hyperkeratosis of the squamous epithelium
d. Stromal infiltration

19. A child of weight 8 Kg has Bitot's spots in both eyes. Which is the most appropriate schedule of vitamin A for the child? *(AIPG)*
a. 2 lakh units IM on days 0,14
b. 1 lakh units IM on days 0, 14
c. 2 lakh units IM on days 0,1,14
d. 1 lakh units IM on days 0,1,14

20. Bitot's spots are seen in: *(Bihar PG 2014)*
a. Conjunctiva b. Cornea
c. Retina d. Vitreous

21. Which of the following is true about pterygium? *(AIIMS 2013)*
a. Probe can be passed underneath the pterygium at the limbus
b. Associated with exposure to infrared radiation
c. Bare sclera technique has 30-80% recurrence
d. Elastotic degeneration with distortion of the Descemet's membrane is seen

22. The histology of pterygium is: *(Manipal)*
a. Elastotic degeneration
b. Epithelial inclusion bodies
c. Precancerous changes
d. Squamous metaplasia of the epithelium

23. Stocker's line is seen in: *(AIIMS 2010)*
a. Pinguecula
b. Pterygium
c. Conjunctival melanosis
d. Conjunctival naevus

24. Which of the following may be used to prevent recurrence after pterygium excision? *(APPG)*
a. Natamycin b. Mitomycin C
c. Amphotericin d. Chlormycetin

25. Subconjunctival haemorrhage is seen in all except: *(JIPMER)*
a. Passive venous congestion
b. Pertussis
c. Trauma
d. High intraocular pressure

26. Which of the following may present as a bluish red nodule resembling conjunctival haemorrhage? *(APPG)*
a. Kaposi sarcoma
b. Ciliary staphyloma
c. Lymphoma
d. Limbal dermoid

27. **Parenchymatous xerosis of the conjunctiva is seen in:** *(PGI)*
    a. Trachoma
    b. Vitamin A deficiency
    c. Vernal conjunctivitis
    d. Phlyctenular conjunctivitis
    e. Alkali burns
28. **True about cornea is/are:** *(PGI 2010)*
    a. Power is 43D
    b. Majority of the refraction occurs at the air-cornea interface
    c. With the rule astigmatism is seen because the vertical meridian is steeper than the horizontal
    d. Spherical in shape
    e. Refractive index is 1.334
29. **In which of the following tissues is long spaced collagen seen?** *(AIIMS 2013)*
    a. Diaphragm
    b. Cornea
    c. Basement membrane
    d. Tympanic membrane
30. **In hypoxic injury, the cornea becomes edematous due to the accumulation of** *(AIIMS 2014)*
    a. Carbon dioxide b. Lactate
    c. Pyruvate d. Glycogen
31. **Which of the following is not true regarding the cornea?** *(PGI 2015)*
    a. Endothelium helps to maintain the cornea in a dehydrated state
    b. Oxygen is derived by the corneal epithelium from the air through the tear film
    c. Glucose supply for the cornea is derived from the aqueous
    d. Thickness of the cornea is more at the centre than the periphery
    e. Richly vascular
32. **Contact lens wear has been shown to have deleterious effects on the corneal physiology. Which of the following statements is incorrect?** *(AIPG)*
    a. The level of glucose availability in the corneal epithelium is reduced
    b. There is reduction in the density of hemidesmosomes
    c. There is increased production of CO_2 in the epithelium
    d. There is reduction in the glucose utilisation by the corneal epithelium
33. **Corneal transparency is maintained by all *except*:** *(AIIMS)*
    a. Relative hydration of the cornea
    b. Arrangement of collagen fibers
    c. Unmyelinated nerve fibers
    d. Mitotic figures at the center of cornea
34. **Corneal transparency is maintained by:** *(AIIMS)*
    a. Keratocytes
    b. Bowman's membrane
    c. Descemet's membrane
    d. Endothelium
35. **Corneal thickness is best measured by:** *(APPG)*
    a. Ophthalmometer
    b. Lensometer
    c. Pachymeter
    d. Focimeter
36. **A patient with conjunctival infection led to corneal perforation. Swabs showed Gram negative cocci which had translucent colonies and were oxidase positive. What would be the most probable causative organism?** *(AIIMS 2013)*
    a. Moraxella catarrahalis
    b. Neisseria gonorrhoae
    c. Pseudomonas aeruginosa
    d. Acinobacter actinatus
37. **Which of the following can penetrate the intact cornea:** *(PGI)*
    a. Gonococcus
    b. Pseudomonas
    c. Diphtheria
    d. Streptococcus
    e. Staphylococcus epidermidis

38. **Ulcer serpens is caused by:** *(DPG)*
    a. Pseudomonas b. Pneumococcus
    c. Gonococcus d. Diphtheria
39. **Which of the following can cause corneal perforation in just 48 hours?** *(AIIMS)*
    a. Staphylococcus b. Pseudomonas
    c. Diphtheria d. Aspergillus
40. **Which of the following is not a feature of fungal corneal ulcer?** *(AIIMS 2014)*
    a. Fixed hypopyon
    b. Ulcer with sloughing margins
    c. Symptoms are more pronounced than signs
    d. Fungal hyphae are seen on KOH mount
41. **Satellite nodule with corneal ulcer is seen in:** *(AIPG)*
    a. Fungal ulcer
    b. Viral keratitis
    c. Bacterial ulcer
    d. Acanthamoeba keratitis
42. **A young man aged 30 years presents with difficulty in vision in the left eye for the past 10–15 days. He gives history of trauma to the eye with vegetative matter 15 days back. On examination there is an ulcerative lesion in the cornea whose base has a soft creamy infiltrate. The margins are feathery with a few satellite lesions. Which is the most probable etiological agent?** *(AIIMS)*
    a. Acanthamoeba
    b. Corynebacterium diphtheria
    c. Fusarium
    d. Streptococcus pneumoniae
43. **Microscopy of a corneal ulcer showed branched septate hyphae. The probable diagnosis is:** *(AIIMS)*
    a. Candida b. Aspergillus
    c. Mucormycosis d. Histoplasma
44. **Which of the following is the drug of choice for fungal corneal ulcers caused by filamentous fungi?** *(AIPG)*
    a. Itraconazole b. Natamycin
    c. Nystatin e. Fluconazole
45. **Which of the following is used in the treatment of fungal keratomycosis?** *(AIIMS 2014)*
    a. Silver sulfadiazine
    b. Linezolid
    c. Vancomycin
    d. Doxycycline
46. **Which of the following is the most important adjuvant therapy for fungal corneal ulcer?** *(AIPG)*
    a. Atropine sulphate
    b. Dexamethasone
    c. Pilocarpine
    d. Lignocaine
47. **Steroids are contraindicated in:** *(AIPG)*
    a. Phlyctenular conjunctivitis
    b. Mooren's ulcer
    c. Vernal keratoconjunctivitis
    d. Dendritic ulcer
48. **Corneal sensations are decreased in:** *(AIPG)*
    a. Herpes simplex
    b. Fungal infection
    c. Trachoma
    d. Mooren's ulcer
49. **Which of the following is an important feature of herpes simplex keratitis?** *(COMEDK 2013)*
    a. Circumciliary congestion
    b. Corneal infiltrate
    c. Pannus
    d. Decrease in corneal sensation
50. **A 56-year-old man has painful rashes over the forehead and upper eyelid along with punctate keratopathy for the past two days. About a year ago, he underwent chemotherapy for non- Hodgkin's lymphoma. What is the most probable diagnosis?** *(AIIMS)*
    a. Impetigo
    b. SLE

c. Herpes zoster
d. Pyoderma gangrenosum

51. In a patient presenting with Herpes Zoster ophthalmicus, all of the following are true *except*: *(WBPG)*

a. It is caused by Varicella zoster
b. The virus is lodged in the Gasserian ganglion and travels down the trigeminal nerve
c. Corneal involvement is seen when the tip and sides of the nose are involved
d. Punctate keratitis may coalesce to form dendritic ulcers like Herpes simplex

52. A 17-year-old girl with severe painful keratitis came to the hospital and Acanthamoeba keratitis was suspected. Which of the following is not a risk factor for the same? *(AIIMS)*

a. Extended wear contact lens
b. Exposure to dirty water
c. Corneal trauma
d. Squamous blepharitis

53. Which of the following statements regarding Acanthamoeba keratitis is true? *(AIPG)*

a. For isolation of the causative agent, the corneal scrapings should be cultured on a nutrient agar plate
b. The causative agent Acanthamoeba is a helminth whose normal habitat is the soil
c. Keratitis due to Acanthamoeba is not seen in immunocompromised host
d. Acanthamoeba does not depend upon human host for the completion of its life cycle

54. A patient using contact lens develops corneal infection. Laboratory diagnosis of Acanthamoeba keratitis is made. The following is the best drug for treatment: *(AIPG)*

a. Propamidine
b. Neosporine
c. Ketoconazole
d. Polyhexamethylene biguanide

55. Kallu, a 25-year-old male patient presented with red eye and complains of pain, photophobia, watering and blurred vision. He gives history of trauma with vegetable matter. Corneal examination shows a dendritic ulcer. Microscopy shows macrophage like cells. On culture in Non-nutrient agar enriched with E.coli, there are plaque formations. Which is the most likely organism? *(AIIMS)*

a. Herpes simplex b. Acanthamoeba
c. Candida d. Adenovirus

56. A person with prolonged usage of contact lens presented with irritation of the left eye. After examination a diagnosis of keratitis was made and corneal scrapings revealed the growth of Pseudomonas aeruginosa. The organisms were found to be multidrug resistant. Which of the following best explains the mechanism of antibiotic resistance in these organisms? *(AIPG)*

a. Ability to transfer resistance genes from adjacent flora
b. Improper contact lens hygiene
c. Frequent and injudicious use of antibiotics
d. Ability of Pseudomonas to produce biofilm

57. Which of the following is/are caused by bacterial infection? *(PGI 2013)*

a. Phlyctenular conjunctivitis
b. Marginal keratitis
c. Mooren's ulcer
d. Vogt-Koyanagi-Harada's disease
e. Hypopyon corneal ulcer

58. Recurrent corneal erosions are seen in: *(PGI)*

a. Keratoglobus
b. Keratoconus
c. Glaucoma
d. Corneal dystrophy

59. In Keratoconus, all are seen *except*: *(PGI)*

a. Munson's sign
b. Thinning of cornea at the center
c. Distortion of the corneal reflex
d. Hypermetropic refractive error

60. Keratoconus is associated with all *except*: *(Manipal 2009)*

a. Down's syndrome
b. Marfan's syndrome
c. Ehlers-Danlos syndrome
d. Usher syndrome

61. True about Keratoconus is/are: *(PGI)*

a. Increased curvature of the cornea
b. Astigmatism is seen
c. Kayser-Fleischer ring is seen
d. Cornea is thick
e. Soft contact lenses are used

62. Acute hydrops is seen in: *(APPG)*

a. Keratoglobus
b. Bullous keratopathy
c. Keratoconus
d. Buphthalmos

63. Fleischer's ring is seen in: *(WBPG)*

a. Pterygium b. Chalcosis
c. Keratoconus d. Trauma

64. Early Keratoconus may be diagnosed by: *(APPG)*

a. Corneal topography
b. Keratometry
c. Pachymetry
d. Ophthalmoscopy

65. Enlarged corneal nerves may be seen in all of the following *except*: *(AIPG)*

a. Keratoconus
b. Herpes simplex keratitis
c. Leprosy
d. Neurofibromatosis

66. Band shaped keratopathy is due to the deposition of: *(AIIMS 2013)*

a. Calcium b. Amyloid
c. Iron d. Melanin

67. Band shaped keratopathy is treated by: *(APPG)*

a. Propamidine isethionate
b. EDTA
c. Polyhexamethylene biguanide
d. Chlorhexidine

68. Corneal dystrophies are: *(AIIMS)*

a. Primarily unilateral
b. Primarily bilateral
c. Primarily unilateral with systemic disease
d. Primarily bilateral with systemic disease

69. Which of the following dystrophies is an autosomal recessive condition? *(AIPG)*

a. Lattice dystrophy
b. Granular dystrophy
c. Macular dystrophy
d. Fleck dystrophy

70. Which of the following is the least common corneal dystrophy? *(AIPG 2010)*

a. Macular dystrophy
b. Lattice type I dystrophy
c. Lattice type II dystrophy
d. Granular dystrophy

71. Which of the following stains is used in Granular dystrophy of cornea? *(AIIMS 2015)*

a. Masson's trichrome
b. Congo-red
c. Colloidal iron
d. PAS

72. A 12-year-old girl with tremors has golden brown discolouration of the Descemet's membrane. The most likely diagnosis is:

a. Fabry's disease
b. Wilson's disease
c. Glycogen storage disease
d. Acute rheumatic fever

73. A 28-year-old male complains of glare in both eyes. The cornea shows whorl like opacities in the epithelium. He gives

history of long term treatment with Amiodarone. The most likely diagnosis is: *(COMEDK)*

a. Terrian's marginal degeneration
b. Cornea verticillata
c. Band shaped keratopathy
d. Arcus juvenilis

74. Pigment deposition on cornea is seen in toxicity of: *(PGI)*

a. Chloroquine b. Digoxin
c. Amiodarone d. Ranitidine
e. Diclofenac

75. Which of the following does not result in amorphous whorl like deposits in the cornea? *(AIIMS 2015)*

a. Chloroquine b. Amiodarone
c. Indomethacin d. Chlorpromazine

76. Dellen is: *(Manipal)*

a. Localized thinning of peripheral cornea
b. Raised lesion at the limbus
c. Marginal keratitis
d. Age related corneal degeneration

77. Neurotrophic keratopathy is caused by: *(AIPG)*

a. Bell's palsy
b. Facial nerve palsy
c. Trigeminal nerve palsy
d. None of the above

78 Exposure keratopathy is due to paralysis of: *(APPG)*

a. Trigeminal nerve
b. Facial nerve
c. Abducens nerve
d. Occulomotor nerve

79. Photophthalmia or Snow blindness is caused by: *(AIPG)*

a. Ultraviolet rays b. Infrared rays
c. Gamma rays d. X-rays

80. Treatment of photophthalmia includes: *(PGI)*

a. Irrigation with saline
b. Cold compress
c. Pad and bandage
d. Analgesics
e. Lubricant eye drops

81. In human corneal transplantation, the donor tissue is: *(AIIMS)*

a. Synthetic polymer
b. Donor tissue from cadaveric human eyes
c. Donor tissue from live human eyes
d. Monkey eyes

82. Donor cornea is harvested from cadaveric donors with what time interval of death? *(Kerala PG 2015)*

a. 3 hours b. 6 hours
c. 2 days d. 16 hours

83. Which of the following is not an absolute contraindication for corneal transplantation? *(AIIMS 2014)*

a. TB Meningitis
b. Rabies
c. Death due to unknown cause
d. SSPE

84. Which of the following statement regarding corneal transplantation is true: *(AIPG)*

a. Whole eye is preserved in tissue culture
b. Donor is not accepted if age is more than 60 years
c. Specular microscopy is used to assess endothelial cell count
d. HLA matching is mandatory

85. Signs of graft rejection are all *except*: *(PGI)*

a. Krachmer's spots
b. Khodadoust line
c. Graft edema
d. Epithelial rejection line
e. Foster's spots

86. Percentage of endothelial cell loss after Descemet's stripping Automated Endothelial Keratoplasty (DSAEK) is: *(AIIMS)*

a. 5% b. 10–20%
c. 30–40% d. 50–60%

87. Sclera is thinnest at: *(AIIMS)*
a. Limbus
b. Equator
c. Anterior to the attachment of superior rectus
d. Posterior to the attachment of superior rectus

88. The most common systemic association of scleritis is: *(AIIMS)*
a. Ehlers-Danlos Syndrome
b. Systemic sclerosis
c. Rheumatoid arthritis
d. Giant cell arteritis

89. In scleritis, all are true *except*: *(Manipal)*
a. Scleromalacia perforans is commonly associated with systemic disease
b. Pain is not a prominent feature
c. Retinal detachment is a known complication
d. Glaucoma may occur

90. Most common cause of anterior staphyloma is: *(APPG)*
a. Perforated corneal ulcer
b. Scleritis
c. Myopia
d. Glaucoma

91. Ciliary staphyloma is due to: *(APPG)*
a. Scleritis
b. Myopia
c. Iridocyclitis
d. Choroiditis

92. Blue sclera is seen in: *(COMEDK)*
a. Alkaptonuria
b. Osteogenesis imperfecta
c. Ehlers-Danlos Syndrome
d. Kawasaki disease

93. A patient presents with history of 'chuna' particles falling in the eye. Which of the following should not be done? *(AIIMS 2014)*
a. Repeated irrigation of the conjunctival sac with normal saline
b. Frequent instillation of Na- citrate drops
c. Thorough slit lamp examination
d. Double eversion of the lids to remove the chuna particles

94. Universal marker for limbal stem cells is: *(AIIMS 2015)*
a. Elastin
b. Keratin
c. Collagen
d. ABCG2

ANSWERS AND EXPLANATIONS

1. **c. Antibiotics are the mainstay of treatment.** The mainstay of treatment in viral conjunctivitis is only lubricants.
2. **a. Adenovirus.**
   Acute haemorrhagic conjunctivitis is caused by Adenovirus, Echovirus, Enterovirus and Coxsakie virus
3. **d. Papilloma virus**
4. **c. Moraxella**
5. **d. Gram-negative diplobacillus**
6. **a. Chlamydia trachomatis**
7. **c. Five or more follicles in the upper tarsal conjunctiva**
8. **b.Trachoma**
9. **c. Trachoma**
10. **a. Trachoma**
11. **c. Ivermectin.**
    The main drug for trachoma is Azithromycin (500 mg single dose). Other drugs which have been found to be useful are Tetracyclines, Erythromycin and Rifampicin.
12. **d. Vernal keratoconjunctivitis**
13. **a. Papillary hypertrophy**
    d. Tranta's spots
14. **b. Spring catarrh**
15. **b. Vernal keratoconjunctivitis**
16. **a. It is most commonly associated with tuberculosis.**
    In our country, Phlyctenular conjunctivitis is most commonly associated with tuberculosis. But in western countries, it is associated with Staphylococcus.
17. **a. Contact lens**
18. **c. Hyperkeratosis of the squamous epithelium.**
    The ocular surface changes associated with Xerophthalmia are due to metaplasia of the non-keratinized epithelium into keratinized epithelium.
19. **c. 2 lakh units IM on days 0,1,14**
20. **a. Conjunctiva**
21. **c. Bare sclera technique has 30–80% recurrence**
    Probe can be passed beneath a pseudopterygium but not a true pterygium.
    It is associated with exposure to UV rays.
    Pterygium is an elastotic degeneration but it does not reach up to the Descemet's membrane. It encroaches only up to the stroma of the cornea.
22. **a. Elastotic degeneration**
23. **b. Pterygium**
24. **b. Mitomycin C**
    Since bare sclera technique of pterygium has high recurrence, the methods used are Pterygium excision with Mitomycin C, amniotic membrane graft or conjunctival autograft
25. **d. High intraocular pressure**

Causes of subconjunctival haemorrhage:

- **Trauma**[Q]
- Foreign body
- **Hypertension**[Q]
- Pertussis
- Bleeding disorders
- Pneumococcus
- Hemorrhagic viral conjunctivitis[Q]

26. a. Kaposi sarcoma

27. a. Trachoma, b. Vitamin A deficiency, e. Alkali burns

28. a. Power is 43D, b. Majority of the refraction occurs at the air-cornea interface, c. With the rule astigmatism is seen because the vertical meridian is steeper than the horizontal

29. b. Cornea

30. b. Lactate

The cornea derives its nutrition from the aqueous humour and the limbal blood vessels. Oxygen supply to the cornea comes mainly from the air. Contact lenses may decrease the oxygen supply to the cornea. As a result, there is increase in anaerobic glycolysis leading to accumulation of lactic acid.

31. d. Thickness of the cornea is more at the centre than the periphery, e. Richly vascular

32. d. There is reduction in the glucose utilisation by the corneal epithelium

33. a. Relative hydration of the cornea

It is the relative dehydration of the cornea that is responsible for transparency, not the hydration

34. d. Endothelium

35. c. Pachymeter

36. b. Neisseria gonorrhoae

The question hints at an organism which causes conjunctivitis and penetrates through an intact cornea. Hence, the answer is Neisseria

37. a. Gonococcus, c. Diphtheria

38. b. Pneumococcus

39. b. Pseudomonas

40. c. Symptoms are more pronounced than signs

41. a. Fungal ulcer

42. c. Fusarium

43. b. Aspergillus

44. b. Natamycin

45. a. Silver sulfadiazine

Though not commonly used, **silver sulfadiazine and iodine** also have been shown to be effective in fungal infections in cornea.

46. a. Atropine sulphate

47. d. Dendritic ulcer

48. a. Herpes simplex

49. **d. Decrease in corneal sensation**
50. **c. Herpes zoster**
51. **d. Punctate keratitis may coalesce to form dendritic ulcers like herpes simplex**
    Herpes Zoster forms Pseudodendrites which look similar to the dendritic ulcers of Herpes simplex. But dendrites have a small terminal bulb at the end of the shaft of the ulcer whereas pseudodendrites are devoid of this terminal bulb.
52. **d. Squamous blepharitis**
53. **d. Acanthamoeba does not depend upon human host for the completion of its life cycle**
    Acanthamoeba are free-living protozoa, not helminth living in water and soil.
    They do not require any host for completion of their life cycle but may cause infection in humans and animals.
    They cause infection in both immunocompetant and immunocompromised hosts.
    Culture should be done in non-nutrient agar enriched with *E. coli*.
54. **d. Polyhexamethylene biguanide**
55. **b. Acanthamoeba**
56. **d. Ability of pseudomonas to produce biofilm**
57. **e. Hypopyon corneal ulcer**
    Phlyctenular conjunctivitis is an allergic keratoconjunctivitis. Marginal keratitis and Mooren's ulcer are immunological in etiology. VKH disease is a panuveitis, also immunological in etiology.
58. **d. Corneal dystrophy.**
    Recurrent corneal erosions are seen in anterior corneal dystrophies, fingernail trauma etc.
59. **d. Hypermetropic refractive error**
60. **d. Usher syndrome**
61. **a. Increased curvature of cornea, b. Astigmatism is seen**
62. **c. Keratoconus**
63. **c. Keratoconus**
64. **a. Corneal topography**
    Keratometry, Pachymetry, Topography and Retinoscopy are all used in the diagnosis of Keratoconus. But early diagnosis is done by Corneal Topography.
65. **a. Keratoconus**

Causes of enlarged corneal nerves
• **Neurofibromatosis**[Q]
• **MEN 2B**[Q]
• Refsum's disease
• Amyloidosis
• Icthyosis
• **Leprosy**[Q]
• HSV keratitis
• **Acanthamoeba keratitis**[Q]

Causes of prominent corneal nerves

- Keratoconus
- Buphthalmos
- Reis-Buckler dystrophy

66. a. Calcium

67. b. EDTA

68. b. Primarily bilateral

69. c. Macular dystrophy

70. a. Macular dystrophy

Most common stromal dystrophy is Lattice dystrophy

71. a. Masson's trichrome

72. b. Wilson's disease

Deposition of Cu in the **Descemet's membrane of the cornea**Q in Wilson's disease gives rise to Kayser-Fleischer (KF) ring. **It first appears close to the superior limbus**Q

73. b. Cornea verticillata

74. a. Chloroquine, c. Amiodarone

75. None. All the mentioned drugs may cause whorl like deposits on the cornea or vortex keratopathy

76. a. Localized thinning of cornea at the limbus

77. c. Trigeminal nerve palsy

78. b. Facial nerve

79. a. Ultraviolet rays

Photophthalmia or snow blindness refers to corneal epithelial erosions due to exposure to intense UV rays. It was seen due to reflection of UV rays from snow in extremely cold areas and hence the name.

Treatment is reassurance, tranquilizers and analgesics. Locally, cold compress, pad and bandage, lubricants and cycloplegics are given.

80. b. Cold compress, c. Pad and bandage, d. Analgesic, e. Lubricant eye drops

81. b. Donor tissue from cadaveric human eyes

82. b. 6 hours

83. a. TB meningitis

84. c. Specular microscopy is used to assess endothelial cell count

Specular microscopy is used to assess the endothelium of the donor tissue prior to transplantation. HLA matching is not mandatory for cornea transplant. Usually only donor cornea is preserved in preservation media. Age is not a criterion for discarding donor tissue.

85. e. Foster's spots

Graft rejection is an immunological response of the host to the donor tissue. It may lead to graft failure if not properly treated. The clinical features of rejection in a graft are:

- Decrease in vision
- Graft edema
- **Krachmer's spots**Q in the epithelium and anterior stroma suggestive of epithelial/stromal rejection

- **Khodadoust line**[Q] in the endothelium suggestive of endothelial rejection

Foster Fuchs spots are seen in the retina in myopia

86. **c. 30-40%**
87. **d. Posterior to the attachment of superior rectus**
88. **c. Rheumatoid arthritis**
89. **b. Pain is not a prominent feature**

Scleritis is associated with moderate to severe pain

90. **a. Perforated corneal ulcer**
91. **a. Scleritis**
92. **b. Osteogenesis imperfecta, c. Ehlers-Danlos Syndrome**
93. **c. Thorough slit lamp examination**

Chemical injury to the eye is an ophthalmic emergency. **Alkali injury due to lime or chuna** is very common in children especially in the rural areas. If not treated properly it may have serious consequences. The features of alkali injury are:

- Necrosis of the conjunctival and corneal epithelium.
- **Loss of limbal stem cells leads to conjunctivalisation and vascularisation of the cornea with loss of corneal transparency**[Q]
- Deep stromal necrosis may lead to perforation of the cornea
- Intraocular inflammation may also be seen.

Treatment includes

- **Thorough irrigation with normal saline for 15–30 minutes till the pH becomes normal**[Q]
- **Double eversion of the lids**[Q] to remove all the lime particles
- Debridement of the necrotic epithelium to allow proper epithelialisation
- Antibiotics
- **Topical steroids are given for 7–10 days**[Q] to decrease inflammation and encourage epithelialisation. They should be stopped after 10 days because they impair the healing process.
- **Ascorbic acid**[Q] to promote healing
- **Tetracyclines**[Q] may be given because they have anti-collagenase activity
- Citrates are given as they have anti-neutrophilic activity and decrease inflammation
- Thorough slit lamp examination may not be possible in acute stage due to severe pain and hence the answer
- Rehabilitation of these patients may involve surface reconstruction procedures like **amniotic membrane grafting followed by keratoplasty**[Q].
- **Keratoprosthesis**[Q] **is the final option** when keratoplasty is not possible due to severely damaged ocular surface.

94. **d. ABCG2**

Chapter 3

Glaucoma

Glaucoma is a type of **optic neuropathy** occurring in response to raised intraocular pressure and characterized by typical changes in the optic disc and the visual field. Glaucomatous optic neuropathy is also called **Cavernous Optic Atrophy**[Q].

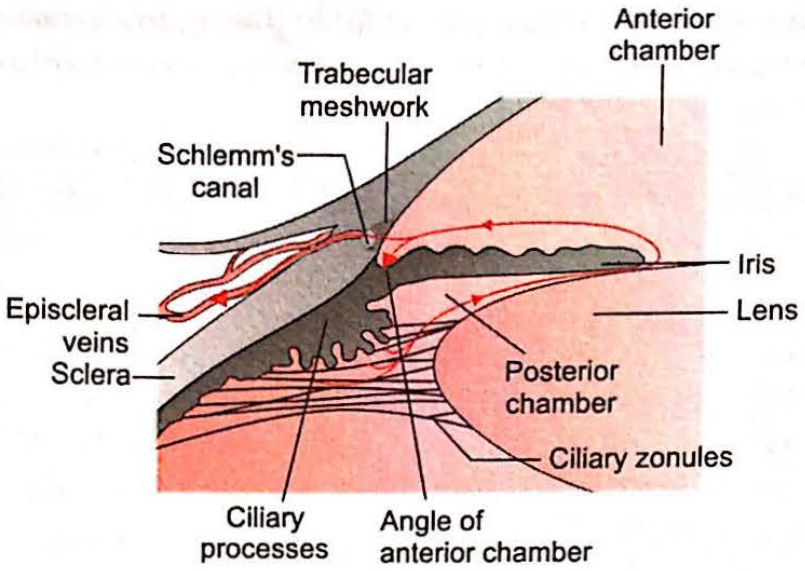
AQUEOUS HUMOUR DYNAMICS

Aqueous humour is basically an ultrafiltrate of the plasma. The site of production is the **ciliary processes of the pars plicata**[Q]. Each ciliary process is lined by two layers of epithelium namely pigmented and non-pigmented. At the centre of the ciliary process is a rich capillary network within the surrounding stroma. Aqueous humour production involves the following steps:

- Formation of a plasma filtrate from the capillary network within the stroma by the process **of ultrafiltration**[Q]
- Transfer of this plasma filtrate from the ciliary stroma into the aqueous compartment across the epithelium. This involves two processes namely **diffusion**[Q] and active **secretion**[Q]
- Rate of aqueous production is **2.3 µL/min**
- Substances whose concentration in aqueous is less than plasma: **Protein, glucose,** urea[Q]
- Substances whose concentration in aqueous is more than plasma: **Ascorbate,** lactate, **pyruvate**[Q]

Aqueous humour flows from the posterior chamber to the anterior chamber through the pupil. From the anterior chamber it has two outflow pathways:

- **Conventional or Trabecular pathway**[Q]: It operates through the angle of anterior chamber and accounts for **90%** of aqueous outflow. The structures in the pathway are **Trabecular meshwork → Schlemm's canal → Collector vessels→ Episcleral veins**[Q]. The site of maximum resistance in this pathway is the **juxtacanalicular part of the trabecular meshwork**[Q]
- **Unconventional or Uveoscleral pathway**[Q]: Accounts for remaining **10%** aqueous outflow:
  - Ciliary body → Suprachoroidal space → Episcleral veins
  - Ciliary body → Suprachoroidal space → Vortex veins



Aqueous outflow through angle of anterior chamber

Evaluation of Glaucoma

- IOP (intraocular pressure)
- Gonioscopy or evaluation of the angle of anterior chamber
- Optic disc evaluation
- Visual field evaluation or Perimetry

1. **Intraocular Pressure (IOP)**
   - **Normal: 11 to 21 mm Hg or 15.5 + 2.5 mm Hg**Q
   - Diurnal variation is less than 5 mm Hg.

Tonometers

i. Indentation tonometer
   - **Schiotz tonometer:** It is widely used because it is portable and the technique is simple to learn. But the main disadvantage is that the reading is affected by **scleral rigidity**Q

ii. Applanation tonometer: It is based on **Imbert Fick**Q law. The law states P= F/A where P = Pressure inside a sphere, F= Force and A= Area. In most applanation tonometers, the area (A) is fixed and the force (F) is variable.

The different types of Applanation tonometers available are:

- **Goldman Applanation tonometer**Q**:** It is the **gold standard tonometer**Q. It gives accurate and reproducible readings. The main disadvantage is that the reading is dependent on **the corneal thickness**. Also its accuracy is decreased in **irregular corneas**
- **Perkin's handheld tonometer:** It is a portable tonometer and **used mainly in children**
- **Tonopen:** It is used in **irregular corneas**Q
- **Mackay-Marg tonometer:** It is used in **irregular corneas**Q
- **Maklakov tonometer:** This is an applanation tonometer with **variable applanation area(A) and fixed force (F)**Q

iii. Non-contact tonometer: This uses a puff of air to flatten the cornea and hence it is free from the risk of transmitting infection.

iv. Newer tonometers
- Pascal's Dynamic Contour Tonometer: It is the most accurate tonometer
- **Rebound Tonometer**: It is a **home care tonometer or self use tonometer**[Q]
- Transpalpebral Tonometer: It is used in uncooperative patients.

2. Gonioscopy

Gonioscopy means visualization of the angle of the anterior chamber. Light travelling from the anterior chamber angle is total internally reflected at the cornea-air interface because it is incident at an angle more than the critical angle for the two media. (**Critical angle for air-cornea interface is 46 degrees**[Q]). Hence, a lens called a gonioscope is required to overcome this total internal reflection There are two types of gonioscopy lenses:
- Direct gonioscopy lenses: Koeppe, Barkan, Thorpe, Swan Jacob
- Indirect gonioscopy lenses: Goldmann, Zeiss, Posner

Angle evaluation is clinically done with gonioscope but there are machines available for the same. They are:
- **Anterior Segment OCT**[Q]
- **Ultrasound Biomicroscopy (UBM)**[Q]

Structures seen on gonioscopy (from anterior to posterior):
- **Schwalbe's line** which represents **the anterior limit of the Descemet's membrane**[Q]
- Trabecular meshwork
- Scleral spur
- Ciliary body and root of iris

Depending on the structures which are visible on gonioscopy, the angle is graded as open, occludable or closed.

Shaffer's grading of Angle width:

Grade	Angle	Structures visible	Configuration	Chances of closure
IV	35–45°	Schwalbe's line to ciliary body	Wide open	Nil
III	20–35°	Schwalbe's line to scleral spur	Open	Nil
II	20°	Schwalbe's line to trabecular meshwork	Moderately narrow	Possible[Q]
I	10°	Schwalbe's line only	Narrow	High
0	0°	None	Closed	Closed

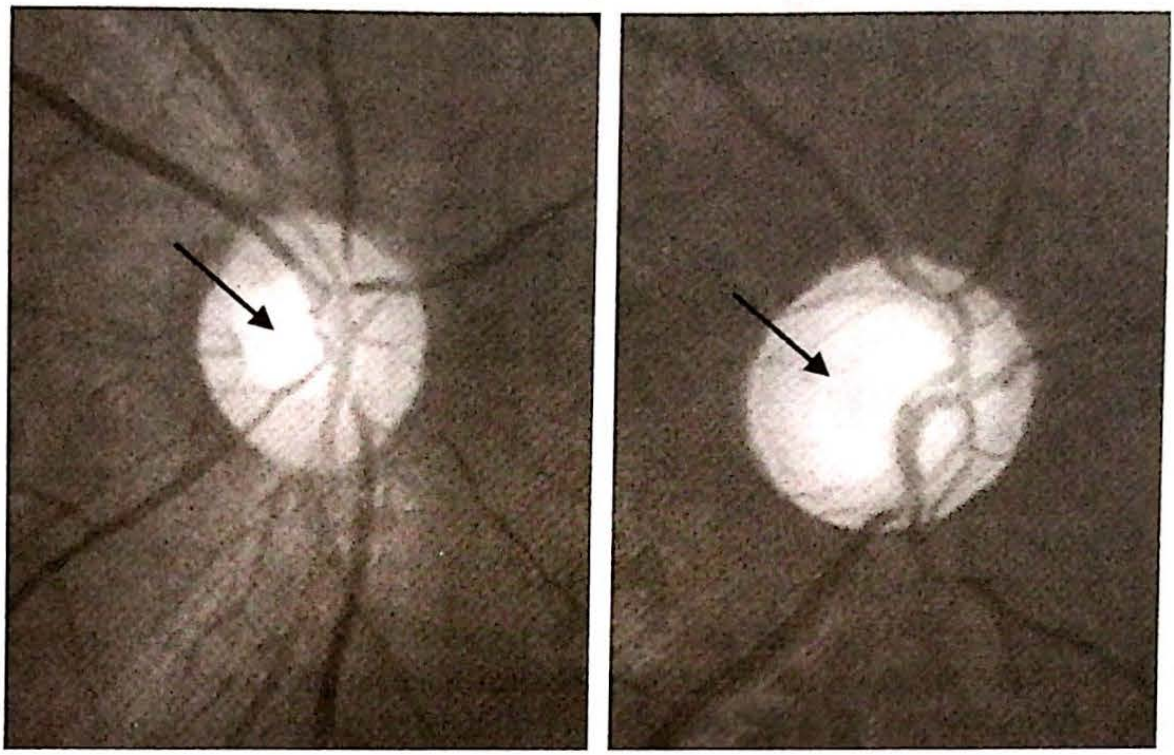
3. Evaluation of Optic Disc

Glaucomatous disc damage is due to:
- Mechanical effect of raised IOP
- Compromised blood supply

Optic disc evaluation is clinically done by **Direct Ophthalmoscope/90D lens/60D lens.** The machines available for the same are:

- **Optical Coherence Tomography(OCT)**Q
- **Confocal Scanning Laser Polarimetry**Q

Glaucomatous optic disc changes



Normal disc **Glaucomatous disc**

- **Increase in Cup: Disc ratio due to enlargement of the optic cup**Q (The physiological cup is a depression at the centre of the disc. The black arrows in the photographs point to the optic cup. As visible in the photographs here, the Cup: Disc ratio in the normal disc is around 0.3:1 whereas in the glaucomatous disc, it is very much increased)
- Asymmetry of >0.2 in the C:D ratio between the two eyes
- Thinning of neuroretinal rim which usually follows the **ISNT** rule (Inferior- Superior-Nasal-Temporal). Hence the upper and lower rims are thinned first making the cup **vertically oval**Q**.**
- **Laminar dot sign**Q**:** The pores of the lamina cribrosa become visible through the optic cup as the cup becomes deep.
- Bayonetting of blood vessels
- **Nasal shifting of vessels**Q
- Nerve fibre bundle defects

4. Visual Field Evaluation or Perimetry

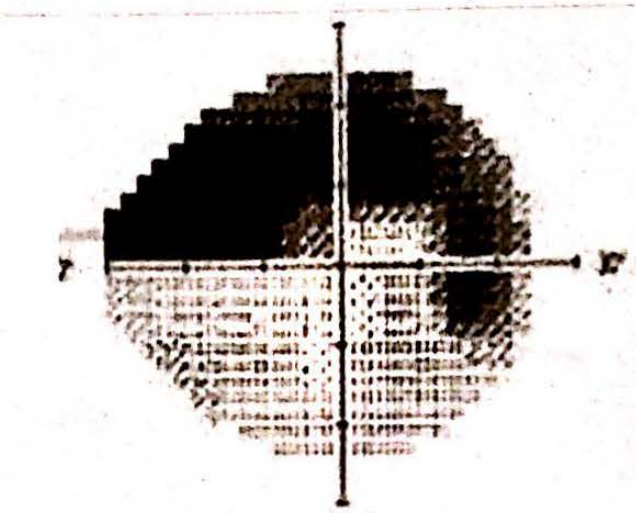
The normal visual field is:

- 50 degrees superiorly
- 60 degrees nasally
- 70 degrees inferiorly
- 100°–110° temporally

Blind spot: Between 10°–20° temporally

Visual field changes in glaucoma

- **Paracentral scotomas (earliest reliable field defect in glaucoma)Q**
- Seidel scotoma
- Nasal step of Roenne
- **Arcuate or Bjerrum scotoma**
- Ring scotoma
- Total scotoma



Arcuate scotoma (characteristic scotoma of glaucoma)

5. **Tonography:** This test is used to measure **the facility of aqueous outflowQ.**

Classification of Glaucoma

1. Primary glaucoma
   - Primary open angle glaucoma (POAG)
   - Primary angle closure glaucoma (PACG)
2. Secondary glaucoma: Glaucoma associated with other ocular and systemic diseases
3. Developmental glaucoma
   - Primary congenital glaucoma or Buphthalmos
   - Developmental glaucoma with associated anomalies.

Primary Open Angle Glaucoma

Primary open angle glaucoma (POAG) is a bilateral disease characterized by:

- Adult onset
- IOP > 21mm Hg or diurnal variation> 8 mm Hg
- Open angles on gonioscopy
- Optic disc changes suggestive of glaucoma
- Visual field changes specific to glaucoma

Risk Factors

- Age more than 65 years of age
- Diabetes mellitus

- Thyroid disorders
- Family history
- Myopia

Genetics: POAG is associated with six different locii on the human genome designated as **GLC1A-F**. The genes identified are **Myocillin**[Q] gene and **Optineurin**[Q] gene.

Mechanism of glaucoma: Trabecular meshwork sclerosis with reduction in intertrabecular spaces increases resistance to aqueous outflow. This results in increase in IOP.

Presentation: Patient is usually asymptomatic in the early stages. The patient may present with non-specific complaints of eye pain, headache and fatigue on near work. **Difficulty in vision at night may be a feature**[Q]**.** Blurring of vision and loss of visual field are complained in the advanced stage.

Diagnosis: IOP evaluation, Gonioscopy, Optic disc, Visual fields

Treatment

- Medical therapy
- **Laser trabeculoplasty**[Q]
- Filtering surgery when maximum tolerable medical therapy is not sufficient to control IOP

POAG has two variants:

- **Ocular hypertension (OHT)**: This is a subset of POAG where IOP > 21 mm Hg with no evident damage to the optic nerve or visual field
- **Normal tension glaucoma (NTG)**[Q]: It is considered to be a subset of POAG wherein the IOP does not rise beyond 21mmHg but optic disc and visual field changes specific to glaucoma are present. **Optic nerve head haemorrhage is seen commonly in NTG**[Q]**.**

Primary Angle Closure Glaucoma

Primary angle closure glaucoma (PACG) is a condition where optic nerve damage and visual field loss have resulted due to primary angle closure. Primary angle closure means elevation of IOP due to obstruction of aqueous outflow when the trabecular meshwork is occluded by the peripheral iris.

Risk Factors

- Age more than 65 years
- Sex – females
- **High hypermetropia**[Q]
- **Nanophthalmos**[Q]
- **Microcornea**[Q]
- Thick lens
- **Plateau iris**[Q]

Pathogenesis

- **Pupillary block:** Dilatation of the pupil drags the iris tissue peripherally leading to crowding of the angle. Dilatation also makes the iris flaccid so that it touches the surface of the lens. This prevents aqueous from travelling from posterior to anterior chamber. Accumulation of aqueous behind the iris pushes the iris forward and closes the angle.
- **Synechiae formation:** Shallow anterior chamber with narrow angles leads to formation of anterior synechiae (adhesions between iris and cornea). This also leads to angle closure.

Stages of PACG

- **Angle closure suspect:** No symptoms are present. During a routine examination, shallow anterior chamber and narrow angles are identified on gonioscopy. This stage indicates a possibility of developing angle closure at a later date. Treatment is **prophylactic laser iridotomy**Q.
- **Angle closure stage:** Intermittent attacks of angle closure are seen. These attacks are **usually precipitated by dim light**Q**, stress, and mydriatic drugs which cause pupillary dilatation.** During the attack, there is sudden pain, redness and blurring of vision in the affected eye. Colored haloes may be present due to corneal epithelial edema. The attack resolves spontaneously when the pupil constricts. Treatment is **prophylactic laser iridotomy**Q.
- **Acute angle closure glaucoma**Q: In this condition, there is complete closure of the angle leading to severe rise in IOP. This is an ophthalmic emergency and the patient presents with the following features:
  - Severe pain in the eye, associated with headache
  - Nausea and vomiting
  - Severe decrease in vision associated with redness of the affected eye
  - Lid edema, chemosis, circumcorneal congestion
  - Cornea is **oedematous and hazy**Q
  - Anterior chamber is very shallow and may be associated with cells and flare.
  - Pupil is **vertically oval, mid dilated, fixed/nonreacting**Q
  - IOP markedly increased to 60–70 mmHg

  Treatment
  - Immediate lowering of IOP with intravenous **Mannitol**Q or oral **Acetazolamide**Q
  - Constriction of the pupil with 1% **Pilocarpine**Q drops to break the attack
  - Aqueous suppressant eye drops
  - **Laser iridotomy**Q: This is done when the acute attack has resolved and the corneal oedema has cleared. It is also done prophylactically in the other eye as the disease is bilateral
- **Chronic angle closure glaucoma:** Formation of peripheral anterior synechiae leads to constantly high IOP with associated damage to optic nerve and visual field loss.
  Vogt's triad is characteristic: Pigment dispersion on corneal endothelium+ Sectoral iris atrophy+ Glaucomaflecken (Ant. capsular lens cataract)Q.
  Treatment is medical management followed by glaucoma filtration surgery

- **Absolute angle closure glaucoma:** This is the stage of a painful blind eye due to permanently high IOP. Treatment is **cyclodestruction** [Q] by laser or cryotherapy

Provocative tests[Q]**:** Prior to the advent of gonioscopy, these tests were used to identify patients with occludable angles. If an increase in IOP >8 mm Hg was seen, the test was considered positive and the angle as occludable. These tests are not done nowadays.

- Mydriatic provocative test
- **Dark Room test**[Q]
- Prone provocative test

Primary Congenital Glaucoma

Primary congenital glaucoma is a type of paediatric glaucoma where developmental anomaly of the angle of anterior chamber leads to obstruction of aqueous outflow and consequent rise in IOP. The distinctive feature of this condition is enlargement of the eyeball in response to the high IOP. This is called **Buphthalmos**[Q]**.**

Symptoms:

- **Lacrimation**[Q]
- **Photophobia**[Q]
- **Blepharospasm**[Q]

This **triad** of symptoms is due to the corneal epithelial oedema that results from increased IOP.

Signs

- **Buphthalmos**
- Increased corneal diameter: (>12 mm)
- Corneal edema is present associated with **Haab's striae**[Q] (Tears in the Descemet's membrane)
- Deep AC
- Antero-posteriorly flat lens, Subluxated lens
- Optic disc shows glaucomatous cupping
- Tonometry shows high IOP

Management: **Surgery is the treatment**[Q]**.** The options are:

- Goniotomy
- Trabeculotomy
- Trabeculectomy

Trabeculotomy combined with Trabeculectomy[Q] is the best modality

Developmental Glaucoma with Associated Anomalies

A. Associated with **irido-corneal dysgenesis**[Q]
   - Axenfeld's anomaly
   - Riegers anomaly
   - Riegers syndrome
   - Peters anomaly

B. Associated with **Aniridia**[Q]
C. Associated with **Ectopia lentis**[Q]: Marfan, Weil-Marchesani and Homocystinuria
D. Associated with congenital ectropion uveae
E. Associated with **Nanophthalmos**[Q]
C. Associated with systemic conditions like
- **Naevus of Ota**[Q]
- Lowe's syndrome
- Phacomatosis like **Sturge-Weber syndrome**[Q]
- **VonRecklinghausen's Disease**[Q]

Secondary Glaucoma

A. Lens induced glaucoma
- **Phacomorphic glaucoma**[Q]**:** It is a type of secondary angle closure glaucoma due to **intumescent cataract**[Q]**:** The swollen cataractous lens pushes the iris forward to occlude the angle.
- **Phacolytic glaucoma**[Q]**:** It is a type of secondary open angle glaucoma due to **hypermature Morgagnian cataract**[Q]**:** It is also called **lens protein glaucoma**[Q]. The liquefied cortical protein matter leaks through the intact capsule and blocks the pores of the trabecular meshwork.
- **Phacotoxic glaucoma:** This is seen in traumatic rupture of the capsule. The lens material in the anterior chamber blocks the pores of the trabecular meshwork.
- **Phacoanaphylactic glaucoma:** This is seen in traumatic rupture of the capsule. The inflammatory reaction to the lens matter blocks the trabecular meshwork.

Treatment of lens induced glaucoma is lowering of IOP with drugs followed by **cataract surgery**[Q].

B. Iridocorneo-endothelial syndrome (ICE)[Q]

This is a condition characterized by **abnormal proliferation of corneal endothelium across the angle of anterior chamber**[Q]. This membrane causes obstruction to aqueous outflow. It has three variants:
- **Progressive iris atrophy**[Q]
- **Chandler's syndrome:** Associated with abnormal corneal endothelium
- **Cogan Reese syndrome:** Associated with iris naevus

Treatment is **medical management and filtering surgery**.

C. Pigmentary glaucoma
- In this condition, there is loss of iris pigments which subsequently get deposited in different ocular tissues like cornea, lens capsule and trabecular meshwork. This is called **pigment dispersion.**
- Glaucoma results due to blockage of trabecular meshwork by these pigments
- It is common in **young males**[Q] between 25–35 years of age with associated **myopia**[Q].
- **Iris heterochromia and transillumination defects**[Q] are seen. **Krukenberg spindle**[Q] (vertical spindle of pigments on corneal endothelium) is a characteristic feature.

Treatment is **medical management, laser trabeculoplasty**[Q]**, filtration surgery.**

D. Pseudoexfoliation glaucoma (Glaucoma capsulare)

Pseudoexfoliation is a condition where an **amorphous, eosinophilic material** produced from the **lens epithelium** is deposited in different ocular tissues like corneal endothelium, lens, zonules, pupillary margin and trabecular meshwork. Glaucoma results from blockage of the trabecular meshwork by this substance.

Treatment is **medical management, laser trabeculoplasty** Q **and filtration surgery.**

E. Neovascular glaucoma

Glaucoma resulting from retinal ischaemic disorders like **Proliferative Diabetic Retinopathy, CRVO, Eales disease, ROP.**

Treatment is **panretinal photocoagulation, glaucoma valve surgery.** (Explained in detail in the chapter on Retina)

F. Inflammatory glaucoma/ Uveitic glaucoma

Glaucoma may be seen in acute or chronic uveitis especially uveitic syndromes like **Fuchs heterochromic iridocyclitis** and **Possner Schlossman syndrome**Q. In these conditions, the inflammation is relatively mild but the rise of IOP is high. This is called **glaucomatocyclitic crisis**Q.

Treatment is **steroids and IOP lowering drugs.** (Explained in detail in the chapter on uveitis)

G. Steroid induced glaucoma

Steroids, mainly by **topical route of administration**Q lead to glaucoma. Steroids increase the **synthesis of glycosaminoglycans**Q which leads to thickening of the trabecular meshwork.

Treatment is to discontinue steroids and medical control of IOP.

H. Traumatic glaucoma

Glaucoma may result from penetrating or blunt trauma. The different mechanisms are:

- **Hyphaema**Q
- Inflammatory glaucoma
- Haemolytic glaucoma
- Haemosiderotic glaucoma
- Ghost cell glaucoma
- **Angle recession glaucoma**Q
- Glaucoma associated with subluxation/dislocation of lens

I. Glaucoma associated with elevated episcleral venous pressure

Seen in **thyroid orbitopathy, orbital inflammations, carotido-cavernous fistula.**

Treatment of the cause

J. Malignant glaucoma/ciliary block glaucoma

- This is a type of **secondary angle closure glaucoma** which is seen after intraocular surgeries like trabeculectomy, cataract etc.
- The cause is **posterior misdirection of aqueous leading to expansion of vitreous volume**Q. The expanded vitreous pushes the iris diaphragm forward causing angle occlusion.

Treatment is **atropine**Q**, laser hyloidotomy and vitrectomy**Q**.**

Antiglaucoma Drugs

Beta Blockers		
Timolol maleate[Q] (nonselective Beta blocker) **0.5% drops bd**	**Aqueous suppressant** Decreases production of aqueous by acting on beta receptors in the ciliary body[Q]	Hypotension Bradycardia Bronchospasm **Dry eye**[Q] **Superficial punctate keratitis**[Q]
Betaxolol (Selective Beta$_1$ blocker) (0.5% drops bd)	Same	Same but bronchospasm is avoided
Adrenergic Agonists		
Brimonidine (0.2%, 0.1% drops bd)[Q]	**Aqueous suppressant**[Q] Decreases aqueous production by acting on alpha$_2$ receptors of the ciliary body Enhances outflow through the trabecular meshwork	**Allergic conjunctivitis**[Q] **CNS depression**[Q] **Cystoid macular edema**[Q] **Lid retraction**[Q]
Apraclonidine (0.5% drops)[Q]	Same	**Same but lid retraction is more**[Q] Tachyphylaxis[Q]
Cholinergics		
Pilocarpine(2% drops tds)[Q]	Increases trabecular meshwork outflow **In angle closure glaucoma, it breaks the pupillary block by miosis**[Q]	**Brow ache**[Q] Myopia **(accommodative spasm)**[Q] Retinal detachment[Q] Miosis **Iris cysts**[Q]
Carbonic Anhydrase Inhibitors		
Acetazolamide (250 mg tablet tds)[Q]	**Aqueous suppressant:** Decreases aqueous production by inhibition of carbonic anhydrase[Q]	**Paresthesias**[Q] GI upset Renal stones **Sulfa sensitivity**[Q] **Metabolic acidosis**[Q] Aplastic anemia
Dorzolamide (2% drops tds)[Q]	Same	**Endothelial decompensation**[Q] Bitter taste
Prostaglandin Analogues		
Latanoprost (0.005% drops od)[Q] **Bimatoprost (0.03% drops od)**[Q] **Travoprost (0.004% drops od)**[Q]	**Increases uveoscleral outflow**[Q]	**Iris pigmentation**[Q] Cystoid macular edema Anterior uveitis **Hypertrichosis**[Q]
Hyperosmotics		
Glycerol 50% oral	**Decreases the vitreous volume by drawing water from the vitreous**[Q]	Nausea, vomiting, Cardiac arrhythmia Cardiac overload
Mannitol 20% intravenous	Same	Electrolyte imbalance Congestive heart failure
Urea intravenous (rarely used)	Same	Same

Laser Procedures

A. Laser Iridotomy

Indications:

- **Angle closure glaucoma**[Q]
- **Prophylactic iridotomy in the fellow eye of ACG**[Q]

Laser used: **Nd: YAG**[Q]

Laser iridotomy is usually done in the superonasal quadrant

B. Argon Laser Trabeculoplasty

Indications:

- **Primary Open angle glaucoma**[Q]
- **Pseudoexfoliation glaucoma**[Q]
- **Pigmentary glaucoma**[Q]

Laser used: **Argon Laser**[Q]

Glaucoma Surgeries

- Goniotomy and Trabeculotomy (for congenital glaucoma)
- Filtration surgery:Trabeculectomy
- Filtration surgery with antimetabolites like Mitomycin C
- Nonpenetrating filtration surgery: Visco- canalostomy, Deep sclerectomy
- Glaucoma valve surgery
- Express shunt surgery[Q]
- Cyclodestructive surgery for absolute glaucoma

Glaucoma Valves

These are used in cases of refractory glaucoma or when filtering surgery has high chances of failure. These implants are placed subconjunctivally and have a drainage tube in the anterior chamber. This tube drains a controlled amount of aqueous to the sub-Tenon space.

Indications:

- Failed trabeculectomy
- Neovascular glaucoma
- Uveitic glaucoma
- Post Keratoplasty glaucoma
- Post retinal surgery glaucoma
- Primary congenital glaucoma (intractable)

Example: Ahmed Glaucoma Valve (AGV)

QUESTIONS

1. **Cells affected in glaucomatous optic neuropathy are:** *(AIIMS 2014/2013)*
   a. Amacrine cells
   b. Bipolar cells
   c. Ganglion cells
   d. Rods and cones
2. **In the conversion of CO_2 and H_2O to form carbonic acid during formation of aqueous humour, the enzyme catalyzing the reaction is:** *(AIIMS)*
   a. Carboxylase
   b. Carbamylase
   c. Carbonic anhydrase
   d. Carbonic dehydrogenase
3. **Regarding aqueous humour, which of the following statements is/are true:** *(PGI)*
   a. It is secreted at a rate of 2-3 microlitre/min
   b. Secreted by ciliary processes
   c. Has less protein than plasma
   d. Provides nutrition
   e. Normal IOP is 5-15 mmHg
4. **Which of the following is used as self-tonometer?** *(AIIMS 2014)*
   a. Diaton palpebral tonometer
   b. Rebound tonometer
   c. Perkin's tonometer
   d. Dynamic contour tonometer
5. **Tonometer used in irregular cornea:** *(AIIMS)*
   a. Mackay-Marg tonometer
   b. Rebound tonometer
   c. Draeger's tonometer
   d. Maklakov tonometer
6. **Tonometer with variation in applanation surface is:** *(AIIMS)*
   a. Maklakov tonometer
   b. Mackay-Marg tonometer
   c. Rebound tonometer
   d. Draeger tonometer
7. **Critical angle of air-cornea interface:** *(AIIMS)*
   a. 46 degrees
   b. 64 degrees
   c. 24 degrees
   d. 36 degrees
8. **Which of the following procedures is not done in dilated pupil?** *(AIIMS 2014)*
   a. Gonioscopy
   b. Fundoscopy
   c. Laser inferometry
   d. Electroretinogram
9. **Schwalbe's line is:** *(DNB)*
   a. The posterior limit of the Descemet's membrane
   b. The posterior limit of the Bowman's membrane
   c. The anterior limit of the Descemet's membrane
   d. The anterior limit of the Bowman's membrane
10. **Visual field abnormalities in the Bjerrum's area are seen in:** *(Kerala PG 2015)*
    a. Cataract
    b. Glaucoma
    c. Keratitis
    d. Proptosis
11. **Tonography is used to determine:** *(AIPG)*
    a. The rate of formation of aqueous
    b. The facility of aqueous outflow
    c. The IOP at different times
    d. None of the above
12. **True about Buphthalmos is/are:** *(PGI)*
    a. Large cornea
    b. Shallow anterior chamber
    c. Haab's striae
    d. High IOP
    e. Medical management is the key
13. **A baby about 30 days old presents with excessive lacrimation and photophobia.**

He has large and hazy cornea in both eyes. His lacrimal system is normal. What is the probable diagnosis? *(AIIMS)*

a. Congenital glaucoma
b. Megalocornea
c. Keratoconus
d. Hunter's syndrome

14. The treatment of congenital glaucoma is: *(AIIMS)*

a. Essentially topical medication
b. Trabeculoplasty
c. Trabeculotomy with trabeculectomy
d. Cyclocryotherapy

15. Which of the following does not cause hazy cornea in a newborn? *(AIPG)*

a. Endothelial dystrophy
b. Mucoplysaccharidosis
c. Sclerocornea
d. Droplet keratopathy

16. Shallow anterior chamber is seen in all *except*: *(TNPG)*

a. Old age
b. Hypermetropia
c. Steroid induced glaucoma
d. Angle closure glaucoma

17. Which statements regarding depth of anterior chamber is/are false: *(PGI)*

a. Depth is less in women than men
b. Depth corresponds to the volume of aqueous humour
c. Depth increases with age
d. Depth is less in hypermetropes
e. Depth is more in myopes

18. All of the following predispose to angle closure glaucoma *except*: *(AIIMS)*

a. Small cornea
b. Flat cornea
c. Shallow anterior chamber
d. Short axial length of the eyeball

19. True about PACG is/are: *(PGI)*

a. More common in females
b. Shallow anterior chamber is a risk factor
c. Deep anterior chamber is a risk factor
d. Small diameter of the cornea is a risk factor
e. More common in myopes

20. A 36-year-old female develops pain in the eyes after prone dark room test. Which of the drugs should be avoided? *(AIIMS 2013)*

a. Acetazolamide
b. Pilocarpine
c. Atropine
d. Timolol

21. Kusumlata presents with acute painful red eye and vertically oval mid-dilated pupil. Most likely diagnosis is: *(AIIMS/AIPG)*

a. Acute retrobulbar neuritis
b. Acute angle closure glaucoma
c. Acute anterior uveitis
d. Severe keratoconjunctivitis

22. A 60-year-old male presents with coloured haloes. On Fincham's test, the haloes split and then reunite. The most probable diagnosis is: *(AIIMS)*

a. Acute congestive glaucoma
b. Open angle glaucoma
c. Senile immature cataract
d. Mucopurulent conjunctivitis

23. A 55-year-old female comes to the casualty with history of severe eye pain, redness and diminution of vision. On examination the visual acuity is 6/60, there is circumcorneal congestion, corneal edema and a shallow anterior chamber. Which is the drug of choice: *(AIIMS)*

a. Atropine ointment
b. Intravenous Mannitol
c. Ciprofloxacin eye drops
d. Betamethasone eye drops

24. First drug to be given in acute angle closure glaucoma: *(AIIMS)*

a. Acetazolamide
b. Atropine

c. Pilocarpine
d. Timolol

25. Drug of choice for acute angle closure glaucoma: *(AIIMS/AIPG)*
a. Pilocarpine
b. Atropine
c. Timolol
d. Acetazolamide

26. Treatment of choice for acute angle closure glaucoma: *(AIPG)*
a. Pilocarpine
b. Laser iridotomy
c. Timolol
d. Trabeculoplasty

27. Drugs used in acute congestive glaucoma are all *except*: *(PGI)*
a. Atropine
b. Pilocarpine
c. Acetazolamide
d. Mannitol
e. Timolol

28. Treatment of choice of fellow eye in acute congestive glaucoma: *(AIIMS/PGI)*
a. Pilocarpine
b. Nd: YAG iridotomy
c. Peripheral iridectomy
d. Careful follow-up

29. Treatment of choice for absolute glaucoma: *(APPG)*
a. Cyclocryotherapy
b. Acetazolamide
c. Trabeculectomy
d. Timolol

30. Open angle glaucoma causes: *(COMEDK)*
a. Sudden loss of vision
b. Difficulty in dark adaptation
c. Amaurosis fugax
d. Uniocular diplopia

31. In POAG, which of the following is not seen: *(PGI)*
a. Vertical cupping
b. Horizontal cupping
c. Bayonetting of vessels
d. Dot sign

32. Earliest field defect in primary open angle glaucoma: *(AIIMS)*
a. Paracentral scotoma
b. Ring scotoma
c. Seidel scotoma
d. Arcuate scotoma

33. A 70-year-old patient presents with progressive deterioration of vision. On examination, the pupillary reaction is sluggish and the IOP is normal. Fundoscopy shows a large and deep cup. Visual field reveals paracentral scotoma. What is the probable diagnosis? *(AIPG)*
a. Primary angle closure glaucoma
b. Normal tension glaucoma
c. Neovascular glaucoma
d. Absolute glaucoma

34. A male patient with history of hypermature cataract presents with sudden onset pain, redness, photophobia in the right eye. On examination, there is a deep anterior chamber with raised IOP. The left eye is normal. What is the likely diagnosis? *(AIIMS)*
a. Phacomorphic glaucoma
b. Phacolytic glaucoma
c. Phacotoxic glaucoma
d. Phacoanaphylactic uveitis

35. Iridocorneoendothelial syndrome is associated with: *(AIIMS)*
a. Progressive iris atrophy
b. Bilateral stromal edema of cornea
c. Deposition of collagen in the Descemet's membrane
d. Deposition of glycosaminoglycan in the Descemet's membrane

36. Malignant glaucoma is seen in: *(PGI)*
a. After intraocular surgery
b. Intraocular malignancy
c. Trauma
d. Thrombosis

37. **Malignant glaucoma, correct statements is/are:** *(PGI)*
    a. Anterior chamber is normal
    b. Misdirected aqueous flow
    c. Pilocarpine is the drug of choice
    d. Management is medical only
    e. Atropine is the drug of choice

38. **Neovascularisation of iris is seen in all *except*:** *(MPPG)*
    a. CRVO
    b. Diabetic retinopathy
    c. Fuchs heterochromic iridocyclitis
    d. Congenital cataract

39. **The laser procedure used for treating rubeosis iridis is:** *(AIIMS)*
    a. Goniophotocoagulation
    b. Panretinal photocoagulation
    c. Laser trabeculoplasty
    d. Laser iridotomy

40. **A 25-year-old patient presents with painless red eye with an IOP of 60 mm Hg. What is the most likely diagnosis?** *(AIIMS 2014)*
    a. Chronic papilloedema
    b. Acute angle closure glaucoma
    c. Glaucomatocyclitic crisis
    d. Acute anterior uveitis

41. **Krukenberg spindle is seen in:** *(DNB)*
    a. Pigmentary glaucoma
    b. Sympathetic ophthalmitis
    c. Retinitis pigmentosa
    d. Chalazion

42. **Krukenberg spindle is seen in:** *(APPG)*
    a. Corneal endothelium
    b. Retina
    c. Lens
    d. Conjunctiva

43. **Which of the following drugs is not used topically for the treatment of glaucoma?** *(AIPG)*
    a. Timolol
    b. Latanoprost
    c. Acetazolamide
    d. Dorzolamide

44. **Contraindications for topical beta-blockers are:** *(PGI)*
    a. Hypertension
    b. Asthma
    c. Tachycardia
    d. Hypotension
    e. Depression

45. **Which of the following anti-glaucoma medications can cause drowsiness?** *(AIPG)*
    a. Latanoprost
    b. Brimonidine
    c. Timolol
    d. Dorzolamide

46. **Which anti-glaucoma medication is unsafe in infants?** *(DPG)*
    a. Timolol
    b. Brimonidine
    c. Dorzolamide
    d. Latanoprost

47. **Latanoprost acts in glaucoma by:** *(AIPG)*
    a. Decreasing aqueous humour production
    b. Increasing uveoscleral outflow
    c. Increasing trabecular outflow
    d. Releasing pupillary block

48. **Which of the following topical drugs causes heterochromia iridis?** *(AIIMS 2015)*
    a. Latanoprost
    b. Prednisolone
    c. Olopatadine
    d. Timolol

49. **Which of the following drugs is not used in a patient of acute congestive glaucoma having a history of sulfa allergy?** *(AIIMS)*
    a. Glycerol
    b. Acetazolamide
    c. Mannitol
    d. Latanoprost

50. **Hyperosmotic agents act by:** *(APPG)*
    a. Increasing aqueous outflow
    b. Decreasing aqueous production

c. Decreasing vitreous volume
d. Increasing uveoscleral outflow

51. Which drug is contraindicated in uveitic glaucoma? *(DNB)*
a. Beta blockers
b. Mydriatic
c. Miotics
d. Carbonic anhydrase inhibitors

52. Which of these drug combinations is not generally used in glaucoma? *(DNB)*
a. Timolol + Latanoprost
b. Timolol+ Brimonidine
c. Timolol+ Pilocarpine
d. Pilocarpine + Latanoprost

53. Laser trabeculoplasty is indicated in: *(AIIMS 2014)*
a. Neovascular glaucoma
b. Pseudoexfoliation glaucoma
c. Chronic angle closure glaucoma
d. Uveitic glaucoma

54. Express shunt in glaucoma is made up of: *(AIIMS 2014)*
a. Silicon
b. Titanium
c. Gold
d. Stainless steel

55. Treatment options for glaucoma are all *except*: *(PGI)*
a. Trabeculotomy
b. Trabeculectomy
c. Visco canalostomy
d. Vitrectomy
e. Iridectomy

56. Hypersecretory glaucoma is seen in: *(AIPG)*
a. Epidemic dropsy
b. Marfan's syndrome
c. Hypertension
d. Diabetes

ANSWERS AND EXPLANATIONS

1. **c. Ganglion cells.** The axons of the ganglion cells of the retina form the nerve fibre layer which continues as the optic nerve. Hence, glaucoma is associated with ganglion cell loss
2. **c. Carbonic anhydrase**
3. **a. It is secreted at the rate of 2–3 microlitres per minute. b. Secreted by ciliary processes. c. Has less protein than plasma. d. Provides nutrition**
   The normal IOP is 10–21 mm Hg
4. **b. Rebound tonometer**
5. **a. Mackay-marg tonometer**
6. **a. Maklakov tonometer**
7. **a. 46 degrees**
8. **a. Gonioscopy**
9. **c. The anterior limit of the Descemet's membrane**
10. **b. Glaucoma**
11. **b. The facility of aqueous outflow**
12. **a. Large cornea c. Haab's striae d. High IOP.**
13. **a. Congenital glaucoma**
14. **c. Trabeculotomy with trabeculectomy**
15. **d. Droplet keratopathy.**
    Causes of Hazy cornea in a child (STUMPED)
    - S- Sclerocornea
    - T- Trauma during birth
    - U- Ulcer
    - M- Mucoplysaccharidosis
    - P- Peter's anomaly
    - E- Endothelial dystrophy
    - D- Dermoid (limbal)

    Also Congenital glaucoma.
16. **c. Steroid induced glaucoma**
17. **b. Depth depends on volume of aqueous humour. c. Depth increases with age**
18. **b. Flat cornea**
19. **a. More common in females b. Shallow anterior chamber is a risk factor. d. Small diameter of the cornea is a risk factor**
20. **c. Atropine.**
    Pain after dark room test indicates that the patient has occludable angles. Hence, atropine is contraindicated because it is a long acting mydriatic and may precipitate angle closure
21. **b. Acute angle closure glaucoma**
22. **c. Senile immature cataract**
    Fincham's test helps to differentiate between coloured haloes in immature cataract and acute congestive glaucoma. When viewed through a stenopaeic slit, the haloes of an immature cataract are broken up into segments but the halo in ACG remains intact.

23. **b. Intravenous Mannitol**
    The first drug to be administered in a case of Acute ACG is intravenous Mannitol or oral Acetazolamide. This helps to reduce the IOP by dehydrating and reducing the vitreous volume.
    Following this Pilocarpine eye drops are used to constrict the pupil and break the acute attack. Thus, **pilocarpine is the definitive drug for Acute ACG.**
    Finally, when the corneal oedema has resolved, the patient has to undergo laser iridotomy to prevent such attacks in future. Laser has to be done in the fellow eye also as the disease is bilateral. Hence, **laser iridotomy is the definitive management of Acute ACG.**
24. **a. Acetazolamide**
25. **a. Pilocarpine**
26. **b. Laser iridotomy**
27. **a. Atropine**
28. **b. Nd- YAG iridotomy**
29. **a. Cyclocryotherapy**
30. **b. Difficulty in dark adaptation**
31. **b. Horizontal cupping**
32. **a. Paracentral scotoma**
33. **b. Normal tension glaucoma**
    The question describes a patient with normal IOP but optic disc changes and visual field changes suggestive of glaucoma. Hence, the answer is normal tension glaucoma.
34. **b. Phacolytic glaucoma**
    - Cataract with glaucoma with shallow anterior chamber: Phacomorphic glaucoma
    - Cataract with glaucoma with normal or deep anterior chamber: Phacolytic glaucoma
35. **a. Progressive iris atrophy**
36. **a. After intraocular surgery**
37. **b. Misdirected aqueous flow. e. Atropine is the drug of choice**
38. **d. Congenital cataract**
39. **b. Panretinal photocoagulation**
40. **c. Glaucomatocyclitic crisis**
41. **a. Pigmentary glaucoma**
42. **a. Corneal endothelium**
43. **c. Acetazolamide**
    It is used orally.
44. **b. Asthma. d. Hypotension**
45. **b. Brimonidine**
46. **b. Brimonidine.** Brimonidine causes CNS depression and drowsiness and hence is contraindicated in children
47. **b. Increasing uveoscleral outflow**

48. **a. Latanoprost.** It causes iris pigmentation and may lead to heterochromia iridis when given unilaterally.

Causes of heterochromia iridis:
• Iris naevus, iris melanoma
• Ocular melanosis
• Fuchs heterochromic iridocyclitis[Q]
• **Siderosis bulbi[Q]**
• Congenital Horner's syndrome
• **Sturge-Weber syndrome[Q]**
• Waardenberg syndrome
• **Latanoprost[Q]**

49. **b. Acetazolamide.** It has the potential to cause SJS in patients of sulfa allergy
50. **c. Decreasing vitreous volume**
51. **c. Miotics.** Miotics should not be given in uveitis as they increase the ciliary spasm
52. **d. Pilocarpine+ Latanoprost.** Both these drugs increase intraocular inflammation and hence the combination is avoided
53. **b. Pseudoexfoliation glaucoma**
54. **d. Stainless steel**
55. **d. Vitrectomy.** Though vitrectomy is a treatment modality for malignant glaucoma, it is the best choice among the available options.
56. **a. Epidemic dropsy**
    Epidemic dropsy is a rare condition seen due to Argemone mexicana poisoning. It is an example where glaucoma is due to excessive production of aqueous humour.

Chapter 4

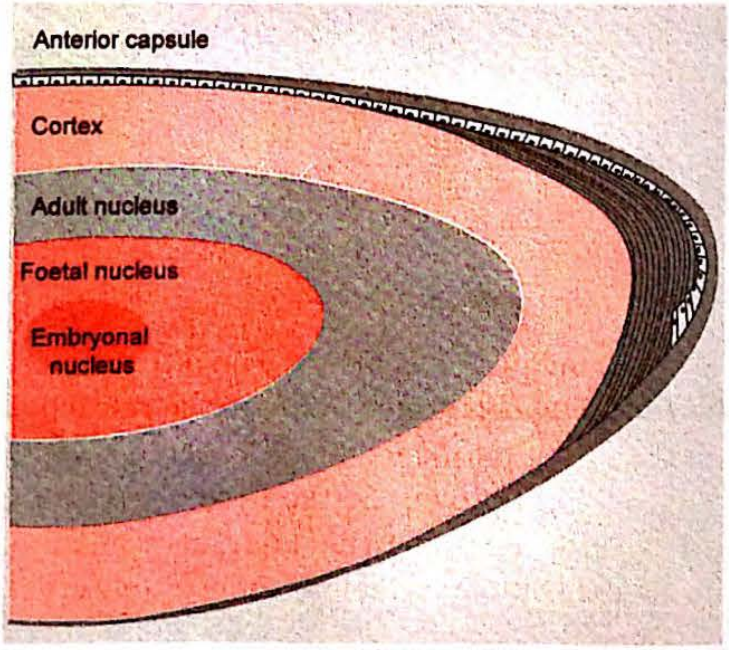
Lens

The lens is a biconvex, transparent crystalline structure which divides the eye into anterior and posterior segments. It is placed in a saucer shaped depression called **patellar fossa**[Q].

- Refractive index: 1.386[Q]
- Refractive power: 14-16 D[Q]
- Equatorial diameter: 10 mm
- It has two surfaces anterior and posterior. **The anterior surface is less convex than the posterior**[Q]**.**
- Radius of curvature:
  - Anterior 10 mm
  - Posterior 6 mm

The lens is made up of:

- Lens capsule: It is a transparent membrane covering the lens. It is **thickest in the pre-equatorial region**[Q] and **thinnest at the posterior pole**[Q]
- Lens epithelium: There is a single layer of epithelium on the anterior surface of the lens but no epithelium on the posterior surface
- Lens fibres: These are protein fibres arranged closely in concentric layers. Lens fibres are formed throughout life so that the oldest fibres are at the centre and the newest fibres are most superficial. Hence, the lens fibres may be divided into



Anatomy of the lens

- **Nucleus**: It is the central part of the lens containing the older fibres. It is again subdivided into **embryonal nucleus** (formed in first 3 months of gestation), **foetal nucleus** (formed till birth), **infantile nucleus** (formed from birth to puberty), **adult nucleus** (formed after puberty)
- **Cortex**: It is the peripheral part containing the newly formed fibres.

The lens is suspended from above and below by the ciliary zonules. The attachment of the vitreous to the posterior surface of the lens is called **Weigert's ligament**[Q]. The potential space between the lens and vitreous is called **retrolental space of Berger**[Q].

Anti-oxidants present in lens

- **Glutathione**[Q]
- **Superoxide dismutase**[Q]
- **Catalase**[Q]
- **Vitamin C**[Q]
- **Vitamin E**[Q]

CONGENITAL AND DEVELOPMENTAL CATARACT

This is due to disturbance in the growth and development of the lens. Disturbance in development during the period of gestation leads to **congenital cataract** and usually affects the **embryonic or foetal nucleus. Developmental cataract** is due to disturbance in development after birth and hence affects the **infantile or adult nucleus and cortex.** The different morphological varieties are:

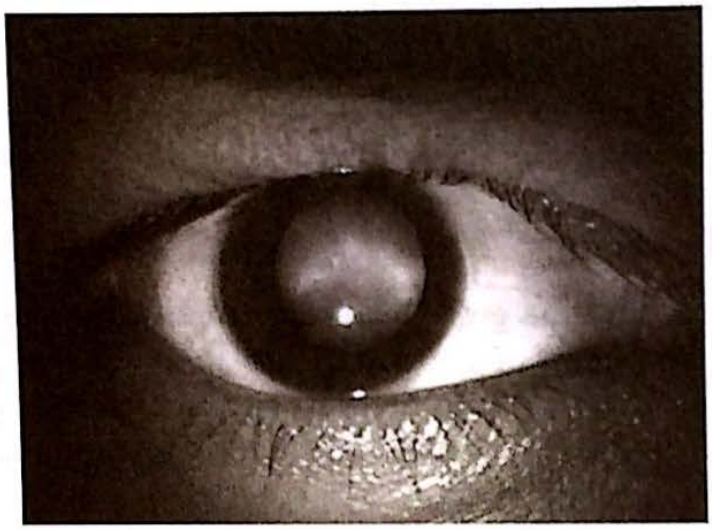
- **Punctate cataract:** It consists of minute white dots through the lens but causes no visual disturbance. It is called **blue dot cataract**[Q]**. It is the most common type of congenital cataract**[Q]
- **Zonular or lamellar cataract: This is the most common type of congenital cataract associated with diminished vision**[Q]. A zone in the foetal nucleus is usually affected. **Hypocalcemia in pregnancy is an important cause**[Q]
- **Nuclear cataract:** This usually affects the **embryonic nucleus. Rubella in pregnancy is an important cause**[Q]
- **Coronary cataract:** It usually occurs around puberty and is characterised by opacities in the periphery of the lens
- Anterior polar cataract
- Posterior polar cataract

SENILE CATARACT

It is the most common type of acquired cataract[Q]. The different morphological types are:

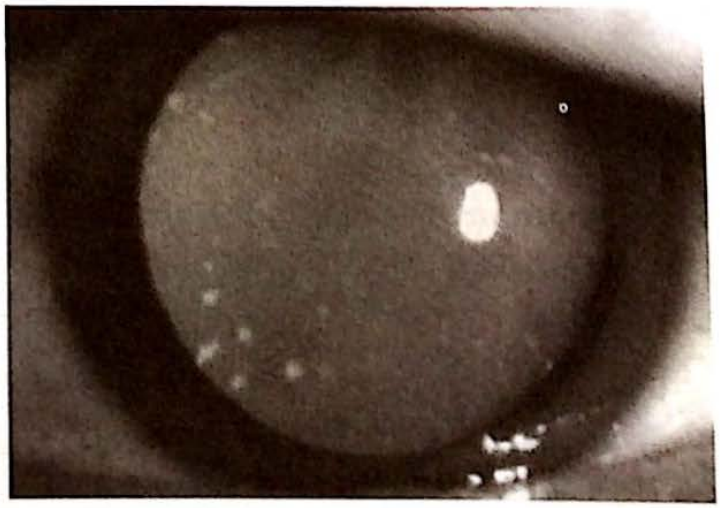
- **Nuclear cataract:** This is an age related increase in the refractive index of the lens nucleus. It causes a myopic shift in refraction known as **index myopia**[Q]. As a result of this myopic shift, the near vision improves. So the need for presbyopic glasses decreases. This is called **second sight of old age**[Q]. The main mechanism of formation of nuclear cataract is conversion of the soluble crystallins to insoluble crystalloids[Q]

- **Cortical cataract** (Cuneiform cataract): This is opacification of the peripheral portion of the lens. Hence, it leads to **visual difficulty in dim light when the pupil is dilated**Q. The main mechanism of formation of cortical cataract is decrease in concentration of proteins and amino acids, increase in sodium with associated hydration of the lens.
- **Subcapsular cataract:** It is usually seen beneath the posterior capsule at the posterior pole. As it lies just at the centre of the visual axis, it is the most visually significant cataract**Q**. It causes **difficulty in vision in bright light**Q and for near workQ.
- **Mature cataract:** When the entire lens has become opacified, it is called mature cataract. **Pearly white**Q **appearance with absence of iris shadow**Q **is seen.** If not operated, it progresses to hypermature cataract



Mature cataract ***(see colour plate 1)***

- **Hypermature cataract:** It has two varieties:
  - Sclerotic cataract: The lens becomes shrunken and small with calcification on the anterior capsule. There is wrinkling of the lens capsule. It predisposes to **subluxation of lens**Q
  - Morgagnian cataract: There is total liquefaction of the cortex as a result of which the nucleus sinks inferiorly. The liquefied cortex leaks through the intact capsule and blocks the trabecular meshwork. This leads to **phacolytic glaucoma**Q **or lens protein glaucoma.** It also causes **phacoanaphylactic uveitis**Q



Morgagnian cataract ***(see colour plate 1)***

- Intumescent cataract: This means a swollen cataract due to excessive hydration. The swollen lens pushes the iris forward leading to closure of the angle. This is called as **phacomorphic glaucoma**Q.

TRAUMATIC CATARACT

It may be seen in different types of injury like:

- **Penetrating injuries as a result of direct injury to the lens:** It is usually **anterior subcapsular or anterior polar cataract**Q.
- **Blunt trauma or concussion injuries:** Due to the impact of the trauma, the iris diaphragm moves backward and touches the lens capsule. As a result, the iris pigments form a ring on the anterior capsule called as **Vossius ring**Q. The typical cataract is called **Rosette cataract**Q
- Thermal injury
- Electrical and radiation injury

METABOLIC CATARACT

Different metabolic diseases are associated with cataract like

- **Diabetes mellitus**: Typical cataract seen in diabetes is called snow flake cataract. Increase in blood glucose leads to increase in glucose content of the lens. This saturates the hexokinase enzyme responsible for directing the glucose to the glycolytic pathway. The excess glucose is diverted to the **sorbitol**Q **pathway. The key enzyme in this pathway is aldose reductase**Q**.** Accumulation of sorbitol causes snow flake cataract.
- **Galactosemia**: It is an autosomal recessive disorder due to deficiency of the enzyme Galactose1-P-uridyl transferase. Accumulation of galactose leads to **oil droplet cataract**Q**.** It is however a **reversible cataract**Q and disappears when galactose is removed from the diet
- Fabry's disease: This is due to deficiency of β-galactosidase A and leads to **spoke-like lens opacities**.
- Lowe's (oculocerebrorenal) syndrome: It is an inborn error of metabolism associated with mental retardation, renal dysfunction, osteomalacia, muscular hypotony, frontal prominence. Ocular features are **posterior lenticonus**Q**, cataract and microspherophakia**Q
- **Wilson's disease**: It is a deficiency of α-2 globulin ceruloplasmin leading to inadequate Cu binding and deposition of Cu in tissues. The ocular features are **Kayser-Fleischer ring (KF)** and **Sunflower Cataract**Q**.** It is also known as **pseudo cataract** because it does not cause visual impairment
- Hypocalcaemia leads to spokes or riders in the cortex of the lens.

TOXIC CATARACT

The drugs causing toxic cataract are:

- **Corticosteroids**: Steroids by systemic route of administrationQ may lead to toxic cataract. The typical morphology is **posterior subcapsular cataract**Q
- Chloroquine

- Chlorpromazine: It causes **star shaped or stellate cataract**
- Miotics may cause **anterior subcapsular cataract**
- **Amiodarone**[Q] may cause **anterior subcapsular cataract**[Q]
- INH and Ethambutol
- Smoking

COMPLICATED CATARACT

This name is given to cataract arising due to inflammatory or degenerative diseases of the eye. It is usually **posterior subcapsular cataract**[Q]. It has a typical **bread crumb appearance**[Q] with **polychromatic lustre**[Q]. The causes are:

- Anterior, intermediate or posterior uveitis
- Retinitis pigmentosa[Q]
- High Myopia[Q]
- Angle closure glaucoma: This is typically **anterior subcapsular cataract**[Q] and is known as **glaucomaflecken**[Q]
- Intraocular tumours

PRESENILE CATARACT

The different conditions associated with presenile cataract are:

- **Myotonic dystrophy**: The typical cataract is **Christmas Tree Cataract**[Q].
- **Syndermatotic cataract:** It is associated with skin disorders like atopic dermatitis, icthyosis and psoriasis
- **Down's syndrome**[Q]
- **Werner's syndrome**
- **Neurofibromatosis**

CLINICAL FEATURES OF CATARACT

- Gradually progressive decrease in vision
- Glare and coloured haloes

  Coloured haloes are also seen in **mucopurulent conjunctivitis** and **acute angle closure glaucoma.** In conjunctivitis, the haloes disappear on washing the eye. For differentiating haloes of cataract and glaucoma, **Fincham's test**[Q] was used. On holding a stenopaeic slit in front of the eye, the haloes due to cataract will break but the haloes due to glaucoma will remain intact.
- Monocular diplopia: This is more common in intumescent cataract.

MANAGEMENT OF CATARACT

The treatment of cataract is cataract surgery. Cataract surgery has evolved over decades to the highest level of predictability and precision today. **The incision in modern day cataract surgery is very small with the benefit of no suture**[Q]**, minimum postoperative astigmatism**[Q] **and quick visual recovery. The intraocular lenses implanted today after**

cataract surgery are foldable, can be implanted through a very small incision and provide visual quality similar to the natural lens.

Preoperative Evaluation of Cataract Patient[Q]

- Systemic evaluation: Diabetes, hypertension, heart disease, lung disease, infections
- **Visual acuity and refraction:** In case of advanced cataract where visual acuity is very poor, **perception of light and projection of rays**[Q] should be assessed. This gives a rough idea about the retina and optic nerve function
- Colour vision: To assess the optic nerve
- Tests for macular function like Potential Acuity Meter (PAM)
- **IOP evaluation by Tonometry**[Q]
- Gonioscopy is done only if IOP is high
- **Pupillary reaction**
- Tests for stereoacuity
- Anterior segment evaluation on slit lamp
- Fundus evaluation: 90D lens, Direct and Indirect Ophthalmoscope
- **Syringing** to check for patency of the lacrimal drainage pathway
- **USG B-Scan** to assess the posterior segment in advanced cataract where fundus cannot be evaluated clinically.
- **OCT:** to assess the macula in case of any pathology
- **Keratometry and Axial length** to calculate IOL power

The different types of cataract surgery are:

- **Intracapsular cataract extraction (ICCE)**: In this technique, the lens along with the capsular bag is removed through a large incision. **The only indication of ICCE today would be subluxated lens**[Q]**.** Intraocular lens has to be iris-fixated, sclera fixated or placed in the anterior chamber.
- **Extracapsular cataract extraction (ECCE)**: In this technique, the lens is removed in toto by making an opening in the anterior capsule. As a result a large limbal incision of 8–9 mm is needed. The capsular bag with an opening in the anterior capsule is left after removal of the lens. **The IOL is placed in the capsular bag**[Q]
- **Small incision cataract surgery (SICS):** The technique is almost similar to ECCE but a corneoscleral tunnel of 6–7mm instead of a limbal incision
- **Phacoemulsification:** In this technique, the lens is emulsified and aspirated by an ultrasound probe after making a circular opening in the anterior capsule. So the incision is small, just enough to permit the entry of the probe **(3.2 mm)**[Q]**. A foldable IOL is implanted in the capsular bag**[Q]
- **Microincision cataract surgery (MICS)**: This is similar to conventional phacoemulsification but the incision is even smaller **(1.8–2.2 mm)**

IOL Power Calculation

Different formulae are used for IOL power calculation:

- **SRK-II** formula[Q]: This is used for emmetropic eyes (Axial length 22–25 mm). The formula states

P = A-2.5L-0.9K (A = constant, L = Axial length, K = keratometry)

- **Hoffer Q** formula: This is used for hypermetropic eyes. (Axial length < 22 mm)
- **SRK-T** formula: This is used for myopic eyes. (Axial length > 25 mm)
- **Haigis/ Holladay II** formula: These are used in post-refractive surgery cases.

Types of Intraocular Lenses

The different types of IOL are:

- Non-foldable lenses: These are usually made up of **Polymethylmethacrylate (PMMA)**. They are posterior chamber IOLs, anterior chamber IOLs, iris-fixated IOLs and sclera fixated IOLs
- Foldable lenses: These are made up of **Acrylic**[Q] **(hydrophobic or hydrophilic)** and hydrogels. They are all posterior chamber IOLs which are placed in the capsular bag. The different types are:
  - **Monofocal IOL**: These provide good distance vision but glasses have to be used for near. (since accommodation is lost after pseudophakia)
  - **Multifocal IOL**: These IOLs have separate zones which focus for distance and near. So they provide good vision both for distance and near without glasses. The main **disadvantage is glare and haloes**[Q]
  - **Accommodative IOLs**: These lenses can move in the capsular bag during accommodation to provide good vision for both distance and near without glasses

Complications of Cataract Surgery

The complications related to surgery are:

- Posterior capsular tear with nucleus drop
- Vitreous loss
- Retinal detachment[Q]
- Postoperative uveitis
- Toxic anterior segment syndrome (TASS)
- Cystoid macular oedema[Q]
- Striate keratopathy
- Pseudophakic bullous keratopathy due to endothelial decompensation
- **Endophthalmitis**[Q]: This is the most dreaded and worst possible complication of cataract surgery. **It is a suppurative inflammation starting from the vitreous which extends to all the parts of the eye except the sclera**[Q]. If the sclera also becomes involved, the condition is then called as **panophthalmitis**[Q]. Postoperative endophthalmitis has the following features:
  - Early onset (within 7 days of surgery): The most common causative organism is **Staphylococcus epidermidis**
  - Late onset: The most common causative organisms are **fungi and Propionibacterium acne**[Q]
  - Prevention is of utmost importance. The main source of infecting organisms is the patient's own flora. Hence, prophylactic topical antibiotics are given 3 days prior

to surgery. **Irrigation of the conjunctival cul-de-sac with povidone iodine prior to surgery is a must**Q.

- Postoperative topical antibiotics are given for 1-2 weeks
- The patient presents with sudden onset, pain, redness and decrease in vision. Exudates are seen in the vitreous on examination
  - Treatment is **intravitreal tap and intravitreal antibiotics**. If the patient does not improve, **pars plana vitrectomy**Q is done
  - Treatment of panophthalmitis is **evisceration**Q

The complications related to IOL are

- Malposition of IOL: The IOL may be decentred, subluxated or dislocated leading to:
  - Sunset syndromeQ: Subluxated inferiorly
  - Sunrise syndrome: Subluxated superiorly
  - Windshield wiper syndrome: Lens keeps moving with movement of head
- **Posterior capsule opacification (PCO)**Q: The posterior capsule behind the lens may opacify months or years after cataract surgery leading to visual impairment.
  - Morphological types are **Elschnig's pearls**Q and **Sommering's ring**Q.
  - Treatment is **Nd: YAG capsulotomy**Q.

MANAGEMENT OF CATARACT IN CHILDREN

- Cataract in children is an important cause for development of **stimulus deprivation amblyopia**Q
- **Unilateral cataract is more dangerous than bilateral cataract because the risk of amblyopia is more**
- The ideal age for cataract surgery in children is **4–6 weeks**Q because 6 weeks is the critical period for visual maturation in children. Thus, early surgery decreases the possibility of amblyopia
- **IOL power selection in children:** In very small children, it is extremely difficult to calculate the IOL power as the axial length and keratometry values are not accurate. In older children, if IOL power is calculated according to the present axial length, there occurs a **myopic shift** as the child grows older. **Hence, undercorrection of IOL power from the calculated value is needed.**
  - In children < 1yr of age, IOL is avoided. Patients are left aphakic and advised post-operative contact lenses. Secondary IOL implantation is planned at a later date when the child is older
  - Between 1–8 years of age, IOL is implanted. The power of the IOL is undercorrected from the calculated value according to the age of the patient
  - Beyond 8 years, IOL power is the same as the calculated value from the formulae.

ANOMALIES OF LENS POSITION

The lens is located in a saucer shaped depression called the patellar fossa between the anterior and posterior chamber of the eye. A lens in abnormal position since birth is called **Ectopia lentis.** Acquired cases are called **subluxation and dislocation.** Subluxation means

that a part of the lens still lies with the patellar fossa. Dislocation means that the lens is completely out of the patellar fossa.

Congenital Causes

- Marfan's syndrome[Q] (associated with **superotemporal**[Q] subluxation)
- Homocystinuria[Q] (associated with **inferonasal**[Q] subluxation)
- Weil Marchesani (microspherophakia with anterior dislocation of lens)
- Ehlers-Danlos syndrome
- Hyperlysinemia
- Stickler's disease
- Sulphite oxidase deficiency

Acquired Causes

- **Trauma (most common)**[Q]
- Buphthalmos
- Megalocornea
- High myopia
- Pseudoexfoliation
- Hypermature sclerotic cataract
- Chronic uveitis
- Endophthalmitis
- Intraocular tumours

QUESTIONS

1. **The capsule of the crystalline lens is thinnest at:** *(UPPG)*
   a. Anterior pole b. Posterior pole
   c. Equator d. None
2. **Ascorbate and Alpha Tochopherol are maintained in the lens in reduced state by:** *(AIIMS 2014)*
   a. Glucose b. Glycoprotein
   c. Glutathione d. Fatty acids
3. **Which of the following does not handle free radicals in the lens?** *(AIPG)*
   a. Vitamin A b. Vitamin C
   c. Vitamin E d. Catalase
4. **The crystalline lens derives its nutrition from:** *(AIIMS)*
   a. Blood vessels
   b. Connective tissue
   c. Aqueous and vitreous
   d. Capsule of the lens
5. **Transport of ascorbic acid in the lens is done by:** *(AIPG)*
   a. Myoinositol b. Choline
   c. Taurine d. Na-K ATPase
6. **Ligament of Weigert is:** *(APPG)*
   a. Attachment of the vitreous to the posterior capsule of the lens
   b. Medial palpebral ligament
   c. Attachment of the superior oblique tendon
   d. Associated with the middle ear
7. **Position of the lens in Marfan's syndrome is:** *(COMEDK)*
   a. Superior b. Superotemporal
   c. Inferior d. Nasal
8. **Typical bilateral inferonasal subluxation of lens is seen in:** *(DNB)*
   a. Marfan's syndrome
   b. Homocystinuria
   c. Hyperlysinemia
   d. Trauma
9. **Ectopia lentis is seen in:** *(PGI)*
   a. Marfan's syndrome
   b. Congenital rubella
   c. Homocystinuria
   d. Sulphite oxidase deficiency
   e. Myotonic dystrophy
10. **Anterior lenticonus is seen in:** *(AIPG)*
    a. Alport's syndrome
    b. Lowe's syndrome
    c. Down's syndrome
    d. William's syndrome
11. **Spontaneous absorption of lens material is seen in:** *(COMEDK)*
    a. Marfan's syndrome
    b. Hallerman Streiff syndrome
    c. Aniridia
    d. Persistent hyperplastic primary vitreous (PHPV)
12. **Gene commonly indicated in congenital cataract:** *(AIIMS 2014)*
    a. PAX-6 b. CRYGS- 3
    c. LMX-1B d. PITX-3
13. **Most common type of congenital cataract is:** *(AIIMS)*
    a. Capsular b. Zonular
    c. Cupuliform d. Blue dot
14. **Most common congenital cataract associated with decreased vision:** *(AIIMS)*
    a. Blue dot b. Cupuliform
    c. Zonular d. Coronary
15. **Riders are seen in:** *(AIPG)*
    a. Blue dot cataract
    b. Zonular cataract
    c. Sutural cataract
    d. Coronary cataract
16. **Commonest type of cataract:** *(PGI)*
    a. Hereditary b. Trauma
    c. Diabetes d. Age related
    e. Radiation

17. **Which of the following is true regarding concentration of proteins in senile cataract?** *(AIIMS 2013)*
    a. More insoluble protein, less soluble protein
    b. More soluble protein, less insoluble protein
    c. Equal concentration of soluble and insoluble protein
    d. None of the above
18. **High molecular weight protein present in cataractous lens in humans:** *(AIIMS 2015)*
    a. HM 1 and HM 2
    b. HM 2 and HM 3
    c. HM 3 and HM 4
    d. HM 4 and HM1
19. **Most visually significant cataract:** *(AIIMS)*
    a. Nuclear cataract
    b. Cortical cataract
    c. Posterior subcapsular cataract
    d. Zonular cataract
20. **Second sight phenomenon is seen in:** *(TNPG)*
    a. Nuclear cataract
    b. Cortical cataract
    c. Posterior subcapsular cataract
    d. Mature cataract
21. **Which of the following is not seen in uncomplicated mature cataract?** *(APPG)*
    a. Absence of iris shadow
    b. Pearly white colour
    c. Normal anterior chamber
    d. Absent light perception
22. **The most common complication of hypermature sclerotic cataract:** *(AIIMS)*
    a. Dislocation of lens
    b. Phacomorphic glaucoma
    c. Uveitis
    d. Neovascularisation of iris
23. **Which type of cataract causes phacomorphic glaucoma?** *(DNB)*
    a. Incipient cataract
    b. Intumescent cataract
    c. Morgagnian cataract
    d. Zonular cataract
24. **Typical appearance of diabetic cataract is:** *(AIIMS)*
    a. Sunflower cataract
    b. Breadcrumb cataract
    c. Polychromatic lustre
    d. Snowflake cataract
25. **Cataract in diabetic patient is due to accumulation of sorbitol. The enzyme responsible is:** *(AIIMS)*
    a. Hexokinase
    b. NADPH dependent Aldose Reductase
    c. Glucokinase
    d. Phosphofructokinase
26. **Fluctuating refractive errors with cataract is seen in:** *(AIIMS)*
    a. Morgagnian cataract
    b. Diabetic cataract
    c. Intumescent cataract
    d. Traumatic cataract
27. **Sunflower cataract is seen in:** *(AIPG)*
    a. Chalcosis
    b. Diabetes
    c. Syphilis
    d. Stargardt's disease
28. **Polychromatic lustre is seen in:** *(AIPG/AIIMS)*
    a. Complicated cataract
    b. Diabetic cataract
    c. Post radiation cataract
    d. Congenital cataract
29. **Steroid induced cataract is:** *(AIPG)*
    a. Posterior subcapsular
    b. Anterior subcapsular
    c. Nuclear cataract
    d. Cupuliform cataract
30. **Rosette cataract is seen in:** *(APPG)*
    a. Blunt trauma
    b. Diabetes

c. Wilson's disease
d. Myopia

31. Anterior polar cataract is seen in: *(PGI)*
a. Diabetes
b. Perforating injury
c. Irradiation
d. Chalcosis

32. Vossius ring is seen in: *(DNB)*
a. Penetrating trauma
b. Concussion injury
c. Iridocyclitis
d. Acute angle closure glaucoma

33. Christmas tree cataract is seen in: *(PGI)*
a. Down's syndrome
b. Rubella
c. Myotonic dystrophy
d. Diabetes

34. Which of the following does not cause complicated cataract? *(WBPG)*
a. Pathological myopia
b. Diabetes mellitus
c. Retinitis pigmentosa
d. Iridocyclitis

35. Select the correct match: *(PGI 2013)*
a. Wilson's disease- Sunflower cataract
b. Alport's syndrome-Posterior lenticonus
c. Amiodarone-Anterior subcapsular cataract
d. Myotonic dystrophy—Christmas tree cataract
e. Down's syndrome—Cortical cataract

36. Specific pattern of cataract is not seen in: *(APPG)*
a. Juvenile diabetes
b. Leprosy
c. Myotonic dystrophy
d. Wilson's disease

37. Recovery after cataract surgery is fastest with: *(MPPG)*
a. ICCE
b. ECCE
c. Phacoemulsification
d. SICS

38. Advantages of ECCE over ICCE are: *(PGI)*
a. Less chance of cystoid macular oedema
b. Less chance of endophthalmitis
c. Can be used in traumatic lens subluxation
d. Less chance of vitreous haemorrhage and retinal detachment
e. Minimal endothelial damage

39. In present scenario, only indication of ICCE is: *(DNB)*
a. Mature cataract
b. Paediatric cataract
c. Subluxated cataract
d. Immature cataract

40. The standard sutureless cataract surgery with phacoemulsification and foldable IOL implantation has an incision of: *(AIIMS)*
a. 1–1.5 mm
b. 2–2.5 mm
c. 3–3.5 mm
d. 4–4.5 mm

41. Phacoemulsification includes: *(PGI)*
a. Hydrodissection
b. Hydrodeliniation
c. Continuous curvilinear capsulorrhexis
d. IOL implantation
e. Lens aspiration

42. Best irrigating fluid for cataract surgery is: *(AIIMS)*
a. Normal saline
b. Ringer lactate
c. Balanced salt solution
d. Balanced salt solution + glutathione

43. Treatment for congenital cataract is: *(DPG)*
a. Pharmacotherapy
b. Combined cataract surgery and goniotomy
c. Cataract surgery with IOL implantation with posterior capsulotomy
d. Pars plana lensectomy with no IOL implantation

44. A child has congenital cataract involving the visual axis which was detected by the parents right at birth. When should the child be operated? *(DPG)*

a. Immediately
b. At 2 months of age
c. At 1 year of age when the globe attains normal size
d. At 4 years when ocular and orbital growth is complete

45. Essential parameters for IOL power calculation are: *(TNPG)*

a. Keratometry and corneal thickness
b. Corneal thickness and axial length of the eyeball
c. Keratometry and axial length of the eyeball
d. Corneal thickness and anterior chamber depth

46. Best site for IOL implantation is: *(DPG)*

a. Iris
b. Anterior chamber
c. Capsular bag
d. Sulcus

47. Modern IOLs are made up of: *(PGI)*

a. PMMA
b. Acrylic acid
c. Glass
d. Silicon
e. Styrene

48. In which of the following conditions is IOL implantation after cataract surgery contraindicated? *(AIPG)*

a. Fuchs heterochromic iridocyclitis
b. Juvenile rheumatoid arthritis
c. Psoriatic arthritis
d. Reiter's syndrome

49. Complications of cataract surgery are: *(PGI)*

a. Endophthalmitis
b. Optic neuropathy
c. Retinal detachment
d. Vitreous loss
e. Lagophthalmos

50. Postoperative endophthalmitis in cataract surgery is decreased by all *except*: *(WBP...*

a. Antibiotic eye drops
b. Intracameral antibiotic at the end of the surgery
c. Postoperative oral antibiotics
d. Sterile operative preparation

51. Which is the most important factor in the prevention of postoperative endophthalmitis? *(AIPG)*

a. Preoperative preparation with povidone iodine
b. One week antibiotic therapy prior to surgery
c. Trimming of eyelashes
d. Use of intravitreal antibiotics

52. Late onset endophthalmitis after cataract surgery is caused by: *(AIPG)*

a. Staphylococcus epidermidis
b. Pseudomonas
c. Streptococcus pyogenes
d. Propionibacterium acne

53. A 56-year-old man presents after 3 days of cataract surgery with history of pain and decrease in vision after an initial improvement. The most likely diagnosis is: *(AIIMS)*

a. Endophthalmitis
b. After cataract
c. Central retinal vein occlusion
d. Retinal detachment

54. Endophthalmitis involves all the layers of the eyeball *except*: *(AIPG)*

a. Cornea
b. Choroid
c. Sclera
d. Retina

55. Most common late complication of cataract surgery: *(DNB)*

a. Cystoid macular oedema
b. Glaucoma
c. Posterior capsule opacification
d. Uveitis

56. Ring of Sommering is seen in: *(DNB)*
a. Diabetes b. Galactosemia
c. After cataract d. Wilson's disease

57. Treatment of posterior capsule opacification is: *(AIIMS)*
a. Krypton laser
b. Argon laser
c. Nd- YAG laser
d. CO_2 laser

58. A 60-year-old patient operated 6 months back for cataract presents with floaters and decrease in vision. The likely diagnosis is: *(AIIMS)*
a. Vitreous haemorrhage
b. Retinal detachment
c. CRAO
d. Cystoid macular oedema

59. How many weeks after cataract surgery are spectacles prescribed? *(AIPG)*
a. 6 weeks b. 10 weeks
c. 12 weeks d. 14 weeks

60. Which of the following is the output indicator of NPCB? *(AIIMS 2014)*
a. Number of cataract surgeries leading to sight restoration
b. Decrease in the prevalence of blindness
c. Number of school children provided with glasses for refractive correction
d. Number of eye surgeons trained

ANSWERS AND EXPLANATIONS

1. **b. Posterior pole**
2. **c. Glutathione**
3. **a. Vitamin A**
4. **c. Aqueous and vitreous**
5. **d. Na-K ATPase**
6. **a. Attachment of the vitreous to the posterior surface of the lens**
7. **b. Superotemporal**
8. **b. Homocystinuria**
9. **a. Marfan's syndrome, c. Homocystinuria, d. Sulphite oxidase deficiency**
10. **a. Alport's syndrome**
    Lenticonus is a congenital condition where the anterior or the posterior surface of the lens is protruded. It may be anterior or posterior.
    Anterior lenticonus is seen in **Alport's syndrome**Q
    Posterior lenticonus is seen in **Lowe's syndrome**Q
11. **b. Hallerman Streiff syndrome**
12. **b. CRYGS-3**
13. **d. Blue dot cataract**
14. **c. Zonular cataract**
15. **b. Zonular cataract**
    Hypocalcemia or Vitamin D deficiency in pregnancy gives rise to spoke like opacities which are called **riders**. It is a type of zonular cataract.
16. **d. Age related cataract**
17. **a. More insoluble protein, less soluble protein**
18. **c. HM 3 and HM 4**
19. **c. Posterior subcapsular cataract**
20. **a. Nuclear cataract**
21. **d. Absent light perception**
    Mature cataract means that the lens is completely opacified. Iris shadow is absent. Unless complicated, the anterior chamber and IOP will be normal. Visual acuity is markedly diminished but light perception with accurate projection of rays will be present in the absence of any other pathology.
22. **a. Dislocation of lens**
23. **b. Intumescent cataract**
24. **d. Snowflake cataract**
25. **b. NADPH dependent Aldose Reductase**
26. **b. Diabetic cataract**
27. **a. Chalcosis**
28. **a. Complicated cataract**
29. **a. Posterior subcapsular cataract**
30. **a. Blunt trauma**

31. **b. Perforating injury**
Anterior polar cataract occurs at the site of trauma to the lens capsule
32. **b. Concussion injury**
33. **c. Myotonic dystrophy**
34. **b. Diabetes mellitus**
Diabetes causes metabolic cataract not complicated cataract.
35. **a. Wilson's disease—Sunflower cataract, c. Amiodarone-Anterior subcapsular cataract, d. Myotonic dystrophy—Christmas tree cataract**
36. **b. Leprosy**
37. **c. Phacoemulsification**
38. **a. Less chance of cystoid macular oedema, b. Less chance of endophthalmitis, d. Less chance of vitreous haemorrhage and retinal detachment, e. Minimal endothelial damage**
All complications of cataract surgery are less with the techniques like ECCE, SICS and Phacoemulsification as compared to ICCE.
Also in ICCE, the capsular bag is removed; hence the IOL has to be iris fixated, scleral fixated or placed in the anterior chamber. **In modern day cataract surgery, IOL is placed in the capsular bag.**
Thus, the only indication of ICCE today would be markedly subluxated cataract where the capsular bag cannot be retained.
39. **c. Subluxated cataract**
40. **c. 3–3.5 mm**
41. **a. Hydrodissection, c. Continuous curvilinear capsulorrhexis, d. IOL implantation**
The steps of Phacoemulsification surgery are:
    - Wound construction
    - Entry into anterior chamber
    - Anterior capsulorrhexis (a circular opening is made in the anterior capsule of the lens)
    - Hydrodissection (This means injecting fluid beneath the capsule to separate the lens from the capsule. Hydrodeliniation is a type of controlled hydrodissection done in posterior polar cataract)
    - Phacoemulsification of the lens
    - Cortical wash
    - IOL implantation
    - Wound closure by hydration
42. **d. Balanced salt solution+ Glutathione**
43. **c. Cataract surgery with IOL implantation with posterior capsulotomy**
In children it has been seen that after cataract surgery there is rapid opacification of the posterior capsule and anterior vitreous. Hence in children, two additional steps are added to the routine cataract surgery. They are:
    - Primary posterior capsulotomy(Opening is made in the posterior capsule at the centre of the visual axis)
    - Anterior vitrectomy (removal of the anterior vitreous)

Needling, discision, lensectomy are old procedures which are not done now.

44. a. Immediately
The dictum for congenital cataract is 'operate as early as possible', best within 4–6 week of birth

45. c. Keratometry and axial length of the eyeball

46. c. Capsular bag

47. a. PMMA, b. Acrylic, d. Silicon

48. b. Juvenile rheumatoid arthritis
Explained in chapter on uveitis

49. a. Endophthalmitis, c. Retinal detachment, d. Vitreous loss

50. c. Postoperative oral antibiotics
Endophthalmitis is the most dreaded and vision threatening complication of cataract surgery. Hence, prevention of infection is of paramount importance. The different steps to prevent endophthalmitis are:

- Preoperative antibiotic eye drops 3 days prior to surgery
- Sterile operative preparation
- Irrigation of conjunctival cul-de-sac with povidone iodine (most important) prior to surgery
- Postoperative antibiotic eye drops

51. a. Preoperative preparation with povidone iodine

52. d. Propionibacterium acne

53. a. Endophthalmitis

54. c. Sclera

55. c. Posterior capsular opacification

56. c. After cataract
After cataract is the old name for posterior capsule opacification. The more common variety is Elschnig's pearls. Sommering's ring is also a type of PCO.

57. c. Nd- YAG laser

58. b. Retinal detachment

59. a. 6 weeks
After cataract surgery steroid eye drops are given in tapering doses over 6 weeks. Glasses are usually prescribed at the end of this period.

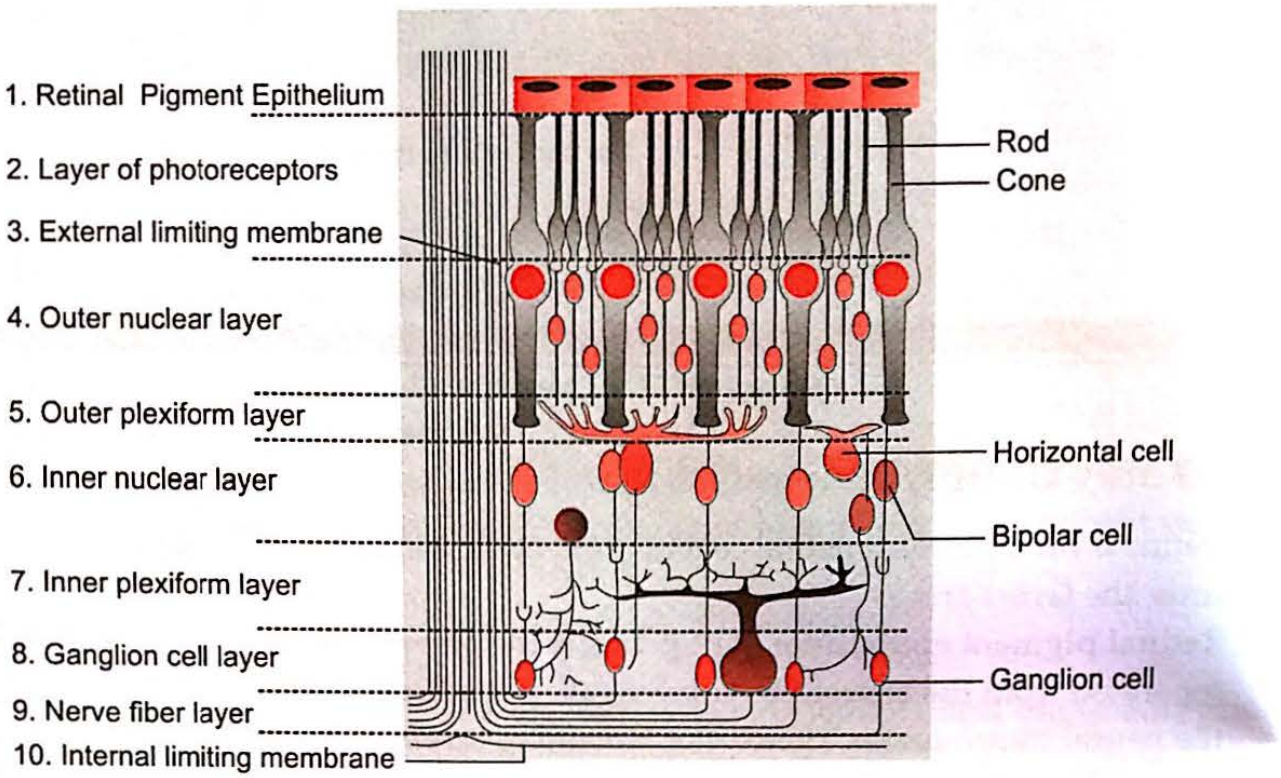
60. a. Number of cataract surgeries leading to sight restoration

Retina

ANATOMY OF RETINA

The retina is the innermost neural coat of the eyeball. It consists of ten layers. From outside inwards, the layers are:

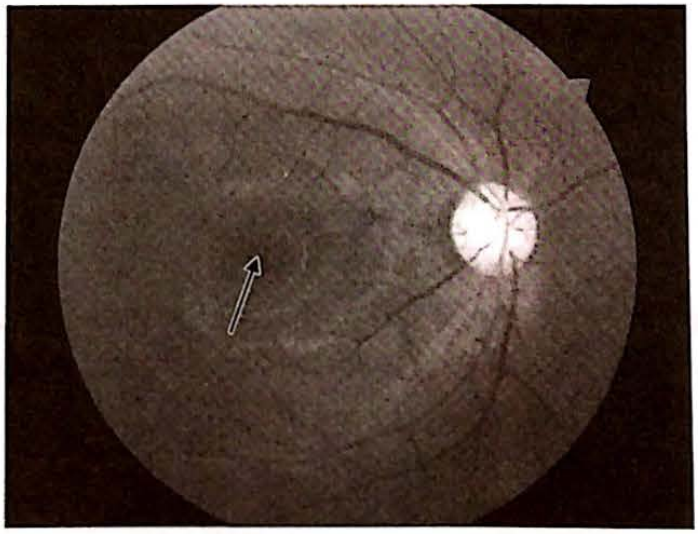
- **Retinal pigment epithelium (RPE):** This is the **outermost layer of the retina** which is separated from the choroid by a layer called the **Bruch membrane**Q. It has no role in the neural transmission. Hence, the remaining layers of the retina which are involved in neural activity are collectively called as **neurosensory retina**Q. The functions of RPE are:
  - It acts as metabolic pump to prevent the accumulation of debris in the subretinal space
  - It provides nutrition to the photoreceptors
- **Layer of photoreceptors:** This contains the **outer segments of the photoreceptors** namely the rods and the cones
  - The **rods** are responsible for vision in dim light or **scotopic vision**Q. About 120 million rod cells are present in the retina with maximum concentration in the mid periphery. Rods are absent at the foveola. They contain a pigment called **rhodopsin**Q
  - The **cones** are responsible for **colour vision** and vision in bright light or **photopic vision**Q. About 6.5 million cones are present in the retina with maximum concentration in the fovea. They contain a pigment called **iodopsin**
- **External limiting membrane**
- **Outer nuclear layer:** It contains the **cell bodies of the photoreceptor cells**Q
- **Outer plexiform layer:** It contains the **synapses between the photoreceptors and bipolar cells**Q
- **Inner nuclear layer:** It contains the **cell bodies of the bipolar cells**Q. It also contains the cells bodies of the horizontal cells, amacrine cells and Muller's cells
- **Inner plexiform layer:** It contains the **synapses between the bipolar cells and ganglion cells**Q
- **Ganglion cell layer-** It contains the cell bodies of the ganglion cells
- **Retinal nerve fibre layer (RNFL) –It is constituted by the axons of the ganglion cells**Q
- **Internal limiting membrane-** It separates the retina from the vitreous and is formed by the foot processes of the Muller's cells



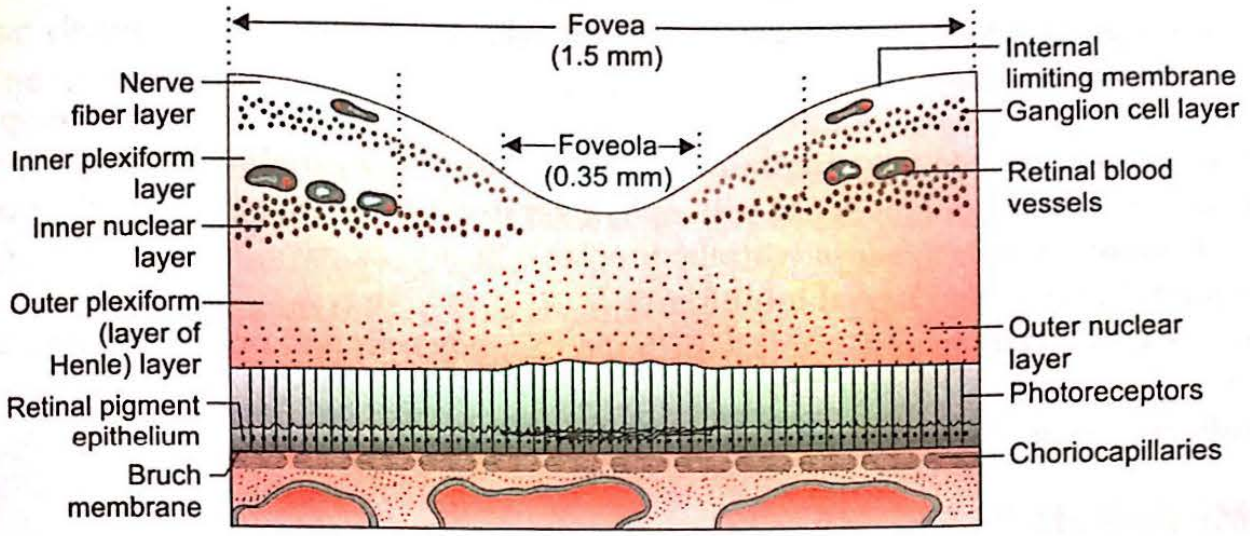
Layers of the retina

IMPORTANT REGIONS OF THE RETINA

- **Optic Disc:** It is also known as the optic nerve head and signifies the point of origin of the optic nerve. It is **1.5 mm**[Q] in diameter and consists of only the nerve fibre layer of the retina. Due to the absence of photoreceptors, no visual response is seen if light falls in this area of the retina. Hence, it also called as the blind spot
- **Macula:** The oval area on the posterior pole of the eye is the macula. It has an overall diameter of **5.5 mm**[Q]. The centre of the macula is the **fovea (1.5 mm in diameter)**[Q]. The centre of the fovea is called the **foveola (0.35 mm).** It has the **highest concentration of cones**[Q]. Rods are absent in this region. The centre of the macula is about **2 disc diameters (3 mm)**[Q] from the margin of the optic disc
- **Ora Serrata:** It is the junction of the retina and ciliary body



Normal Fundus (arrow points to the fovea) ***(see colour plate 1)***

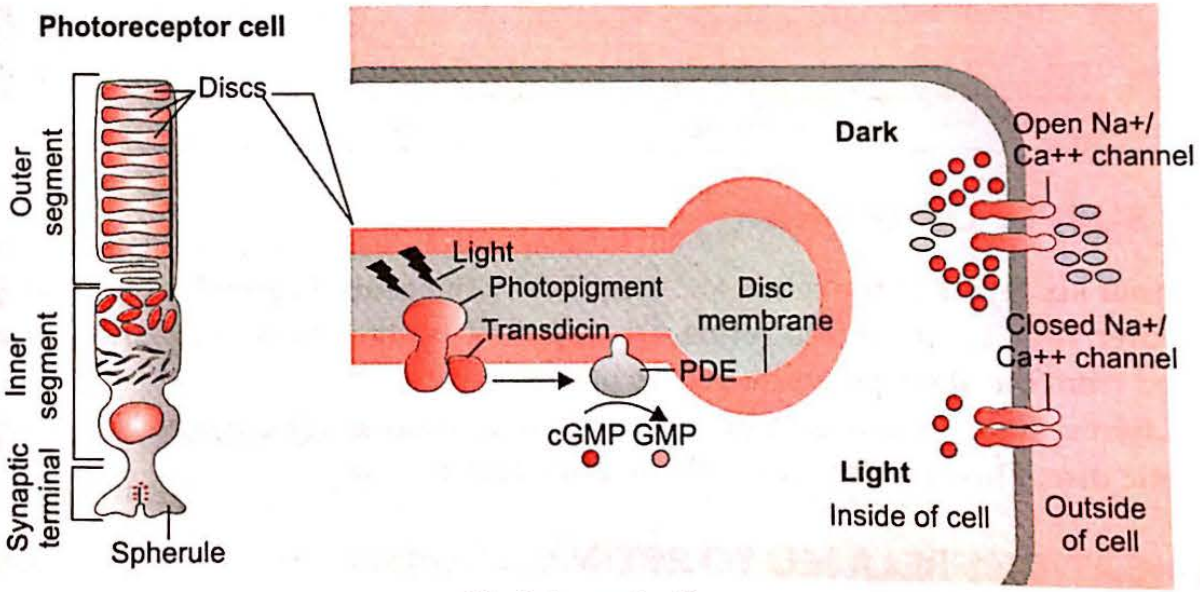


Layers at the fovea

Important Points to Remember

- The fovea, which is the centre of the macula is a depression called as **foveal pit**[Q]
- It is a pit because many layers of the retina are absent here. At the fovea, the layers present are RPE, layer of photoreceptors, external limiting membrane, outer nuclear layer and internal limiting membrane.

TRANSMISSION OF VISUAL IMPULSE



Phototransduction

- The visual impulse is generated in the photoreceptors in response to light. Each photoreceptor has an **inner segment** containing the **nucleus and cytoplasmic organelles.** The **outer segment** of the photoreceptor contains discs with **photopigments** which are activated in response to light.
- The activated photopigment attaches to **transducin** protein and activates the **phosphodiesterase enzyme(PDE)**[Q].
- Phosphodiestrase leads to conversion of **cGMP to GMP**[Q].

- Decrease in the concentration of cGMP leads to **closure of Na+/ Ca++ channels** resulting in **hyperpolarisation**[Q]
- This generates a visual impulse which is transmitted serially from the **photoreceptors to the bipolar cells to the ganglion cells**. The **axons of the ganglion cells** which form the nerve fibre layer of the retina subsequently transfer the impulse to the **optic nerve**[Q]
- The **horizontal cells** synapse with the bipolar cells and prevent lateral spread of the impulse. This is called **lateral inhibition**[Q]
- The **amacrine cells** mainly synapse with the ganglion cells and provide **feedback input**[Q]
- **Mullers cells** are **glial cells** providing structural support.

EXAMINATION OF RETINA

- Indirect Ophthalmoscope: It is used to visualize peripheral retina.
- Direct Ophthalmoscope, 90D lens: Used to visualize the posterior pole
- Goldman Three Mirror Lens
  - Central Part: Used to visualize the posterior pole
  - Equatorial Mirror: Used to visualize the area surrounding the equator
  - Peripheral Mirror: Used to visualize the peripheral retina up to the ora serrata

	Direct Ophthalmoscope	**Indirect Ophthalmoscope**
Magnification	15 times[Q]	5 times[Q]
Field of view	Limited: about 2 disc diameter	Large: Up to ora serrata
Hazy media	Not much useful	Useful because of bright illumination
Type of image	Virtual and erect[Q]	Real and inverted[Q]

BLOOD SUPPLY OF RETINA

- The **inner six layers** of the retina are supplied by the **central retinal artery (CRA)**[Q]
- The **outer four layers** of the retina are supplied by the choriocapillaries which are derived from the **short posterior ciliary arteries**[Q]
- These two systems of blood vessels form an anastomotic circle around the margin of the optic disc. This is called the **circle of Zinn-Haler**[Q]

INVESTIGATIONS RELATED TO RETINA

- **Fundus Fluorescein Angiography (FFA)**: It is used to study the normal physiology of retinal and choroidal circulation. 5 ml of 10% solution of sodium fluorescein dye is injected in the **antecubital vein**[Q]. Serial fundus photographs are taken. There are typically four phases in FFA:
  - Pre-arterial phase: Dye is seen in the choroidal vessels
  - Arterial phase: Dye is seen in the retinal arteries
  - Arterio-venous phase: There is complete filling of retinal arteries and capillaries. Lamellar blood flow appears in the veins
  - Venous phase: Dye is seen in the retinal veins only

FFA helps to identify areas of retinal hypoperfusion, leakage and neovascularisation.

- **Indocyanine green angiography (ICGA):** This is a type of angiography done with Indocyanine green dye. It helps to study the **choroidal vasculature**[Q]
- **Optical Coherence Tomography (OCT):** This is an optical scan of the retinal layers. It is useful in evaluating retinal thickness, especially in macular oedema
- **Ocular electrophysiology:** The different tests are:
  - **Electroretinogram (ERG):** This is a mass response of the retina to light. It helps to identify gross retinal pathologies like **retinal dystrophies**. ERG has three waves, namely:
    - a wave: It arises from the photoreceptors[Q]
    - b wave: It arises from the bipolar cells
    - c wave: It arises from the RPE

  Pattern ERG: Special ERG which is used for macular diseases

  Multifocal ERG: Special ERG used for localized retinal diseases
  - **Electroculogram:** This test is used to evaluate the function of RPE. It is measured in terms of a ratio called **Arden's ratio**[Q]. (Normal value > 1.85[Q])
  - **Visual evoked potential (VEP):** This test is used to evaluate the visual pathway, mainly the optic nerve. The main wave is P100 wave. Increase in latency and decrease in amplitude of this wave indicates dysfunction of the visual pathway, mainly the optic nerve.

RETINAL DETACHMENT

Separation of the Neurosensory Retina from Retinal Pigment Epithelium (RPE).

Types

- Rhegmatogenous retinal detachment
- Tractional retinal detachment
- Exudative retinal detachment

Rhegmatogenous RD

Pathophysiology

The essential lesion is a **rhegma** (retinal break) through which the liquefied vitreous gains access to the retina. This liquefied vitreous accumulates beneath the neurosensory retina leading to RD. This is commonly seen in:

- **High myopes:** Peripheral retinal degenerations like **lattice degeneration**[Q], holes and breaks with liquefaction of overlying vitreous predispose to RD
- **Retinoschisis:** This is a condition where there is splitting of the neurosensory retina and vitreous degeneration. It is of two types:
  - Typical retinoschisis: Split at the level of **Outer Plexiform Layer**[Q]
  - Reticular retinoschisis: Split at the level of **Nerve Fibre Layer**[Q]. Seen more commonly in hypermetropes in the inferotemporal periphery of retina

- **Trauma**
- **Aphakia**[Q]
- **Pseudophakia**[Q]

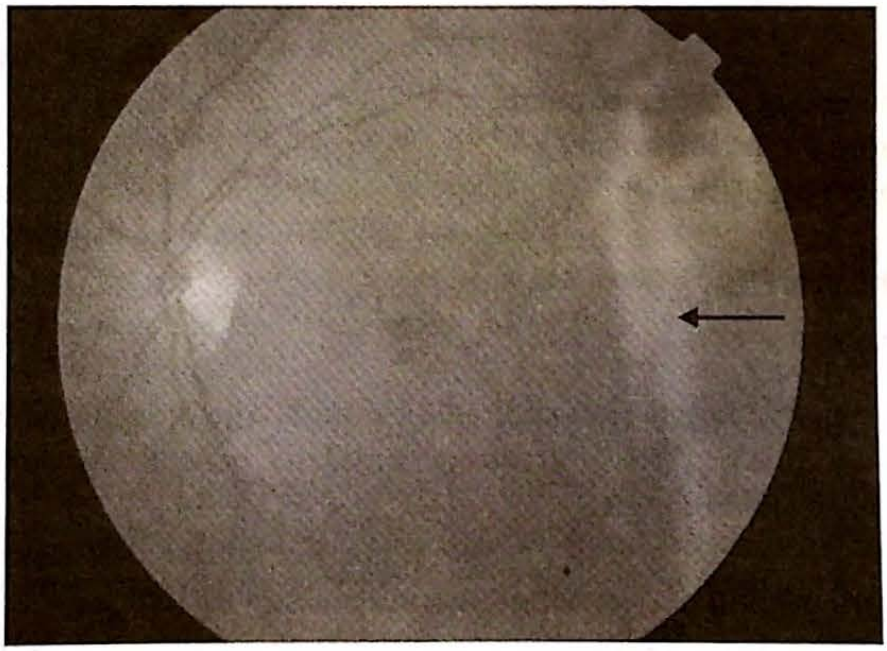
> - Most dangerous type of retinal break: **Horseshoe tear**[Q]
> - Most dangerous location of retinal break: **Superotemporal retina**[Q]

Clinical Features

- Premonitory features:
  - **Photopsia**[Q]: Flashes of light due to vitreoretinal traction at site of break
  - Floaters: Due to associated vitreous haemorrhage
- **Visual field defect** which is described by the patient as **a black curtain or veil**[Q] in front of the eye
- Loss of vision when the detachment involves the macula.

Signs

- Decrease in visual acuity
- **Hypotony:** The liquefied vitreous in the subretinal space is absorbed through the RPE leading to hypotony
- **Shafer's sign**[Q]: Pigments in anterior vitreous (tobacco dusting) is a feature of **fresh RD**[Q]
- Detached retina is **convex** in configuration. Surface is **corrugated** with free undulation. Break is identified at the periphery.



Retinal detachment ***(see colour plate 1)***

Treatment

- **Prophylactic laser barrage:** It is done in:
  - Symptomatic break (associated with photopsia and floaters)

- Horseshoe tear[Q]
- Superior, especially supero-temporal tears
- Aphakia[Q]
- One eyed patient

- **Surgery for Retinal Detachment**
  - **Scleral buckling:** In this procedure, the sclera is indented by attaching an explant known as a buckle. This pushes the RPE inwards the neurosensory retina. Subretinal fluid is drained and the break is closed by laser or **cryotherapy**[Q]
  - **Pneumatic Retinopexy**[Q]**:** In this procedure, the neurosensory retina is pushed towards the RPE by injecting an expansile gas in the vitreous cavity. The break is then sealed with laser. The gases commonly used are **Sulphur Hexafluoride(SF6)**[Q] and **Perfluoropropane (C3F8)**
  - **Pars plana vitrectomy**

Tractional RD

Pathophysiology

Fibrovascular membranes[Q] in the vitreous due to long standing vitreous hemorrhage exert traction on the retina. This pulls the retina forward leading to retinal detachment.

Causes

- Proliferative diabetic retinopathy (PDR)[Q]
- Central retinal vein occlusion
- Eales' disease
- Retinopathy of prematurity (ROP)

Signs

- Detached retina is **concave** in configuration with highest elevation at the site of the tractional band. No breaks are seen.
- Minimal mobility

  Treatment: Pars plana vitrectomy and **endophotocoagulation**[Q]

Exudative RD

Pathophysiology

Exudative fluid, mainly from choroid, collects in the subretinal space leading to retinal detachment.

Causes

- Inflammatory conditions like choroiditis, choroidal vasculitis, posterior scleritis[Q]
- Choroidal tumours like melanoma, hemangioma, metastasis[Q]
- Toxemia pregnancy[Q]

- Malignant hypertension[Q]
- Coat's disease[Q]

Clinical Features

- Floaters due to vitritis but no photopsia
- Visual field defect
- Loss of vision due to involvement of macula

Signs

- Detached retina has a **convex** configuration. Surface is **smooth.** No break is seen
- **Shifting fluid is seen**[Q].

Treatment

- Systemic steroids
- Treatment of the cause

RETINAL VASCULAR DISORDERS

Diabetic Retinopathy

Risk Factors

- **Duration of the diabetes (most important risk factor)**[Q]
- Poor glycemic control
- Hypertension
- Hyperlipidemia
- Pregnancy
- Nephropathy

Pathophysiology

- The hallmark of diabetic retinopathy is the alteration in the structure and cellular composition of the microvasculature
- Hyperglycemia leads to the formation of advanced glycation end products (AGE) which are deposited in the walls of the retinal blood vessels. AGE cause **loss of pericytes**[Q] and damage to the endothelial cells. This in turn leads to **microaneurysm**[Q] formation due to weakening of the vessel walls
- Endothelial cells are responsible for maintenance of the blood retinal barrier. Hence, endothelial cell damage leads to abnormal capillary permeability and leakage
- Retinal leucostasis is another important factor in the development of diabetic retinopathy. Increase in inflammatory cytokines causes influx of leucocytes which adhere to the vascular endothelium and cause a decrease in capillary perfusion
- Retinal hypoperfusion leads to increase in angiogenic mediators like **Vascular Endothelial Growth Factor (VEGF)** which stimulate **neovascularization.**

Classification

- **Non-proliferative diabetic retinopathy (NPDR)**
- **Proliferative diabetic retinopathy (PDR)**
- **Diabetic maculopathy.**

Non-proliferative Diabetic Retinopathy

- **Microaneurysms:** They are the **first detectable lesions of DR**Q. They are dilated capillaries present at level of **Inner Nuclear Layer**Q of the retina due to **loss of pericytes**Q in the capillary walls
- **Intraretinal hemorrhages:** Rupture of the weakened capillaries leads to intraretinal haemorrhages. They are of two types: Superficial flame shaped haemorrhages and dot blot haemorrhages. **Flame shaped haemorrhages are located in the Nerve Fibre Layer**Q**. Dot blot haemorrhages are located in the Inner Nuclear Layer**Q
- **Hard exudates:** These are **lipoproteins**Q leaking from the damaged retinal blood vessels. They are mainly located in the **Outer Plexiform Layer**Q
- **Cotton Wool Spots:** Occlusion of the small retinal blood vessels leads to infarcts mainly in the retinal nerve fibre layer. These are called cotton wool spots or soft exudates
- **Intraretinal microvascular abnormalities (IRMA):** Bending, looping, beading and dilatation of the veins.

Proliferative Diabetic Retinopathy

- Widespread retinal ischaemia leads to increase in a mediator called **Vascular Endothelial Growth Factor (VEGF)**Q which causes neovascularisation
- Neovascularisation may occur at the disc **(NVD)** or along the arcades, in which case it is called Neovascularization elsewhere **(NVE)**
- The new blood vessels are friable and may rupture leading to **vitreous haemorrhagen (VH)**
- Long standing vitreous haemorrhage leads to fibrous organization. The fibrous bands in the vitreous exert traction on the retina leading to **Tractional Retinal Detachment (TRD)**Q
- Effect of VEGF in the anterior segment is neovascularisation of the iris called **rubeosis iridis**
- New vessels at the angle of anterior chamber lead to **neovascular glaucoma.**

Diabetic Maculopathy

Maculopathy includes microaneurysm, haemorrhages and hard exudates at the macula leading to macular thickening or oedema. This may due to **focal leakage or diffuse leakage from the capillary bed.** But maculopathy needs to be treated only when it becomes **Clinically Significant Macular Edema (CSME)**Q. The criteria for CSME are:

- Retinal oedema or thickening within 500 microns of the centre of the macula
- Hard exudates within 500 microns of the centre of the macula
- One or more disc diameters of retinal thickening, part of which is within one disc diameter of the centre of the macula

Investigations

- Systemic investigations like blood sugar and lipid profile
- Fundus fluorescein angiography (FFA): It helps to identify vascular leakage, nonperfusion and abnormal new vessels
- Optical Coherence Tomography(OCT): It helps to evaluate the macular thickness.

Treatment

- Control of systemic parameters like hypertension, hyperglycemia and hyperlipidemia.
- **Panretinal photocoagulation (PRP)**[Q]: This is done in **Proliferative Diabetic Retinopathy (PDR)**[Q]. The commonly used laser is **Argon**[Q].

(Note: The mechanism of action of laser is described as **conversion of hypoxia** to **anoxia**[Q]. This means that the laser converts a hypoxic dying peripheral retinal tissue to an anoxic dead tissue. This decreases the level of VEGF and helps the remaining central retinal tissue to survive).

- **For Diabetic Maculopathy**
  - In Focal Leakage : Focal Laser
  - **In Diffuse Leakage: Macular Grid Laser**[Q]
- Intravitreal Injections
  - **Steroids:** Triamcinolone, Dexamethasone

(Note: Intravitreal Dexamethasone is available in the form of an implant containing **700 micrograms**[Q] of the drug which is slowly released over a period of **6 months**)

  - **Anti VEGF:** Bevacizumab, Ranibizumab

(Note: Only **Ranibizumab is FDA approved** for intravitreal use. Bevacizumab is used as an off- label drug)

- Surgery- **Pars plana Vitrectomy**[Q]. Indications for surgery are:
  - **Tractional retinal detachment**[Q]
  - Non-resolving vitreous haemorrhage
  - Patients not responding to laser

- Most common cause of severe vision loss in diabetes: **Vitreous Haemorrhage**[Q]
- Most common cause of moderate vision loss in diabetes: **Diabetic Maculopathy**[Q]

RETINAL VEIN OCCLUSION

Predisposing Factors

- Systemic hypertension
- Atherosclerosis
- Hypercoagulative conditions
- Venous stasis, periphlebitis
- Raised IOP

Retinal vein occlusion can either be **Central Retinal Vein Occlusion (CRVO)** or **Branch Retinal Vein Occlusion (BRVO).**

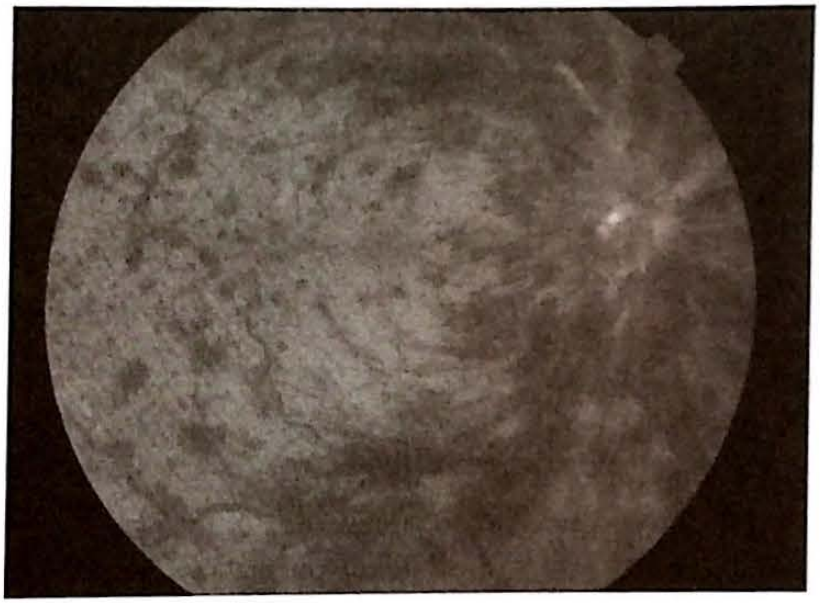
Central Retinal Vein Occlusion (CRVO)

Symptoms

Sudden painless loss of vision

Signs

- Visual acuity: Decrease in visual acuity, usually less than 6/60
- Relative afferent pupillary defect (RAPD)
- Anterior segment is normal
- Fundus changes
  - Marked tortuosity and dilatation of the veins
  - Extensive haemorrhages in both the central and peripheral retina. This is called **Splashed Tomato Appearance**[Q] **or Blood and Thunder Appearance**
  - Cotton wool spots
  - Disc oedema
  - Macular oedema



Splashed tomato appearance (CRVO) ***(see colour plate 1)***

Branch Retinal Vein Occlusion (BRVO)

Features are similar to CRVO but fundus changes are localised along the involved blood vessel only.

Complications

- Occlusion of the vein results in decrease in forward blood flow through the retinal arteries leading to widespread ischaemia. This, in turn, causes increase in the level of vascular endothelial growth factor (VEGF) which leads to neovascularisation. Thus the complications of venous occlusion are **NVD, NVE, Vitreous haemorrhage, Tractional RD** and **Neovascular Glaucoma.**

Treatment

- Panretinal photocoagulation
- Intravitreal injection: Steroids and Anti VEGF
- Pars plana vitrectomy

- Most common complication of **BRVO: Vitreous haemorrhage**
- Most common complication of **CRVO: Neovascular glaucoma**
- Neovascular glaucoma in **CRVO is also called 100 day glaucoma**

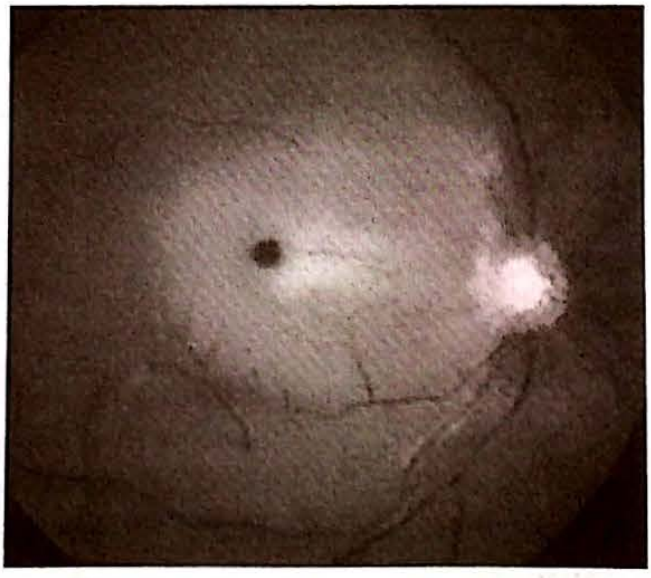
CENTRAL RETINAL ARTERY OCCLUSION

Predisposing Factors

- Hypertension, Atherosclerosis, Hypercoagulative conditions
- Atrial fibrillation
- Emboli

Symptoms and Signs

- Marked decrease in visual acuity
- Relative afferent pupillary defect (RAPD)
- Anterior segment is normal
- Fundus
  - White opaque retina due to ischaemia of the inner retinal layers mainly the nerve fibre layer
  - **Cherry Red Spot**[Q]: The fovea which is devoid of the inner retinal layers receives blood supply from the choriocapillaries. Hence, it appears bright red in contrast to the surrounding ischaemic retina
  - Marked narrowing of the retinal arterioles
  - **Sludging and segmentation of blood column (Cattle Track Sign)**[Q]



Cherry red spot ***(see colour plate 1)***

Treatment

- CRAO is an **ocular emergency** whose window period of treatment is **3 hours**
- Ocular massage for15 min –This leads to lowering of IOP which in turn increases the blood flow and may dislodge the thrombus
- IV Mannitol
- Aspiration of aqueous humour from the anterior chamber called paracentesis.

HYPERTENSIVE RETINOPATHY

In chronic hypertension, the changes that are seen in the retinal vasculature can be grouped as:

- **Arteriolar narrowing** which may be diffuse or focal.: Severe narrowing leads to the development of infarcts or **cotton wool spots**[Q]
- **Vascular leakage**: Damage to microvasculature leads to intraretinal haemorrhages and **hard exudates**[Q]
- Arteriosclerosis: Thickening of the vessel wall characterized by hypertrophy and hyalinization of the vessel wall. Arteriosclerotic changes are classified as:
  - Grade I: Broadening of the arteriolar light reflex
  - Grade II: Deflection at the AV crossing junction (Salu's sign)
  - Grade III: Copper wiring of arterioles
  - Grade IV: Silver wiring of the arterioles

Keith-Wagener-Barker Classification

- Grade I: Mild generalized arteriolar attenuation
- Grade II: Severe Grade I + Focal arteriolar attenuation
- Grade III: Flame shaped haemorrhages and cotton wool spots
- Grade IV: Papilloedema

In acute hypertension or **malignant hypertension** the changes seen are:

- Extensive flame shaped haemorrhages
- Hard exudates in a ring around the macula also called as **macular star**[Q]
- Cotton wool spots
- Choroidal infarcts which are called **Elschnig's spots**[Q]
- Papilloedema
- **Exudative Retinal detachment**[Q].

RETINOPATHY OF PREMATURITY (ROP)

- Retinopathy of prematurity (ROP) is a proliferative retinopathy seen in premature infants of low birth weight, especially those who have been exposed to ambient oxygen
- The important risk factors which predispose to development of ROP include:
  - **Prematurity**[Q]
  - Low birth weight[Q]

- Oxygen therapy
- Sepsis

Screening for ROP

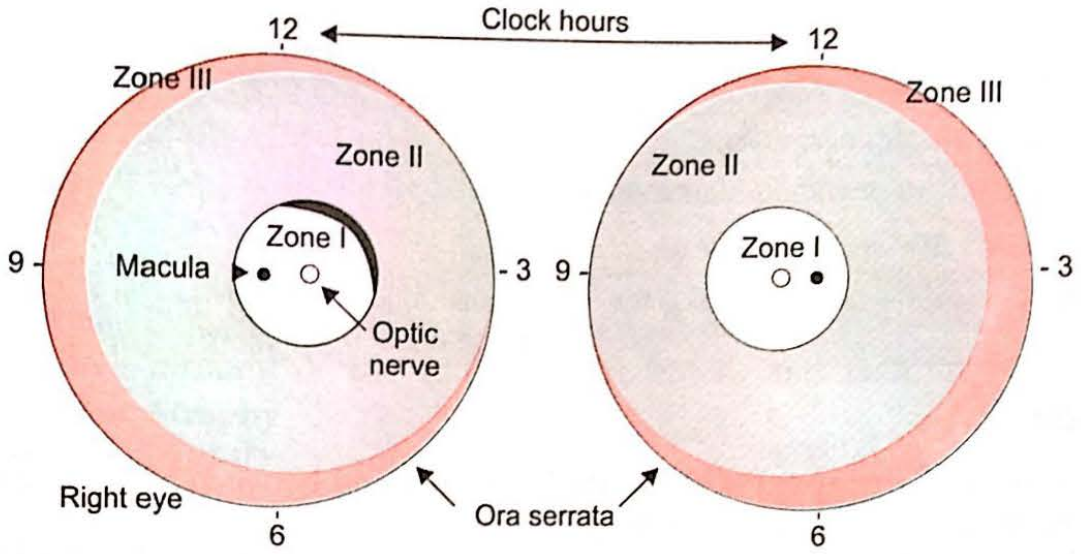
- Neonates born **at ≤ 32 week of gestation or birth weight< 1500 gm must be screened for ROP**[Q]
- Neonates born at **> 32 weeks weighing between 1500–2000 gm** who have been exposed to oxygen are also screened
- Screening is done at 4 weeks from birth or 32 weeks whichever is later.

Pathogenesis of ROP

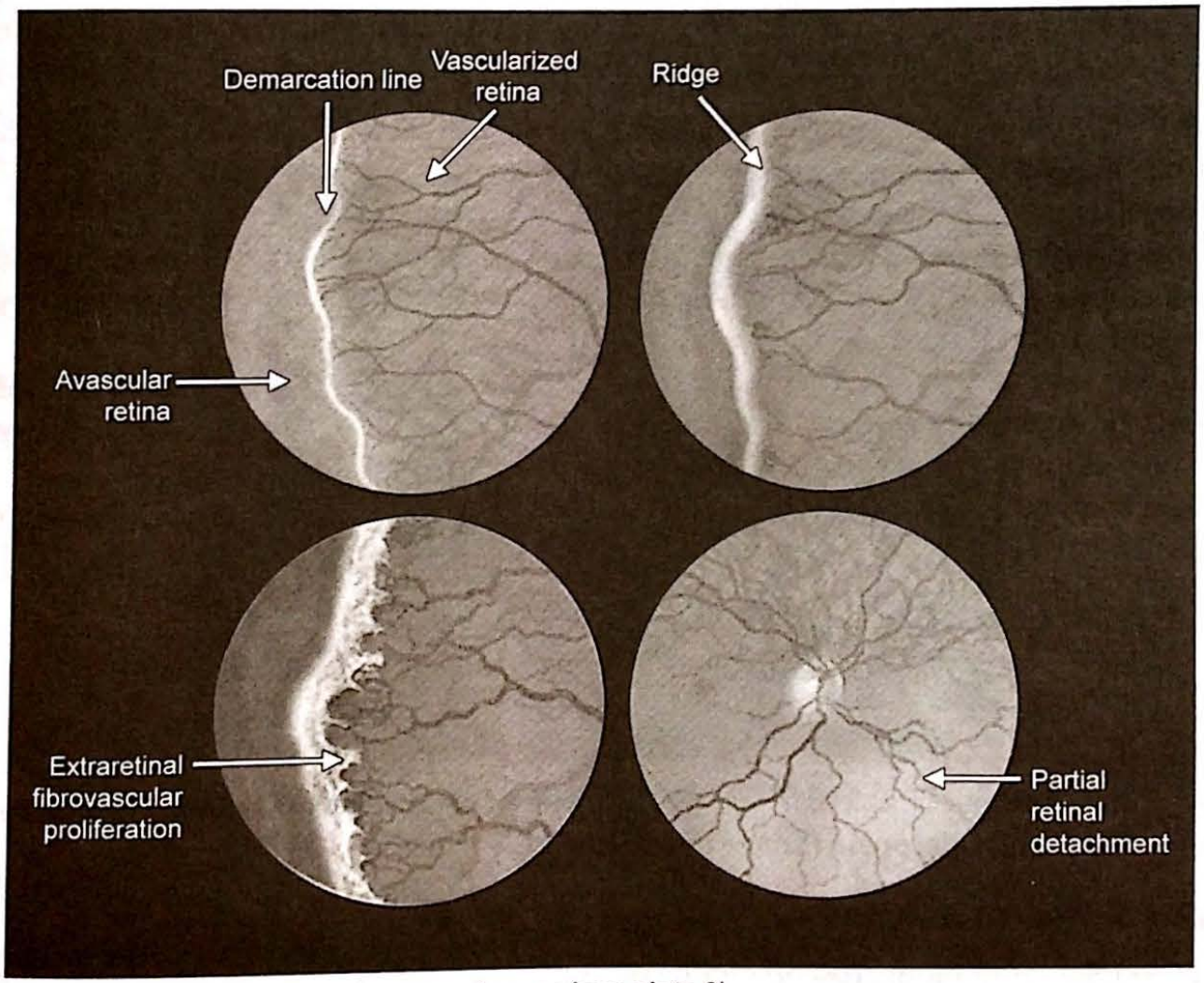
- The retinal vasculature starts developing at around the fourth month of gestation. At about the eighth month, the vessels have reached the nasal retina but they do not reach the temporal periphery till the tenth month (1 month after delivery of a term baby)
- Premature infants, thus, have an incompletely vascularised retina which is dependent on an optimum level of VEGF for their vessel migration
- With oxygen therapy, there is suppression of the basal VEGF which causes halting of the vessel migration
- Thus, the normal development of the retinal vasculature is disturbed. Later on, this incompletely vascularised hypoxic retina starts generating excess VEGF which leads to proliferative retinopathy or ROP.

Classification of ROP		
1. Location	Zone I	It is a circle with the **optic nerve at the centre** and a radius of twice the distance from optic nerve to macula
	Zone II	It extends from the edge of Zone I to the **ora serrata nasally and equator temporally**
	Zone III	It is a crescent shaped area from Zone II to ora-serrata temporally
2. Severity	Stage 1	Presence of a thin **white demarcation line**[Q] separating the vascular from avascular retina
	Stage 2	The line becomes prominent to form **a ridge**[Q]
	Stage 3	**Extra retinal fibrovascular proliferation**[Q] occurs. Abnormal vessels and fibrous tissue arise from the ridge and extend into the vitreous
	Stage 4	**Partial retinal detachment**[Q]; not involving macula (4A) or involving macula (4B)
	Stage 5	**Total retinal detachment**[Q]
3. Plus disease[Q]		Dilatation and tortuosity of posterior retinal vessels[Q] is seen

International Classification of Retinopathy of Prematurity (ICROP) zones



Stages of ROP



(see colour plate 2)

Based on results of ETROP (Early Treatment of Retinopathy of Prematurity) study, the **treatment** of ROP is described as:

Type	Treatment
Type 1 ROP or threshold ROP[Q]	Peripheral retinal ablation
Zone I, any stage ROP with plus disease	
Zone I, stage 3 ROP with or without plus disease	
Zone II, stage 2 or 3 ROP with plus disease.	
Type II ROP: ROP not meeting the criteria for threshold ROP	Regular follow-up is advised till the development of the retina is completed

Prevention

- Judicious oxygen therapy: PaO2 should be maintained between 50–80 mm Hg. SaO2 should be maintained between 89–95%
- Judicious use of blood transfusions
- Prenatal steroids prevent respiratory distress and intraventricular hemorrhage which are two important risk factors of ROP.

EALES DISEASE

- Bilateral idiopathic, occlusive, peripheral retinal periphlebitis with neovascularisation
- Commonly seen in young males
- Strong association with **Tuberculosis**[Q]
- The disease is characterized by the occlusion of peripheral retinal veins associated with vasculitis. Retinal hypoperfusion leads to increase in VEGF which causes retinal neovascularisation.
- As the disease is mainly localized to the peripheral retinal veins, presentation is usually late. Presentation is usually with **vitreous haemorrhage**[Q]**.** This is the **most common cause of spontaneous vitreous haemorrhage in young adult**[Q]
- Retinal photocoagulation is the treatment. Anti VEGF injection, Pars plana vitrectomy are other options.

- Most common cause of vitreous haemorrhage in adult: **Diabetes**[Q]
- Most common cause of vitreous haemorrhage in young adults: **Trauma**[Q]

ACQUIRED MACULOPATHIES

The main symptoms of macular disease: Blurring of central vision associated with **metamorphopsia**[Q] **and micropsia** (image distortion)

Examination of Macula

- Direct Slit lamp biomicroscopy with 90D lens
- **Amsler Grid**[Q]
- Photostress test

- Two Light Discrimination Test
- Laser Interferometry (LI)
- **Potential Acuity Meter (PAM)**[Q]

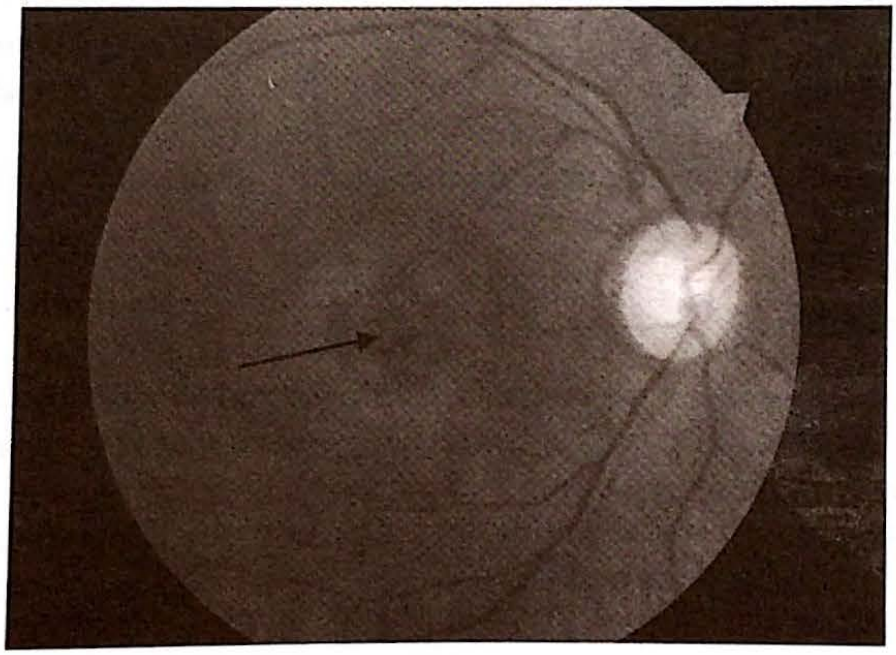
AGE-RELATED MACULAR DEGENERATION (ARMD)

Age-related macular degeneration is an age related atrophy of the RPE and the photoreceptors associated with choroidal neovascularization. It is of two types:

- Non-exudative/Dry ARMD
  - Accumulation of debris between the retinal pigment epithelium and Bruch's membrane of the choroid. These are called as **Drusens**
  - Progressive atrophy of the RPE, photoreceptors and choroid which is called **Geographic Atrophy**
- Exudative/Wet ARMD
  - RPE Detachment
  - Breaks in the Bruch membrane allow the choriocapillaries to grow into the retina. This is called **Choroidal Neovascular Membrane (CNVM)**[Q]
  - Disciform Scar

Investigation: **Indocyanine Green Angiography (ICGA)**[Q]

Note: ICGA is useful for evaluation of the choroidal vasculature whereas FFA is useful for the study of the retinal vasculature.



Exudative ARMD/CNVM (indicated by black arrow) ***(see colour plate 2)***

Treatment

- Anti-VEGF injection[Q]
- Photodynamic therapy (PDT)[Q]
- Transpupillary thermotherapy (TTT)
- Low vision Aids—For advanced burnt out disease

CENTRAL SEROUS RETINOPATHY (CSR)

Central Serous Retinopathy is an idiopathic detachment of the neurosensory retina in the region of the macula due to the accumulation of serous fluid.

- More common in males aged between 20-40 years
- Risk Factors: **Stress**[Q], **smoking**[Q] **and steroids**[Q]
- Seen in **Type A personalities**[Q]
- Sudden onset decrease in vision associated with micropsia and metamorphopsia
- Localized detachment of neurosensory retina at the posterior pole leading to **acquired hypermetropia** (effective shortening of the globe)
- Fundus fluorescein angiography: The classical patterns are:
  - **Smoke Stack Pattern**[Q] or Mushroom/Umbrella pattern
  - **Ink Blot Pattern**[Q] (Enlarging Dot Sign)
- Treatment:
  - Wait and watch[Q] (as spontaneous resolution occurs in about **12 weeks)**
  - Photocoagulation, anti-VEGF injections are options where resolution does not occur in three months:

CYSTOID MACULAR OEDEMA (CME)

- CME is defined as accumulation of fluid in the **Outer Plexiform Layer**[Q] of the retina in the area of the macula due to defect in the **inner blood retinal barrier**[Q]
- Cause of CME is intraocular inflammation like **pars planitis**, anterior uveitis, intraocular surgery etc
- Fundus Fluorescein Angiography: The typical pattern is called **flower petal appearance**[Q]
- Treatment is **steroids**, nonsteroidal anti-inflammatory drugs, anti- VEGF.

RETINAL DYSTROPHIES

Retinitis Pigmentosa

Retinitis Pigmentosa (RP) is a type of retinal dystrophy predominantly affecting the rod photoreceptor cells of the retina with subsequent degeneration of the cones.

- **Sporadic: Most common**[Q]
- Inheritance may be **Autosomal Dominant (AD)**, Autosomal Recessive (AR) or X-Linked Recessive (least common)

Symptoms

Night blindness or Nyctalopia[Q]

Signs

The fundus picture of RP is described as triad:

- Arteriolar attenuation[Q]
- Waxy disc pallor[Q]
- Mid peripheral pigmentary changes described as bony spicules[Q]

Other features are Myopia, Keratoconus, Glaucoma, Posterior Subcapsular Cataract[Q], Cystoid Macular Oedema

Investigations

- **Scotopic ERG: Decrease in the amplitude of the 'a' wave**[Q]
- Visual field assessment shows midperipheral **Ring Scotoma**[Q] **or Annular Scotoma**[Q]. There is gradual constriction of the visual field from the periphery with a **central tubular** vision remaining in the advanced stages of the disease.

Atypical RP

- RP sine pigmento: Inconspicuous pigmentary change.
- Retinitis punctata albescens: Scattered white dots are seen in the fundus

Atypical RP Syndromes

- **Refsum's disease**[Q]**:** Peripheral neuropathy, Cerebellar ataxia, Deafness
- **Usher's syndrome**[Q]**:** Sensorineural deafness
- **Cockayne's syndrome:** Deafness, Nystagmus, Ataxia, Mental Retardation
- **Kearns sayre syndrome**[Q]**:** Chronic progressive external ophthalmoplegia, Heart Block
- Laurence-Moon-Biedl syndrome[Q]: Mental retardation, Polydactyl, Obesity, Hypogonadism.
- **Barren-kornzweig syndrome**
- **Friedrich's ataxia**
- **NARP:** Neuropathy, Ataxia, Retinitis Pigmentosa

BEST'S DISEASE (JUVENILE VITELLIFORM DYSTROPHY)

- **Autosomal dominant (AD)**[Q]
- Dystrophy of **RPE layer**[Q] at the macula
- **Deposition of lipofuscin**[Q] **pigment at the macula**
- Typically has the following stages
  - Vitelliform Stage: **Egg yolk appearance of macula**
  - Pseudohypopyon Stage
  - Vitelloruptive Stage: **Scrambled eggs appearance of macula**
- Investigation: **EOG**[Q]

STARGARDT'S DISEASE AND FUNDUS FLAVIMACULATUS

- **Autosomal Recessive(AR) inheritance**
- Stargardt's disease is a retinal dystrophy with predominant macular involvement, hence also called **Juvenile Macular Dystrophy**
- Presents in first-second decade with decreased central vision

- Mottled appearance of the macula is seen. It is typically described as **beaten bronze appearance**Q
- Adult variant is Fundus Flavimaculatus where involvement is predominantly in the peripheral retina. Hence, patients may be asymptomatic

CONGENITAL STATIONARY NIGHT BLINDNESS

- Group of retinal disorders characterized by infantile onset nyctalopia which is non-progressive
- May have autosomal dominant, Autosomal Recessive or X-linked Recessive inheritance
- The fundus may be normal or abnormal in appearance. Two characteristic types are:
  - Fundus albipunctatus
  - **Oguchi's disease**Q**:** The fundus has an unusual golden-yellow colour in light adapted condition which becomes normal after prolonged dark adaptation. This is called **Mizuo phenomenon**Q**.**

LEBER'S CONGENITAL AMAUROSIS

- This condition is associated with very poor vision since birth or very early childhood
- Fundus shows **salt and pepper retinopathy**Q
- Characteristic feature is **oculodigital syndrome** where constant rubbing of the eyes by the child due to poor vision leads to **enophthalmos.**

RETINOBLASTOMA

Most common primary malignant intraocular tumor of childhoodQ

- Seen in 1:17000 live births and accounts for 3% of all childhood cancers
- Familial cases: 6%
- Sporadic cases: 94%

Genetics

- Gene for retinoblastoma is Rb gene (oncosuppressor gene), the locus of which is **13q14**Q
- **Knudson's two hit hypothesis**Q is used to explain the genetic pattern of retinoblastoma

Clinical Features

The common age of presentation is **18–24 months**Q. The presenting features are:

- **Leukocoria (Amaurotic cat's eye reflex): Most common presenting feature**Q
- **Strabismus**Q**:** Second most common presenting feature
- Proptosis
- Keratitis/Perforated ulcer
- Hyphaema/Hypopyon
- Rubeosis iridis
- Complicated cataract
- Endophthalmitis

- Retinal detachment
- Orbital cellulitis
- Phthisis bulbi

Types

- **Exophytic:** Grows outwards into the subretinal space resulting in total retinal detachment
- **Endophytic:** Projects from the retina into the vitreous cavity. It is associated with secondary calcification which gives cottage cheese appearance.

Mode of Spread

- Intraocular extension
- Extraocular extension– **Most common mode of spread is via the Optic Nerve**[Q]. It spreads to the brain via the optic nerve and to the orbit via emissary veins.
- Metastasis

Associated Tumours

- **Osteosarcoma**[Q] **(most common)**
- Malignant melanoma
- Testicular Ca
- Ewing's tumour
- Wilms' tumour

Trilateral Retinoblastoma: Bilateral Retinoblastoma + Pinealoblastoma[Q]

Investigations

- USG B-Scan: **Presence of intraocular mass with calcification**[Q]
- CT Scan
- MRI: Investigation of choice. It confirms the diagnosis and detects the extent of the disease
- **Aqueous humour paracentesis: Aqueous: Plasma LDH > 1**[Q]

Treatment

- Cryotherapy: Small tumor anterior to the equator
- Photocoagulation: Small tumors posterior to the equator
- Brachytherapy: Small tumors anterior to the equator
- Enucleation: **Tumours involving more than 50% of the globe** have to be enucleated with **a minimum optic nerve stump of 10 mm**[Q]
- External Beam Radiotherapy: Adjuvant therapy after enucleation in tumours with orbital and intracranial extension.
- Chemotherapy: Metastatic disease is treated with palliative chemotherapy. Extraocular disease is treated with neo-adjuvant chemotherapy prior to enucleation to downstage the disease. **Carboplatin, Etoposide,Vincristine**[Q] are the drugs used in chemotherapy

- Other modalities
  - Thermochemotherapy
  - Photodynamic therapy (PDT)

LEUCOCORIA[Q]

- Retinoblastoma
- Cataract
- Coloboma
- Persistent hyperplastic primary vitreous (PHPV)
- Toxocariasis
- Metastatic endophthalmitis
- Retinal atrocytoma
- Coats' disease
- ROP
- Retinal detachment

COATS' DISEASE

- This is a vascular disorder of the retina associated with **intraretinal telangiectasia**
- It is a **unilateral** condition seen in **boys** between **4–10 years of age**
- In this condition, abnormal telangiectatic and leaky blood vessels are seen most commonly in the **infero-temporal quadrant**[Q] of the retina. These blood vessels cause severe intraretinal and subretinal exudation leading to exudative retinal detachment[Q]
- Presentation is with **leucocoria** and **strabismus**[Q] associated with vision loss
- On examination exudation with retinal detachment is seen
- Treatment is laser photocoagulation of the telangiectatic vessels
- A variant of Coats' disease is called **Idiopathic Juxtafoveal Telangiectasia**[Q].

PERSISTENT HYPERPLASTIC PRIMARY VITREOUS (PHPV)

- It is a congenital condition where the primary vitreous fails to regress. It is of two types–anterior and posterior. **Anterior PHPV is more common**
- In anterior PHPV, the persistent primary vitreous presents as a retrolental mass into which elongated ciliary processes are inserted. The contraction of the ciliary body pulls these processes and tears the capsule of the lens.
- Presents with **leucocoria in a microphthalmic eye**[Q]. It is always **unilateral**[Q].
- The features of anterior PHPV are summarized as ABC:
  - Decreased **axial length** or microphthalmos
  - **Bands** in the vitreous
  - Elongated **ciliary body**
- Posterior PHPV is a rare condition where a dense white membrane is seen to extend from the disc to the peripheral retina. **It presents with retinal detachment.**

QUESTIONS

1. **Which of the following is not true about direct ophthalmoscopy?** *(AIIMS 2015)*
   a. 2 disc diameters field
   b. Image is virtual and erect
   c. Magnification is 5 times
   d. Self-illuminated device
2. **Which of the following is false about indirect ophthalmoscopy?** *(AIIMS 2013)*
   a. Convex lens is used
   b. Image is virtual and erect
   c. Magnification is 4-5 times
   d. It is so bright that regular haziness is penetrated
3. **There is a retained intraocular foreign body in the eye. Which is the most important test for monitoring vision?** *(AIIMS 2014)*
   a. Dark adaptometry
   b. Visual evoked potential
   c. ERG
   d. EOG
4. **Normal value of Arden index is** *(AIIMS 2014)*
   a. 1
   b. 1.5
   c. Less than 185%
   d. More than 185%
5. **'b' wave in ERG arises from:** *(APPG 2014)*
   a. Rods and cones
   b. Bipolar cells
   c. Ganglion cells
   d. Retinal pigment epithelium
6. **In fluorescein angiography of retina, the dye is injected in:** *(AIPG)*
   a. Femoral artery
   b. Antecubital vein
   c. Ophthalmic artery
   d. Internal carotid artery
7. **The retina receives its blood supply from all *except*:** *(AIPG)*
   a. Posterior ciliary arteries
   b. Central retinal artery
   c. Retinal arteries
   d. Circle of Zinn haler
8. **The distance of the fovea from the temporal margin of the optic disc is:** *(AIPG)*
   a. 1 disc diameter
   b. 2 disc diameters
   c. 3 disc diameters
   d. 4 disc diameters
9. **Regarding the fovea, which of the following is true:** *(PGI)*
   a. Has the lowest threshold for light
   b. Contains only rods
   c. Contains only cones
   d. Maximum visual acuity
   e. Located on the optic nerve
10. **Which area of the retina has the highest concentration of rods?** *(Bihar PG 2014)*
    a. Parafoveal region
    b. Optic disc
    c. Fovea
    d. Ora serrata
11. **The retina develops from:** *(COMEDK 2013)*
    a. Neuroectoderm
    b. Surface ectoderm
    c. Endoderm
    d. Mesoderm
12. **The risk of rhegmatogenous retinal detachment is increased in all of the following *except*:** *(AIIMS 2013)*
    a. Pseudophakia
    b. Trauma
    c. Hypermetropia
    d. Lattice degeneration
13. **Which of the following is not true about rhegmatogenous retinal detachment?** *(AIIMS 2014)*
    a. It is caused due to fibrous bands in the vitreous

b. It presents with floaters and photopsia
c. It may extend up to ora serrata
d. Surgery is the primary treatment

14. Subretinal demarcation line or watershed line is seen in: *(AIIMS 2014)*
a. Fresh rhegmatogenous retinal detachment
b. Old rhegmatogenous retinal detachment
c. Retinopathy of prematurity
d. Retinitis pigmentosa

15. Pneumatic retinopexy is an outpatient procedure where retinal detachment is sealed with air insufflation. Which is the gas used in the process? *(AIIMS 2014)*
a. Carbon dioxide
b. Sulphur hexafluoride
c. Nitrous oxide
d. Oxygen

16. A young patient with history of using glasses for the past 10 years comes with complains of photopsia and sudden loss of vision in the right eye. Which of the following tests should be done. *(AIIMS)*
a. Cycloplegic refraction
b. Gonioscopy
c. Direct ophthalmoscopy
d. Indirect ophthalmoscopy

17. A patient presents with sudden onset floaters and perception of a curtain or veil in front of his right eye. What is the most probable diagnosis? *(AIIMS 2011)*
a. Vitreous haemorrhage
b. Retinal detachment
c. Eales' disease
d. Glaucoma

18. Causes of exudative retinal detachment are: *(PGI)*
a. Scleritis
b. Toxemia pregnancy
c. Dysthyroid eye disease
d. Sickle cell retinopathy

19. Causes of exudative retinal detachment are: *(PGI)*
a. Central retinal artery occlusion
b. Harada's disease
c. Hypertensive retinopathy
d. Coats' disease

20. Retinal detachment may be treated by: *(Bihar PG 2014)*
a. Cryosurgery
b. Enucleation
c. Evisceration
d. Ranibizumab

21. A person is diagnosed to have diabetes on his 45th birthday. When will you recommend a dilated fundus examination for him? *(AIIMS 2014)*
a. Immediately
b. Before his 50th birthday
c. After his 50th birthday
d. When he complains of decrease in vision

22. Microaneurysms are the earliest feature of diabetic retinopathy. In which layer of retina are they seen: *(AIIMS 2014)*
a. Outer plexiform layer
b. Inner nuclear layer
c. Layer of rods and cones
d. Retinal pigment epithelium

23. Which of the following is not a feature of diabetic retinopathy? *(DPG)*
a. Microaneurysm
b. Cotton wool spots
c. Hard exudates
d. Choroidal neovascularisation

24. Features of non-proliferative diabetic retinopathy are all *except*: *(PGI)*
a. Neovascularisation
b. Hard exudates
c. Soft exudates
d. Microaneurysm

25. Diabetic retinopathy can lead to: *(PGI)*
a. Vitreous haemorrhage
b. Retinal detachment
c. IIIrd, IVth and VIth cranial nerve palsies

d. Rubeosis iridis
e. Hypermetropia

26. Grid photocoagulation is indicated in *(COMEDK)*
a. Ischaemic maculopathy
b. Clinically significant macular oedema
c. Macular hole
d. Proliferative diabetic retinopathy

27. Panretinal photocoagulation is indicated in: *(DPG)*
a. Clinically significant macular edema
b. Retinal break
c. Proliferative diabetic retinopathy
d. Tractional retinal detachment

28. A patient with Clinically Significant Macular Edema (CSME) was treated with macular grid photocoagulation. After 3 months the OCT showed persistent vitreo-retinal traction. What is the next line of management? *(AIIMS 2011)*
a. Wait and watch
b. Intravitreal bevacizumab
c. Pars plana vitrectomy
d. Repeat macular grid photocoagulation

29. All of the following are involved in the pathogenesis of diabetic macular oedema *except*: *(AIIMS)*
a. Retinal pigment epithelium dysfunction
b. Oxidative stress
c. Increase in VEGF
d. Increase in protein kinase C

30. ETDRS chart is used for vision evaluation in diabetic patients. What does ETDRS stand for? *(AIIMS)*
a. Extended treatment for diabetic retinopathy study
b. Early treatment for diabetic retinopathy study
c. Emergency treatment for diabetic retinopathy study
d. Emerging treatment for diabetic retinopathy study

31. Treatment of advanced diabetic retinopathy with vitreo-retinal fibrosis and tractional retinal detachment include all the following *except*: *(AIPG)*
a. Removal of epiretinal membrane
b. Pars plana vitrectomy
c. Reattachment of retina
d. Exophotocoagulation

32. A 35-year-old patient of Insulin Dependent Diabetes Mellitus (IDDM) on insulin for the past 10 years complains of gradual progressive, painless loss of vision. What is the possible diagnosis? *(AIIMS)*
a. Cataract
b. Vitreous haemorrhage
c. Rhegmatogenous retinal detachment
d. Tractional retinal detachment not involving the macula

33. A young patient presents with sudden, painless loss of vision in one eye. Ocular examination reveals visual acuity of perception of light and cherry red spot on the fundus. A systolic murmur is heard on the chest. The probable diagnosis is: *(AIIMS)*
a. Central retinal artery occlusion
b. Central retinal vein occlusion
c. Multifocal choroiditis with infective endocarditis
d. Central serous retinopathy

34. Cherry red spot is seen in all *except*: *(AIPG)*
a. Niemann-Pick disease
b. CRAO
c. Tay-Sachs disease
d. CRVO

35. A 20-year-old presents with history of tennis ball injury to the right eye. On examination a red spot is seen on the macula. The most likely diagnosis is: *(AIIMS)*
a. Macular hole
b. Berlin's oedema

c. Macular tear
d. Macular haemorrhage

36. Cherry red spot in children after trauma is seen in: *(AIIMS 2015)*
a. CRAO
b. CRVO
c. Berlin's oedema
d. Niemann-Pick disease

37. Premature baby weighing 1000 gms or less is most likely to suffer from *(AIIMS 2013)*
a. Cataract
b. Glaucoma
c. Retinopathy of prematurity
d. Retinal detachment

38. A 28 week old baby suffered from respiratory distress syndrome at birth. On day 14, he developed sepsis. At what postnatal age should the baby undergo retinal evaluation for ROP? *(AIIMS 2014)*
a. 2 weeks b. 4 weeks
c. 6 weeks d. 8 weeks

39. A premature baby on examination shows bilateral ROP (Zone 1, Stage II with plus disease). How will you manage the patient? *(AIIM 2011)*
a. Examine the patient again after 1 week
b. Laser photocoagulation of both eyes
c. Laser photocoagulation of worse eye
d. Vitreoretinal surgery

40. A 25-year-old male presents with sudden painless loss of vision in one eye. There is no history of trauma. On examination, the anterior segment is normal but there is no fundal glow. Which of the following is the most likely cause? *(AIPG)*
a. Vitreous haemorrhage
b. Developmental cataract
c. Optic atrophy
d. Acute angle closure glaucoma

41. Which of the following is not a feature of vitreous haemorrhage? *(Jipmer 2015)*
a. Sudden loss of vision
b. Floaters
c. Metamorphopsia
d. Absence of fundal glow

42. A young male patient presents with recurrent vitreous haemorrhage. Probable diagnosis is: *(PGI)*
a. Eales' disease
b. CRVO
c. Coat's disease
d. Proliferative vitreoretinopathy
e. Episcleritis

43. A 30-year-old male with history of headache is sent for fundus evaluation. On examination he was found to have generalised arterial attenuation with multiple cotton wool spots and flame shaped haemorrhages in both eyes. The probable diagnosis is: *(AIIMS)*
a. Diabetic retinopathy
b. Hypertensive retinopathy
c. Central retinal artery occlusion
d. Temporal arteritis

44. Hard exudates are seen in all except: *(PGI)*
a. Diabetic retinopathy
b. Retinitis pigmentosa
c. Eales' disease
d. Retinal artery macroaneurysm
e. Choroidal neovascular membrane

45. Cotton wool spots are seen in *(PGI)*
a. Diabetic retinopathy
b. Hypertensive retinopathy
c. AIDS
d. Retinoblastoma
e. Toxemia pregnancy

46. Which of the following is not associated with rubeosis iridis? *(DNB)*
a. Proliferative diabetic retinopathy
b. Retinopathy of prematurity
c. Central serous retinopathy
d. Eales' disease

47. Ocriplasmin is a recombinant protease used to treat *(AIIMS 2013)*
a. Retinal break
b. Diabetic macular oedema

c. Uveovitreal membrane
d. Submacular bleeding

48. **Retinitis pigmentosa is not associated with:** *(AIIMS)*
a. Usher syndrome
b. Barren- Kornzweig syndrome
c. Kearne Sayre syndrome
d. Marfan syndrome

49. **Retinitis pigmentosa is a feature of all *except*:** *(AIIMS 2010)*
a. Refsum's disease
b. Hallervorden- Spatz disease
c. NARP
d. Abetalipoproteinemia

50. **All are true about Retinitis pigmentosa except** *(AIIMS)*
a. It may have X-linked inheritance
b. Early diagnosis and treatment prevent progression of the disease
c. Visual acuity is preserved even in advanced stage of the disease
d. Associated with systemic abnormalities

51. **Most common type of cataract seen in Retinitis pigmentosa is:**
a. Nuclear cataract
b. Posterior subcapsular cataract
c. Cortical cataract
d. Anterior polar cataract

52. **Ring scotoma is a feature of:** *(AIIMS)*
a. Blue dot cataract
b. Nuclear cataract
c. Retinitis pigmentosa
d. Diabetic retinopathy

53. **All of the following are true about Retinitis pigmentosa *except*:** *(DNB)*
a. Presence of pigments in the retina
b. Narrowing of the arterioles
c. Pale waxy disc
d. Normal ERG

54. **A retinal disease is characterised by progressive rod-cone dystrophy. It presents with pale disc and retinal vessel attenuation. What is the third feature of the triad?** *(COMEDK 2015)*
a. Macular degeneration
b. Bony spicule pigmentary changes
c. Pre-retinal haemorrhages
d. Cotton wool spots

55. **Idiopathic nyctalopia is due to hereditary:** *(AIIMS)*
a. Absence of rod function
b. Absence of cone function
c. Absence of both rod and cone function
d. Decrease in function of bipolar cells

56. **Night blindness is seen in all *except*:** *(WBPG)*
a. Retinitis pigmentosa
b. Cone dystrophy
c. Oguchi's disease
d. Vitamin A deficiency

57. **Mizuo phenomenon is seen in:** *(AIIMS)*
a. Oguchi's disease
b. Fundus albipunctatus
c. Fundus flavimaculatus
d. Retinitis pigmentosa

58. **A young patient presents with loss of central vision. There is no significant family history. Both ERG and EOG are normal. Which is the most likely diagnosis?** *(AIPG)*
a. Retinitis pigmentosa
b. Rod-cone dystrophy
c. Stargardt's disease
d. Best's disease

59. **A young patient presents with loss of central vision. ERG is normal but EOG is abnormal. What is the likely diagnosis?** *(AIPG)*
a. Retinitis pigmentosa
b. Rod-cone dystrophy
c. Stargardt's disease
d. Best's disease

60. **Ideal diagnostic test for Best's disease is:** *(JIPMER)*
a. Dark adaptometry
b. ERG
c. EOG
d. Perimetry

61. **Which of the following has Autosomal Dominant inheritance pattern?** *(AIIMS)*
    a. Best's disease
    b. Gyrate atrophy
    c. Laurence-Moon- Biedl syndrome
    d. Barren-Kornzweig syndrome

62. **Pigmentary changes between the posterior pole and equator known as ' salt and pepper retinopathy' is seen in all *except*:** *(AIIMS 2013)*
    a. Resolving retinal detachment
    b. Phenothiazine toxicity
    c. Congenital rubella
    d. Fundus flavimaculatus

63. **A young male presents with central scotoma in the left eye. His right eye showed 6/6 vision. On examination there is focal foveal detachment in the left eye. What should be the next step?** *(AIIMS 2014)*
    a. Examine retrolental cells
    b. Inquire about use of steroids
    c. Inquire about trauma to the other eye
    d. Examination on slit lamp

64. **A 25-year-old executive presents with metamorphopsia in his right eye. On examination, there is a shallow detachment at the macula. FFA shows smokestack appearance. Which of the following should be the line of management?** *(AIIMS)*
    a. Topical antibiotic steroid
    b. Systemic steroids
    c. Pulse methyl prednisolone
    d. Wait and watch for spontaneous recovery

65. **Enlarging dot sign on FFA is seen in:** *(PGI)*
    a. Cystoid macular edema
    b. Central serous retinopathy
    c. Clinically significant macular edema (CSME)
    d. Coats' disease

66. **ICGA is primarily indicated in:** *(AIPG)*
    a. Minimal classic CNVM
    b. Occult CNVM
    c. Angioid streaks
    d. Polypoidal choroidal vasculopathy

67. **Choroidal neovascular membrane (CNVM) is seen in all *except*:** *(AIIMS)*
    a. Hypermetropia
    b. Myopia
    c. Traumatic choroidal rupture
    d. Angioid streaks

68. **Photodynamic therapy is used for the treatment of:**
    a. Cataract
    b. Glaucoma
    c. Wet ARMD
    d. Uveitis

69. **Which of the following is not used in the treatment of neovascular ARMD?** *(COMEDK 2015)*
    a. Alemtuzumab
    b. Bevacizumab
    c. Ranibizumab
    d. Pegabtanib sodium

70. **Angioid streaks are seen in:** *(AIIMS)*
    a. Pseudoxanthema elasticum
    b. Tendinous xanthoma
    c. Xanthelasma
    d. Eruptive xanthoma

71. **Most common clinical presentation of retinoblastoma is:** *(AIIMS 2013)*
    a. Leucocoria+ Heterochromia iridis
    b. Leucocoria+ Pseudohypopyon
    c. Leucocoria+ Hyphaema
    d. Leucocoria + Strabismus

72. **Hereditary retinoblastoma is associated with which chromosomal segment:** *(AIPG)*
    a. 13q14 b. 13p14
    c. 14q13 d. 14p13

73. **Knudson's two hit hypothesis describes the occurrence of:** *(PGI)*
    a. Glaucoma
    b. Retinoblastoma

c. Optic nerve glioma
d. Meningioma

74. Familial retinoblastoma *(PGI)*
a. Has autosomal recessive inheritance
b. Usually bilateral
c. Occurs due to mutation of Rb gene
d. More common than sporadic retinoblastoma
e. Poorer prognosis than sporadic retinoblastoma

75. The most common second malignancy in survivors of retinoblastoma is: *(AIPG)*
a. Thyroid cancer
b. Osteosarcoma
c. Chondrosarcoma
d. Pinealoblastoma

76. Increased LDH in aqueous humour suggests a diagnosis of: *(AIPG)*
a. Galactosemia
b. Glaucoma
c. Retinoblastoma
d. Gyrate atrophy

77. Most common route of spread of retinoblastoma: *(AIIMS 2015)*
a. Lymphatics b. Optic nerve
c. Direct spread d. Vascular

78. As regards to retinoblastoma, which of the following statements is false? *(AIIMS)*
a. 94% of the cases are sporadic
b. Patients with sporadic disease do not pass the genes to their offspring
c. Calcification in the tumour is detected on USG B-scan
d. Reese- Ellsworth classification is useful for predicting prognosis after radiotherapy

79. A 2-year-old child presents with leucocoria in the right eye since 2 months. On examination total retinal detachment is seen. USG B-scan reveals the presence of a subretinal mass with calcification. What is the most probable diagnosis? *(AIIMS)*
a. Coats' disease
b. Retinoblastoma
c. Toxocariasis
d. Retinal tuberculoma

80. A one year old child having leucocoria was diagnosed to have large unilateral retinoblastoma filling half the globe. What is the management of the patient? *(AIPG)*
a. Enucleation
b. Chemotherapy followed by local therapy
c. Photodynamic therapy
d. Radiotherapy followed by chemotherapy

81. A 5-year-old boy is diagnosed to have bilateral retinoblastoma. In the right eye there is advanced retinoblastoma almost filling the globe whereas in the left eye, a few small lesions are seen in the periphery. What is the management? *(AIPG)*
a. Enucleation of both eyes
b. Enucleation of right eye and focal therapy of left eye
c. Radiotherapy
d. Six cycles of chemotherapy

82. Ideal treatment of bilateral advanced retinoblastoma *(PGI/ JIPMER)*
a. Enucleation
b. Chemotherapy
c. Radiotherapy
d. Photocoagulation

83. Pseudo rosettes are seen in *(PGI)*
a. Ophthalmia nodosum
b. Retinoblastoma
c. Trachoma
d. Phacolytic glaucoma

84. Poor prognostic factors for retinoblastoma are: *(PGI 2015)*
a. Size >4 mm
b. Size >2 mm
c. Associated glaucoma

d. Undifferentiated tumour cells
e. Scleral involvement

85. **Enucleation means** *(PGI)*
a. Removal of the contents of the globe
b. Removal of the entire globe with portion of the optic nerve
c. Removal of the contents of the orbit
d. Removal of the globe leaving a frill of sclera around the optic nerve

86. **Evisceration is contraindicated in:** *(AIIMS 2015)*
a. Malignancy
b. Panophthalmitis
c. Severe ocular trauma
d. Expulsive choroidal haemorrhage

87. **Leucocoria is seen in all *except*: *(AIIMS)***
a. Persistent hyperplastic primary vitreous
b. Congenital glaucoma
c. Endophthalmitis
d. Retinoblastoma

88. **A 7-year-old male presents with 6/6 vision in the right eye and hand movements close to face vision in the left eye. On fundoscopy, the right eye was normal. The left eye showed retinal detachment, subretinal yellowish exudates and telangiectatic vessels. What is the most probable diagnosis?** *(AIIMS2014)*
a. Coats' disease
b. Sympathetic ophthalmitis
c. Familial exudative vitreoretinopathy
d. Retinopathy of prematurity

89. **All of the following are true about Idiopathic Juxtafoveal Telangiectasia *except*:** *(AIIMS)*
a. It is a variant of Coat's disease
b. It is associated with macular telangiectasia
c. It is associated with structural abnormalities of the retinal blood vessels
d. It is associated with peripheral retinal telangiectasia

90. **Which of the following is associated with Persistent hyperplastic primary vitreous (PHPV)?** *(AIPG)*
a. Patau syndrome
b. Edward syndrome
c. Trisomy 14
d. Down's syndrome

91. **Which of the following does not show calcification?** *(AIIMS 2014)*
a. Persistent hyperplastic primary vitreous
b. Choroidal osteoma
c. Optic nerve head drusen
d. Retinoblastoma

92. **Acute loss of vision in a case of alcoholic pancreatitis** *(AIPG)*
a. Purtscher's retinopathy
b. Acute congestive glaucoma
c. Central retinal artery obstruction
d. Optic neuritis

93. **Roth's spots are seen in:** *(PGI)*
a. Hypertension
b. Diabetes
c. Bacterial endocarditis
d. Central retinal artery occlusion

94. **Bull's eye maculopathy is seen in toxicity of:** *(PGI/AIIMS)*
a. Chloroquine b. Dapsone
c. Rifampicin d. Ethambutol

95. **Mucopolysaccharide hyaluronic acid is present in:** *(AIIMS)*
a. Vitreous humour
b. Cornea
c. Lens
d. Blood vessels

96. **A vitreous sample has been collected at 9 pm. What advice would you like to give to the staff on duty regarding the overnight storage of the sample?** *(AIPG)*
a. The sample should be stored at 4 degrees Celsius
b. The sample should be incubated at 37 degrees Celsius
c. The sample should be kept in the deep freezer

d. The sample should be refrigerated for the initial 3 hours and then incubated

97. When compared to blood, vitreous humour has higher concentration of: *(AIIMS 2015)*

a. Glucose
b. Sodium
c. Potassium
d. Ascorbate

98. Which of the following is not an ocular emergency? *(PGI 2015)*

a. Ocular trauma
b. Sympathetic ophthalmitis
c. CRAO
d. CRVO
e. Endophthalmitis

ANSWERS AND EXPLANATIONS

1. **c. Magnification is 5 times**
2. **b. Image is virtual and erect**
3. **c. ERG**
   Electroretinogram is an important prognostic tool for intraocular metal foreign body, especially iron. In siderosis bulbi, the early change in ERG is an increase in the amplitude of the negative a wave with a normal b wave. Later the amplitude of the b wave also decreases. In the end stage disease, ERG becomes completely extinguished.
4. **d. More than** 185%
5. **b. Bipolar cells**
6. **b. Antecubital vein**
7. **a. Posterior ciliary arteries**
   This is a slightly controversial question because all the mentioned vessels are involved in the blood supply to the retina. I would choose posterior ciliary artery as the answer because the short posterior ciliary arteries do not directly supply the retina. They form the choriocapillaries from where the outer layers of retina are supplied.
8. **b. 2 disc diameters**
9. **a. Has the lowest threshold for light, c. Contains only cones, d. Maximum visual acuity**
10. **a. Parafoveal region**
    The highest concentration of rods is in the midperiphery of the retina. But among the options here the most appropriate is parafoveal
11. **a. Neuroectoderm**
    Explained in the chapter on Ocular Embryology
12. **c. Hypermetropia**
    Rhegmatogenous RD is associated with high myopia and not hypermetropia
13. **a. It is caused due to fibrous bands in the vitreous**
    Fibrous bands in the vitreous are responsible for causing tractional RD, not rhegmatogenous.
14. **b. Old rhegmatogenous retinal detachment**
15. **b. Sulphur hexafluoride**
16. **d. Indirect ophthalmoscopy**
    The question hints at a possible myopic patient presenting with complaints of flashes and floaters. This may be due to a peripheral retinal break and has the potential to progress to Rhegmatogenous RD. Hence, this patient should undergo a peripheral retinal screening with indirect ophthalmoscopy. If a break is identified, laser barrage of the break should be done to prevent RD.
17. **b. Retinal detachment**
18. **a. Scleritis, b. Toxemia of pregnancy**
19. **b. Harada's disease, c. Hypertensive retinopathy, d. Coats' disease**
    Causes of exudative RD
    - Inflammatory disorders like choroiditis, choroidal vasculitis, posterior scleritis
    - Choroidal tumours

- Vogt-Koyanagi-Harada's disease (This is a panuveitis with multifocal choroiditis leading to exudative RD)
- Malignant hypertension/Toxemia pregnancy
- Coats' disease (Retinal telengiectasia with leaking retinal blood vessels leading to exudative RD).

20. a. Cryosurgery

Rhegmatogenous retinal detachment is treated by Scleral Buckling surgery. The important steps of the surgery are:

- Application of the buckle
- Drainage of the subretinal fluid
- Cryotherapy to the breaks

Hence, we choose the answer cryosurgery (though the term is not completely appropriate).

21. a. Immediately

Screening for Diabetic Retinopathy

- For Insulin Dependent Diabetes Mellitus (IDDM) or juvenile-onset DM: Screening should be started 5 years after diagnosis[Q]
- For Non-insulin Dependent Diabetes Mellitus (NIDDM) or late-onset diabetes: Screening should be started at the time of diagnosis[Q]

The patient in the question belongs to the NIDDM group.

22. b. Inner nuclear layer

23. d. Choroidal neovascularisation

Proliferative diabetic retinopathy is associated with retinal neovascularisation

24. a. Neovascularisation

Neovascularisation is a feature of PDR not NPDR

25. a. Vitreous haemorrhage, b. Retinal detachment, c. IIIrd, IVth, VIth cranial nerve palsies, d. Rubeosis iridis

Diabetes may lead to myopic shift and frequent changes in refraction due to fluctuation in blood sugar levels but hypermetropia is not seen.

26. b. Clinically significant macular oedema

27. c. Proliferative diabetic retinopathy

28. c. Pars plana vitrectomy

This question is an example of a case where the macular oedema is caused by a tractional band at the macula. Hence, the treatment is pars plana vitrectomy.

29. d. Increase in protein kinase C

30. b. Early treatment for diabetic retinopathy study

31. d. Exophotocoagulation

The different procedures that are done in diabetic vitrectomy are:

- Pars plana vitrectomy
- Release of vitreous membranes
- Removal of epiretinal membranes

(The removal of the tractional bands will cause the retina to settle)

- Endophotocoagulation
- Silicon oil injection (This is a vitreous substitute used to reform the vitreous cavity)

32. **a. Cataract**
    Diabetes is associated with snow flake cataract. It may also lead to rapid progression of senile cataract.
33. **a. Central retinal artery occlusion**
34. **d. CRVO**

Important causes of **Cherry Red Spot**
• **CRAO/BRAO**[Q]
• **Berlin's oedema (blunt trauma)**[Q]
• **Gaucher's disease**[Q]
• **Niemann-Pick's disease**[Q]
• **Tay-Sach disease**[Q]
• **Metachromatic leukodystrophy**[Q]
• Hurler's syndrome
• Sialodosis
• Multiple sulfatase deficiency

35. **b. Berlin's oedema**
36. **c. Berlin's oedema**
    Berlin's oedema is seen after blunt trauma or concussion injury. It is retinal oedema mainly seen in the nerve fibre layer. As a result, the entire retina appears pale except the fovea where the nerve fibre layer is absent. This gives the appearance of cherry red spot.
37. **c. Retinopathy of prematurity**
38. **b. 4 weeks**
39. **b. Laser photocoagulation**
    The question describes a case of Threshold ROP in both eyes. Hence, the treatment is laser photocoagulation of both eyes.
40. **a. Vitreous haemorrhage**
    The question describes a young male with sudden painless vision loss. Absence of fundal glow means dense media opacity. So this is a case of Eales' disease presenting with vitreous haemorrhage
41. **c. Metamorphopsia**
42. **a. Eales' disease**
43. **b. Hypertensive retinopathy**
44. **b. Retinitis pigmentosa**
    Hard exudates are lipoproteins leaking from the retinal blood vessels due to damage to the endothelium.

Causes of **Hard exudates**
• **Diabetes Mellitus**[Q]
• **Hypertension (macular star)**[Q]
• **Retinal artery macroaneurysm**[Q]

Contd...

Contd...

• **Capillary haemangioma of the retina (Von Hippel-Lindau disease)Q**
• **Retinal vascular occlusions like CRVO, BRVOQ**
• Coat's disease
• Choroidal neovascular membrane
• Radiation retinopathy
• Neuroretinitis (macular star)

45. **a. Diabetic retinopathy, b. Hypertensive retinopathy, c. AIDS, d. Toxemia pregnancy**
Cotton wool spots are infarcts in the retinal nerve fibre layer due to microvascular occlusion. They were previously called soft exudates.

Causes of **Cotton wool spots**
• **Diabetes MellitusQ**
• **Hypertension/ Toxemia pregnancyQ**
• **Retinal ischaemic disorders like CRVO, BRVO, ocular ischaemic syndromeQ**
• Embolic disorders
• Infections like **HIVQ**, leptospirosis, fungal infections
• Collagen vascular disorders like SLE
• Neoplastic disorders like lymphoma, leukaemia
• **Radiation retinopathyQ**

46. **c. Central serous retinopathy**
Causes of rubeosis iridis
- Proliferative diabetic retinopathy
- CRVO
- Eales' disease
- Retinopathy of prematurity
- Sickle cell retinopathy
- Chronic iritis
- Iris tumours

47. **c. Uveovitreal membrane**
48. **d. Marfan's syndrome**
49. **b. Hallervorden-Spatz disease**
50. **b. Early diagnosis and treatment prevents progression of the disease**
Progression is the natural course of the disease and cannot be prevented. However, visual acuity in bright light in good till advanced stage of the disease.
51. **b. Posterior subcapsular cataract**
52. **c. Retinitis pigmentosa**
53. **d. Normal ERG**

54. b. Bony spicule pigmentary changes

The classical triad of Retinitis pigmentosa is:

- Waxy disc pallor
- Arteriolar attenuation
- Bony spicules or retinal pigmentary changes

55. a. Absence of rod function

56. b. Cone dystrophy

Causes of **Night blindness**
• **Vitamin A deficiency**[Q]
• **Retinitis pigmentosa**[Q]
• Congenital stationary night blindness (Oguchi's disease, Fundus albipunctatus)
• Choroideremia
• Advanced glaucoma
• Pathological myopia

57. a. Oguchi's disease

58. c. Stargardt's disease

Stargardt's disease being localised to the macula does not produce any appreciable change in the routine Flash ERG. It shows decrease in the amplitude of Pattern ERG.

59. d. Best's disease

60. c. EOG

61. a. Best's disease

62. a. Resolving retinal detachment

Salt and pepper retinopathy refers to areas of pigmentation along with areas of hypopigmentation in large areas of retina.

Important causes of **Salt and Pepper Retinopathy**
• **Congenital syphilis**[Q]
• **Congenital rubella**[Q]
• Retinitis pigmentosa
• **Leber's congenital amaurosis**[Q]
• **Fundus flavimaculatus**[Q]
• Albinism
• Cystinosis
• **Phenothiazine toxicity**[Q]

63. b. Inquire about the use of steroids

The question describes features suggestive of Central Serous Retinopathy (CSR). Steroids are one of the risk factors for CSR and hence the answer.

64. d. Wait and watch for spontaneous recovery

The question describes a case of CSR. Treatment for CSR is to wait for spontaneous resolution for 3 months. Hence the answer

65. **b. Central serous retinopathy**

66. **b. Occult CNVM**

Indocyanine Green Angiography (ICGA) is used to visualise the choroidal vasculature. One of its main uses is to differentiate an Occult CNVM from a Classic CNVM when the clinical picture is not completely clear. The regimen of Anti-VEGF injection is different for the two types. Prognosis is worse for Occult CNVM.

Idiopathic Polypoidal Choroidal Vasculopathy (IPCV) is a relatively uncommon condition where the clinical picture is similar to CNVM but the prognosis is poor. Diagnosis of IPCV is also confirmed by ICGA.

67. **a. Hypermetropia**

Causes of CNVM are:

- Age-related macular degeneration (ARMD)Q
- High myopiaQ
- Traumatic choroidal ruptureQ
- Angioid streaksQ

68. **c. Wet ARMD**

69. **a. Alemtuzumab**

The drugs used for Wet/Exudative/Neovascular AMD are Bevacizumab and Ranibizumab. Pegabtanib was rarely used before.

70. **a. Pseudoxanthema elasticum**

Angioid Streaks are idiopathic breaks in the Bruch membrane. (Membrane separating choroid from the RPE). Conditions associated with angioid streaks are:

- Pseudoxanthema elasticumQ
- Ehlers–DanlosQ
- Paget's disease
- Sickle cell disease

71. **d. Leucocoria + strabismus**

The most common presentation of Rb is leucocoria followed by strabismus.

The common age of presentation is 18-24 months.

72. **a. 13q14**

73. **b. Retinoblastoma**

74. **b. Usually bilateral, c. Occurs due to mutation of Rb gene, e. Poor prognosis as compared to sporadic retinoblastoma**

75. **b. Osteosarcoma**

76. **c. Retinoblastoma**

77. **b. Optic nerve**

78. **b. Patients with sporadic disease do not pass the gene to their offsprings**

Sporadic retinoblastoma accounts for 94% cases and only 6% cases are familial. But even patients with sporadic disease can transmit the defective gene to the offspring. However, the prognosis is better than the familial variety.

79. **b. Retinoblastoma**

2-year old child, leucocoria, intraocular mass with calcification—Diagnosis of Retinoblastoma

80. a. Enucleation

In the section on Retinoblastoma we have discussed the different treatment modalities available for the disease. Indications for different treatment modalities are:

A. Enucleation
- Intraocular retinoblastoma involving more than 50% of the globe
- Intraocular retinoblastoma extending to anterior segment
- Retinoblastoma with orbital extension (after chemoreduction)

B. Focal therapy: Photocoagulation, Cryotherapy, Brachytherapy
- Small intraocular tumours
- Larger tumours may be treated with focal therapy after chemoreduction (to avoid enucleation)

C. Chemotherapy
- Palliative therapy for retinoblastoma with metastasis
- Bilateral advanced retinoblastoma (to avoid bilateral enucleation)
- Chemoreduction

D. Radiotherapy
- Advanced cases with intracranial extension
- Orbital radiotherapy for cases with extension to orbital wall

81. b. Enucleation of the right eye and focal therapy of the left eye

82. b. Chemotherapy

83. b. Retinoblastoma

Retinoblastoma arises as a malignant proliferation of the primitive retinal cells called retinoblasts. Histopathological examination shows typical arrangement of tumour cells called rosettes. The different types are:
- Flexner-Wintersteiner rosettes (highly specific to Rb)[Q]
- Homer-Wright rosettes[Q]
- Pseudo rosettes[Q]
- Flurettes[Q]

84. a. Size > 4 mm, c. Associated glaucoma, d. Undifferentiated tumour cells, e. Scleral involvement

85. b. Removal of the entire globe with portion of the optic nerve

There are three types of destructive ocular procedures
- **Enucleation**: Removal of the entire globe with a portion of the optic nerve. Absolute indication is intraocular malignancy[Q] (retinoblastoma, malignant melanoma). Relative indications are painful blind eye, anterior staphyloma, microphthalmos, and phthisis bulbi. Absolute contraindication is panophthalmitis[Q]
- **Evisceration**: Removal of the contents of the globe leaving behind the sclera and the extraocular muscles. Absolute indication is panophthalmitis[Q]. Absolute contraindication is intraocular malignancy[Q]
- **Exanteration**: It is a radical procedure involving removal of the globe, extraocular muscles, adnexa and a portion of the bony orbit. It is rarely done today.

86. a. Malignancy

87. b. Congenital glaucoma

88. **a. Coats disease**
The question describes a male child between 4-10 years of age with telangiectatic retinal vessels and exudative retinal detachment only in one eye. The other eye is normal. Hence the answer is Coats disease.

89. **d. It is associated with peripheral retinal telangiectasia**
Idiopathic Juxtafoveal Telangiectasia is a variant of Coats disease. In Coats disease, the telangiectatic vessels are peripherally located along the inferotemporal arcade. In juxtafoveal telangiectasia, the abnormal vessels are macular.

90. **a. Patau syndrome**

91. **a. Persistent hyperplastic primary vitreous**

Causes of **intraocular calcification**
• **Optic nerve head drusen**Q
• **Retinoblastoma**Q
• **Choroidal osteoma**Q **(Tuberous sclerosis)**
• **Choroidal angioma**
• **Coat's disease**Q
• **Retinopathy of prematurity**Q
• Healed chorioretinitisQ (mainly toxoplasmosis)
• Dystrophic calcification: Phthisis bulbi
• Metastatic calcification: Systemic hypercalcemia

92. **a. Purtscher's retinopathy**
It is a blockage of the retinal capillaries by emboli leading to multiple haemorrhages and cotton wool spots throughout the retina.
Causes are acute pancreatitisQ, head and chest trauma, SLE, chronic renal failure.

93. **c. Bacterial endocarditis**
Roth's spots are retinal haemorrhages with pale or white centres. The important causes are:
- Bacterial endocarditis
- Diabetes, hypertension
- Leukaemia
- Pernicious anaemia
- HIV (rarely)

94. **a. Chloroquine**
Bull's eye maculopathy is a term given to a condition where the macula shows a central area of hyperpigmentation surrounded by a zone of hypopigmentation. Chloroquine and less commonly Hydroxychloroquine are associated with this type of maculopathy when given for long periods of type as in rheumatoid arthritis.
Dose of chloroquine causing maculopathy: 250 mg/day for 3 years, cumulative dose of 300 gmQ

Other causes of **Bull's eye maculopathy**
• Cone dystrophy
• Cone-rod dystrophy
• Stargardt's disease
• Atypical retinitis pigmentosa (Bardet-Biedl syndrome)
• Benign concentric annular macular dystrophy
• Fenestrated sheen macular dystrophy
• Central areolar choroidal atrophy
• Lipofuscinosis
• Chronic macular hole, ARMD

95. **a. Vitreous humour**
96. **a. The sample should be stored at 4 degrees Celsius**
97. **d. Ascorbate**
98. **d. CRVO**

Chapter 6

Uveal Tract

Uvea is the middle vascular coat of the eyeball comprising of the following parts:

- Iris
- Ciliary body: It is divided into pars plicata and pars plana
- Choroid

UVEITIS

Uveitis refers to the inflammation of the uveal tract.

Anatomical Classification

Depending upon the anatomical part of the uveal tract which is involved, uveitis can be classified into:

- Anterior uveitis: It involves the iris and pars plicata of the ciliary body, hence known as **iridocyclitis**[Q]
- Intermediate uveitis: It involves the pars plana of the ciliary body and the surrounding vitreous, hence known as **pars planitis**[Q]
- Posterior uveitis: It involves mainly the choroid with associated inflammation of the overlying retina and vitreous, hence called **chorioretinitis.**

Pathological Classification

- Non-granulomatous
- **Granulomatous**- Important causes of granulomatous uveitis are:
  - **Tuberculosis**[Q]
  - **Leprosy**[Q]
  - **Syphilis**[Q]
  - Herpetic uveitis
  - Brucellosis
  - Histoplasmosis, Cryptococcosis
  - **Sarcoidosis**[Q]
  - **Vogt-Koyanagi-Harada's disease**[Q]
  - **Sympathetic Ophthalmitis**[Q]
  - **Lens induced uveitis**[Q]
  - **Foreign body uveitis**[Q]

Clinical Classification

- Acute: Sudden onset uveitis with limited duration
- Recurrent: Repeated episodes of uveitis separated by periods of inactivity lasting at least 3 months
- Chronic: Persistent inflammation which promptly recurs within 3 months when treatment is stopped.

CLINICAL FEATURES

Anterior Uveitis

Clinical features of acute anterior uveitis are:

- Pain, photophobia, redness associated with decrease in vision
- Circumciliary congestion
- **Keratic precipitates (KPs)**[Q]: These are proteinaceous deposits on the back of the cornea. They are usually present in a triangular area in the lower part of the cornea, known as **Arlt's triangle**[Q]. They are divided into:
  - **Mutton fat KPs:** Large KPs seen in **granulomatous uveitis**[Q]. They are composed of **epithelioid cells and macrophages**[Q]
  - Medium and small sized KPs: Seen in non-granulomatous uveitis. They are composed of lymphocytes
  - Pigmented KPs: Seen in old uveitis
  - Red KPs: Seen in haemorrhagic uveitis
- Anterior chamber
  - Aqueous cells: They are inflammatory cells in the aqueous humour
  - Aqueous flare: This is due to the disruption of the blood-aqueous barrier and increase in the protein content of the aqueous humour. The flare becomes clinically detectable due to a phenomenon known as **Tyndall effect**[Q]
- Pupillary signs
  - Constriction of the pupil due to ciliary spasm
  - **Posterior synechiae:** Segmental adhesions between the iris and the anterior capsule of the lens occurs due to prolonged inflammation. Dilatation of this pupil gives a distorted appearance known as **festooned pupil**[Q]
  - **Ring synechiae:** When synechiae are formed around 360 degrees of the pupillary margin, it is called ring synechiae or **seclusio pupillae**[Q]. When the pupillary area is covered by an inflammatory membrane it is known as **occlusio pupillae**[Q]
- Iris nodules: Seen in **granulomatous inflammation**
  - **Koeppe's nodules**[Q]: Seen at the pupillary border
  - **Bussaca's nodules**[Q]: Seen at the base of the iris.

Intermediate Uveitis

- Patient mainly complains of floaters
- Anterior segment: May be quiet or associated with mild inflammation

- Posterior segment: Whitish vitreous exudates are seen surrounding the pars plana, more prominent inferiorly. These exudates are known are **snow-ball opacities.** They coalesce to form an inflammatory plaque around the pars plana which is referred to as **snow-banking**[Q].

Posterior Uveitis

- Decrease in vision is the common complaint. Pain, photophobia and redness are generally absent
- Anterior chamber is generally quiet
- Posterior chamber: It shows the following features:
  - Vitritis
  - Choroiditis: It may be unifocal or multifocal
  - Periphlebitis
  - Neuroretinitis

Complications

- **Complicated cataract:** Most common complication of **recurrent anterior uveitis**[Q]
- Secondary glaucoma
  - Pupillary block glaucoma: Formation of seclusio or occlusio pupillae does not allow the aqueous to move to the anterior chamber. As the result the iris bulges forward forming **iris bombe** leading to secondary angle closure
  - Open angle glaucoma: This occurs due to blockage of the trabecular meshwork by the inflammatory cells
- **Cystoid macular oedema: It is mainly seen in intermediate uveitis**[Q]
- **Exudative retinal detachment: It is seen in posterior uveitis**[Q]
- Band shaped keratopathy: It is seen in chronic cases
- Iris atrophy, rubeosis: It is seen in chronic cases
- Phthisis bulbi: It is seen in chronic cases

Treatment

- Anterior uveitis
  - **Topical steroids: Drug of choice**[Q]
  - Periocular steroid in the form of sub-Tenon injection is given in severe or unresponsive cases
  - Mydriatric-cycloplegic like **atropine:** This relieves the **ciliary spasm**[Q] and **prevents the formation of posterior synechiae**[Q]
- Intermediate uveitis
  - Periocular steroid
  - Systemic steroids
- Posterior uveitis
  - Systemic steroids

- Panuveitis
  - Systemic steroids
  - Cytotoxic agents and Immunomodulators: These are given in cases like **Sarcoidosis, Sympathetic Ophthalmitis, VKH**[Q].

CAUSES OF UVEITIS

Different causes of uveitis affect the eye in different ways. The basic presentation is that of uveitis but with certain special characteristics for each. We have to memorize these salient features for MCQ examinations.

JUVENILE RHEUMATOID ARTHRITIS

- It has three varieties:
  - Pauciarticular: **Pauciarticular Seronegative**[Q] variety has the **most common** association with uveitis
  - Polyarticular
  - Systemic: **Systemic variety**[Q] or **Still's disease** has **least common** association with uveitis
- Presents as **bilateral non-granulomatous anterior uveitis**
- This anterior uveitis is not associated with circumcorneal congestion, hence also known as **white eye uveitis**[Q]
- Associated with **complicated cataract**, extensive posterior synechiae and band shaped keratopathy
- **IOL insertion is generally avoided**[Q] after cataract surgery in these cases due to possibility of severe inflammatory reaction

HLA B-27 ASSOCIATED UVEITIS

- It is associated with diseases like **Ankylosing spondylitis**[Q], Reiter's syndrome and Psoriatic arthritis
- **Acute severe recurrent non-granulomatous anterior uveitis**[Q] is seen.

FUCHS' HETEROCHROMIC IRIDOCYCLITIS

- It is a distinctive entity seen in young females
- Associated with **unilateral non-granulomatous anterior uveitis**
- **Diffusely distributed stellate white keratic precipitates**[Q]
- **Absence of posterior synechiae**[Q]
- **Spontaneous hyphaema** due to rupture of small filiform vessels seen in the angle. This is called **Amsler's sign**[Q]
- Iris heterochromia is seen

POSSNER SCHLOSSMAN SYNDROME

- It is a distinctive entity seen in young males
- **Mild non-granulomatous anterior uveitis is associated with severe rise of IOP (about 50–60 mm Hg)**Q
- It is also called **glaucomatocyclitic crisis**Q.

BEHCET'S DISEASE

- It is associated with **HLA B-5**Q
- **Bilateral non-granulomatous anterior and posterior uveitis**
- **Associated with transient hypopyon**Q
- Features of posterior segment involvement like vitritis, periphlebitis, retinitis may be present.

TOXOPLASMOSIS

- Toxoplasma is an obligate intracellular parasite where cat is the definitive host and human being is the intermediate host
- Infection occurs usually in the foetal life
- **Unilateral non-granulomatous anterior and posterior uveitis is seen**
- In the posterior segment, there is severe vitritis associated with **a focal necrotising retinochoroiditis**Q**, usually at the macula**. This is called **headlight in fog appearance**Q
- Treatment is Clindamycin/Cotrimoxazole/Pyrimethamine along with steroids.

SARCOIDOSIS

- **Bilateral chronic granulomatous panuveitis is seen. Sometimes it may present as only intermediate uveitis**
- Vitritis with **snow ball opacities**
- Periphlebitis: Sheathing of vessels is seen due to periphlebitis. This is known as **candle wax dripping**Q
- Posterior segment nodules known as **Lander's sign**Q may be seen
- Choroidal granuloma may be seen
- Optic nerve head granuloma may also be seen.

VOGT-KOYANAGI-HARADA'S DISEASE

- It is an **oculo-neuro-cutaneous** condition. It is associated with **HLA DR4**
- The cutaneous features are:
  - Alopecia
  - Vitiligo
  - Poliosis
- The neurological features are:
  - Meningismus

- Encephalopathy
- Tinnitus

- The ocular features are:
  - **Bilateral granulomatous panuveitis**[Q]
  - Bilateral multiple areas of exudative retinal detachment
  - Perilimbal vitiligo, known as **Suguira's sign**
  - In chronic cases, the fundus has a dull yellow colour known as **sunset glow** fundus.

SYMPATHETIC OPHTHALMITIS

- It is a distinctive condition where **severe penetrating trauma**[Q] to the area of the ciliary **body**[Q] **in one eye** leads to **bilateral granulomatous panuveitis**
- The eye which has suffered the trauma is called the **exciting eye** and the other eye is called as the **sympathising eye**
- **The first symptom of sympathising ophthalmitis is blurring of near vision**[Q] **due to loss of accommodation**
- **The first sign is retrolental flare**[Q]
- Anterior and intermediate uveitis are seen
- In the posterior segment, yellowish white subretinal granulomas called as **Dalen Fuchs' nodules**[Q] are seen
- To prevent sympathetic ophthalmia, severely traumatised eyes with no visual potential may be **enucleated.**

HIV ASSOCIATED UVEITIS

- HIV associated uveitis is a **bilateral non-granulomatous panuveitis**[Q].
- Anterior and intermediate uveitis may be present but involvement is mainly posterior
- In the posterior segment, the most commonly seen feature is **HIV microangiopathy**[Q] which presents as **cotton wool spots**[Q]
- Treatment is anti-retroviral therapy associated with steroids
- HIV is associated with certain opportunistic infections which also give rise to various types of uveitis. These are:
  - **Cytomegalovirus retinitis (CMV)**: This mainly causes **posterior uveitis.** It is characterised by severe retinal vasculitis, haemorrhages and opacification. This is called **sauce and cheese retinopathy**[Q] or **pizza-pie retinopathy.** The vasculitis extends along the blood vessels relentlessly like a fire to reach the optic nerve head, hence also called **brushfire retinopathy.** The treatment is intravenous Gancyclovir/Cidofovir/Foscarnet. Intravitreal Gancyclovir and Cidofovir are also given.
  - **Pneumocystitis carinii associated chorioretinitis**
  - **Progressive outer retinal necrosis (PORN):** This is a type of **necrotising retinochoroiditis** seen in HIV patients due to fulminant infection by **varicella zoster virus.** It initially involves only the outer retinal layers but rapidly progresses to full-thickness involvement and necrosis. Treatment is intravenous ganciclovir

- **Cryptococcus associated chorioretinitis**
- **Tuberculosis, leprosy, syphilis** associated **panuveitis** is also seen in HIV patients.

HERPES SIMPLEX ASSOCIATED UVEITIS

- **Granulomatous anterior uveitis**, usually associated with high IOP
- It is associated with **extensive iris atrophy**[Q]
- Posterior uveitis is also seen
- **Acute retinal necrosis (ARN):** It is a typical **necrotising retinitis** seen in young male patients due to HSV infection. It is a retinal vasculitis with multiple focii of chorioretinitis which rapidly progresses to necrosis. The posterior pole is usually spared. Treatment is intravenous acyclovir along with steroids.

WHITE DOT SYNDROMES

- This is a broad term which encompasses different entities which are associated with **posterior uveitis** giving rise to multiple white dots in the chorioretina. These are actually **microgranulomas** composed of lymphocytes and macrophages. The important causes of white dot syndromes are:
  - Acute posterior multifocal placoid pigment epitheliopathy
  - Serpiginous choroidopathy
  - Birdshot retinochoroidopathy
  - Punctuate inner choroidopathy
  - Progressive subretinal fibrosis and uveitis
  - Presumed ocular histoplasmosis syndrome (POHS)
  - Multiple evanescent white dot syndrome (MEWDS)

CAUSES OF ANTERIOR UVEITIS

- Juvenile rheumatoid arthritis[Q]
- HLA B27 associated uveitis[Q]
- Fuchs' heterochromic iridocyclitis[Q]
- Possner Schlossman syndrome[Q]
- Inflammatory bowel disease
- Herpes simplex
- Lens induced uveitis
- Behcet's disease

CAUSES OF INTERMEDIATE UVEITIS

- Sarcoidosis
- Toxocariasis
- Candidiasis
- Multiple sclerosis

CAUSES OF POSTERIOR UVEITIS

- Toxoplasmosis
- Herpes simplex associated **acute retinal necrosis**
- Behcet's disease
- HIV associated uveitis
- CMV retinitis
- White dot syndromes

CAUSES OF PANUVEITIS

- Sarcoidosis[Q]
- Vogt-Koyanagi-Harada's disease[Q]
- Sympathetic ophthalmitis[Q]
- Tuberculosis[Q]
- Leprosy[Q]
- Syphilis[Q]
- Herpes zoster[Q]

MASQUERADE SYNDROME

These are conditions which mimic uveitis. They are:

- Intraocular malignancies like retinoblastoma, choroidal melanoma, choroidal metastasis
- Leukaemia
- Lymphoma
- Amyloidosis

- **Ophthalmia nodosum**: Granulomatous uveitis due to caterpillar hair[Q].

QUESTIONS

1. **True about ciliary body is:** *(PGI 2013)*
   a. Located about 10 mm from the corneoscleral junction
   b. Consists of pars plana and pars plicata
   c. Contraction of the ciliary body helps in accommodation
   d. Secretes aqueous humour
   e. Derives its blood supply from the short posterior ciliary arteries
2. **Granulomatous uveitis is seen in:** *(PGI)*
   a. Vogt-Koyanagi-Harada's disease
   b. Fuchs' heterochromic iridocyclitis
   c. Behcet's disease
   d. Sarcoidosis
   e. Psoriatic arthritis
3. **Koeppe's and Busacca's nodules are characteristic of:** *(JIPMER)*
   a. Granulomatous uveitis
   b. Non-granulomatous uveitis
   c. Recurrent uveitis
   d. Chronic uveitis
4. **Mutton fat keratic precipitates are seen in:** *(APPG)*
   a. Non-granulomatous uveitis
   b. Granulomatous uveitis
   c. Posterior uveitis
   d. Intermediate uveitis
5. **Mutton fat keratic precipitates are not seen in:** *(Manipal)*
   a. Tuberculosis
   b. Fuchs' heterochromic iridocyclitis
   c. Sarcoidosis
   d. Fungal infections
6. **Anterior uveitis is characterized by all *except*:** *(AIIMS)*
   a. Aqueous flare
   b. Shallow anterior chamber
   c. Circumcorneal congestion
   d. Miosis
7. **The type of synechiae in iris bombe is:** *(AIPG)*
   a. Ring b. Total
   c. Filiform d. Goniform
8. **What is the most common complication of recurrent anterior uveitis?** *(AIIMS)*
   a. Staphyloma
   b. Cataract
   c. Glaucoma
   d. Vitreous haemorrhage
9. **Drug of choice for acute iridocyclitis:** *(DPG)*
   a. Steroids b. Acetazolamide
   c. Atropine d. Antibodies
10. **Primary objective of use of atropine in anterior uveitis is:** *(AIIMS)*
    a. Relaxation of ciliary muscle
    b. Increase blood flow
    c. Prevent posterior synechiae formation
    d. Increase supply of antibodies
11. **Which drug should not be used in raised IOP with uveitis:** *(PGI)*
    a. Timolol b. Pilocarpine
    c. Atropine d. Acetazolamide
12. **Snow banking is seen in:** *(PGI)*
    a. Pars planitis b. Endophthalmitis
    c. Coats' disease d. Eales' disease
13. **In a patient of anterior uveitis, decrease in vision due to posterior segment involvement may be because of:** *(AIIMS)*
    a. Vitreous floaters
    b. Inflammatory disc oedema
    c. Exudative retinal detachment
    d. Cystoid macular oedema
14. **Iridocyclitis is a feature of:** *(AIIMS)*
    a. Juvenile rheumatoid arthritis with systemic features
    b. Seropositive pauciarticular JRA
    c. Seronegative pauciarticular JRA
    d. Seropositive polyarticular JRA

15. A 25-year-old man has pain, redness and mild diminution of vision in one eye for the past 3 days. There is also history of low backache for the past one year. On examination there is circumcorneal congestion, few keratic precipitates on the corneal endothelium, 2+ cells in the anterior chamber. Intraocular pressure is within normal limits. The patient is likely to be suffering from: *(AIIMS)*
    a. Acute angle closure glaucoma
    b. HLA B-27 associated anterior uveitis
    c. JRA associated anterior uveitis
    d. Herpetic keratitis
16. All of the following are true regarding anterior uveitis in ankylosing spondylitis *except*: *(SGPGI)*
    a. More common in female
    b. Recurrent attacks are seen
    c. Fibrinous reaction in anterior chamber is seen
    d. Narrowing of joint spaces in sacroiliac joints is a feature
17. HLA B5 is associated with: *(APPG 2014)*
    a. Vogt-Koyanagi-Harada's disease
    b. Possner Schlossman syndrome
    c. Behcet's disease
    d. Reiter's syndrome
18. In Fuchs' heterochromic iridocyclitis, true is: *(Manipal)*
    a. 60% cases develop glaucoma
    b. Show a good response to steroids
    c. Lens implantation following cataract surgery is contraindicated
    d. Hyphaema during cataract surgery is seen
19. A young patient presents with gradual blurring of vision in the left eye. Slit lamp reveals fine stellate keratic precipitates, aqueous flare and posterior subcapsular cataract. No posterior synechiae are seen. The most likely diagnosis is:
    a. Intermediate uveitis
    b. Heerfordt's disease
    c. Heterochromic iridocyclitis of F...
    d. Subacute iridocyclitis
20. Skin depigmentation, bilateral uve... and tinnitus is a feature of: *(AIIM...*
    a. Waardenberg syndrome
    b. Vogt-Koyanagi-Harada disease
    c. Alport's syndrome
    d. Werner's syndrome
21. A 32-year-old male presents with bl... ring of vision in the right eye. On exa... nation there is mid iritis, severe vitr... and a focal necrotic lesion on the mac... The most likely diagnosis is: *(AIIM...*
    a. Multiple evanescent white dot s... drome
    b. Ocular toxoplasmosis
    c. Multifocal choroiditis
    d. Ocular sarcoidosis
22. Most rapid and accurate method to dia... nose CMV retinitis: *(AIIMS 20...*
    a. Virus isolation in intraocular fluid
    b. Viral antigen detection in vitreous
    c. Viral nucleic acid detection in intra... ocular fluid
    d. Viral antibody in blood by ELISA
23. Ocular manifestations of HIV are all *except*: *(PGI...*
    a. Predisposition to bacterial, viral an... fungal infections
    b. Kaposi sarcoma
    c. CMV retinitis
    d. Cotton wool spots
    e. Intraocular lymphoma
24. Sauce and cheese retinopathy is seen in: *(DNB...*
    a. Toxoplasmosis b. CMV retinitis
    c. Tuberculosis d. Sarcoidosis
25. Headlight in fog appearance is characteristic of: *(DNB...*
    a. CMV retinitis b. Tuberculosis
    c. Toxoplasmosis d. Sarcoidosis
26. In which of these conditions is intraocular pressure very high with minimum inflammation? *(Maharashtra...*

a. Acute iridocyclitis
b. Glaucomatocyclitic crisis
c. Acute angle closure glaucoma
d. Hypertensive uveitis

27. **Sympathetic ophthalmitis is:** *(AIIMS)*
a. U/L suppurative uveitis
b. B/L suppurative uveitis
c. U/L non-suppurative uveitis
d. B/L non-suppurative uveitis

28. **Earliest symptom of sympathetic ophthalmitis is:** *(AIIMS)*
a. Photophobia
b. Pain
c. Loss of near vision
d. Loss of distant vision

29. **A 20-year-old man complains of difficulty in reading newspaper in the right eye 4 weeks after gunshot injury in the left eye. The likely diagnosis is:** *(AIPG)*
a. Macular oedema
b. Sympathetic ophthalmitis
c. Optic nerve avulsion
d. Delayed vitreous haemorrhage

30. **First sign in sympathetic ophthalmitis :** *(AIIMS)*
a. Aqueous flare
b. Keratic precipitates
c. Retrolental flare
d. Constriction of the pupil

31. **Dalen Fuchs' nodules are seen in:** *(AIIMS/PGI)*
a. Sympathetic ophthalmitis
b. Myopia
c. Spring catarrh
d. Retinal detachment

32. **Which of the following is incorrect regarding phthisis bulbi?** *(AIPG)*
a. The intraocular pressure is increased
b. Calcification of the globe is common
c. Sclera is thickened
d. Size of the globe is reduced

33. **Uveal effusion syndrome is associated with all *except*:** *(AIPG)*
a. Myopia
b. Ciliochoroidal detachment
c. Structural defects in the sclera
d. Nanophthalmos

ANSWERS AND EXPLANATIONS

1. **b. Consists of pars plicata and pars plana, c. Contraction of ciliary body helps in accommodation, d. Secretes aqueous humour**
   The ciliary body begins about 1.5 mm posterior to the limbus in the horizontal meridian and 2 mm posterior to the limbus in the vertical meridian. It ends about 7-8 mm posterior to the limbus (7 mm superiorly, nasally, inferiorly and 7.5-8 mm temporally). It is supplied by the long posterior ciliary arteries and the anterior ciliary arteries which form the major arterial circle at the root of the iris.
2. **a. Vogt-Koyanagi-Harada's disease, d. Sarcoidosis**
3. **a. Granulomatous uveitis**
4. **b. Granulomatous uveitis**
5. **b. Fuchs' heterochromic iridocyclitis**
6. **b. Shallow anterior chamber**
7. **a. Ring**
8. **b. Cataract**
9. **a. Steroids**
10. **a. Relaxation of the ciliary muscle**
    Atropine is the adjuvant drug that must be given in all cases of uveitis. The main role of atropine is to relieve the ciliary spasm which makes the patient comfortable.
11. **b. Pilocarpine**
    Pilocarpine causes miosis and ciliary spasm. It also increases intraocular inflammation. Hence, it is not preferred in inflammatory glaucoma.
12. **a. Pars planitis**
13. **d. Cystoid macular oedema**
    Cystoid macular oedema is more common with intermediate uveitis but may also occur with anterior uveitis.
14. **c. Seronegative pauciarticular JRA**
15. **b. HLA-B27 associated anterior uveitis**
16. **a. More common in female**
17. **c. Behcet's disease**
18. **d. Hyphaema during cataract surgery is seen**
19. **c. Heterochromic iridocyclitis of Fuch**
20. **b. Vogt-Koyanagi-Harada's disease**
21. **b. Ocular toxoplasmosis**
    This is a classical description of ocular toxoplasmosis or headlight in fog appearance
22. **c. Viral nucleic acid detection in intraocular fluid**
    PCR for detection of viral nucleic acid in intraocular fluid is the best investigation for CMV retinitis
23. **e. Intraocular lymphoma**
    HIV may be associated with intraorbital not intraocular lymphoma.
24. **b. CMV retinitis**

25. **c. Toxoplasmosis**
26. **b. Glaucomatocyclitic crisis.** (explained in the chapter on glaucoma)
27. **d. B/L non-suppurative uveitis**
28. **c. Loss of near vision**
29. **b. Sympathetic ophthalmitis**
30. **c. Retrolental flare**
31. **a. Sympathetic ophthalmitis**
32. **a. The intraocular pressure is increased**

Pthisis bulbi occurs as the end stage of severe ocular disease. It is described as a shrunken and hypotonous globe with atrophic and disorganised ocular tissue.

Causes are chronic uveitis, perforating injuries, panophthalmitis, multiple ocular surgeries etc.

Features are:

- No visual potential
- Globe is small and shrunken with disorganised ocular tissue so that ocular structures cannot be identified clearly
- Ciliary body shutdown leading to hypotony
- Thickening of the sclera
- It may be associated with intraocular calcification

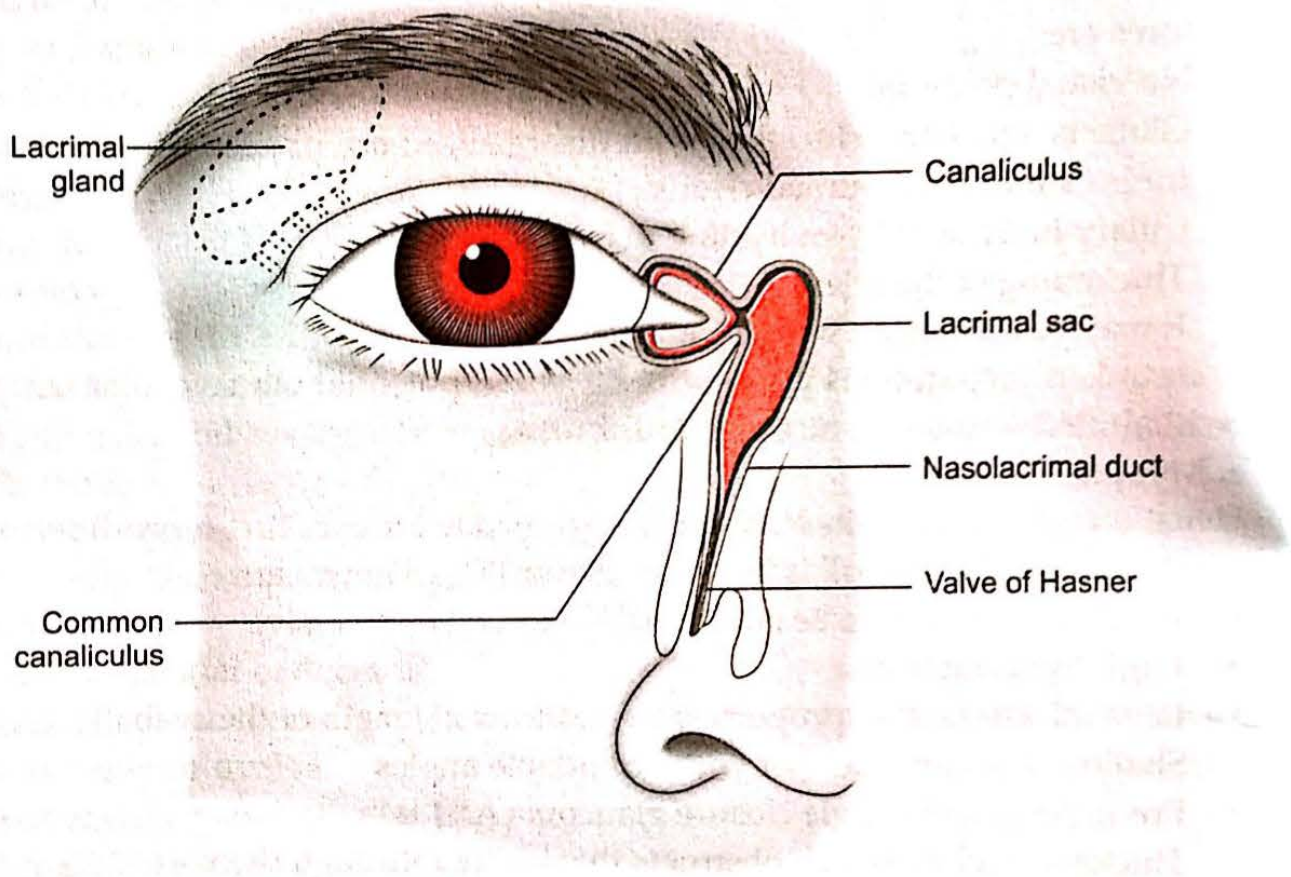
A shrunken, hypotonous globe with nil visual potential where ocular structures can be identified is called as **atrophic bulbi.**

33. **a. Myopia**

Uveal effusion is seen in Nanophthalmos or Dwarf eye. It is a condition where the axial length of the eyeball is less than 19 mm. The features are:

- Familial variety may be autosomal dominant or recessive
- **High hypermetropia**
- Lens thickness is disproportionate to the axial length of the eyeball
- Shallow anterior chamber with occludable angles
- Predisposition to angle closure glaucoma
- Thickened sclera which obstructs the outflow through the vortex veins leading to uveal effusion
- Ciliochoroidal detachment
- Exudative retinal detachment

LACRIMAL SYSTEM



Anatomy of the lacrimal system

Anatomy

The lacrimal system consists of the lacrimal glands and the lacrimal drainage system.

- **Main lacrimal gland:** It lies in the **superotemporal part of the orbit**Q. It has two parts which lie above and below the LPS muscle. It is mainly responsible for **reflex tear secretion**Q. The ducts from this gland end in the superior and inferior fornix
- **Accessory lacrimal glands**: They are the glands of **Krause and Wolfring**Q which are concerned with the **basal tear secretion**Q. They are located in the plica, inferior fornix and infra-orbital region
- **Lacrimal drainage system:** The components are:
  - **Puncta**: There are two puncta, upper and lower on the papilla lacrimalis in medial part of the lid

- **Canaliculi**: From the puncta arises the canaliculus. Each canaliculus has a short **vertical portion (2 mm)** and a long **horizontal portion (8 mm)**.The upper and lower canaliculi join together to form the common canaliculus which leads to the lacrimal sac. The opening is guarded by the **valve of Rosenmuller**[Q]
- **Lacrimal sac**: It lies in the lacrimal fossa between the anterior and posterior lacrimal crests in medial wall of orbit formed by lacrimal bone and frontal process of maxilla.
- **Nasolacrimal duct (NLD)**: The NLD connects the lacrimal sac to the **inferior meatus of the nose**[Q]. It is about 18 mm long of which the upper 12 mm is osseous and the lower 6 mm is membranous. It passes **downwards, backwards and laterally**[Q]. Its opening at the nose is guarded by the **valve of Hasner**[Q].

Tear Film

The tear film is made of three layers namely:

- Outer lipid layer: This is an oily layer secreted by the **meibomian glands**[Q] of the eyelid. Its function is to **prevent evaporation**[Q] of the aqueous layer
- Middle aqueous layer: This is the main layer of the tear film produced by the main and accessory lacrimal glands
- Inner mucin layer: This layer is produced by the **goblet cells**[Q] of the conjunctiva. This layer converts the hydrophobic corneal surface to a **hydrophilic** surface so that the aqueous layer can spread over it.

Dry Eye

Deficiency of the tear film is referred to as dry eye. It may be of three types depending upon the layer of the tear film which is deficient:

- **Evaporative dry eye:** This is caused due to deficiency of the lipid layer or conditions causing excessive evaporation. Possible causes are:
  - Meibomian gland disease, Posterior blepharitis[Q]
  - Lagophthalmos[Q]
  - Proptosis[Q]
  - Air-conditioned room
- **Aqueous layer deficiency dry eye (Keratoconjunctivitis sicca**[Q]**)**: The possible causes are:
  - Sjogren's syndrome
  - Collagen vascular diseases like rheumatoid arthritis, SLE etc
- **Mucin layer deficiency dry eye:** This is due to parenchymatous damage to the conjunctiva in conditions like
  - Chemical and thermal burns[Q]
  - Stevens-Johnson syndrome[Q]
  - Ocular cicatricial pemphigoid
  - Herpes zoster[Q]
  - Trachoma[Q]

Symptoms

Irritation, burning and foreign body sensation

Signs

- There is a decrease in the height of the marginal tear strip with strands of mucous and debris on the corneal surface
- Punctate epithelial erosions may be seen.

Tests

- Tear film break up time (TBUT): Normal value is > 10 seconds
- **Schirmer's test**: Normal 10–25 mm, Borderline 5–10 mm, Impaired < 5 mm
- Vital dye staining
  - Rose Bengal: It has an affinity for devitalized epithelial cells and mucus
  - Fluorescein: It stains the punctuate epithelial erosions
- Conjunctival impression cytology
- Lysozyme assay
- Tear osmolarity assay
- Conjunctival biopsy

Treatment

- Tear conservation by decreasing room temperature, use of humidifiers
- Tear substitutes: The common tear substitutes are **polyvinyl alcohol, carboxymethyl cellulose, hydroxypropyl methyl cellulose, and hyaluronic acid**Q
- Mucolytics like acetylcysteine which disperse mucus filaments and plaque
- **Steroid and Cyclosporine** eye drops for keratoconjunctivitis sicca. Systemic steroids, immunosuppressants may also be used in severe cases
- Reduction of tear drainage by **punctal occlusion**
- **Tarsorrhaphy**Q is done in cases of lagophthalmos and proptosis
- **Mucous membrane grafting and amniotic membrane grafting**Q are options for parenchymatous conjunctival damage.

Epiphora

Epiphora means watering. The causes of epiphora may be classified as:

- Hyperlacrimation: Conditions which increase reflex tearing like conjunctivitis, keratitis, foreign body etc.
- Inadequate drainage: This may be due **anatomical obstruction** in the drainage pathway or **functional obstruction due to lacrimal pump failure**Q.

Tests for lacrimal drainage

- Syringing and probing
- Jones' dye test
- Fluorescein dye disappearance test

- **Dacryocystography (DCG): Confirmatory test for anatomical obstruction**Q
- Radionucleotide testing (**Dacryoscintigraphy): Confirmatory test for functional obstruction**Q

Congenital Dacryocystitis

- The NLD is a solid tube during its development. It becomes hollow and canalized by the time of birth
- Failure of canalisation of the lower end of NLD leads to congenital dacryocystitis. **The site of obstruction is the valve of Hasner** Q.
- The child is brought with complains of watering and discharge
- Ocular examination is normal. **Regurgitation of mucoid fluid is seen on pressing over the lacrimal sac, suggestive of obstruction in the NLD**
- The treatment options are:
  - **Hydrostatic massage (Crigler's massage)**Q: When done properly, NLD obstruction is relieved in more than 90% infants
  - Syringing and probing: It is usually done if massage is ineffective after **1 year of age**Q
  - Balloon dacryocystoplasty Q
  - Lacrimal intubation
  - **Dacryocystorhinostomy(DCR): It is usually done after 3 years of age**Q.

Acute Dacryocystitis

- This is an acute suppurative inflammation of the lacrimal sac. The cause is obstruction in the NLD leading to stasis of secretion in the sac and secondary infection
- The patient presents with sudden onset pain and swelling in the area of the sac. It may be associated with systemic features like fever
- Complications
  - Lacrimal abscess
  - Lacrimal fistula
  - **Orbital cellulitis**Q
- Treatment
  - Systemic antibiotics and anti-inflammatory drugs
  - Hot compression and antibiotics are given locally
  - **DCR is done 4–6 weeks after the resolution of the acute episode**Q.

Chronic Dacryocystitis

- This is a low-grade inflammation in the sac due to stasis of secretion as a result of NLD obstruction
- The patient presents with complains of watering and discharge
- **Regurgitation test is positive**Q
- **Syringing test shows mucoid regurgitation from other punctum suggestive of NLD obstruction**Q

- Complications
  - Mucocoele
  - Encysted mucocoele
  - Pyocoele
  - Acute on chronic dacryocystitis
  - Fibrosed sac
- Treatment is DCR

Dacryocystorhinostomy (DCR)

Anastomosis made between **lacrimal sac and middle meatus of nose**[Q]. This bypasses the obstruction of NLD.

Indications

- Chronic dacryocystitis/NLD block
- Atonic lacrimal sac
- Mucocoele
- Lacryolith
- Congenital dacryocystitis, when other measures have failed.

Contraindications

- Acute dacryocystitis
- Tuberculosis of sac
- Malignancy of sac
- Obstructive lesions in the nose like nasal polyp, deviated nasal septum, atrophic rhinitis
- Obstruction above the sac like canalicular obstruction
- Very old patients.

Dacryocystectomy (DCT)

In this procedure, the lacrimal sac is removed. It is done in old patients where a prolonged surgery like DCR may be difficult.

EYELIDS

Basic Anatomy

The layers of the eyelid from outside inwards are:

- Skin and subcutaneous tissue
- Layer of striated muscle (LPS and orbicularis oculi)
  - The **LPS** which is the **main elevator of the eyelids**[Q] arises from the **sphenoid** at the apex of the orbit. It passes above the superior rectus along the roof of the orbit to

reach the eyelids. It is then converted to a thin aponeurosis. This aponeurosis splits into two layers. **The superficial layer inserts into the skin of the lid forming the lid crease. The deep layer inserts into the tarsal plate**Q.

- The **orbicularis oculi** is responsible for **lid closure.** The palpebral part of the muscle arises from the frontal process of maxilla and lacrimal bone and inserts into the lateral palpebral raphe

- Fibrous tissue: It consists of the **tarsal plate** at the centre and the **orbital septum** in the periphery
- Layer of non-striated muscle (**Muller's muscle**): It arises from the LPS and gets inserted into the tarsus. It is an **accessory lid elevator**Q
- Palpebral conjunctiva

The glands of the eyelid are:

- **Meibomian glands**: They are **modified sebaceous glands**Q. They are located in the tarsal plate but their ducts open at the lid margin. **They secrete the outer lipid layer of the tear film**Q
- **Glands of Zeis**: They are **modified sebaceous glands**Q located at the base of the lash follicle
- **Glands of Moll**: They are **modified sweat glands**Q located at the lid margin between two lash follicles

Inflammation of the eyelids

- **External hordeolum or Stye**Q**:** This is an acute suppurative inflammation of the **gland of Zeis**Q caused mainly by **Staphylococcus aureus**. The patient presents with a painful lid swelling where maximum tenderness is at the base of the involved lash follicle. Treatment is hot compression, local antibiotics, systemic antibiotics and anti-inflammatory drugs. **Epilation may also be done.**
- **Internal hordeolum**Q**:** This is an acute suppurative inflammation of the **Meibomian gland**Q caused mainly by **Staphylococcus aureus**. The patient presents with a painful lid swelling. Treatment is hot compression, local antibiotics, systemic antibiotics and anti-inflammatory drugs.
- **Chalazion:** This is a **chronic lipogranulomatous inflammation**Q of the **Meibomian gland**Q**.** It presents as a painless lid swelling. It is more common on the upper lid as the number of Meibomian glands are more on the upper lid. Treatment is **intralesional steroid injection** followed by **Incision and Curettage (I and C)**Q
- **Seborrhoeic blepharitis:** It is a chronic inflammation of the anterior lid lamina involving the glands of Zeis and Moll associated with seborrhoeic dermatitis or dandruff. Treatment is lid hygiene and local antibiotic-steroid.
- **Staphylococcal anterior blepharitis:** It is a chronic inflammation of the anterior lamina of the lids involving the glands of Zeis and Moll. It is associated with scaling and crusting of the lid margin. The crusts on removal may leave small bleeding ulcers on the lid margin. **Long standing cases may lead to madarosis, poliosis, trichiasis and thickening of the lid margins**Q. Treatment is lid hygiene and local antibiotic steroid.

Ptosis

Drooping of the eyelid is referred to as ptosis. The causes of ptosis are divided into:

- Neurogenic
  - Third nerve palsy
  - Horner's syndrome
  - **Marcus-Gunn jaw-winking syndrome**[Q]: This is a condition where aberrant communication between the **third nerve and fifth nerve**[Q] leads to synchronous movement of the lid with movement of the jaw.
- Myogenic
  - Simple congenital ptosis: Due to dystrophic LPS muscle
  - **Blepharophimosis syndrome**: This is a congenital condition associated with bilateral **ptosis, telecanthus, epicanthus inversus and lateral ectropion**[Q]
  - Ocular myopathies
  - Myasthenia gravis
- Aponeurotic: This is involutional ptosis due to weakness of LPS aponeurosis
- Mechanical: This is ptosis associated with lid tumours.

Treatment

- **Fasanella-Servat**[Q] operation: Procedure of choice for ptosis due to Horner's syndrome
- **LPS resection**[Q]**:** This may be done through conjunctival route **(Blaskovic's operation)**[Q] or cutaneous route **(Everbuch's operation)**[Q]. It is done in cases of moderate ptosis with good LPS function.
- **Fascia lata sling/ Frontalis sling**[Q]**:** Procedure of choice for severe ptosis with poor LPS function
- Procedure of choice for Marcus-Gunn jaw-winking syndrome: **LPS excision with fascia lata sling**[Q].

Entropion

It is the inward turning of the lid margin leading to rubbing of the eyelashes on the cornea. It is of the following types:

- **Involutional**: It is more common in the lower lid due to thinning of the tarsus, laxity of the canthal tendons, and weakness of the lower lid retractors. The surgeries for correction are:
  - Transverse lid sutures
  - Modified Wheeler's operation
  - Weis' procedure
  - **Lester Jones procedure: For severe, recurrent cases**[Q]
- **Cicatricial**: It is more common on the upper lid due to scarring of the palpebral conjunctiva. The causes are trachoma, herpes, Steven Johnson syndrome, ocular cicatricial pemphigoid, chemical and thermal burns. The surgeries for correction are:
  - Wedge resection of tarso-conjunctiva

  - **Tarsal fracture**[Q]
  - Mucous membrane grafting
- Congenital: It is seen more commonly in the lower lid. It usually resolves spontaneously in 1-2 years
- Acute spastic: It is due to spasm of the orbicularis in essential blepharospasm.

Ectropion

It is the outward turning of the lid margin. It causes epiphora. The different types are:

- **Involutional:** It affects the lower lid and is caused by weakness of orbicularis and laxity of the medial and lateral canthal tendons. The corrective surgeries are:
  - Ziegler's cautery: For medial ectropion
  - Medial conjunctivoplasty: For medial ectropion
  - Lazy-T procedure: for medial ectropion
  - **Modified Kuhnt-Szymanowski procedure**[Q]: For severe cases involving both medial and lateral side of eyelid
- **Paralytic ectropion:** This is due to facial nerve palsy and may lead to exposure keratopathy. The treatment is:
  - Lubricant eye drops and tarsorrhaphy
  - **Medial canthoplasty**

Trichiasis

- Inward turning or misdirection of eyelashes which rub on the surface of the cornea
- Treatment is **epilation(temporary), electrolysis or cryotherapy (permanent)**[Q]

Distichiasis

- Presence of a second row of eyelashes along the opening of the meibomian glands
- Treatment is required only if the lashes disturb the cornea. Options are the same as trichiasis.

Causes of Trichomegaly (increase in the length of eyelashes)
Drug induced-**Phenytoin**[Q], **Topical prostaglandin analogues**[Q], Cyclosporine
Malnutrition
AIDS
Porphyria
Hypothyroidism
Certain rare congenital conditions like Hermanasky-Pudlak syndrome, Cornelia de Lange syndrome, Oliver McFarlane syndrome

Causes of Madarosis (loss of eyelashes)	**Causes of poliosis** (whitening of eyelashes)
Chronic anterior blepharitis[Q]	Chronic anterior blepharitis[Q]
Infiltrating lid tumours	Sympathetic ophthalmitis
Burns	Vitiligo
Radiotherapy and chemotherapy	Vogt-Koyanagi-Harada's disease[Q]
Leprosy[Q]	Waardenberg syndrome
Myxoedema[Q]	Tuberous sclerosis
SLE	Albinism
Generalised alopecia, psoriasis	Marfan's syndrome(rarely)

Causes of lid retraction
Thyroid eye disease[Q]
Contralateral ptosis
Upper lid scarring
Surgical overcorrection of ptosis
Third nerve misdirection
Duane's retraction syndrome
Perinaud's syndrome (Collier's sign)
Infantile hydrocephalus (Setting sun sign)
Uraemia

ORBIT

Anatomy

The orbit has four walls, roof, and floor, medial and lateral. The medial walls are parallel whereas the lateral walls make an angle of 90 with each other. The base of the orbit is at the orbital margin and the apex is at the optic foramen.
Volume of orbit is 30 cc.

Walls of the Orbit

	Roof	**Medial wall**	**Floor**	**Lateral wall**
1.	Orbital plate of frontal bone	Frontal process of Maxilla	Maxilla	Zygomatic bone
2.	Lesser wing of sphenoid[Q]	Lacrimal bone	Zygomatic bone	Greater wing of sphenoid[Q]
3.		Ethmoid	Palatine bone[Q]	
4.		Body of sphenoid[Q]		

- **Floor is the most frequently fractured wall of orbit in trauma**[Q]
- **Medial wall is the weakest wall**[Q]
- **Lateral wall is the strongest wall**[Q].

Optic Canal/Optic Foramen

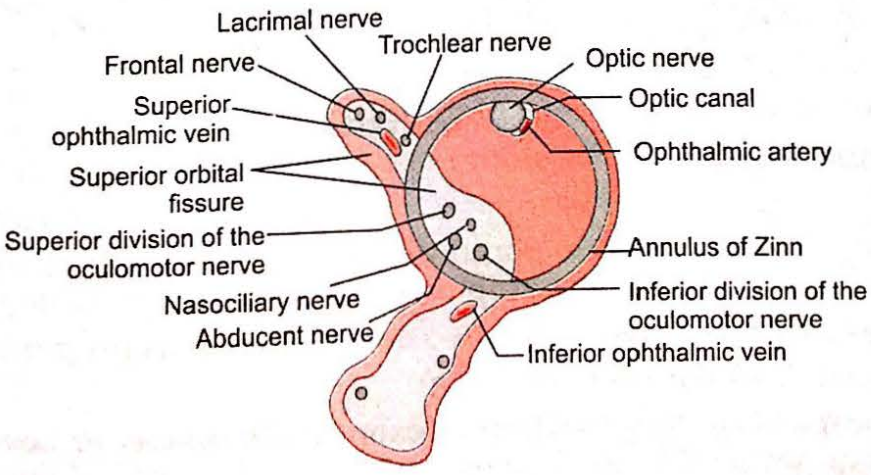
- It is formed by the **lesser wing and body of sphenoid**Q
- It lies between **the roof and medial wall of the orbit**Q
- Vertically oval. Length 6–11 mm, Diameter 4–6 mm
- It transmits
  - **Optic nerve with its coverings**Q
  - **Ophthalmic artery**Q
- Best view for imaging optic canal is **Rheese**Q view.

Superior Orbital Fissure

- It lies between **lesser and greater wing of sphenoid**Q
- It lies between **the roof and lateral wall of the orbit**Q
- It is situated lateral to optic foramen at the orbital apex
- It is comma shaped, approximately 22 mm long.
- It is divided into three parts by the tendinous ring called the **Annulus of Zinn**Q
- The structures passing above the annulus are:
  - **Lacrimal nerve**Q
  - **Frontal nerve**Q
  - **Trochlear nerve**Q
  - Superior ophthalmic vein
- The structures passing through the annulus are:
  - **Two divisions of Oculomotor nerve**Q
  - **Abducens nerve**Q
  - **Nasociliary nerve**Q
- The structures passing below the annulus are:
  - Inferior ophthalmic vein

Spaces of the orbit

- Subperiosteal space: between the orbital wall and the periorbita
- Extraconal space: Between the periorbita and the extraocular muscles
- Intraconal space: Enclosed on all sides by extraocular muscles

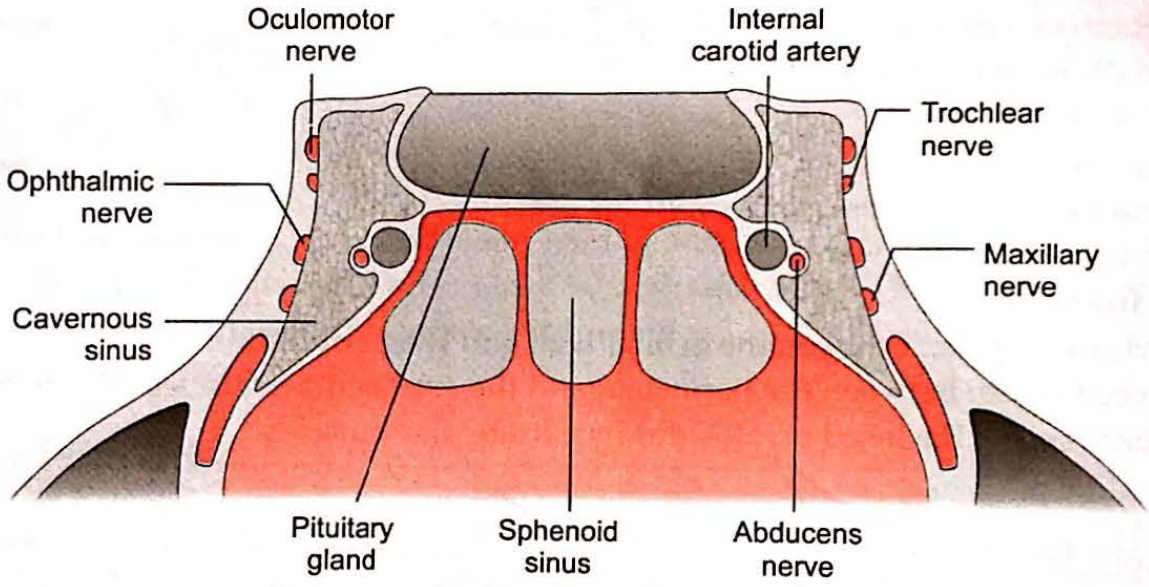


Superior Orbital Fissure

Another structure whose anatomy we must know while studying orbital diseases is the cavernous sinus.

Cavernous Sinus

- It is a venous sinus in the brain. There are two sinuses located on either side of the optic chiasma. Each sinus has valveless communication with the facial veins and so infection in the dangerous area of the face has the potential to reach the cavernous sinus
- It also communicates with the cavernous sinus of the other side, so that involvement is frequently **bilateral**Q
- **Within the cavernous sinus lie the internal carotid artery and the VIth cranial nerve**Q
- In the lateral wall of the sinus lie
  - **IIIrd cranial nerve**Q
  - **IVth cranial nerve**Q
  - **Ophthalmic branch of Trigeminal nerve**Q
  - **Maxillary branch of Trigeminal nerve**Q
- Thus involvement of cavernous sinus presents with
  - Involvement of **IIIrd, IVth, VIth, Ophthalmic and Maxillary nerves**Q
  - **Proptosis**Q (due to engorgement of the orbital veins which communicate with the cavernous sinus)
  - Conjunctival congestion and chemosis.



Cavernous sinus

PROPTOSIS

Protrusion of the eyeball is known as proptosis. An absolute protrusion of > 21 mm or a relative difference of >2 mm between the two eyes is known as proptosis. It may be axial or non-axial (associated with ocular deviation).

Measurement of proptosis is done by **Hertel's exophthalmometer or Luedde's exophthalmometer (in children)**Q.

Causes of axial proptosis	Causes of non-axial proptosis
Thyroid ophthalmopathy[Q]	Lacrimal gland tumour
Orbital cellulitis[Q]	Frontal mucocoele
Carotido-cavernous fistula[Q]	Ethmoidal mucocoele
Cavernous sinus thrombosis	Carcinoma maxillary sinus
Retinoblastoma	Encephalocoele
Optic nerve glioma	Meningomyelocoele
Ophthalmic artery aneurysm	Rhabdomyosarcoma
Optic nerve meningioma	Metastatic neuroblastoma
Cavernous haemangioma[Q]	Chloroma
Pseudotumour	Orbital varix

Causes of pulsatile proptosis	Causes of bilateral proptosis
Carotido-cavernous fistula[Q]	**Thyroid ophthalmopathy**[Q]
Ophthalmic artery aneurysm	Carotido-cavernous fistula[Q]
Encephalocoele[Q]	Cavernous sinus thrombosis[Q]
Meningomyelocoele[Q]	Metastatic neuroblastoma[Q]
	Chloroma[Q]

Causes of intermittent proptosis	Causes of Pseudoproptosis
Encephalocoele	High myopia
Meningomyelocoele	Buphthalmos
Orbital varix	Microphthalmos, phthisis in other eye

- Most common cause of unilateral proptosis in children: **Orbital cellulitis**[Q]
- Most common cause of bilateral proptosis in children: **Chloroma**[Q]
- Most common cause of unilateral proptosis in adults: **Thyroid ophthalmopathy**[Q]
- Most common cause of bilateral proptosis in adults: **Thyroid ophthalmopathy**[Q]

Thyroid Ophthalmopathy

This is also known as Ophthalmic Graves' disease (OGD). It is a bilateral condition but it starts unilaterally. The thyroid status of the patient may be **hyperthyroid, euthyroid or hypothyroid,** but it is usually hyperthyroid. Thyroid ophthalmopathy has two stages.

- Active stage: In this stage, there is infiltration of the orbit with inflammatory cells resulting in orbital soft tissue oedema and proliferation. Thus, the signs in this stage are due to inflammation and oedema.
- Quiescent stage: In this stage, there is resolution of the orbital oedema with secondary fibrosis.

Eyelid Signs

- **Dalrymple's sign**[Q]: Upper lid retraction (generally the 1st sign to appear)
- **Von Graefe's sign**[Q]: Upper lid lag on down gaze

- **Kocher sign**[Q]: Staring & frightened look
- **Rosenbach sign**[Q]: Tremor of closed lids
- **Stellwag sign**[Q]: Infrequent blinking
- **Mobius sign**[Q]: Poor convergence

Soft Tissue Signs

- Lid oedema
- Conjunctival congestion and chemosis
- **Superior limbic keratitis**[Q]

Proptosis

Bilateral proptosis, axial, non-pulsatile[Q]**.**

Dysthyroid Optic Neuropathy

This is due to direct compression of the optic nerve and its blood supply by raised intraorbital pressure.

Restrictive Thyroid Myopathy

Inferior rectus is the first muscle to be involved[Q]**.**

Treatment

- Lubricant eye drops
- Systemic steroids
- Systemic immunosuppressants
- Orbital radiotherapy
- Orbital decompression is done in cases not responding to medical therapy.
  Indications are:
  - Proptosis leading to exposure keratopathy
  - Dysthyroid optic neuropathy.

Carotido-cavernous Fistula

- It is an abnormal communication between **the internal carotid artery and the cavernous sinus**[Q]
- It may be **post-traumatic** or spontaneous (due to hypertension, atherosclerosis, aneurysm)
- Patient presents with **bilateral proptosis**[Q] which begins unilaterally. **Proptosis is axial, pulsatile. It is associated with thrill and bruit which may be abolished by pressing on the ipsilateral carotid**[Q]
- It is associated with **congestion and chemosis**[Q] due to dilatation of orbital veins

- It may also lead to **third, fourth and sixth cranial nerve palsy**[Q], usually starting with the sixth nerve
- Treatment is surgical.

Cavernous Sinus Thrombosis

- **Cavernous sinus thrombosis develops due to uncontrolled infection in the dangerous area of the face like orbital cellulitis, dacryocystitis**[Q]
- Patient presents with high fever and headache.
- There is **bilateral proptosis**[Q] which begins unilaterally. **Proptosis is axial and non-pulsatile**[Q]
- Engorgement of orbital veins leads to **conjunctival congestion and chemosis**
- There may also be associated **palsy of third, fourth and sixth cranial nerves**[Q] starting with the sixth nerve
- Treatment is high dose intravenous antibiotics.

Orbital Cellulitis

- Orbital cellulitis is usually due to spread of infection from nasopharynx, ethmoidal, frontal and maxillary sinuses
- The causative organisms are **Haemophilus influenzae, Streptococcus pneumoniae, Staphylococcus aureus, Streptococcus pyogenes**
- The patient presents with sudden onset pain and swelling of the eye associated with fever.
- **The signs are lid oedema, chemosis and proptosis. Proptosis is axial and non-pulsatile. Limitation of ocular movements is seen**[Q]
- Complications are subperiosteal abscess, cavernous sinus thrombosis, intracranial spread
- Treatment is systemic antibiotics

Blow-out fracture of the Orbit

- This is a **fracture of the orbital floor**[Q] without involving the rim of the orbit
- It is associated with blunt trauma like fist injury and cricket ball injury
- The features are:
  - Peri-orbital ecchymosis
  - Crepitus on palpation (subcutaneous emphysema)
  - Anaesthesia in the area of the cheek due to injury to the infraorbital nerve
  - **Enophthalmos**[Q]
  - **Diplopia due to entrapment of the inferior rectus. Diplopia is present both in upgaze and downgaze and it is called double gaze diplopia**[Q].
- Treatment is orbital floor reconstruction

DESTRUCTIVE OCULAR SURGERIES

There are three main types of destructive ocular surgeries:

- **Enucleation**: It is the removal of the eyeball with a part of the optic nerve. So at the end of the procedure a stump of the optic nerve is left behind. Indications are
  - **Intraocular malignancies like retinoblastoma (absolute indication)**[Q]
  - Severely traumatized eye (to prevent sympathetic ophthalmia in the other eye)
  - Microphthalmos, Phthisis bulbi
  - Painful blind eye

Absolute contraindication: **Panophthalmitis**[Q]

- **Evisceration:** In this procedure, a corneal button is removed. The intraocular contents are removed by an evisceration spoon. A frill of sclera attached with the optic nerve is left at the end of the procedure. Indications are:
  - **Panophthalmitis (absolute indication)** [Q]
  - Microphthalmos, phthisis bulbi
  - Painful blind eye

Absolute contraindication: **Intraocular malignancies**[Q]

- **Exanteration:** This procedure is rarely done now. It involves removal of the globe, orbital soft tissues, periosteum of the orbital wall, part or whole of the lids. Indications are:
  - Orbital malignancies
  - Mucormycosis

QUESTIONS

1. **An elderly female presented with recurrent swelling of the upper eyelid. Histopathological evaluation revealed it to be a chalazion. What would be the histopathological finding?** *(AIIMS 2013)*
   a. Lipogranuloma
   b. Suppurative granuloma
   c. Foreign body granuloma
   d. Xanthogranuloma
2. **Lipogranulomatous inflammation is seen in:** *(AIIMS 2014)*
   a. Fungal infection
   b. Tuberculosis
   c. Chalazion
   d. Viral infection
3. **Which of the following are true regarding chalazion?** *(PGI)*
   a. Mucous cyst
   b. Sebaceous cyst
   c. It is due to staphylococcal infection
   d. Recurrence may imply malignancy
   e. Occlusion of the meibomian gland
4. **Treatment of chalazion:** *(PGI)*
   a. Incision and drainage
   b. Intralesional steroid
   c. Curettage
   d. Pressure bandage
   e. Antibiotics
5. **A recurrent chalazion should be subjected to histopathological examination to rule out the possibility of:** *(AIIMS)*
   a. Squamous cell Ca
   b. Sebaceous cell Ca
   c. Malignant melanoma
   d. Basal cell Ca
6. **Fasanella Servat operation is indicated in:** *(AIPG)*
   a. Congenital ptosis
   b. Traumatic ptosis
   c. Myasthenia gravis
   d. Horner's syndrome
7. **A patient with ptosis presents with retraction of the ptotic eyelid on chewing. This is called:** *(AIPG)*
   a. Marcus Gunn jaw winking syndrome
   b. Third nerve misdirection syndrome
   c. Abducens palsy
   d. Oculomotor palsy
8. **Bilateral ptosis is not seen in:** *(AIPG)*
   a. Marfan's syndrome
   b. Myasthenia gravis
   c. Myotonic dystrophy
   d. Kearne Sayre syndrome
9. **The operation of plication of inferior lid retractors is indicated in:** *(AIPG)*
   a. Senile ectropion
   b. Senile entropion
   c. Cicatricial entropion
   d. Paralytic ectropion
10. **Fibrous attachment of the lid to the eyeball is called:** *(WBPG)*
    a. Symblepharon
    b. Entropion
    c. Ectropion
    d. Anklyoblepharon
11. **Most common malignant tumour of the eyelids:** *(AIIMS)*
    a. Sebaceous cell Ca
    b. Basal cell Ca
    c. Squamous cell Ca
    d. Malignant melanoma
12. **Telecanthus means:** *(AIIMS)*
    a. Widened interpupillary distance
    b. Widened root of nose with normal interpupillary distance
    c. Widely separated medial orbital wall
    d. Widely separated canthi
13. **Structures passing through superior orbital fissure are:** *(PGI)*
    a. IInd cranial nerve
    b. IIIrd cranial nerve
    c. IVth cranial nerve
    d. VIth cranial nerve
    e. Lacrimal nerve

14. **Purulent inflammation of the tissues of the orbit is called:** *(Kerala PG 2015)*
    a. Orbital cellulitis
    b. Endophthalmitis
    c. Panophthalmitis
    d. Dacryocystitis
15. **Most common cause of unilateral proptosis is :** *(AIIMS)*
    a. Thyrotoxicosis
    b. Retinoblastoma
    c. Intraocular haemorrhage
    d. Raised IOP
16. **Commonest cause of bilateral proptosis in children:** *(AIPG)*
    a. Cavernous haemangioma
    b. Chloroma
    c. Fibrous histiocytoma
    d. Rhabdomyosarcoma
17. **Most common cause of bilateral proptosis in children:** *(AIIMS 2013)*
    a. Rhabdomyosarcoma
    b. Lymphoma
    c. Retinoblastoma
    d. Neuroblastoma
18. **Kamla, aged 45 years presents with unilateral mild axial proptosis. There is no redness or pain. Investigation of choice is:** *(AIIMS)*
    a. T3 and T4 to rule out thyrotoxicosis
    b. CT scan to rule out meningioma
    c. Doppler to rule out haemangioma
    d. USG to rule out orbital pseudotumour
19. **Features of thyroid ophthalmopathy are:** *(PGI)*
    a. External ophthalmoplegia
    b. Internal ophthalmoplegia
    c. Proptosis
    d. Enlargement of extraocular muscle
    e. Lid lag
20. **First muscle to be involved in thyroid ophthalmopathy:** *(AIPG)*
    a. Medial rectus
    b. Inferior rectus
    c. Lateral rectus
    d. Superior rectus
21. **Infection from the dangerous area of the face spreads to the cavernous sinus via which of the following veins?** *(COMEDK 2015)*
    a. Maxillary veins
    b. Retromandibular veins
    c. Superficial temporal vein
    d. Ophthalmic veins
22. **Paralysis of IIIrd, IVth and VIth cranial nerves with involvement of ophthalmic division of the Vth cranial nerve localises the lesion to:** *(AIPG)*
    a. Cavernous sinus
    b. Apex of the orbit
    c. Brainstem
    d. Base of the skull
23. **A 19-year-old young girl with previous history of repeated pain over medial canthus and chronic use of nasal decongestants presented with abrupt onset of fever and chills and rigor, diplopia on lateral gaze, moderate proptosis and chemosis. On examination, optic disc is congested. Most likely diagnosis is:** *(AIIMS)*
    a. Cavernous sinus thrombosis
    b. Orbital cellulitis
    c. Acute ethmoidal sinusitis
    d. Orbital apex syndrome
24. **All of the following could result from infection with right cavernous sinus *except*:** *(AIIMS)*
    a. Constricted pupil in response to light
    b. Engorgement of retinal veins seen on ophthalmological examination
    c. Ptosis of right eyelid
    d. Right ophthalmoplegia
25. **A retrobulbar intraconal mass with well defined capsule presenting with slowly progressive proptosis in the 2nd to 4th decade- Most likely diagnosis:** *(AIIMS)*

a. Capillary haemangioma
b. Cavernous haemangioma
c. Lymphangioma
d. Haemangiopericytoma

26. A patient presents with unilateral proptosis which is compressible and increases on bending forward. No thrill or bruit is present. MRI shows retrobulbar mass with enhancement. The likely diagnosis is: *(AIIMS)*

a. AV malformation
b. Orbital varix
c. Orbital encephalocoele
d. Neurofibromatosis

27. An 8-year-old boy presents with proptosis in the left eye for 3 months. CT scan reveals intraorbital extraconal mass lesion. Biopsy shows embryonal rhabdomyosarcoma. Metastatic workup is normal. The standard line of management is: *(AIIMS)*

a. Chemotherapy
b. Wide local excision
c. Chemotherapy and radiotherapy
d. Enucleation

28. Most common type of optic nerve glioma is: *(AIIMS)*

a. Gemiocytic b. Fibrous
c. Protoplasmic d. Pilocytic

29. All of the following types of lymphoma may be seen in the orbit *except*: (AIIMS)

a. Non-Hodgkin's lymphoma, mixed lymphocytic and histiocytic
b. Non-Hodgkin's lymphoma, poorly differentiated
c. Burkitt's lymphoma
d. Hodgkin's lymphoma

30. Blow out fracture of the orbit involves: *(DNB)*

a. Floor b. Medial wall
c. Roof d. Lateral wall

31. True about blow out fracture of the orbit is/are: *(PGI)*

a. Herniates into maxillary antrum
b. Extraocular movements are restricted
c. Looking down is easy
d. Diplopia is present
e. Orbital floor reconstruction is the treatment

32. Most common cause of fracture of roof of orbit is: *(AIIMS)*

a. Blow on back of head
b. Blow on the forehead
c. Blow on the parietal bone
d. Blow on upper jaw

33. Mucin layer deficiency of tear film is seen in: *(AIIMS)*

a. Keratoconjunctivitis sicca
b. Lacrimal gland removal
c. Canalicular block
d. Herpes zoster

34. Epiphora means: *(AIPG)*

a. Cerebrospinal fluid running from nose after fracture of anterior cranial fossa
b. A presenting feature of a cerebral tumour
c. An abnormal flow of tears due to obstruction of the lacrimal duct
d. Eversion of lower eyelid following injury

35. A two month old child presents with epiphora and regurgitation: The likely diagnosis is: *(DNB)*

a. Mucopurulent conjunctivitis
b. Congenital dacryocystitis
c. Buphthalmos
d. Encysted mucocoele

36. Most common site of obstruction in congenital NLD obstruction: *(PGI 2013)*

a. Upper canaliculus
b. Lower canaliculus
c. Common canaliculus

d. Valve of Hasner
e. Middle turbinate

37. Initial treatment of congenital dacryocystitis is: *(PGI)*

a. Massage
b. Probing
c. DCR
d. Antibiotics
e. No treatment is needed

38. Treatment of chronic dacryocystitis is: *(DNB)*

a. Dacryocystorhinostomy
b. Antibiotics
c. Probing
d. Massage

39. A 60-year-old man presents with watering from his right eye since 1 year. Syringing revealed a patent drainage system. Rest of the ocular examination was normal. provisional diagnosis of lacrimal pump failure was made. Confirmation of diagnosis is done by:

a. Dacryoscintigraphy
b. Dacryocystography
c. Pressure syringing
d. Canaliculus irrigation test

ANSWERS AND EXPLANATIONS

1. **a. Lipogranuloma**
2. **c. Chalazion**
3. **b. Sebaceous cyst, d. Recurrence may imply malignancy, e. Occlusion of the meibomian gland**
4. **b. Intralesional steroid, c. Curettage**
5. **b. Sebaceous cell Ca**
6. **d. Horner's syndrome**
7. **a. Marcus-Gunn jaw winking syndrome**
8. **a. Marfan's syndrome**

Causes of bilateral ptosis
Senile/Involutional/Aponeurotic
Congenital
Blepharophimosis syndrome^{Q}
Myasthenia gravis^{Q}
Chronic progressive external ophthalmoplegia^{Q}
Ocular myopathies
Myotonic dystrophy

9. **b. Senile entropion**
10. **a. Symblepharon**
11. **b. Basal cell Ca**
    Most common lid malignancy: Basal cell Ca
    Most common lid malignancy in recurrent chalazion: Sebaceous cell Ca^{Q}
12. **b. Widened root of the nose with normal interpupillary distance**
13. **b. IIIrd cranial nerve, c. IVth cranial nerve, d. VIth cranial nerve, e. Lacrimal nerve**
14. **a. Orbital cellulitis**
15. **a. Thyrotoxicosis**
16. **b. Chloroma**
17. **d. Neuroblastoma**
    Since chloroma is not present among the options, we have chosen neuroblastoma as the answer.
18. **a. T3 and T4 to rule out thyrotoxicosis**
    The most common cause of proptosis in adult is thyroid ophthalmopathy, hence investigation should rule out thyroid disease as the first step
19. **c. Proptosis, d. Enlargement of the extraocular muscles, e. Lid lag**
    Thyroid orbitopathy leads to intraorbital infiltration of inflammatory cells and orbital soft tissue oedema. This leads to enlargement of the extraocular muscles. When this oedema resolves, there is secondary fibrosis leading to restriction of ocular movements. This is called **restrictive ophthalmopathy not ophthalmoplegia.**
20. **b. Inferior rectus**

21. **d. Ophthalmic veins**

22. **a. Cavernous sinus**

Involvement of the cavernous sinus presents with

- Proptosis
- Chemosis
- Paralysis of third, fourth and sixth cranial nerves starting with the sixth nerve.

23. **a. Cavernous sinus thrombosis**

The question describes a patient with sinusitis who develops sudden onset fever, chills associated with proptosis. This may be due to orbital cellulitis or cavernous sinus thrombosis. The question however mentions diplopia in lateral gaze suggestive of involvement of the sixth nerve; hence the more probable answer is cavernous sinus thrombosis.

24. **a. Constricted pupil in response to light**

Involvement of the cavernous sinus leads to third nerve palsy. Thus, the patient will have dilatation and not constriction of the pupil. (See features of third nerve palsy in the chapter on Neuro-ophthalmology)

25. **b. Cavernous haemangioma**

The most common benign orbital tumour in adult is **Cavernous haemangioma**[Q]. It is an intraconal mass presenting with gradually progressive axial proptosis

Most common benign orbital tumour in children: **Dermoid**[Q]

Most common orbital malignancy in adult: **Lymphoma**[Q]

Most common orbital malignancy in children: **Rhabdomyosarcoma**[Q]

26. **b. Orbital varix**

Orbital varix is a cause of non-axial proptosis, usually intermittent. The proptosis becomes evident on coughing, straining and bending forward. Orbital encephalocoele also has similar features but it is pulsatile and does not show enhancement with contrast. Hence, the answer.

AV malformation or Carotido-cavernous fistula gives rise to pulsatile, axial proptosis associated with thrill and bruit.

27. **c. Chemotherapy and radiotherapy**

Rhabdomyosarcoma is the most common orbital malignancy in children[Q]. It presents with non-axial proptosis which is sudden in onset and very rapidly progressive. Hence, it is also called **malignant proptosis.**

Most common variety of Rhabdomyosarcoma: Embryonal[Q]

Least common variety: Pleomorphic[Q]

Best prognosis: Pleomorphic[Q]

Worst prognosis: Alveolar[Q]

Treatment is chemotherapy with radiotherapy.

Causes of malignant proptosis:

- Rhabdomyosarcoma[Q]
- Orbital cellulitis[Q]
- Chocolate cyst[Q]

28. **d. Pilocytic**

Features of optic nerve glioma

- It is seen in young girls less than 10 years of age
- It is associated with **Neurofibromatosis I**[Q]
- Commonest histological variety is **pilocytic astrocytoma**[Q]
- It presents with gradual onset, slowly progressive proptosis which is axial, non-pulsatile
- It is associated with vision loss.

29. **d. Hodgkin's lymphoma**

30. **a. Floor**

31. **a. Herniates into the maxillary antrum, b. Extraocular movements are restricted, d. Diplopia is present, e. Orbital floor reconstruction is the treatment**

32. **b. Blow on the forehead**

33. **d. Herpes zoster**

34. **c. An abnormal flow of tears due to obstruction of the lacrimal duct**

35. **b. Congenital dacryocystitis**

36. **d. Valve of Hasner**

37. **a. Massage**

38. **a. Dacryocystorhinostomy**

39. **a. Dacryoscintigraphy**

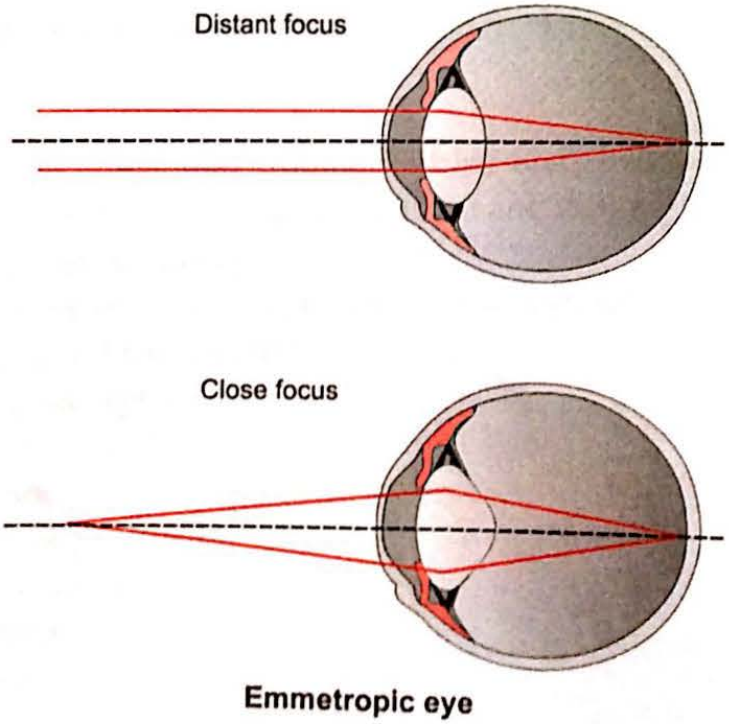
Lacrimal pump failure means inability of the lid muscles to push the tears towards the punctum for drainage. It is also called **functional obstruction** and is confirmed by Dacryoscintigraphy.

Chapter 8

Optics and Refraction

BASICS OF REFRACTION IN THE EYE

- The **total refractive power** of the eye is **58–60 dioptres**Q
- The refractive power of the **cornea** is **43–45 dioptres**Q
- The refractive power of the **lens** is **15–16 dioptres**Q
- The refractive indices of the different ocular media are
  - Cornea: 1.376Q
  - Aqueous humour: 1.336
  - Lens: 1.386Q
  - Vitreous humour: 1.336
- The eye may be considered as a convex or converging lens of 58–60 D power and the retina is like a screen which is located at its principal focus. So the parallel rays of light coming from infinity are brought to focus at a point on the retina. But the eye is capable of adjusting its refractive power according to the distance at which an object is to be viewed. For example, rays of light coming from a near object are divergent rays. So more refractive power is needed by the eye to converge these rays to the retina. This ability to increase the refractive power is called accommodation.



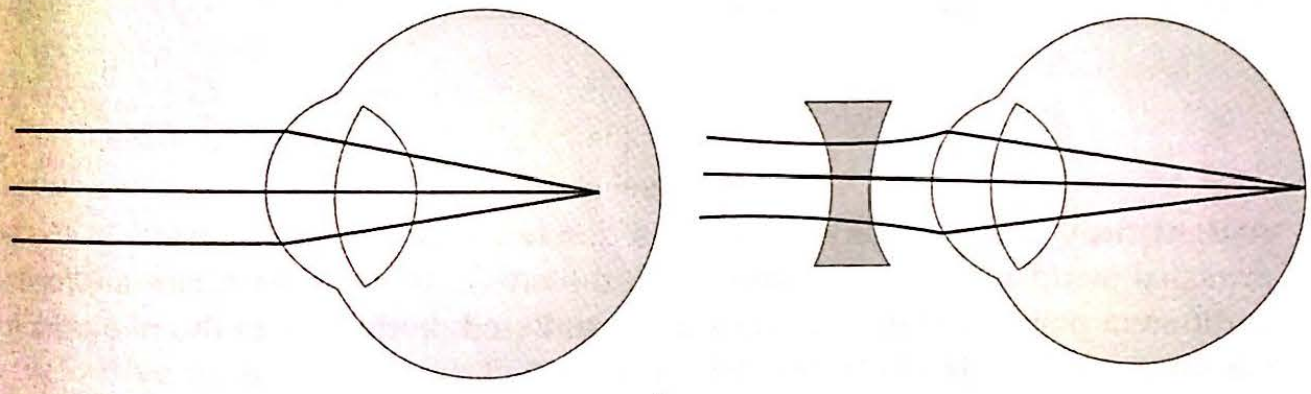
Emmetropic eye

- **Accommodation**: The changes that occur during accommodation are:
  - Contraction of the ciliary muscle
  - Relaxation of the ciliary zonules
  - Increase in the anterior curvature of the lens
  - Increase in the refractive power of the eye
- **Presbyopia**: It is defined as a **physiological decrease in the amplitude of accommodation with age**[Q] which results in difficulty in near vision. The factors responsible are:
  - Decrease in the tone of the ciliary zonules[Q]
  - Rigidity of the zonules with age[Q]
  - Thickening of the lens with age which decreases its flexibility to change its shape[Q]

Hence, presbyopes are to be given **convex lenses for near work**[Q].

Errors of Refraction

A. **Myopia**: In myopia, parallel rays of light coming from infinity, in the absence of accommodation, converge to a point in front of the retina. This means that the refractive power of the myopic eye is more than what it needs. Hence, myopia is treated with **minus or concave lenses**[Q]. The different types of myopia are
   - Axial: Due to increased axial length
   - Curvatural: Due to increase in corneal curvature, as in keratoconus
   - Index: Due to increase in refractive index of the eye, as in nuclear cataract.



Myopic eye

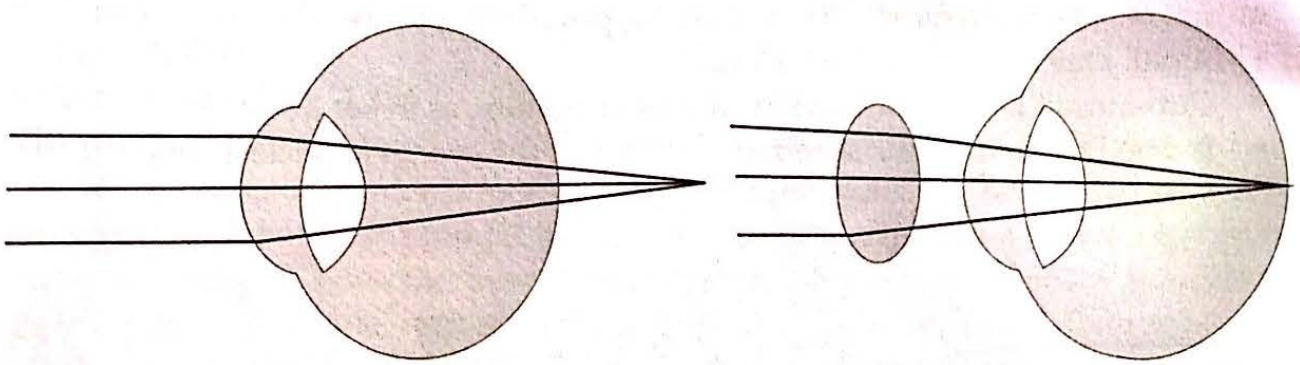
Childhood myopia can be broadly divided into:
- Simple myopia: Refractive error is usually less than -6D with no degenerative changes in the eye
- Pathological myopia: High refractive error with degenerative changes in the eye

Changes in the myopic eye[Q]
- Pseudoproptosis due to large eyeball
- Exotropia or divergent squint[Q]
- Thin cornea[Q]
- Increased risk of open angle glaucoma
- Subluxation and dislocation of lens[Q]
- Complicated cataract

- Vitreous degeneration
- Fundus changes
  - Myopic crescent temporal to the disc due to stretching of the eyeball
  - **Posterior staphyloma**[Q]
  - **Foster Fuchs' maculopathy**[Q]
  - **Lacquer cracks**[Q] **and choroidal neovascular membrane (CNVM)**
  - Peripheral retinal degenerations like lattices, breaks and holes
  - Rhegmatogenous retinal detachment[Q]

B. Hypermetropia: In this condition, the parallel rays of light coming from infinity, in the absence of accommodation, converge to a point behind the retina. Thus, a hypermetropic eye has less refractive power than it needs and so it is treated with **plus lenses or convex lenses**[Q]. Hypermetropia is divided into:

- Latent hypermetropia: This is masked by the tone of the ciliary body
- Facultative hypermetropia: This is masked by the voluntary accommodative effort of the individual
- Absolute hypermetropia: This is the hypermetropia which is apparent.



Hypermetropic eye

C. Astigmatism: In this condition, there is a difference in refractive powers in the two principal meridians of the eye. As a result the rays in the two meridians are focused at different points leading to a blurred image. Based on the axis of the two principal meridians, astigmatism is of two main types:

- Regular astigmatism: Here the two meridians are perpendicular to each other. If the horizontal axis is steeper than vertical, it is called **"against-the-rule"** astigmatism. If the vertical is steeper than horizontal, it is called **"with-the-rule"** astigmatism. If the axes are perpendicular but obliquely inclined, it is called **oblique astigmatism.**
- Irregular astigmatism: Here the two meridians are not perpendicular to each other.

Astigmatism is treated with **cylindrical lenses**[Q] **(lenses with power only in one axis)**.

D. Anisometropia: This is a condition where there is a significant difference in the refractive power of the two eyes. As a result of this, **there is a significant difference in the image size of the two eyes. This is called aniseikonia**[Q]. To avoid this, high anisometropia should be treated with contact lenses (Contact lenses produce less change in image size as compared to spectacles).

Treatment of refractive errors

A. Spectacles are corrective lenses worn in a frame about 12 mm in front of the eye for the correction of refractive error

B. Contact lenses: Contact lenses are lenses worn on the surface of the eye for the correction of refractive error. The different types of contact lenses are:
   - **Soft lenses**: These are made up of **hydroxyethylmethacrylate (HEMA)**[Q] and used for the correction of myopia, hyperopia and low astigmatism
   - **Rigid gas permeable lenses (RGP)**: These are made up of **cellulose acetate butyrate (CAB)**[Q], **styrene** etc. They are used for **high astigmatism.** Irregular astigmatism has to be corrected with RGP lenses.

Advantages of contact lenses
- Less change in image size, hence useful in high degree of anisometropia
- Useful for correcting irregular astigmatism which is not amenable to spectacles
- Greater field of vision
- High power spectacles have aberrations like spherical, chromatic, prismatic etc. These are minimum with contact lenses.

Complications with contact lenses
- **Giant papillary conjunctivitis**[Q]
- Corneal hypoxia with vascularisation—The corneal epithelium receives its oxygen from the air and glucose from the aqueous and limbal vessels. **Contact lenses reduce the availability of oxygen to the cornea**[Q]. As a result, the metabolism shifts from aerobic to anaerobic. **So the content of lactate and pyruvate in the cornea are increased**[Q].
- Corneal abrasion
- Microbial infections: Most common organism is **Pseudomonas**[Q]
- Overwear syndrome also called as contact lens warpage.

Other uses of contact lenses
- Bandage contact lenses are used in non-healing epithelial defects of the cornea, excessive thinning of the cornea, impending perforation
- Contact lenses are also used as a vehicle for drug delivery

C. Refractive surgery: Surgeries to correct refractive errors may be cornea based or lens based. The cornea based surgeries are more common.

Cornea based surgeries
- **LASIK** (laser assisted in situ keratomileusis)—In LASIK, a flap of the cornea is raised and the underlying stromal bed is ablated with laser. This **photoablation** is done by **Excimer laser** [Q]. Hence, at the end of the procedure, **the shape of the cornea is altered, the thickness is decreased** and the refractive error is corrected. The **eligibility criteria**[Q] for LASIK are:
  - Age >18 years
  - Stability of refraction for at least 6 months
  - **Minimum corneal thickness of 500 microns**[Q]
  - **Residual stromal depth of 250 microns**[Q]
  - **Absence of any other corneal pathology like keratoconus**
  - Absence of other ocular pathologies like glaucoma, retinal degenerations etc

- **Photorefractive keratectomy (PRK):** This procedure is used for patients with low to moderate myopia (-2 to -6), hyperopia and astigmatism whose corneal thickness is not adequate for LASIK. Here the corneal epithelium is manually removed instead of making a flap so that more stroma is available for ablation. **Main disadvantages are** pain, **delayed postoperative recovery, corneal haze and regression of refractive error**
- **LASEK (laser assisted surface epithelial keratomileusis)** Used in patients with thin corneas which makes them ineligible for LASIK
- **Femtosecond LASIK:** This is the most recent advancement in laser refractive surgery

Lens based surgeries

- This type of refractive surgery is considered in patients with very high refractive errors when they are not eligible for corneal refractive surgery
- In this procedure, an artificial lens is placed in the eye in front of the patient's own crystalline lens. This is hence called **phakic IOL**
- The main eligibility criteria would be:
  - Age> 18 years with stable refraction for at least 6 months
  - Not eligible for cornea based surgery
  - Open angles on gonioscopy
  - Minimum anterior chamber depth of 2.8 mm
  - No evidence of cataract.

Errors of Accommodation

- Presbyopia: Described earlier
- **Pseudomyopia**[Q]: This is also called **spasm of accommodation**. It is seen in children especially during their examination when they are doing excessive near work. Due to excess accommodation, there is a myopic shift. **So the near vision is good but the distance vision is poor**[Q]. Treatment is to relax the accommodation with cycloplegics
- Inertia of accommodation: In this condition, there is difficulty in changing the focus from distance to near and vice versa. This is because the accommodative system is slow in making a change.
- Paralysis of accommodation: The possible causes are cycloplegics, third nerve palsy etc.
- Insufficiency of accommodation: This is a condition where the amplitude of accommodation is less than the normal physiological limit according to the patient's age.

Refraction

The process of determining and correcting the refractive error of a patient is called refraction. At first an **objective assessment of the refractive error**[Q] is done by **retinoscopy**[Q]. Then, verification of retinoscopy is done by subjective acceptance.

Method of retinoscopy

- Retinoscopy is a method of determining the refractive status of an individual by the method of neutralisation. With the help of a retinoscope, light is shown in the patient's eye and the examiner observes the red reflex in the patient's pupillary area. The reflex

moves along with the movement of the retinoscope mirror. The direction of movement of the reflex depends upon the refractive error of the patient.
  - Movement of reflex in the direction of the mirror: Hypermetropia or myopia <1D^{Q}
  - Movement of reflex opposite to the direction of the mirror: Myopia>1D^{Q}
- Next, the movement of the reflex is neutralized by using plus lenses (with movement) or minus lenses (against movement). The final readings are noted. The same process is carried out in both meridians
- Estimation of refractive error from retinoscopy is done by adjusting for the distance and the cycloplegic used.
  - **For a distance of 1m, the deduction made is 1^{Q}**
  - **For a distance of 2/3m, the deduction made is 1.5^{Q}**
  - The closer the examiner moves to the patient, greater is the deduction
  - **For atropine as cycloplegic, the deduction made is 1**
  - For cyclopentolate, the deduction is 0.75
  - For homatropine, the deduction is 0.5
  - Thus, if retinoscopy done at 1m under atropine gives reading of +5, the corrected reading would be +5-1(for 1m distance)-1(for atropine)=+3

Cycloplegics in Refraction

- Cycloplegics are used to relax the accommodation of the patient so as to get an accurate assessment of the refractive error in retinoscopy. The important cycloplegics are:
  - **Atropine (1% ointment)^{Q}:** It is the **longest acting^{Q}** dilator—cycloplegic with duration of action of about **10–14 days.** It is used for refraction in children less than 3 years of age and patients with squint
  - **Tropicamide (0.5% eye drops):** It is the **shortest acting^{Q}** dilator- cycloplegic with a duration of action of **4–6 hours.** It is used for refraction in adults
  - **Homatropine (2% eye drops):** It has a duration of action of 3–4 days and is used in school going children
  - **Cyclopentolate (1% eye drops):** It has a duration of action of 12–24 hrs
  - **Phenylephrine:** This is a **dilator with no cycloplegic action^{Q}.**

QUESTIONS

1. **Normal power of the reduced eye:** *(Maharashtra)*
   a. + 6D b. + 17D
   c. + 43D d. + 60D
2. **Most important factor determining convergence of light rays on the retina is:** *(AIPG)*
   a. Length of the eyeball
   b. Refractive power of the lens
   c. Curvature of the cornea
   d. Physical state of the vitreous
3. **Which component of the eye has maximum refractive index:** *(AIIMS)*
   a. Anterior surface of lens
   b. Posterior surface of lens
   c. Centre of lens
   d. Cornea
4. **True statements about accommodation:** *(PGI 2013)*
   a. Mainly occurs due to increase in posterior curvature of the lens
   b. Helps to improve stereopsis
   c. It is abolished by sympathomimetic drugs
   d. Mainly due to increase in anterior curvature of the lens
   e. Elasticity of the capsule has a bearing on accommodation
5. **Most common type of refractive error in older children:** *(COMEDK 2015)*
   a. Hypermetropia b. Myopia
   c. Presbyopia d. Astigmatism
6. **The most common cause of myopia is:** *(DPG)*
   a. Increase in axial length of the eyeball
   b. Increase in thickness of the lens
   c. Increase in viscosity of aqueous humour
   d. Increase in viscosity of vitreous humour
7. **Which of the following is a serious complication of degenerative myopia?** *(UPSC)*
   a. Retinal detachment
   b. Posterior staphyloma
   c. Myopic crescent
   d. Vitreous liquefaction
8. **Which of the following is true about degenerative myopia?** *(AIIMS 2013)*
   a. More common in males as compared to females
   b. Myopic degeneration can lead to retinal detachment
   c. It is seen in <6 dioptres of myopia
   d. Retinal tear is less common and is a late complication
9. **Foster Fuchs' spots are seen in:** *(Kerala PG)*
   a. Myopia
   b. Hypermetropia
   c. Sympathetic ophthalmia
   d. Astigmatism
10. **Pseudopapillitis is seen in:** *(DPG)*
    a. Hypermetropia b. Myopia
    c. Squint d. Presbyopia
11. **True about presbyopia:** *(PGI)*
    a. Age related error of refraction
    b. Age-related defect in accommodation
    c. Concave lens is used
    d. Convex lens is used
    e. Cylindrical lens is used
12. **Aniseikonia means:** *(AIIMS)*
    a. Difference in axial length in the two eyes
    b. Difference in the curvature of the cornea in the two eyes
    c. Difference in the size of the pupil in the two eyes
    d. Difference in the size of the image formed by the two eyes

13. **Treatment options for myopia are:** *(PGI)*
    a. Radial keratotomy
    b. LASIK
    c. Epikeratophakia
    d. Keratoplasty
    e. Photorefractive Keratectomy (PRK)

14. **Soft contact lens is made up of:** *(APPG)*
    a. PMMA b. HEMA
    c. Silicon d. Glass

15. **Laser used in the treatment of myopia:** *(DNB)*
    a. Nd- YAG b. Excimer
    c. Argon d. Krypton

16. **A lady wants LASIK surgery for her daughter. Which of the following is not an eligibility criteria for LASIK?** *(AIPG)*
    a. Myopia of 4 D
    b. Age of 15 years
    c. Stable refraction for 1 year
    d. Corneal thickness of 600 microns

17. **Concentration of tropicamide:** *(AIIMS 2013)*
    a. 0.01 b. 0.02
    c. 0.03 d. 0.04

18. **Which of the following is not a cycloplegic?** *(AIPG)*
    a. Phenylephrine b. Atropine
    c. Tropicamide d. Homatropine

19. **Mydriatic to be used in a 3-year-old child for refraction:** *(Maharashtra PG)*
    a. 1% Atropine drops
    b. 1% Atropine eye ointment
    c. 0.5% Tropicamide eye drops
    d. 2% Homatropine eye drops

20. **Objective assessment of refraction is termed as:** *(COMEDK)*
    a. Gonioscopy
    b. Retinoscopy
    c. Ophthalmoscopy
    d. Keratoscopy

21. **In retinoscopy, for a distance of 1m the correction factor is -1D. What is the correction factor for retinoscopy done at 66 cm?** *(Kerala PG)*
    a. - 2 b. - 1
    c. - 0.5 d. - 1.5

22. **On performing retinoscopy using a plane mirror in a patient who has a refractive error of -3D sphere with - 2 cylinder at 90 degrees from a distance of 1m, the reflex would move:** *(AIIMS)*
    a. With the movement in the horizontal axis and against the movement in the vertical axis
    b. With the movement in both the axes
    c. Against the movement in both the axes
    d. With the movement in the vertical axis and against the movement in the horizontal axis

23. **On performing retinoscopy using plane mirror in a patient with myopia of 0.5D from a distance of 1m, the reflex will move:** *(AIIMS)*
    a. Move with the mirror
    b. Move opposite to the mirror
    c. No movement
    d. May move to either side

24. **Cross cylinder is:** *(PGI)*
    a. One plus cylinder and one minus cylinder of equal strength
    b. One plus and one minus cylinder of unequal strength
    c. Two plus cylinders
    d. Both minus cylinders
    e. One spherical and one cylindrical lens

25. **A 35 year old man has 6/5 vision in each eye unaided. His cycloplegic refraction is 0.00. He complains of blurring of newsprint at 30 cm which clears up in about 2 minutes. The probable diagnosis is:** *(AIPG)*
    a. Hypermetropia
    b. Presbyopia
    c. Accommodative inertia
    d. Cycloplegia

26. A 35-year-old man has normal distance vision but complains of difficulty in near vision. His retinoscopy shows +2D sphere. The probable diagnosis is: *(AIIMS)*

a. Hypermetropia
b. Presbyopia
c. Myopia
d. Accommodative inertia

27. A 10-year-old boy is brought to the doctor with complains of squeezing his eyes to see the blackboard in school. What is the probable diagnosis? *(Manipal)*

a. Hypermetropia
b. Myopia
c. Astigmatism
d. Accommodative inertia

28. A 9-year-old boy is brought with complaints of difficulty in vision for distance. On examination, the visual acuity on Snellen's chart is 6/36 but it improves to 6/6 with pin-hole. What is the diagnosis?

a. Malingering
b. Refractive error
c. Developmental cataract
d. Amblyopia

29. Treatment of choice for aphakia: *(AIPG)*

a. Spectacles
b. Contact lenses
c. IOL
d. Laser therapy

ANSWERS AND EXPLANATIONS

1. **d. + 60D**
2. **c. Curvature of the cornea**
   The total refractive power of the eye is about 60D of which 44-45D comes from the cornea. Thus, the cornea is the most important refracting medium responsible for convergence of light rays to the retina
3. **c. Centre of the lens**
4. **d. Mainly due to increase in anterior curvature of the lens, e. Elasticity of the capsule has a bearing on accommodation**
5. **b. Myopia**
6. **a. Increase in axial length of the eyeball**
7. **a. Retinal detachment**
8. **b. Myopic degeneration can lead to retinal detachment**
   Myopia > 6 D is associated with peripheral retinal holes, tears and degenerations like lattice. This may predispose the patient to develop rhegmatogenous retinal detachment
9. **a. Myopia**
10. **a. Hypermetropia**
    In hypermetropia, the optic disc appears small and hyperemic. The disc margins also appear slightly blurred, giving a false impression of papillitis. Hence, the name pseudopapillitis
11. **b. Age-related defect of accommodation, d. Convex lens is used**
12. **d. Difference in the size of the image formed by the two eyes**
13. **a. Radial keratotomy, b. LASIK, e. Photorefractive keratectomy (PRK)**
    Radial keratotomy is an old and abandoned procedure for myopia correction. In this, radial cuts are made in the cornea of about 90% depth and extending from the paracentral region to the limbus. These cuts lead to corneal flattening and correction of myopia. The complications are:
    - Corneal perforation
    - Epithelial ingrowth
    - Irregular corneal scarring leading to irregular astigmatism
    - Unpredictable results
    - Regression of myopia after about 10 years

    Hence, this procedure is not done nowadays. Thus options for myopia correction now are LASIK, PRK, LASEK, Femtosecond LASIK and Phakic IOL.
    Epikeratophakia is a surgery for hypermetropia where a lenticule from a donor cornea is placed on the recipient cornea. It is rarely done.
14. **b. HEMA**
15. **b. Excimer**
16. **b. Age of 15 years**
17. **a. 0.01**
    The concentration of Tropicamide is usually 0.5-1% (0.005–0.01)
18. **a. Phenylephrine**

19. **b. 1% Atropine eye ointment**
20. **b. Retinoscopy**
21. **a. – 1.5**
22. **c. Against the movement in both the axes**
    As the refractive error is myopia >1D in both axes, the movement of the reflex will be against the mirror in both the axes
23. **a. Move with the mirror**
    As the refractive error is myopia <1 D, the reflex will move with the mirror
24. **a. One plus cylinder and one minus cylinder of equal strength, e. One spherical and one cylindrical lens**
    Cross cylinder is an instrument used to refine the power and axis of cylinder during subjective refraction. It consists of one plus and one minus cylinder of same power but in opposite axis
    The combination of a cylinder and a sphere of opposite and double power produces the same effect.
25. **c. Accommodative inertia**
    The question suggests that the patient has good distance and near vision but has difficulty in adjusting from one distance to another. This is accommodative inertia.
26. **a. Hypermetropia**
27. **b. Myopia**
    The question suggests that the boy has poor distance vision and so he tries to squeeze his eyes to see better. Hence the answer is myopia
28. **b. Refractive error**
    A pinhole is a blocker with a small aperture of diameter 1mm at the centre. When placed in front of the eye, it allows only the central rays to pass whereas all the paraxial rays are blocked.
    In refractive errors, visual acuity improves with pinhole.
    In macular disorders, visual acuity decreases with pinhole
29. **c. IOL**
    Aphakia means absence of crystalline lens. **The refractive status in aphakia is high hypermetropia^{Q} (+10 to +12 dioptres).**
    In the early days of cataract surgery, the lens was removed in toto and the patient was left aphakic. Postoperatively, the patient was advised high convex lenses. But these glasses have some important drawbacks. They are:
    - **High magnification**: The high power convex lenses cause a magnification of the image size by **25–30%^{Q}**. These glasses cannot be prescribed in unilateral aphakia because they cause aneisikonia.
    - Limited field of vision
    - Spherical and chromatic aberration
    - **Prismatic aberration^{Q}**: This causes **roving-ring scotoma or Jack in the box^{Q}** effect as a result of which objects keep appearing and disappearing in the field of view.
    - **Pin cushion effect^{Q}**:Straight lines appear curved and images are distorted

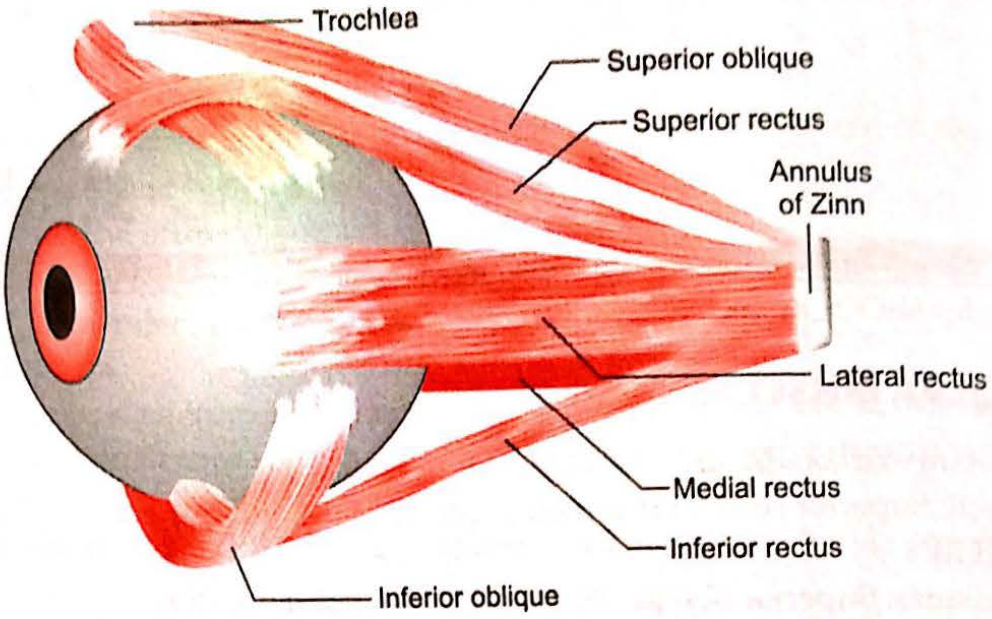
    Aphakia may be corrected by contact lenses but the **treatment of choice is IOL^{Q}.**

Strabismus

EXTRAOCULAR MUSCLES

- The important extraocular muscles are:
  - Four recti: Superior rectus (SR), medial rectus (MR), inferior rectus (IR) and lateral rectus (LR)
  - Two obliques: Superior oblique (SO) and inferior oblique (IO)
- **Origin of muscles:**
  - All the recti and SO arise from **the apex of the orbit**Q.
  - The four recti arise from the **Annulus of Zinn**Q (common tendinous ring at orbital apex).
  - The SO arises from the **body of sphenoid**Q at the apex of the orbit
  - The IO arises from the orbital plate of maxilla **at the floor of the orbit**Q
- **Insertion of the muscles:**
  - Rectus muscles are inserted into the sclera at different distances away from the limbus. The distances from limbus are
    MR: 5.5 mm
    IR: 6.5 mm
    LR: 6.9 mm
    SR: 7.7 mm
  - **The Spiral of Tillaux is the line joining the points of insertion of the rectus muscles**Q
  - The SO after originating from the apex of the orbit first travels superomedially, and then turns backward at the trochlea. It then travels posterolaterally to insert in the sclera in the **upper temporal quadrant of the globe**Q
  - The IO moves backwards and laterally to insert into the **lower temporal quadrant of the globe**Q.
- **Few important facts**
  - Rectus muscle closest to limbus: MRQ
  - Rectus muscle farthest from limbus: SRQ
  - Longest extraocular muscle: SO
  - Shortest extraocular muscle: IO
- **Nerve supply**
  - LR is supplied by sixth cranial nerve
  - SO is supplied by fourth cranial nerve
  - MR, IR, SR, IO are supplied by third cranial nerve

- Remembered as **SO4 LR6** Q



Extraocular muscles

- **Actions of the extraocular muscles:**

Muscle	Action			Nerve supply
	Primary	**Secondary**	**Tertiary**	
MR	Adduction			III
IR	**Depression**	Extorsion	Adduction	III
LR	Abduction			VI
SR	**Elevation**	Intorsion	Adduction	III
SO	**Intorsion**	Depression	Abduction	IV
IO	**Extorsion**	Elevation	Abduction	III

Important points to remember:

- **SIN meaning Superiors are Intorters**Q (SR and SO)
- **RAD meaning Recti are Adductors**Q (SR and IR)
- The superior oblique functions **only as depressor in adducted position** and only as **intorter in abducted position**Q
- The inferior oblique functions **only as elevator in adducted position** and only as **extorter in abducted position**Q.

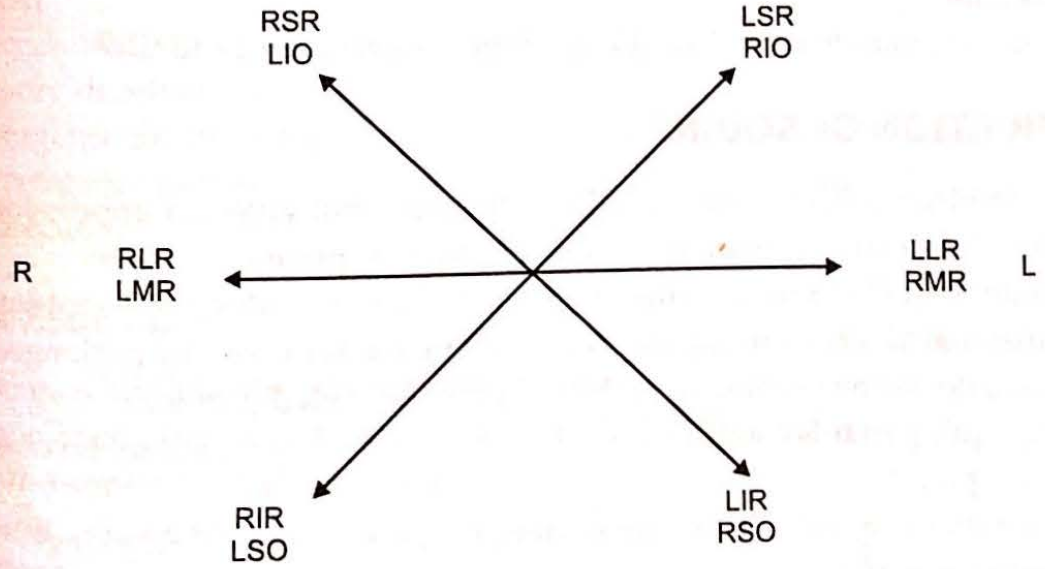
OCULAR MOVEMENTS

- Ductions: These are monocular movements like adduction, abduction, elevation and depression
- Versions: These are binocular conjugate movements (in the same direction)
  - Dextroversion (right sided gaze)
  - Levoversion (left sided gaze)
  - Dextroelevation (up and right gaze)

- Levoelevation (up and left gaze)
- Dextrodepression (down and right gaze)
- Levodepression (down and left gaze)

- Vergence: These are binocular disjugate movements (both eyes move in opposite directions) like convergence. Convergence is the ability of the two eyes to move inwards. It has two components: voluntary and reflex. Reflex convergence again has four components.
  - Tonic convergence: Due to basal tone of muscle
  - Proximal convergence: Induced by psychological awareness of a near object
  - Fusional Convergence
  - **Accommodative Convergence**: It is induced by the effort of accommodation. For each diopter of accommodation, a fairly constant increase in accommodative convergence occurs **(AC/A Ratio).** AC/A ratio denotes the amount of convergence measured in **prism diopter** per unit change in accommodation. **Normal value is 4:1 (1 D of accommodation is associated with 4 prism diopters of convergence)**Q. High AC/A ratio leads to excessive convergence and esotropia. Low AC/A ratio leads to exotropia
- Laws of ocular motility
  - Agonist-antagonist: Muscles of same eye which have opposite functions. For example: MR and LR of the same eye
  - **Synergist**Q**:** Muscles of the same eye which move the eye in the same direction. For example SR and IO of the same eye are synergists in elevation
  - **Yoke muscles**Q**:** A pair of muscles of opposite eyes which produce a conjugate ocular movement. For example LR of right eye and MR of left eye are yoke muscles for dextroversion.

YOKE MUSCLES FOR CARDINAL POSITIONS OF GAZE



- **Hering's law of equal innervation**Q**:** During any conjugate movement, equal and simultaneous innervation flows to a pair of yoke muscles.
- **Sherrington's law of reciprocal innervation**Q**:** Increase in innervation and contraction of a muscle is associated with reciprocal decrease in innervation and relaxation of its antagonist.

BINOCULAR SINGLE VISION (BSV)

BSV is achieved by use of the two eyes together.

Grades of Binocular Vision

- **Grade I: Simultaneous Macular Perception (SMP)**[Q]
- **Grade II: Fusion**[Q]
- **GradeIII: Stereopsis**[Q]**:** Ability to obtain an impression of depth.

DOUBLE VISION/DIPLOPIA

The pre-requisite for binocular single vision is perfect ocular alignment. Strabismus or any ocular deviation hence may result in diplopia. But the individual undertakes certain adaptations in order to overcome this diplopia. The compensatory mechanisms vary according to the type of diplopia and the age of the individual.

Compensatory Mechanisms to Prevent Double Vision

- **Suppression:** It occurs in children with squint where the image formed on the retina by the squinting eye is suppressed by the visual cortex. This leads to the development of **amblyopia.**
- **Compensatory head posture**: This is a feature of paralytic strabismus where the head is turned in the direction of field of action of the weak/paralysed muscle. The type of head posture depends upon the type of diplopia and the muscle involved
  - For horizontal muscle palsy like LR palsy, the patient develops a **face turn**[Q]
  - For vertical muscle palsy like SR or IR, the patient develops **chin elevation** or chin **depression**[Q]
  - For oblique muscle palsy like SO, the patient develops a **head tilt**[Q]

CLASSIFICATION OF SQUINT

- Pseudostrabismus: This is not actually a deviation but gives an impression of ocular deviation. It is seen in prominent **epicanthic fold, hyperteleorism** etc.
- Latent squint: In this condition, the tendency of the eyes to deviate is kept under control by the fusional ability of the individual. If the fusion is broken by prolonged occlusion of one eye, deviation becomes manifest. This is also called phoria.
- Manifest squint: In this condition, the deviation of the eye is evident on observation. It is of two types:
  - **Concomitant squint**: The degree of deviation is the **same in all gazes**[Q]
  - **Incomitant squint**: The common type is **paralytic squint** where there is paralysis of one or more extraocular muscle. **The deviation is maximum in the direction of action of the paralysed muscle**[Q]**.**

	Concomitant squint	Incomitant squint
Deviation	Same in all gazes	**Maximum in the direction of gaze of the affected muscle**[Q]
Extraocular movements	Normal	Limitation in the direction of action of the affected muscle
Amblyopia	May be **present in children**[Q]	Absent
Head posture	Usually absent	Usually present
Primary(P) and Secondary deviation(S)	Both are same	**Secondary deviation > Primary Deviation**[Q]

Important Points

- **Primary deviation is the deviation of the affected eye**[Q].
- **Secondary deviation is the deviation of the good eye under cover**[Q]
- Secondary deviation> Primary deviation in paralytic squint is explained by Herring's law

(Paralytic squint is covered in detail in the chapter on Neuro-ophthalmology under Cranial Nerve Palsies)

ESOTROPIA/CONVERGENT SQUINT

- Essential infantile esotropia
- Accommodative esotropia—It is of three types:
  - Refractive
  - Non-refractive
  - Mixed
- Non-accommodative esotropia
  - Sensory deprivation
  - Divergence insufficiency
  - Convergence excess or spasm
  - Consecutive

Infantile Esotropia

- It presents within the **first six months of birth**[Q]
- Common in children with hydrocephalus and cerebral palsy
- **Large and constant angle of squint**[Q]
- **Minimum refractive error**[Q]
- Alternate fixation in primary position and **cross fixation**[Q] in side gaze
- **Nystagmus**[Q]
- Management is surgical

Accommodative Esotropia

It is esotropia associated with abnormality in the process of accommodation. It usually **manifests at the age of 2–3 years**Q.

- **Refractive:** It is associated with high **hypermetropia**Q. Due to excessive accommodative effort by the patient, there is excessive convergence leading to esotropia. The angle **of deviation is almost equal for both distance and near**Q.
- **Non-refractive:** In this condition, there is minimum refractive error but an abnormally **high AC/A ratio**Q. This means that there is excessive convergence for a normal accommodative effort. **There is minimum deviation for distance but significant esotropia for near**Q
- **Mixed**: This is a combination of high hypermetropia and high AC/A ratio
- Management
  - Spectacles for refractive type
  - Surgery for non-refractive type

SPECIAL SQUINTS

I. **Duane's retraction syndrome:** It is a syndrome where there is failure of innervation of the lateral rectus by the sixth nerve and abnormal innervation of the lateral rectus by the third nerve. It has three types:
   - **Type I: Esotropia with limitation of abduction with relatively normal adduction**Q
   - **Type II: Exotropia with limitation of adduction with relatively normal abduction**Q
   - **Type III: Limitation of both abduction and adduction with minimal deviation**Q

Associated features are:

- On attempted adduction there is retraction of the globe and narrowing of the palpebral aperture

Treatment is not needed in most cases because deviation is not much and there is no amblyopia.

II. **Brown's superior oblique tendon sheath syndrome:** Limitation of elevation in adduction and normal elevation in abduction
III. **Mobius syndrome:** Congenital aplasia of VI, VII, IX, XII cranial nerve nuclei
IV. **Double elevator palsy:** Paresis of SR and IO of the same eye.

PRINCIPLES OF SQUINT SURGERY

The basic principle of squint surgery is weakening of the stronger muscle and strengthening of the weaker muscle. The weakening and strengthening procedures are:

- **Weakening Procedures**
  - Recession: The insertion of the muscle is moved posteriorly towards its origin.
  - Myectomy: Muscle is severed from its insertion but not reattached
- **Strengthening Procedures**
  - Resection: Pull of muscle is enhanced by making it shorter

- Advancement: Muscle is disinserted and advanced closer to the limbus. It is rarely done.

AMBLYOPIA

Amblyopia is a unilateral or bilateral reduction of best corrected visual acuity in the absence of any organic cause.

Types

Amblyopia can be of different types:

- **Strabismic amblyopia**: The protective mechanism of suppression leads to amblyopia. **This type of amblyopia has the best prognosis**Q.
- **Refractive amblyopia:** This is due to uncorrected refractive error. This may be of the following types:
  - Anisometropic amblyopia: It develops due to difference in refractive error between the two eyes. This leads to amblyopia in the eye with the larger refractive error if not appropriately corrected. **Hypermetropes are more prone to develop anisometropic amblyopia**Q.
  - Bilateral emetropic amblyopia can occur if the refractive error is high in both eyes and is not corrected
  - Meridional amblyopia is the term used when amblyopia affects only one meridian due to high astigmatic error
- **Stimulus deprivation amblyopia**: It is seen in cases having media opacity in the form of cataract or corneal opacity from early childhood. These children develop amblyopia due to visual form deprivation. This type of amblyopia has the **worst prognosis**Q.

Clinical Features

- Unilateral or bilateral reduced vision
- **Crowding phenomenon**Q may be seen: Small letters presented separately are identified by the patient but not when presented in a group.
- Neutral density filter test (NDF): When the patient is asked to read through a NDF, the amblyopic eye shows no change but a decrease in visual acuity is seen in the normal eye.

Treatment

- **Occlusion**Q**:** Occlusion of the good eye forces the child to see with the amblyopic eye and helps in improving vision.
- **Penalization**Q: If a child is not co-operative to occlusion, penalization by instillation of atropine in the good eye is the next option
- Pleoptic therapy
- CAM stimulator

Amblyopia treatment should be tried till the child is 12 years old. **Best results are seen up to 8 years of age**Q.

QUESTIONS

1. **Functions of superior oblique muscle are:** *(PGI)*
   a. Intortion
   b. Extortion
   c. Abduction
   d. Adduction
   e. Depression

2. **Function of superior oblique muscle is:** *(AIPG)*
   a. Elevation with inward rotation
   b. Elevation with outward rotation
   c. Depression with inward rotation
   d. Depression with outward rotation

3. **Which of the following muscles is an intorter?** *(AIIMS)*
   a. Inferior rectus
   b. Inferior oblique
   c. Superior rectus
   d. Lateral rectus

4. **The superior oblique is supplied by:** *(AIPG)*
   a. IIIrd cranial nerve
   b. IVth cranial nerve
   c. VIth cranial nerve
   d. Vth cranial nerve

5. **Primary function of superior oblique is:** *(APPG)*
   a. Elevation b. Depression
   c. Intortion d. Extortion

6. **IIIrd cranial nerve supplies:** *(PGI)*
   a. Lateral rectus
   b. Levator palpebrae superioris
   c. Superior oblique
   d. Superior rectus
   e. Medial rectus

7. **Action of right superior oblique is:** *(PGI)*
   a. Dextroelevation
   b. Dextrodepression
   c. Levodepression
   d. Levoelevation

8. **Functions of superior rectus are:** *(DNB)*
   a. Elevation, extortion, adduction
   b. Elevation, intortion, adduction
   c. Depression, intortion, adduction
   d. Depression, extortion, adduction

9. **Which of the following muscles does not have adduction function?** *(WBPG)*
   a. Medial rectus
   b. Superior rectus
   c. Inferior oblique
   d. Inferior rectus

10. **The yoke muscle of right superior oblique is:** *(DNB)*
    a. Right inferior oblique
    b. Left inferior oblique
    c. Right inferior rectus
    d. Left inferior rectus

11. **Left superior oblique and left inferior rectus are:** *(Kerala PG)*
    a. Yoke muscles b. Agonists
    c. Antagonists d. Synergists

12. **The reciprocal inhibition of antagonist muscle is explained by:** *(AIIMS)*
    a. Sherrington's law
    b. Laplace's law
    c. Hick's law
    d. Herring's law

13. **Equal and simultaneous innervation of yoke muscles is explained by:** *(DPG)*
    a. Sherrington's law
    b. Fick's law
    c. Herring's law
    d. Von Graefe's law

14. **Which of the following is not a grade of binocular single vision?** *(Maharashtra PG)*
    a. Simultaneous macular perception
    b. Retinal correspondence
    c. Fusion
    d. Stereopsis

15. **Hirschberg test is used to detect:** *(APPG)*
    a. Diplopia
    b. Squint
    c. Refractive error
    d. Glaucoma
16. **Cover test is used to detect:** *(DNB)*
    a. Manifest squint
    b. Paralytic squint
    c. Latent squint
    d. Pseudosquint
17. **Pseudo convergent squint is seen in:** *(PGI)*
    a. Thyrotoxicosis
    b. Broad epicanthus
    c. Abducens palsy
    d. Narrow interpupillary distance
18. **Features of infantile esotropia are:** *(PGI)*
    a. Present since birth
    b. Large angle esotropia
    c. Inferior oblique overaction
    d. Surgery is the treatment
    e. High refractive error
19. **Which of the following are true about infantile esotropia?** *(PGI 2015)*
    a. Onset after 1 year of age
    b. Amblyopia may develop
    c. Angle of deviation is large and fixed
    d. Surgery should be done only after 2 years
    e. Minimum refractive error
20. **Treatment of refractive accommodative esotropia is:** *(AIIMS)*
    a. Surgery
    b. Occlusion therapy
    c. Convergence exercises
    d. Correction of refractive error
21. **True regarding accommodative esotropia :** *(PGI)*
    a. Glasses are used when miotics are ineffective
    b. Miotics are used when glasses are ineffective
    c. Miotics are used when AC/A ratio is high
    d. Surgery is the only treatment
22. **A 3-year-old child has esotropia in the right eye. On retinoscopy there is +4.5D hyperopia in right eye and +4D hyperopia in the left eye. The AC/A ratio is normal. What is the probable diagnosis?** *(JIPMER)*
    a. Infantile esotropia
    b. Refractive accommodative esotropia
    c. Non-refractive accommodative esotropia
    d. Duane's retraction syndrome
23. **A 10-year-old complains of headache. His best corrected visual acuity in the right eye is 6/36 and in the left eye is 6/6. Retinoscopy shows +5D in right eye and +1D in left eye. All other ocular examination is normal. What is the possible diagnosis?** *(DNB)*
    a. Optic neuritis
    b. Cortical blindness
    c. Amblyopia
    d. Malingering
24. **Secondary deviation> Primary deviation is a feature of:** *(WBPG)*
    a. Accommodative squint
    b. Paralytic squint
    c. Infantile esotropia
    d. Alternate exotropia
25. **Secondary deviation > Primary deviation in paralytic squint is explained by which law?** *(WBPG)*
    a. Sherrington's law
    b. Herring's law
    c. Park's law
    d. Fick's law
26. **Which of the following is not a feature of paralytic squint?** *(DNB)*
    a. Diplopia
    b. Compensatory head posture
    c. Amblyopia
    d. Secondary deviation is more than primary deviation

27. Amblyopia is best corrected by: *(AIPG)*

a. < 5 years b. < 8 years
c. < 15 years d. < 20 years

28. Treatment of choice for amblyopia is: *(AIIMS)*

a. Occlusion therapy
b. Orthoptic exercises
c. Spectacles
d. Surgery

29. Which of the following is true regarding Duane's retraction syndrome Type I: *(WBPG)*

a. Defective abduction with normal adduction
b. Defective adduction with normal abduction
c. Both adduction and abduction are defective
d. Elevation is defective

30. Limitation of both adduction and abduction is seen in: *(AIIMS)*

a. Duane's Type I
b. Duane's Type II
c. Duane's Type III
d. Double elevator palsy

31. A-V pattern squint: Which of the following is/are true? *(PGI 2015)*

a. The terms 'A' or V pattern squint are labelled when the amount of deviation in squinting eye varies by more than 10° and 15°, respectively, between upward and downward gaze.
b. The terms 'A or V pattern squint are labeled when the amount of deviation in squinting eye varies by more than 20° and 25° respectively, between upward and downward gaze.
c. Usually, overaction of the inferior oblique or weakness of superior oblique leads to A pattern and overaction of the superior oblique or weakness inferior oblique to V pattern
d. Usually, overaction of the inferior oblique or weakness of superior oblique leads to V pattern & over action of the superior oblique or weakness of inferior oblique to A pattern
e. Oblique muscle dysfunction is the commonest cause of AV pattern

ANSWERS AND EXPLANATIONS

1. **a. Intortion, c. Abduction, e. Depression**
2. **c. Depression with inward rotation**
3. **c. Superior rectus**
   Just remember SIN meaning Superiors are Intorters.
   So **superior rectus** and **superior oblique** are **intorters**
4. **b. IVth cranial nerve**
5. **c. Intortion**
6. **b. Levator palpebrae superioris, d. Superior rectus, e. Medial rectus**
   The muscles supplied by the IIIrd cranial nerve are:
   - Levator palpebrae superioris
   - All recti except lateral rectus
   - Inferior oblique
   - Sphincter pupillae and ciliary muscles
7. **c. Levodepression**
   The muscles involved in levodepression are left inferior rectus and right superior oblique
8. **b. Elevation, intortion, adduction**
9. **c. Inferior oblique**
   Just remember RAD meaning recti are adductors except lateral rectus
10. **d. Left inferior rectus**
11. **d. Synergists**
12. **a. Sherrington's law**
13. **c. Herring's law**
14. **b. Retinal correspondence**
15. **b. Squint**
    Clinical tests for squint
    - **Hirschberg test**: This test is used to measure the angle of **manifest squint or tropia**º. Light is shown on the eye. If there is no squint, the reflex should appear centrally on the pupil in both eyes. If there is esotropia or medial deviation of the eye, the reflex will fall temporally. If there is exotropia or lateral deviation of the eye, the reflex will fall nasally. If the reflex falls at the pupillary margin, the angle of deviation is around 15 degrees. If it falls at the limbus, the angle of deviation is around 45 degrees.
    - **Krimsky test or Prism Bar test**: This test is also used to measure the angle of **manifest squint**º by using prisms
    - **Cover-Uncover test**: This test is used to detect **latent squint or heterophoria**º. Here one eye is covered in order to break the fusion. When the cover is removed, the eye under cover will show deviation if there is a phoria.
    - **Alternate cover test**: In this test the two eyes are alternately covered to detect phoria in either eye
    - **Maddox rod test**: This test is used to detect **torsional deviation**º.
    - **Hess charting:** This test is used to confirm the paretic eye in cases of **paralytic squint**º.

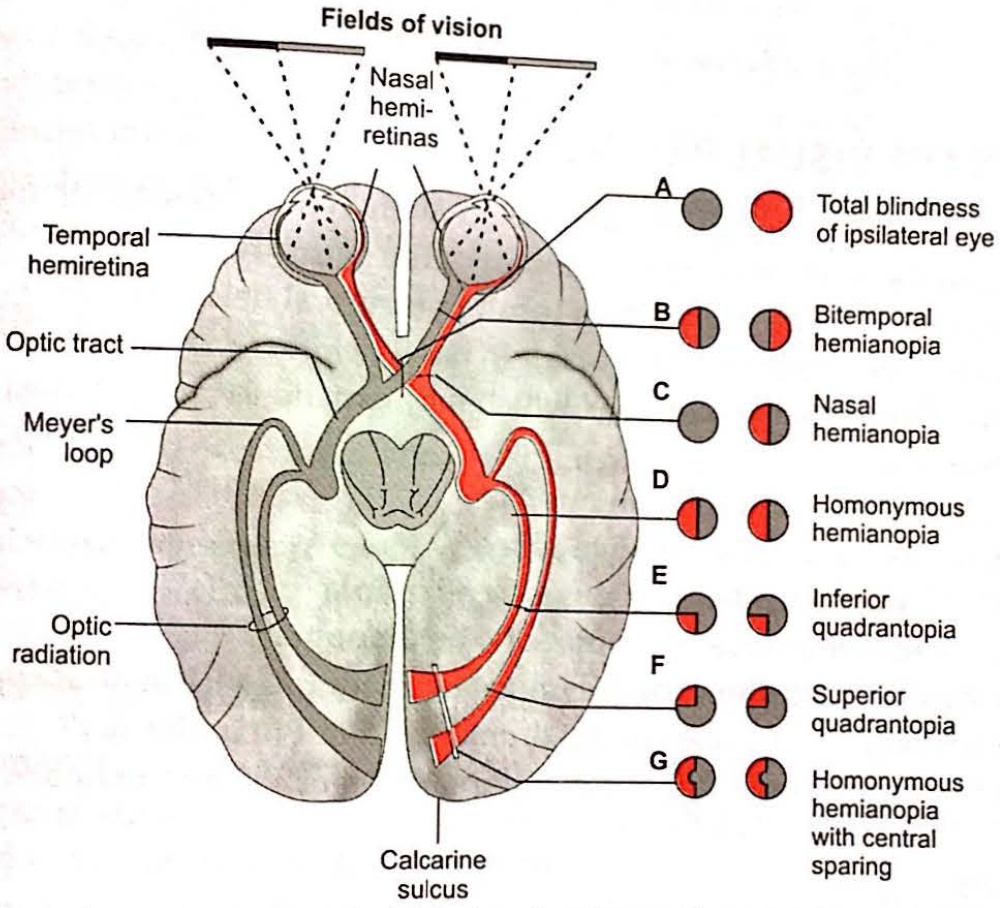
16. **c. Latent squint**
17. **b. Broad epicanthus**
18. **b. Large angle esotropia, c. Inferior oblique overaction, d. Surgery is the treatment**
19. **b. Amblyopia may develop, c. Angle of deviation is large and fixed, e. Minimum refractive error**
    Surgery should be done as early as possible in infantile esotropia to restore ocular alignment and prevent the development of strabismic amblyopia.
20. **d. Correction of refractive error**
21. **c. Miotics are used when AC/A ratio is high**
    Miotics like pilocarpine may be used as a modality of management in non-refractive accomodative esotropia. But it is not a very popular method.
22. **b. Refractive accommodative esotropia**
    The question describes a patient with the following features:
    - Age 3 years
    - Esotropia
    - Hypermetropic refractive error
    - Normal AC/A ratio

    Hence, the answer is refractive accommodative esotropia
    In infantile esotropia, there is minimum refractive error and associated features like cross fixation, nystagmus will be present
    In non-refractive accommodative esotropia, there will be minimum refractive error with high AC/A ratio.
23. **C. Amblyopia**
    In this question, the patient has anisometropia because the refractive error in the right eye is +5D and in the left eye is +1D. The vision in right eye is 6/36 but the ocular examination is normal. This means that there is no organic cause for decreased vision in the right eye. Hence the answer is amblyopia. This is a type of refractive amblyopia (anisometropic).
24. **b. Paralytic squint**
    Paralytic squint is a type of incomitant squint. In incomitant squint secondary deviation is more than primary deviation.
25. **b. Herring's law**
26. **c. Amblyopia**
27. **b. <8 years**
28. **a. Occlusion therapy**
29. **a. Defective abduction with normal adduction**
30. **c. Duane's Type III**
31. **a. The terms 'A' or V pattern squint are labelled when the amount of deviation in squinting eye varies by more than 10° and 15°, respectively, between upward and downward gaze.**
    d. Usually, overaction of the inferior oblique or weakness of superior oblique leads to V pattern and overaction of the superior oblique or weakness of inferior oblique to A pattern
    e. Oblique muscle dysfunction is the commonest cause of AV pattern
    The above described features are seen in pattern strabismus.

Chapter 10

Neuro-ophthalmology

VISUAL PATHWAY



The diagram shows the anatomy of the visual pathway. Note that the nasal hemiretina visualises the temporal field and the temporal hemiretina visualises the nasal field. Thus, lesions involving the nasal fibres of a particular eye affect the temporal field whereas lesions involving the temporal fibres affect the nasal field

The visual impulse travels along the pathway described below

- The visual impulse travels from the **rods and cones of the retina** via the **bipolar cells, ganglion cells** and **nerve fibre layer** to the **optic nerve.** Beyond the optic nerve lies the **optic chiasma.**
- At the level of the chiasma, the fibres from the **nasal part of the retina cross over to the other side whereas the temporal fibres continue on the same side.**
- Beyond the optic chiasma is the **optic tract**. Thus, the optic tract contains the temporal fibres from the same side and the nasal fibres which have crossed over from the opposite side.

- Beyond the optic tract, the fibres pass through the **lateral geniculate body** and the **optic radiation. The inferior fibres of the optic radiation pass through the temporal lobe (Meyer's loop) whereas the superior fibres pass through the parietal lobe of the brain** Q.
- Finally the fibres reach the **occipital lobe** of the brain where the **primary visual area (area no 17)** Q and the accessory visual areas (18 and 19) are located.
- Thus the visual pathway may be summarized as **Optic nerve → Optic chiasma→ Optic tract→ Lateral geniculate body→ Optic radiation→ Occipital cortex**Q

First order neuron	**Bipolar cells in retina**Q
Second order neuron	**Ganglion cells in the retina**Q
Third order neuron	**Lateral geniculate body**Q

LESIONS IN THE VISUAL PATHWAY

Lesions in the visual pathway are associated with typical defects of the visual field depending upon the fibres present in that part of the pathway.
(Note: Lesions involving the nasal fibres result in temporal field defect.
Lesions involving the temporal fibres result in nasal field defect)

- Optic nerve: **Centrocaecal scotoma**Q which may expand to total scotoma
- Optic chiasma:
  - Central lesion: **Bitemporal hemianopia**Q
  - Peripheral lesion: **Binasal hemianopia**Q
- Optic tract: **Incongruous homonymous hemianopia**Q
- Optic radiation: **Congruous homonymous hemianopia**Q
- Occipital cortex: **Congruous homonymous hemianopia with central sparing**Q
- Lesion in temporal lobe: Superior quadrantanopia or **Pie in the sky**Q
- Lesion in the parietal lobe: Inferior quadrantanopia or **Pie on the floor**Q.

Optic Nerve

The optic nerve is about 50 mm long from the globe to the optic chiasma and is divided into the following parts

- Intraocular - 1 mm
- Intraorbital - 30 mm
- Intracanalicular - 6 mm
- Intracranial - 10 mm

Sheaths are continuous with brain meninges.
Nerve fibres proximal to optic disc are myelinated.
Optic nerve function is assessed by:

- **Visual evoked potential (VEP):** Increased latency and decreased amplitude of the response is seen in optic nerve dysfunction
- Visual field assessment: Centrocaecal scotoma is seen in optic nerve disease

- **Colour vision:** Impaired red-green colour vision is seen in all optic nerve diseases except glaucoma where blue- yellow defect is seen.

DEVELOPMENTAL ANOMALIES AND MALFORMATIONS

Optic Disc Pit

- Optic disc is larger than normal
- Contains a pit of variable size in the inferotemporal quadrant
- Serous detachment of macula is a common complication.

Optic Disc Coloboma

- Autosomal dominant
- Discrete white excavation located inferiorly in the disc producing a superior field defect
- Serous detachment of macula is an association
- Charge syndrome(Coloboma, Heart defects, Choanal atresia, Retarded growth, Genital, Ear anomalies) is an association.

Morning Glory Syndrome

- Rare unilateral condition
- Large funnel shaped excavation of the disc surrounded by an annulus of chorioretinal disturbance
- **The blood vessels emerge from the disc like the spokes of a wheel** Q
- Frontonasal dysplasia, NF-2, Phace syndrome are associations.

Optic Nerve Hypoplasia

- Usually unilateral
- Small disc surrounded by concentric chorioretinal atrophy (double-ring sign)
- Associated with midline defects of the brain
- de Morsier syndrome associated with absence of septum pellucidum, agenesis of corpus callosum, is seen in 10%cases.

DISEASES OF THE OPTIC NERVE

Optic Neuritis

Inflammatory. infective or demyelinating disorder affecting the optic nerve
Etiology:

- **Demyelinating diseases like multiple sclerosis**Q
- Viral infections like Measles, Mumps, Chickenpox in children
- Granulomatous inflammation like Sarcoidosis, T.B., Syphilis
- Infection of adjacent structures like meninges, sinuses, orbit.

Anatomically it is classified into:

- Papillitis: Inflammation of the optic disc
- Neuroretinitis: Inflammation of the optic disc and surrounding retina
- Retrobulbar neuritis: Inflammation of the optic nerve behind the globe.

Symptoms:

- Visual Loss : It is **unilateral and sudden**[Q]
- It may be preceded by pain. Painful ocular movements are seen in retrobulbar neuritis
- **Uhthoff's phenomenon:** Impairment of vision increases with increase in body temperature like a hot bath.

Signs:

- Decrease in visual acuity and contrast sensitivity
- Impairment of red green colour vision[Q]
- Pupil shows RAPD[Q]
- Fundus: The fundus picture depends upon the anatomical type.
  - In papillitis, there is **hyperemia** and swelling of optic disc with **blurring** of the disc **margins**. Dilatation and tortuosity of the vessels is also seen.
  - In retrobulbar neuritis, **fundus is normal**[Q]
  - In neuroretinitis, there are features of papillitis with a ring of exudates around the macula. This is called **macular star.**

Investigations:

- Visual field: Centrocaecal scotoma[Q]
- VEP: Increase in the latency of response
- MRI brain and orbit: Helps to rule out multiple sclerosis

Clinical course: There is spontaneous visual recovery in 90% patients over a period of 4-6 weeks. Only about 10% patients may go on to develop chronic optic neuritis. Prognosis is very good.

Treatment: High dose IV Methyl Prednisolone (1 gm daily in two divided doses) for 3 days followed by oral Prednisolone (1 mg/kg/day) for 11 days. Total duration of therapy is 14 days[Q].

Leber's Hereditary Optic Neuropathy (LHON)

- **Maternally inherited mitochondrial DNA mutation**[Q]
- Seen in males between 15-35 years of age
- **Presentation is just like a unilateral case of papillitis but the other eye becomes affected in a few weeks**[Q]
- The disc is hyperemic and oedematous with blurred margins. Dilated vessels extend from the disc to the surrounding retina. This is called **telengiectatic microangiopathy**[Q] and is a distinctive feature of LHON
- Minimal response to steroids is seen with subsequent development of optic atrophy
- Prognosis is very poor
- Vitamin B_{12} injections along with steroids is given.

Anterior Ischaemic Optic Neuropathies (AION)

AION is an important cause of visual loss in the middle age and elderly. It is caused by partial or total infarction of the optic nerve due to occlusion of the **short posterior ciliary arteries**Q. It is of two main types:

- Non-Arteritic AION
- Arteritic AION

Non-Arteritic AION

It is seen in individuals between 45–65 years of age.

Predisposing factors are **atherosclerosis, hypertension, hypercholesterolemia, SLE, PAN, migraine.**

Symptoms

- Sudden, severe, painless loss of vision, with no premonitory visual symptoms
- Unilateral.

Signs:

- Visual acuity and color vision are affected
- Pupil shows RAPD
- Fundus: **Diffuse or sectoral oedema of the disc with disc pallor.** Splinter haemorrhages around the disc may be seen.

Investigation:

- VEP: shows increased latency and decreased amplitude
- Visual field: **Altitudinal scotoma**Q**.**

Treatment: No effective treatment is known.

Arteritic AION

It is seen in patients above 65 years of age and is associated with Giant Cell Arteritis (GCA)Q.

Symptoms:

- Visual loss : Sudden, severe unilateral vision loss accompanied by periocular pain
- May be preceded by **Amaurosis fugax**Q
- **Associated systemic features of GCA like temporal headache, jaw claudication and polmyalgia rheumatica may be present.**

Signs:

- Visual acuity is severely impaired. Colour vision usually cannot be tested due to poor vision
- Pupil shows RAPD
- **Fundus shows severe pallor of the disc with disc oedema. Splinter hemorrhages in the peripapillary region are seen.**

Investigations:

- VEP shows increased latency and decreased amplitude. May be severely extinguished or unrecordable

- ESR: Very High >100 mm/hr
- C-Reactive protein is raised
- Temporal artery biopsy: It is the confirmatory test for diagnosis.

Treatment:

- **IV Methyl Prednisolone (1 gm daily for 3 days) followed by oral Prednisolone** (60-80 **mg/day). It is tapered by 10 mg weekly**
- Maintenance dose of 5-10 mg may be required indefinitely.

Toxic Optic Neuropathy

- It is a bilateral optic neuropathy occurring in response to different drugs and toxins
- It was earlier called toxic amblyopia
- The important toxins and drugs associated are:
  - **Tobacco**
  - **Ethyl alcohol, methyl alcohol**
  - **Ethambutol**[Q]
  - **INH**[Q]
  - **Quinine**
  - **Digoxin.**
- Presentation is bilateral impairment of visual acuity and colour vision. In early stages the disc may be clinically normal but later shows pallor
- Visual fields show bilateral centrocaecal scotoma.

Papilloedema

It is a passive swelling of the optic nerve head secondary to raised intracranial pressure. It is bilateral but may be asymmetrical

Pathogenesis

- The meningeal sheaths of the optic nerve are continuous with the meninges of the brain. So when the ICT increases, there is transmission of pressure to the optic nerve head
- As a result, there is impairment of axoplasmic flow from the optic nerve towards the brain
- There is also associated venous stasis.

Causes:

- Intracranial tumours
- Intra cranial abscess
- Subarachnoid haemorrhage
- Aneurysm
- Benign intracranial hypertension
- Malignant hypertension.

Symptoms:

- Headache, worse on coughing and straining, associated with vomiting
- Transient obscuration of vision (Amaurosis fugax). No definitive visual loss[Q] except in long standing cases

- Sometimes diplopia may be present due to sixth nerve palsy seen as a consequence of raised ICT.

Signs:
- Visual acuity and colour vision are normal
- Pupillary reflexes are normal
- Fundus:
  - In the early stage, there is **blurring of nasal, superior, inferior and then temporal disc margins**[Q]**. Loss of venous pulsation is also seen**[Q]**.**
  - In the established stage, there is hyperemia with severe disc oedema and obliteration of the physiological cup. Veins are dilated, tortuous and engorged. Multiple flame shaped haemorrhages, cotton wool spots are seen in the peripapillary region. Hard exudates are seen at the fovea.

Investigations:
- Visual field: May be normal or **enlargement of the blind spot may be seen**[Q]**.**

Treatment is lowering of ICT by medical, surgical intervention.

Foster Kennedy Syndrome[Q]

- Unilateral papilloedema
- Seen in olfactory lobe tumours
- Ipsilateral optic atrophy due to compression by the tumour
- Contralateral papilloedema due to raised ICT.

Benign Intracranial Hypertension

- It is defined as a condition where raised ICT is seen in the **absence of intracranial space occupying lesion**[Q]. Normal sized ventricles and normal CSF composition is seen
- Seen commonly due to:
  - **Steroid withdrawal**
  - **Oral contraceptive pills**[Q]
  - **Vitamin A toxicity**[Q]
  - **Amiodarone**
  - **Outdated tetracyclines.**
- May also be seen in certain endocrine disorders like hypothyroidism, hypoparathyroidism, Addison's disease, etc.
- Ophthalmic presentation is with **papilloedema.**

Optic Atrophy

Optic atrophy refers to degeneration of the fibres of the optic nerve. It is classified into four types:

- Primary optic atrophy: It is defined as optic atrophy without preceding swelling of the optic disc. The disc is pale with clearly defined disc margins. Causes are:
  - **Hereditary optic neuropathy**
  - **Traumatic optic neuropathy**

- **Toxic optic neuropathy**
- **Recurrent retrobulbar neuritis**
- **Compressive optic neuropathy.**

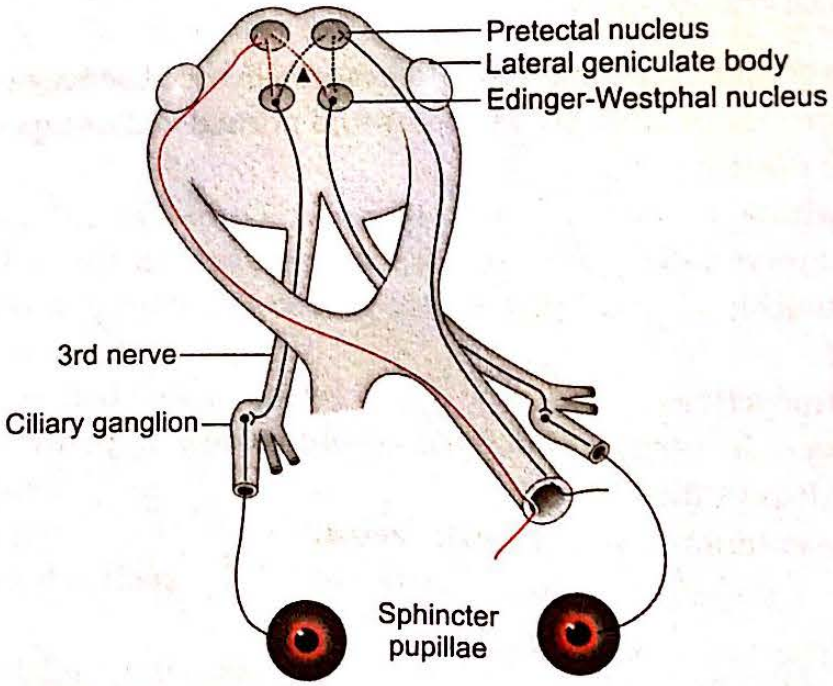
- Secondary optic atrophy: It is optic atrophy with preceding oedema of the optic disc. The disc is pale and dirty looking with ill-defined gliotic margins. Causes are:
  - **Papillitis**[Q]
  - **AION**[Q]
  - **Chronic papilloedema**[Q].
- Consecutive optic atrophy: It is secondary to retinal diseases like **Retinitis Pigmentosa**[Q], **CRAO**[Q].
- Glaucomatous optic atrophy: It is associated with glaucoma and is called as cavernous optic atrophy[Q]

PUPIL

There are two important reflexes involving the pupil:

- Light reflex: Constriction of bilateral pupils occurs when light is shown on one eye. The eye on which light falls, shows **direct reflex** whereas the other eye shows **consensual reflex**
- Near reflex: This has three components namely **constriction of the pupil, convergence and accommodation.**

Light Reflex



Anatomical pathway of light reflex

- The impulse from the retina travels via the **optic nerve, optic chiasma and optic tract**[Q] to reach the **pretectal nucleus**[Q] in the **dorsal midbrain**[Q], which is the centre for the

reflex. At the level of the chiasma, the fibres from the nasal retina cross over to the other side but the temporal fibres remain on the same side

- From the pretectal nucleus, the internuncial neurons connect to the **Edinger-Westphal nucleus (parasympathetic nucleus of oculomotor nerve)**[Q] of both sides
- The efferent impulse travels via the **oculomotor** nerve of both sides up to the **ciliary ganglion**
- Beyond the ciliary ganglion, these parasympathetic fibres are carried by the **short ciliary nerves**[Q] which supply the **sphincter pupillae**[Q] to produce pupillary constriction.

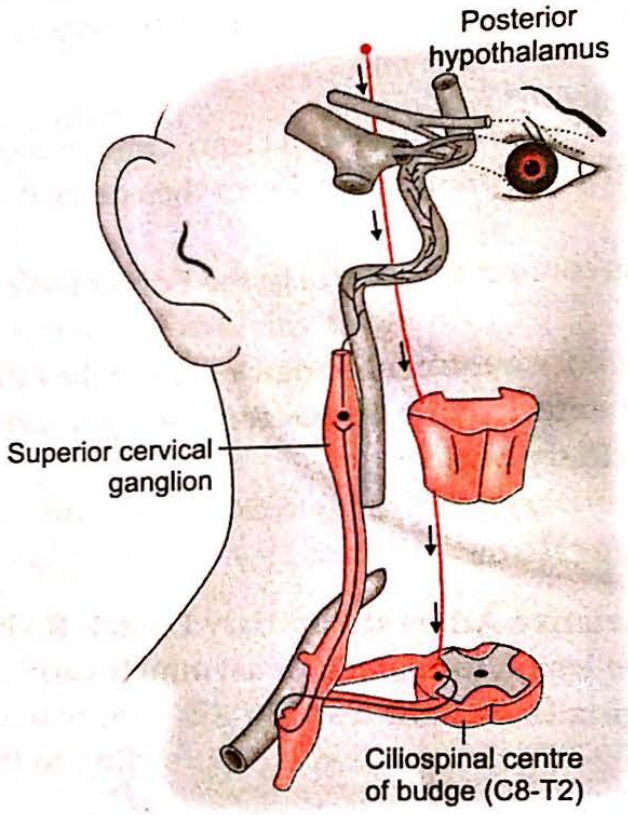
Near Reflex

- The afferent pathway for the near reflex is not clearly understood. The impulse travels from the optic nerve to the occipital cortex. From the cortex it connects to a centre in the **ventral midbrain**[Q]
- The internuncial neurons connect this centre to the **Edinger-Westphal** nucleus of both sides
- The efferent impulse travels via the **oculomotor nerve** to the **ciliary ganglion**. Beyond the ciliary ganglion, **the short ciliary nerves** carry the parasympathetic fibres to the **sphincter pupillae and ciliary muscle.**

Abnormal Pupillary Reactions

- **Marcus Gunn**[Q] pupil **(Relative Afferent Pupillary Defect- RAPD):**
  - Caused by **optic nerve lesion (unilateral or asymmetrical)**[Q]
  - Clinical test is **swinging flash light test**[Q]. In this test, when light is swung to the normal eye, both pupils constrict. When light is swung to the abnormal eye, both pupils dilate[Q]
- **Light-near dissociation:** Light reflex is absent but near reflex is present[Q]. The important examples are:
  - Argyll-Robertson pupil[Q]: Damage to the pretectal nucleus in Neurosyphilis[Q]
  - Perinaud's syndrome: Lesion in the dorsal midbrain
    - Light near dissociation
    - Vertical gaze palsy (Upgaze palsy with normal downgaze)
    - Lid retraction (Collier's sign)
    - Convergence retraction nystagmus.
- **Wernicke's hemianopic pupil:** It is seen in optic tract lesion[Q]. There is impaired pupillary constriction when light is shown on the nasal half of same side and temporal half of the other side
- **Adie's tonic pupil:**
  - Denervation of post-ganglionic supply to the sphincter pupillae and ciliary muscle
  - Both light reflex and near reflex are impaired
  - Anisocoria is seen due to large dilated pupil on the affected side
  - Denervation hypersensitivity: Constriction of affected pupil by 0.125% pilocarpine.

- Horner's syndrome: It is due to disturbance of the sympathetic nerve supply to the dilator pupillae muscle. The sympathetic pathway starts from the **posterior hypothalamus**. The descending fibres reach the **Ciliospinal Centre of Budge** in the spinal cord. From here, the fibres travel to the **superior cervical ganglion** in the neck. The post ganglionic fibres join the **ophthalmic branch of trigeminal nerve**[Q] and reach the dilator pupillae via **the long ciliary nerves**[Q]. Along with the dilator pupillae, these nerves also supply the **Muller's muscle** of the lids and the **sweat gland.**



Anatomical pathway of sympathetic nerve supply

Clinical features:

- Mild ptosis due to paralysis of Muller's muscle[Q]
- Apparent enophthalmos
- Miosis[Q]
- Diminished sweating or anhydrosis[Q]: This is seen in pre-ganglionic cases (Lesion in the pathway prior to the superior cervical ganglion)
- Heterochromia iridis in long standing and pediatric cases.

Pharmacological tests:

- **Cocaine test: Confirms Horner's syndrome**[Q]. On instillation of the drug, normal pupil dilates but Horner's pupil does not
- **Hydroxyamphetamine test: Confirms pre-ganglionic Horner's syndrome.** On instillation of the drug, the pupil dilates in preganglionic Horner's syndrome but it does not dilate in post-ganglionic cases

- **Adrenaline test: Confirms post-ganglionic Horner's syndrome.** On instillation of adrenaline, the dilatation is very prominent and quick in post-ganglionic cases due to denervation hypersensitivity.

CRANIAL NERVE PALSIES

Oculomotor (Third) Nerve Palsy

Anatomy: The course of the nerve from the midbrain to the muscles can be divided into the following parts.

- Nucleus: The nuclear complex is located in the midbrain at level of the superior colliculus. It has both unpaired and paired sub-nuclei.
  - An **unpaired sub-nucleus**[Q] supplies the **bilateral levator muscles**[Q].
  - The **paired superior rectus** sub-nucleus controls the **contralateral muscle**[Q].
  - **Paired sub-nuclei of medial rectus, inferior rectus and inferior oblique control the ipsilateral muscles**
  - **The Edinger-Westphal nucleus supplies parasympathetic input to the sphincter pupillae and ciliary muscles.**
- Fasciculus: The fibres pass through the red nucleus and medial aspect of cerebral peduncle. It is also associated with the cerebellar peduncle. Lesion of the fasciculus leads to:
  - **Benedict syndrome**[Q]: Damage to dorsal part of fasciculus as it passes through the **red nucleus**. Features are **ipsilateral third nerve palsy with contralateral flapping tremors**[Q]
  - **Weber syndrome**[Q]: Damage to ventral part of fasciculus as it passes through the **cerebral peduncle**. Features are **ipsilateral third nerve palsy with contralateral hemiplegia**[Q]
  - **Nothnagel syndrome**[Q]: Damage to the fasciculus in association with **cerebellar peduncle**. Features are **ipsilateral third nerve palsy with ataxia**[Q]
  - **Claude syndrome**: Combination of **Benedict and Nothnagel** syndromes.
- Basilar part: It starts as a series of rootlets which join to form a trunk which traverses the subarachnoid space unaccompanied by cranial nerves. So, **isolated III N palsy is usually basilar in origin**[Q]. This part of the nerve is closely associated with the **posterior communicating artery**
- Intracavernous part: The nerve enters the cavernous sinus and lies in the lateral wall above the fourth nerve. In the anterior part of the cavernous sinus, the nerve divides into superior and inferior divisions which enter the orbit through the superior orbital fissure
- Intraorbital part: The superior division supplies the levator and superior rectus. Inferior division supplies the medial, inferior recti and inferior oblique. It also contains parasympathetic fibres to sphincter pupillae and ciliary muscle.

Pupillomotor fibres which lie in the superficial part of the nerve are supplied by pial blood vessels while main trunk of the nerve is supplied by vasa nervorum. Hence vascular causes of third nerve palsy which affect the vasa nervorum are pupil sparing[Q].

Causes of third nerve palsy:

- **Vascular** causes like hypertension, diabetes **(most common cause in adults)**[Q]
- **Trauma (most common cause in children)**[Q]
- Tumours
- Aneurysm of posterior communicating artery.

Clinical features:

- **Ptosis**[Q] due to involvement of levator palpebrae superioris muscle
- The eye is positioned **down and out**[Q] due to the action of the unaffected muscles, superior oblique and lateral rectus
- Fixed dilated pupil due to involvement of the sphincter pupillae
- Weakness of accommodation due to ciliary muscle involvement.

Trochlear (Fourth) Nerve Palsy

Anatomy: The course of the nerve from the midbrain to the muscle is divided into the following parts:

- Nucleus: Located in the midbrain at the level of the inferior colliculus. It controls the **contralateral superior oblique muscle**
- Fasciculus: The fibres decussate in the anterior medullary velum
- Basilar part: It leaves the brainstem **from the dorsal aspect**[Q]. It curves around the brainstem, passes beneath the tentorial edge, pierces the dura and enters the cavernous sinus
- Intracavernous part: It lies in the lateral wall of the cavernous sinus between the third nerve and ophthalmic nerve. It passes through the superior orbital fissure to enter the orbit
- Orbital part: It innervates the superior oblique **from the orbital surface**[Q].

Causes of fourth nerve palsy: **Trauma (most common)**[Q].

Clinical features:

- Vertical/torsional diplopia[Q]
- **Diplopia is maximum in downgaze** (direction of action of superior oblique muscle)[Q]
- Eye is **deviated upwards** due to underaction of superior oblique (depressor). This is called **hypertropia**[Q]
- **Hypertropia and diplopia worsen in opposite gaze and head tilt to same side**[Q]
- **Compensatory head posture is head tilt to opposite side**[Q]
- Clinical test for superior oblique palsy: **Bielschowsky test**[Q] **and Park's three step test**[Q].

Abducens (Sixth) Nerve Palsy

Anatomy: The course of the nerve from the pons to the muscle is divided into the following parts:

- Nucleus: It lies in the mid portion of **pons** and is closely related to the **facial nerve**
- Fasciculus: It passes ventrally and leaves the brain stem at the pontomedullary junction, lateral to the pyramidal prominence. It is also closely related to the seventh nerve. Lesion in the fasciculus causes:
  - **Millard-Gubler syndrome**[Q]: Lesion involving the fasciculus and pyramidal tract. Features are **ipsilateral sixth and seventh nerve palsy with contralateral hemiplegia**[Q]

- **Basilar part:** It leaves the brain stem at the ponto-medullary junction. This part of the nerve comes in contact with important structures like the tip of the petrous bone and inferior petrosal sinus, before it enters the cavernous sinus. Lesions affecting this part are:
  - **Raised intracranial pressure**: In posterior fossa tumors or pseudotumour cerebri, there is herniation of the brain stem. As a result, the sixth nerves of both sides are stretched over the petrous tip leading to **bilateral sixth nerve palsy (false localising sign)**
  - **Gradenigo syndrome**Q: Inflammation of the petrous apex leads to involvement of fifth, sixth, seventh and eighth cranial nerves.
- Intracavernous part: It lies in the substance of the cavernous sinus in close association with the internal carotid artery and the sympathetic plexus
- Intraorbital part: It enters the orbit through the superior orbital fissure and supplies the lateral rectus.

Causes of sixth nerve palsy:

- **Vascular** causes like diabetes **(most common cause in adults)**Q
- **Trauma(most common cause in children)**Q
- Tumours
- Petrositis, mastoiditis.

Clinical features:

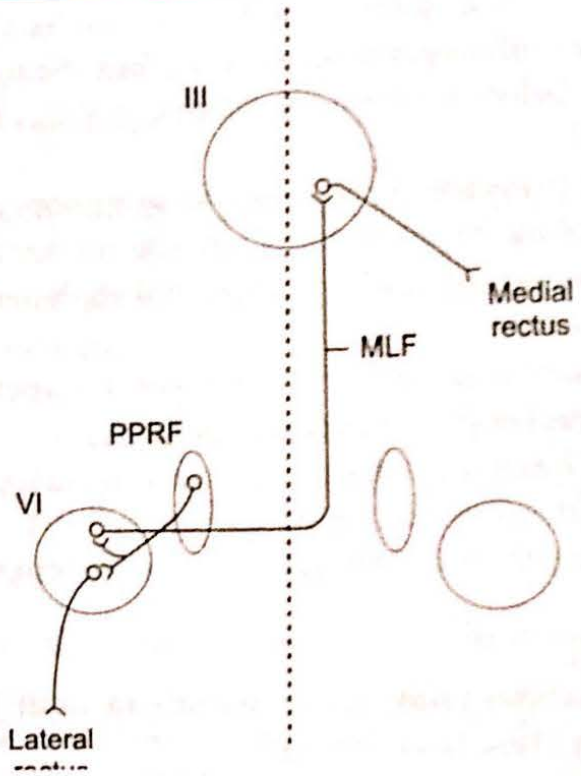
- **Horizontal uncrossed diplopia**Q
- Diplopia is maximum on lateral gaze to the affected side(direction of action of the muscle)Q
- Medial deviation of eyeball or **esotropia**Q
- **Face turn to the same side is the compensatory posture**Q

SUPRANUCLEAR CONTROL OF OCULAR MOVEMENTS

Conjugate eye movements: They are binocular movements where both eyes move synchronously in the same direction. They are called **versions** and are controlled at the cerebral and brainstem levels. **Supranuclear disturbances produce gaze palsies characterised by absence of diplopia.** The important types of conjugate movements are:

- **Saccades:** These are **fast** movements which move the eye from one object to another. They are controlled by the **contralateral frontal lobe**Q
- **Pursuits:** These are **slow** following movements to maintain fixation on one particular object of interest. They are controlled by the **ipsilateral occipital lobe**Q.

Horizontal gaze pathway: Horizontal gaze involves two muscles namely, the ipsilateral lateral rectus and the contralateral medial rectus. The centre for the horizontal gaze is the **PPRF (parapontine reticular formation)**Q. **From here, the neurons connect to the ipsilateral sixth nerve nucleus.** Next the neurons pass through the **medial longitudinal fasciculus (MLF)** to the **contralateral medial rectus subnucleus.**



Horizontal gaze pathway

Lesions in this pathway result in:

- **Horizontal gaze palsy:** This is caused due to lesion in the **PPRF** of the same side[Q]
- **Internuclear ophthalmoplegia (INO):** This is caused by lesion in the **MLF**[Q]
- **One and a half syndrome:** This is caused by lesion in both **PPRF and MLF** of the same side[Q].

Vertical gaze: The centre for the vertical gaze is the rostral interstitial nucleus of the dorsal **midbrain** (riMLF). The important types of vertical gaze palsy are:

- **Perinaud's syndrome:** Upgaze palsy
- **Steel Richardson syndrome:** Downgaze palsy.

NYSTAGMUS

Involuntary, regular, repetitive, rhythmic oscillation of the eyes.

Clinical types:

- **Physiological nystagmus:**
  - **End point nystagmus:** It is seen in extremes of gaze
  - **Optokinetic nystagmus:** It is a jerk nystagmus induced by repetitive moving stimuli like an optokinetic drum (Catford drum)
  - **Vestibular nystagmus:** It is seen when the external auditory canal is stimulated by cold or warm water. It is a jerk nystagmus where the direction of the fast component is remembered by the mnemonic **COWS** meaning "cold opposite warm same".
- **Sensory deprivation nystagmus:** It is seen due to poor vision since early childhood usually less than 2 years of age. Causes are congenital cataract, macular hypoplasia, albinism, etc.

- **Motor imbalance nystagmus:** The important types are:
  - **Ataxic nystagmus:** It occurs in the abducting eye in **Inter nuclear ophthalmoplegia (INO)**[Q]
  - **Down Beat nystagmus:** It is seen in lesions at cervicomedullary junction[Q]
  - **Upbeat nystagmus:** It is seen in phenytoin intoxication and posterior fossa lesions
  - **Sea-Saw nystagmus of Maddox:** It is a special type of nystagmus where one eye elevates and intorts whereas the other eye depresses and extorts. It is seen in **optic chiasma lesions**[Q].

QUESTIONS

1. **Optic nerve is:** *(DPG)*
   a. First order neuron
   b. Second order neuron
   c. Third order neuron
   d. Fourth order neuron
2. **The visual pathway consists of all *except*:** *(PGI)*
   a. Optic tract
   b. Geniculocalcarine tract
   c. Inferior colliculus
   d. Lateral geniculate body
   e. Pretectal nucleus
3. **Bitemporal hemianopia is characteristic of:** *(AIIMS)*
   a. Glaucoma
   b. Optic neuritis
   c. Pituitary tumour
   d. Retinal detachment
4. **Homonymous hemianopia is seen in lesion of:** *(PGI)*
   a. Optic tract
   b. Optic nerve
   c. Optic radiation
   d. Optic chiasma
   e. Occipital cortex
5. **Visual field defect in optic chiasma lesions is/are:** *(PGI)*
   a. Bitemporal hemianopia
   b. Binasal hemianopia
   c. Homonymous upper temporal hemianopia
   d. Heteronymous upper temporal hemianopia
   e. Homonymous upper nasal hemianopia
6. **A homonymous upper quadrantic field defect is due to lesion of:** *(AIIMS)*
   a. Parietal lobe b. Temporal lobe
   c. Occipital lobe d. Optic chiasma
7. **A lesion of the optic radiation involving the Meyer's loop causes:** *(AIIMS)*
   a. Homonymous hemianopia
   b. Centrocaecal scotoma
   c. Superior quadrantanopia
   d. Inferior quadrantanopia
8. **Macular sparing is a feature of lesion in:** *(JIPMER)*
   a. Optic nerve b. Optic tract
   c. Optic radiation d. Occipital cortex
9. **The primary visual cortex is located in:** *(COMEDK 2013)*
   a. Parieto-occipital sulcus
   b. Superior temporal sulcus
   c. Posterior part of calcarine sulcus
   d. Central sulcus
10. **True about cortical blindness is:** *(AIIMS)*
    a. Direct and consensual reflexes are present in both eyes
    b. Direct and consensual reflexes are absent in both eyes
    c. Direct reflex is present but consensual reflex is absent on the normal side
    d. Direct reflex is absent but consensual reflex is present on the normal side
11. **A patient presents with headache and difficulty in vision. On perimetry, right eye shows superotemporal quadrantanopia and left eye shows centrocaecal scotoma. Likely site of lesion is:** *(AIPG)*
    a. Left optic nerve and chiasma
    b. Left optic tract and chiasma
    c. Right optic nerve and chiasma
    d. Right optic tract and chiasma
12. **A female patient presents with loss of vision in both eyes. On examination normal pupillary responses are seen with normal fundus. VER shows extinguished response. The most likely diagnosis is:** *(AIIMS)*
    a. Hysteria
    b. Retrobulbar neuritis

c. Cortical blindness
d. Retinal detachment

13. **Components of the pupillary light reflex are:** *(PGI)*
a. Retina
b. Pretectal nucleus
c. Lateral geniculate body
d. Edinger-Westphal nucleus
e. Calcarine sulcus

14. **Marcus-Gunn pupil is due to:** *(Maharashtra PG)*
a. Defect anterior to chiasma
b. Defect at the optic chiasma
c. Defect posterior to the chiasma
d. Defect in the ciliary muscle

15. **In RAPD, when light is moved from normal to affected eye, there is:** *(AIPG)*
a. Dilatation of both pupils
b. Constriction of both pupils
c. Dilatation in affected eye and constriction in normal eye
d. Dilatation in normal eye and constriction in affected eye

16. **Wernicke's hemianopic pupil is due to lesion in:** *(AIPG/DNB)*
a. Optic tract
b. Optic radiation
c. Optic chiasma
d. Lateral geniculate body

17. **Pupil which responds to convergence but not light is:** *(DPG)*
a. Adie's pupil
b. Argyll-Robertson pupil
c. Hutchison pupil
d. Myotonic pupil

18. **All of the following are true regarding Argyll-Robertson pupil *except*:** *(AIPG)*
a. Near reflex is normal
b. Direct light reflex is absent
c. Consensual light reflex is present
d. Visual acuity is normal

19. **Argyll-Robertson pupil is seen in:** *(DNB)*
a. Multiple sclerosis
b. Midbrain tumour
c. Neurosyphilis
d. All of the above

20. **Dilator pupillae is supplied by:** *(WBPG)*
a. Cholinergic fibres of oculomotor nerve
b. Adrenergic fibres of oculomotor nerve
c. Trigeminal nerve
d. Facial nerve

21. **Which of the following is seen in Horner's syndrome?** *(DNB)*
a. Anhydrosis b. Conjunctivitis
c. Blepharitis d. Optic neuritis

22. **Horner's syndrome causes all *except*:** *(DNB)*
a. Enophthalmos
b. Mydriasis
c. Anhydrosis
d. Narrow palpebral aperture

23. **All of the following are true regarding optic neuritis *except*:** *(AIPG)*
a. Decreased visual acuity
b. Decreased pupillary reaction
c. Abnormal electroretinogram
d. Abnormal visual evoked potential

24. **Optic neuritis is characterised by all of the following *except*:** *(APPG 2014)*
a. Strongly associated with demyelinating disease
b. Subacute unilateral vision loss
c. Pain in exacerbated by ocular movements
d. Optic disc is always abnormal in the acute stage

25. **A child presents with sudden loss of vision in the right eye with painful ocular movements. There are no obvious signs on ophthalmoscopy. The most likely diagnosis is:** *(AIPG)*
a. Optic nerve glioma
b. Retrobulbar neuritis
c. Craniopharyngioma
d. Papillitis

26. A young male patient presents with blurring of vision in the right eye followed by the left eye after 3 months. The disc is hyperemic and oedematous with circumpapillary telengiectasia. Perimetry shows centrocaecal scotoma. The likely diagnosis is: *(AIIMS)*

a. Optic neuritis
b. Acute papilloedema
c. Toxic optic neuropathy
d. Leber's hereditary optic neuropathy (LHON)

27. The most common inherited blindness due to mitochondrial anomaly is: *(AIPG)*

a. Retinitis pigmentosa
b. Leber's congenital amaurosis
c. Leber's hereditary optic neuropathy (LHON)
d. Retinopathy of prematurity

28. Leber's hereditary optic neuropathy (LHON). True is: *(Manipal)*

a. Typically presents in the fourth decade
b. Males do not transmit the disease
c. Is inherited in X-linked fashion
d. The optic nerve becomes pale early in the disease

29. All of the following are true about papilloedema *except*: *(AIPG)*

a. Collection of extracellular fluid
b. Disruption of neurofilaments
c. Stasis of axoplasmic transport
d. Swelling of the axons

30. Fundoscopic features of papilloedema include all of the following *except*: *(AIPG)*

a. Ill-defined disc margins
b. Deep physiological cup
c. Absent venous pulsations
d. Bending of the blood vessels

31. All of the following are true regarding papilloedema *except*: *(AIPG)*

a. It is a non-inflammatory phenomenon
b. Transient loss of vision occurs
c. First sign is blurring of nasal disc margin
d. Sudden loss of vision with painful ocular movements is seen

32. Papilloedema is characterised by all of the following *except*: *(AIIMS)*

a. Loss of venous pulsations
b. Transient obscuration of vision
c. Sudden painless loss of vision
d. Disc oedema

33. Which of the following is/are seen in papilloedema? *(PGI 2013)*

a. Normal blind spot
b. Normal visual acuity even in the last stage
c. Loss of venous pulsations at the disc
d. Sluggish pupillary reaction
e. Normal colour vision

34. Unilateral papilloedema with optic atrophy on the other side is a feature of: *(DPG)*

a. Foster-Kennedy syndrome
b. Fischer syndrome
c. Vogt-Koyanagi-Harada disease
d. WAGR syndrome

35. A 40-year-old lady presents with headache and papilloedema. CT scan of brain shows normal ventricles. Diagnosis is: *(AIPG)*

a. Benign intracranial hypertension
b. Malignant hypertension
c. Papillitis
d. Raised intraocular pressure

36. Disc oedema is seen in: *(DNB)*

a. CRVO
b. CRAO
c. BRVO
d. BRAO

37. Papilloedema is seen in all *except*: *(DNB)*

a. Pseudotumour cerebri
b. CRVO
c. Raised ICT
d. Hypervitaminosis B

38. **Altitudinal field defect is seen in:** *(DNB)*
    a. Ischaemic optic neuropathy
    b. CRVO
    c. CRAO
    d. Papilloedema

39. **Enlargement of the blind spot is seen in:** *(AIPG)*
    a. Papillitis
    b. Papilloedema
    c. Avulsion of the optic nerve
    d. Retinal detachment

40. **A patient with right brow injury due to RTA presents with sudden loss of vision in the right eye. The pupil shows absent direct reflex but normal consensual reflex in the right eye. The fundus is normal. The treatment of choice is:** *(AIPG)*
    a. Intensive intravenous corticosteroids as prescribed for spinal injuries to be instituted within six hours
    b. Pulse methyl prednisolone 250 mg four times a day for three days
    c. Oral prednisolone 1.5 mg/kg body weight
    d. Emergency optic canal decompression

41. **All of the following drugs can cause optic neuropathy *except*:** *(AIPG)*
    a. Rifampicin b. Digoxin
    c. Chloroquine d. Ethambutol

42. **Vitamin B_{12} deficiency causes:** *(AIPG)*
    a. Bitemporal hemianopia
    b. Binasal hemianopia
    c. Heteronymous hemianopia
    d. Centrocaecal scotoma

43. **Optic atrophy is not seen in:** *(AIIMS 2013)*
    a. Retinitis pigmentosa
    b. Methanol poisoning
    c. Central retinal artery occlusion
    d. Polypoidal choroidal vasculopathy

44. **Consecutive optic atrophy is seen in:** *(UPPG)*
    a. Papilloedema
    b. Papillitis
    c. Retinal detachment
    d. Retinitis pigmentosa

45. **A 15-year-old boy has bilateral optic atrophy with diabetes mellitus and diabetes insipidus. The likely diagnosis is:** *(AIPG)*
    a. Kjer syndrome
    b. Behr syndrome
    c. Wolfram syndrome
    d. Leber's congenital amaurosis

46. **Which of the following can cause third nerve palsy:** *(PGI)*
    a. Posterior communicating artery aneurysm
    b. Tolosa-Hunt syndrome
    c. Midbrain infarct
    d. Pons infarct
    e. Lateral medullary syndrome

47. **Oculomotor nerve palsy causes all *except*:** *(AIIMS)*
    a. Miosis
    b. Ptosis
    c. Outward deviation of eyeball
    d. Diplopia

48. **Isolated third nerve palsy with pupillary sparing is seen in:** *(AIPG)*
    a. Aneurysmal rupture
    b. Trauma
    c. Diabetes
    d. Raised ICT

49. **The frequent cause of isolated IIIrd, IVth and VIth cranial nerve palsies in adults is:** *(COMEDK 2015)*
    a. Microvascular ischaemia
    b. Oligodendroglioma
    c. Posterior cerebral artery aneurysm
    d. Brain stem infarction

50. **A 72-year-old patient presents with diplopia. Which of the following features**

is suggestive of IIIrd nerve palsy due to posterior communicating artery aneurysm? *(JIPMER 2015)*

a. Convergent squint
b. Pupil not reacting to light
c. Constricted pupil
d. Exophthalmos

51. Lateral rectus palsy is characterized by: *(AIPG)*

a. Crossed diplopia
b. Uncrossed diplopia
c. Downward deviation of the eyeball
d. Upward deviation of the eyeball

52. In right lateral rectus palsy, all are true *except*: *(AIIMS)*

a. Face turn to left
b. Medial convergent squint
c. Inability to abduct right eye
d. Horizontal diplopia

53. Feature of left sided sixth nerve palsy: *(AIIMS)*

a. Accommodative paresis of left eye
b. Ptosis of left eye
c. Adduction weakness of left eye
d. Diplopia in left gaze

54. A patient presents with head tilted to the right side. On examination, he has left hypertropia which increases on looking to the right side. The muscle most likely to be paralysed is: *(AIIMS)*

a. Left superior oblique
b. Left inferior oblique
c. Right superior oblique
d. Right inferior oblique

55. Diplopia in superior oblique palsy is described as: *(AIPG)*

a. Vertical on looking down
b. Vertical on looking up
c. Horizontal on looking in
d. Horizontal on looking out

56. A patient has moderate ptosis with restriction of ocular movements in all directions but no squint or diplopia. The diagnosis is: *(AIIMS)*

a. Thyroid ophthalmopathy
b. Chronic progressive external ophthalmoplegia
c. Myasthenia gravis
d. Multiple cranial nerve palsies

57. Left sided lateral gaze is affected in lesion of: *(AIPG)*

a. Left frontal lobe
b. Left occipital lobe
c. Right frontal lobe
d. Right occipital lobe

58. Horizontal gaze palsy is due to lesion in: *(PGI)*

a. Parapontine reticular formation
b. Pretectal nucleus
c. Medial longitudinal fasciculus
d. Occipital lobe

59. Internuclear ophthalmoplegia is due to lesion in: *(AIIMS)*

a. Occipital lobe
b. Pretectal nucleus
c. Medial longitudinal fasciculus
d. Parapontine reticular formation

60. One and a half syndrome is due to lesion in: *(JIPMER)*

a. Parapontine reticular formation (PPRF)
b. Medial longitudinal fasciculus (MLF)
c. Both PPRF and MLF
d. Occipital lobe

61. A patient presents with diplopia. On examination, adduction deficit is seen in one eye and abducting saccades in the other eye. Convergence is preserved. What is the likely diagnosis? *(AIIMS)*

a. Partial third nerve palsy
b. Internuclear ophthalmoplegia
c. Duane's retraction syndrome
d. Absence of medial rectus muscle

62. Downbeat nystagmus is a feature of: *(PGI)*

a. Cerebellar lesion
b. Arnold-Chiari malformation
c. Pontine lesion
d. Optic neuritis

63. **A patient has right homonymous hemianopia with defective optokinetic nystagmus. The lesion is most likely to be in:** *(AIIMS)*
    a. Frontal lobe b. Occipital lobe
    c. Parietal lobe d. Temporal lobe

64. **Ophthalmoplegic migraine means:** *(AIPG/AIIMS)*
    a. Headache with irreversible loss of optic nerve function
    b. Recurrent third nerve palsy associated with headache
    c. Headache associated with third, fourth and sixth nerve palsy
    d. Headache associated with optic neuritis

65. **Lamina cribrosa is absent in:** *(AIPG)*
    a. Morning glory syndrome
    b. Nanophthalmos
    c. Coloboma of retina
    d. Optic nerve agenesis

66. **Colour vision is checked by which one of the following?** *(Kerala PG 2015)*
    a. Snellen's chart
    b. Goldman's three mirror lens
    c. Slit lamp
    d. Ishihara's isochromatic charts

67. **Holmgren's wool matching is used for assessment of:** *(Bihar PG 2014)*
    a. Visual field b. Visual acuity
    c. Colour vision d. Refraction

ANSWERS AND EXPLANATIONS

1. **b. Second order neuron**
   The second order neurons of the visual pathway are the ganglion cells. The optic nerve is formed by the axons of the ganglion cells, hence the answer.
2. **c. Inferior colliculus, e. Pretectal nucleus**
3. **c. Pituitary tumour**
4. **a. Optic tract, c. Optic radiation, e. Occipital cortex**
5. **a. Bitemporal hemianopia, b. Binasal hemianopia, d. Heteronymous upper temporal hemianopia**
   - At the centre of the optic chiasma, the fibres from the nasal retina of both eyes cross over to the other side. Hence any lesion affecting the centre of the chiasma will cause **Bitemporal hemianopia or Heteronymous hemianopia**
   - Sometimes, in early cases the hemianopia may be incomplete either **heteronymous upper temporal or lower temporal**
   - In the periphery of the chiasma are the fibres from the temporal retina of both eyes. Thus a peripheral lesion may lead to **Binasal hemianopia** (rare).
6. **b. Temporal lobe**
7. **c. Superior quadrantanopia**
8. **d. Occipital cortex**
9. **c. Posterior part of calcarine sulcus**
   The primary visual area (Area no 17) is located in the calcarine sulcus in the occipital lobe of the brain. The accessory visual areas are Area 18 and Area 19.
10. **a. Direct and consensual reflexes are present in both eyes**
    Cortical blindness means loss of vision due to lesion in occipital cortex. Since the centre for the pupillary reflex is located in the pretectal nucleus in the midbrain, this reflex remains unaffected in cortical blindness.
11. **a. Left optic nerve and chiasma**
    This is a slightly tricky question. The question says that the patient has left centrocaecal scotoma. So the left optic nerve is affected. The patient also has an upper temporal field defect on the right side probably due to an early chiasma lesion.
12. **c. Cortical blindness**
    Cortical blindness means loss of vision due to lesion in the occipital cortex like infarct, haemorrhage, etc. The features are:
    - Sudden painless loss of vision
    - Normal pupillary reflex
    - Normal fundus.

    So, in order to rule out hysteria or malingering, VEP is done. In cortical blindness, VEP will show a decreased or extinguished response whereas it will be normal in hysteria or malingering.
13. **a. Retina, b. Pretectal nucleus, d. Edinger-Westphal nucleus**
14. **a. Defect anterior to chiasma**
15. **a. Dilatation of both pupils**

16. **a. Optic tract**
17. **b. Argyll-Robertson pupil**
18. **c. Consensual light reflex is present**
19. **c. Neurosyphilis**
20. **c. Trigeminal nerve**
21. **a. Anhydrosis**
22. **b. Mydriasis**
23. **c. Abnormal electroretinogram**
24. **d. Optic disc is always abnormal in the acute stage.**
    In retrobulbar neuritis, the optic disc is normal in the acute stage
25. **b. Retrobulbar neuritis**
26. **d. Leber's hereditary optic neuropathy (LHON)**
27. **c. Leber's hereditary optic neuropathy (LHON)**
28. **b. Males do not transmit the disease**
29. **b. Disruption of neurofilament**
    In papilloedema, there is back transmission of the subarachnoid pressure to the optic nerve. This leads to axonal stasis and swelling. There is also impaired venous drainage. But there is no disruption of any neuronal fibres in papilloedema.
30. **b. Deep physiological cup**
    There is obliteration of the physiological cup due to disc swelling.
31. **d. Sudden loss of vision with painful ocular movements is seen**
    In papilloedema, transient obscuration of vision or Amaurosis fugax may be seen but there is no significant vision loss.
32. **c. Sudden painless loss of vision**
33. **c. Loss of venous pulsations at the disc, e. Normal colour vision**
    Visual acuity is usually normal but in chronic end stage papilloedema, it may be decreased.
34. **a. Foster-Kennedy syndrome**
35. **a. Benign intracranial hypertension**
36. **a. CRVO**
    Causes of disc oedema are: (unilateral)
    - Papillitis
    - CRVO
    - Optic nerve head drusen
    - Foster-Kennedy syndrome.
37. **d. Hypervitaminosis B**
    Hypervitaminosis A leads to BIH
38. **a. Ischaemic optic neuropathy**
39. **b. Papilloedema**
40. **a. Intensive intravenous corticosteroids as prescribed for spinal injuries to be instituted within six hours**
    The case described is of traumatic optic neuropathy. The treatment is high dose intravenous corticosteroids in the same regimen prescribed for spinal injuries.

41. **a. Rifampicin**
42. **d. Centrocaecal scotoma**
    Vitamin B_{12} deficiency is an important cause of nutritional optic neuropathy.
43. **d. Polypoidal choroidal vasculopathy**
    Polypoidal choroidal vasculopathy is a variant of ARMD and is not associated with the optic nerve.
44. **d. Retinitis pigmentosa**
45. **c. Wolfram syndrome**
    The important types of hereditary optic neuropathies are:
    - Leber's hereditary optic neuropathy (LHON)
    - Kjer's optic neuropathy: It is an autosomal dominant optic atrophy which presents with vision loss in early childhood. It has no significant systemic features
    - Wolfram syndrome: It is an autosomal recessive disorder described by the eponym DIDMOAD meaning diabetes insipidus, diabetes mellitus, optic atrophy, deafness
    - Behr syndrome: It is an autosomal recessive disorder with features of optic atrophy, pyramidal tract signs, ataxia, mental retardation, urinary incontinence, pes cavus
46. **a. Posterior communicating artery aneurysm, b. Tolosa-Hunt syndrome, c. Midbrain infarct**
47. **a. Miosis**
48. **c. Diabetes**
49. **a. Microvascular ischaemia**
    Microvascular ischaemia due to diabetes, hypertension, etc. frequently causes isolated nerve palsies.
50. **b. Pupil not reacting to light**
51. **b. Uncrossed diplopia**
52. **a. Face turn to the left**
53. **d. Diplopia in left gaze**
54. **a. Left superior oblique**
55. **a. Vertical on looking down**
56. **b. Chronic progressive external ophthalmoplegia**
    Chronic progressive external ophthalmoplegia (CPEO) is a condition which presents with gradually progressive bilateral ptosis and involvement of multiple extraocular muscles. Clinically there is minimal squint and no diplopia. The possible causes are:
    - Kearne-sayre syndrome[Q]
    - Oculopharyngeal dystrophy
    - Myotonic dystrophy[Q]
    - Vitamin E deficiency.

    Episodic ophthalmoplegia is seen in:
    - Myasthenia gravis
    - Eaten-Lambert syndrome
    - Botulism
    - Organophosphorus poisoning

- Snake bite
- Familial periodic paralysis.

57. **c. Right frontal lobe**
The centre for the lateral gaze is the ipsilateral PPRF. But the PPRF is under the control of the contralateral frontal lobe. Hence for the left gaze, the centre is the left PPRF which is controlled by the right frontal lobe.

58. **a. Parapontine reticular formation**

59. **c. Medial longitudinal fasciculus**

60. **c. Both PPRF and MLF**

61. **b. Internuclear ophthalmoplegia**
Please refer to the text on pathway of horizontal gaze
The pathway for horizontal gaze may be summarized as
Parapontine reticular formation (PPRF)
↓
Sixth nerve nucleus of the same side
↓ MLF
Third nerve nucleus of the opposite side
Internuclear ophthalmoplegia (INO) is a defect of the horizontal gaze which is caused due to lesion in the medial longitudinal fasciculus (MLF). **The MLF connects the sixth nerve nucleus to the third nerve nucleus of the opposite side.** Thus, in INO, during lateral gaze the following features are seen:
- The adducting eye fails to move because the third nerve nucleus does not receive any input
- The abducting eye moves laterally but suffers from abducting saccades
- Convergence is normal as the pathway for convergence is completely different.

62. **b. Arnold-Chiari malformation**
Downbeat nystagmus is due to lesion at cervicomedullary junction. Arnold- Chiari malformation is one such condition.

63. **c. Parietal lobe**
Lesion in parietal lobe leads to **Pie on the floor or Inferior quadrantanopia (incomplete homonymous hemianopia).** Parietal lobe lesion also leads to defective optokinetic nystagmus. Hence the answer.

64. **c. Headache with third, fourth and sixth cranial nerve palsies**
- Ophthalmoplegic migraine is defined as headache with migranous characteristics accompanied or followed within 4 days by paresis of one or more ocular nerves namely third, fourth and sixth. (most commonly the third cranial nerve). At least two attacks meeting the criterion are required for diagnosis
- Posterior fossa, parasellar and orbital lesions should have been ruled out by appropriate investigations.

65. **a. Morning glory syndrome**

66. **d. Ishihara's isochromatic chart**

67. c. Colour vision

Tests for colour vision are:

- Ishihara's isochromatic chart
- Farnsworth Munsell 100 hue test
- Holmgren's wool matching test
- Lantern test
- Nagel's anomaloscope.

Chapter 11

Ocular Manifestations of Systemic Diseases

Neurofibromatosis I

- Plexiform neurofibromas on the eyelids (S-shaped eyelid)
- **Enlarged corneal nerves**[Q]
- Congenital glaucoma
- Congenital ectropion uveae
- **Lisch nodules**[Q]
- Choroidal naevus
- Retinal astrocytoma
- **Optic nerve glioma**[Q]
- Spheno-orbital encephalocoele

Neurofibromatosis II

- Posterior subcapsular cataract
- Hamartomas of retinal pigment epithelium and retina

Sturge-Weber Syndrome

- Episcleral haemangioma
- Iris heterochromia[Q]
- Ipsilateral glaucoma

Tuberous Sclerosis

- Atypical iris coloboma
- Iris hypopigmentation
- Retinal astrocytoma

Von Hippel–Lindau Syndrome

- Retinal haemangiomas
- Optic nerve haemangioma

Myasthenia Gravis

- Bilateral ptosis
- Extraocular muscle weakness leading to diplopia
- Nystagmoid movements

Myotonic Dystrophy

- Bilateral ptosis
- Pupillary light near dissociation
- **Presenile cataract (Christmas tree cataract)**[Q]
- Pigmentary retinopathy
- Low pressure

Multiple Sclerosis

- **Retrobulbar neuritis**[Q]
- **Internuclear ophthalmoplegia**[Q]
- Nystagmus
- Extraocular muscle palsies
- **Intermediate uveitis**[Q]

Marfan Syndrome

- High myopia
- Megalocornea
- **Keratoconus**[Q]
- Cornea plana
- Angle anomaly and glaucoma
- **Ectopia lentis**[Q]
- **Microspherophakia**[Q]
- Retinal detachment

Chapter 12

Miscellaneous Topics

BLINDNESS

WHO definition	BCVA less than	BCVA equal to or more than	NPCB definition
Normal vision	------	6/18	Normal vision
Low vision	6/18	6/60	Low vision
Low vision	6/60	3/60	**Economic blindness**[Q]
Blindness	3/60	1/60	**Social blindness**[Q]
Blindness	1/60	Perception of light	**Manifest blindness**[Q]
Blindness	No light perception	--------	Absolute blindness

- BCVA means Best Corrected Visual Acuity in the better eye
- NPCB means National Program for Control of Blindness

Thus according to **WHO, definition of blindness is:**

- **BCVA in the better eye less than 3/60 which means inability to count fingers under good illumination at 3 meters**[Q]
- **Visual field less than 10 degrees in the better eye**[Q]

According to **NPCB, the definition of blindness is:**

- **BCVA in the better eye less than 6/60 (Snellen's chart)**[Q]
- **Visual field less than 20 degrees in the better eye**[Q]

Most common cause of blindness in India: Cataract[Q]

Most common cause of low vision or ocular morbidity in India: Refractive errors[Q]**.**

VISION 2020

- **Vision 2020: Right to Sight** is a global initiative by the WHO to eliminate avoidable blindness.
- It was launched at Geneva in 1999. The important global partners are:
  - International agency for the prevention of blindness
  - Christoffel-blinden mission
  - Hellen Keller International
  - Sight Savers International
  - ORBIS international
  - Rotary International

- International organization against trachoma
- International association of Lions' Club
- International Council of Ophthalmology
- World Council of Optometry

- The diseases targeted globally under Vision 2020 are:
  - **Cataract**[Q]
  - **Refractive errors**[Q]
  - **Onchocerciasis**[Q]
  - **Childhood blindness**[Q]
  - **Trachoma**[Q]
- The Indian chapter of Vision 2020 was launched at Goa in 2001. The diseases targeted under Vision 2020 India are:
  - Cataract
  - Refractive errors
  - Childhood blindness
  - Trachoma
  - **Glaucoma**[Q]
  - **Diabetic retinopathy**[Q].

CAUSES OF LOSS OF VISION

Causes of sudden painful loss of vision
Trauma
Acute keratitis
Acute angle closure glaucoma
Acute iridocyclitis

Causes of sudden painless loss of vision
Vitreous haemorrhage
Central retinal artery obstruction (CRAO)
Branch retinal artery obstruction (BRAO)
Central retinal vein obstruction (CRVO)
Branch retinal vein obstruction (BRVO)
Retinal detachment
Central serous retinopathy
Optic neuritis (pain usually only on ocular movements)
Cortical blindness

Causes of gradual painless loss of vision
Refractive errors
Pterygium
Keratoconus
Corneal dystrophies/degenerations
Open angle glaucoma
Cataract
Diabetic retinopathy
Retinal dystrophies like Retinitis Pigmentosa, Stargardt's disease etc
Age related macular degeneration (ARMD)
Compressive lesions in the brain

QUESTIONS

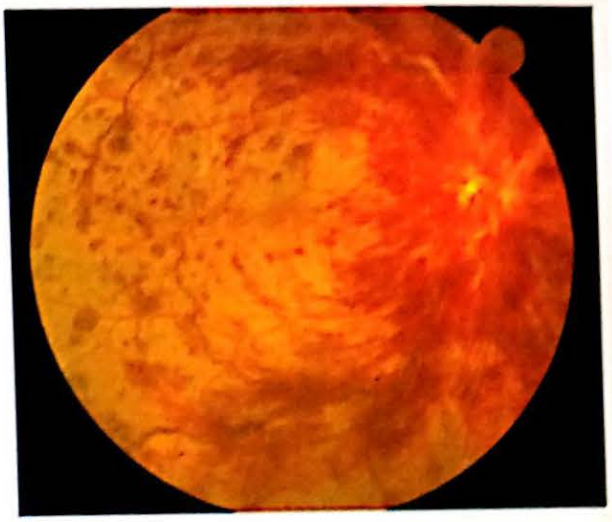
1. **Which of the following is not included under global Vision 2020 Program?** *(AIIMS)*
   a. Cataract b. Refractive error
   c. Trachoma d. Glaucoma
2. **The visual acuity used as cut off for school screening program is:** *(AIIMS)*
   a. 6/12 b. 6/9
   c. 6/6 d. 6/18
3. **Most common cause of ocular morbidity in India:** *(AIPG)*
   a. Cataract
   b. Refractive error
   c. Trachoma
   d. Vitamin A deficiency
4. **WHO criteria for blindness means visual acuity less than:** *(AIIMS)*
   a. 6/18 b. 6/60
   c. 3/60 d. 1/60
5. **A lady presents with visual acuity of less than 6/60 in the right eye and 3/60 in the left eye. According to NPCB, her level of impairment is:** *(AIIMS)*
   a. Social blindness
   b. Economic blindness
   c. Absolute blindness
   d. Manifest blindness

ANSWERS AND EXPLANATION

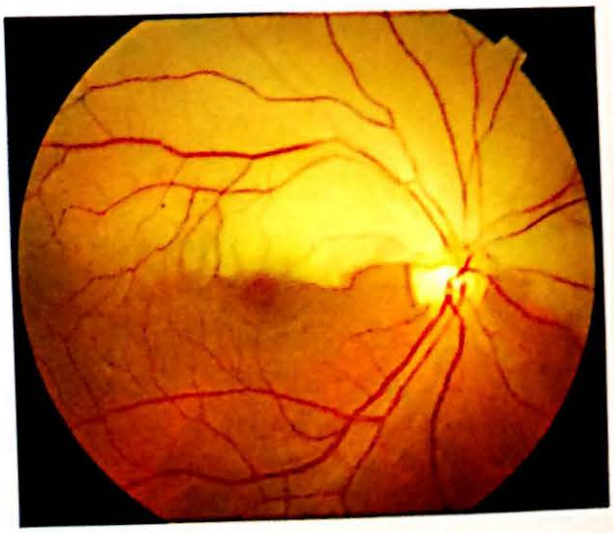
1. **d. Glaucoma**
2. **b. 6/9**
   Children with visual acuity less than 6/9 are referred for further evaluation. The initial screening is done by the school teachers and then referred to the Para Medical Ophthalmic Assistants (PMOA) at the upgraded PHC.
3. **d. Refractive error**
4. **c. 3/60**
5. **b. Economic blindness**

Image-based Questions

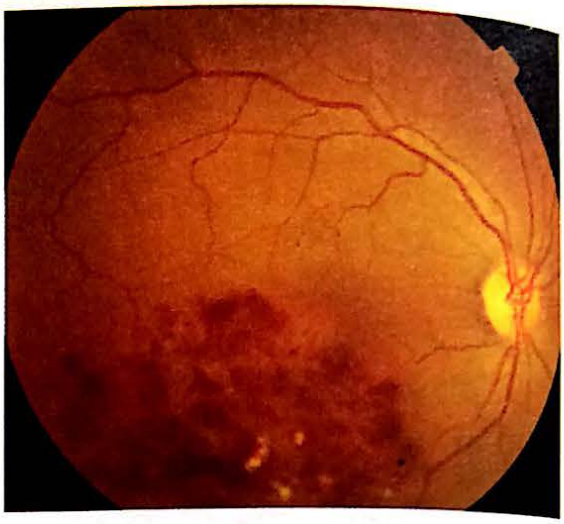
1. **This is the fundus picture of a 60-year-old male patient who presented with sudden loss of vision in the right eye. He is a known hypertensive and diabetic. What is the possible diagnosis?**
   a. Central retinal artery obstruction
   b. Central retinal vein obstruction
   c. Hypertensive retinopathy
   d. Diabetic retinopathy



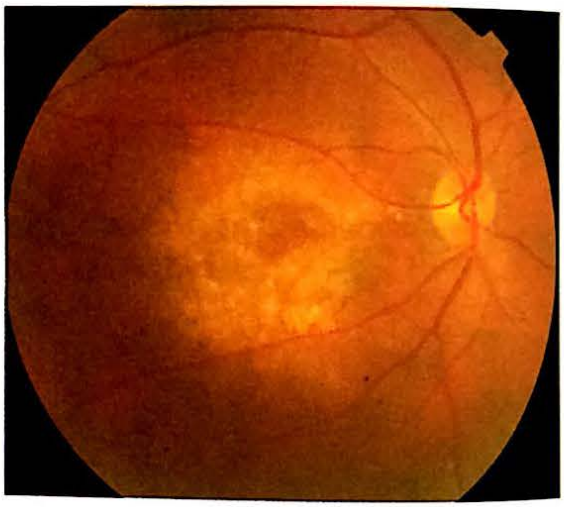
2. **This is the fundus picture of a 60-year-old patient with history of heart disease who presented with sudden onset loss of vision in the right eye. What is the likely diagnosis?**
   a. Central retinal artery occlusion
   b. Branch retinal artery occlusion
   c. Central retinal vein occlusion
   d. Optic neuritis



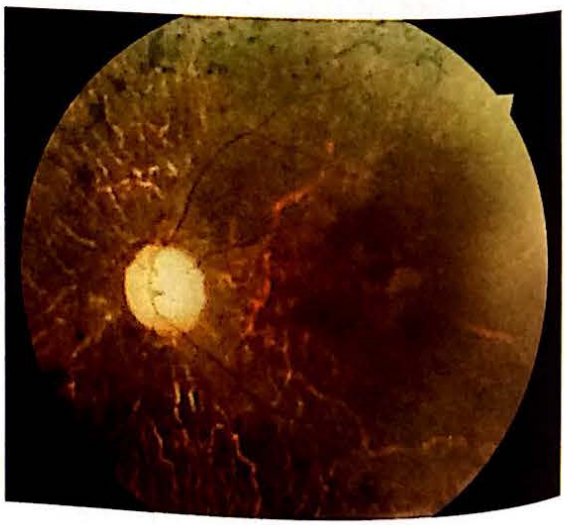
3. **This is the fundus picture of a 50-year-old patient with history of hypertension and diabetes. What is the diagnosis?**
   a. Central retinal vein occlusion
   b. Diabetic retinopathy
   c. Hypertensive retinopathy
   d. Branch retinal vein occlusion



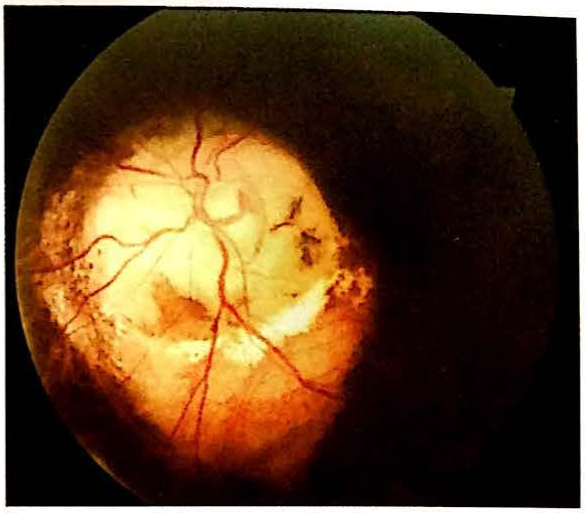
4. **This is the fundus photo of an 80 year old man who presents with gradually progressive decrease in vision in both eyes. On examination he is pseudophakic in both eyes. The fundus in both eyes looks as shown in the photo above. What is the probable diagnosis?**
   a. Central serous retinopathy
   b. Cystoid macular oedema
   c. Age-related macular degeneration
   d. Stargardt's disease



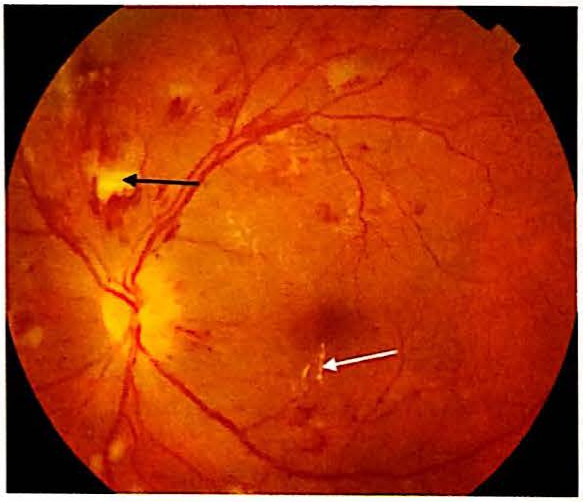
5. **This is the fundus photo of a 25 year-old-man who presents with gradually progressive decrease in vision in both eyes, more in dim light and at night. His father and two other siblings have the same problem. What is the diagnosis?**
   a. Eales' disease
   b. Retinitis pigmentosa
   c. Stargardt's disease
   d. Best's disease



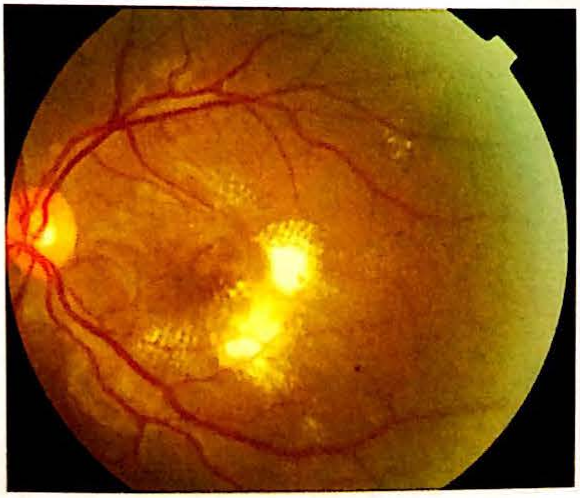
6. **Identify this anomaly:**
   a. Nanophthalmos
   b. Persistent hyperplastic primary vitreous
   c. Morning glory syndrome
   d. Optic disc hypoplasia



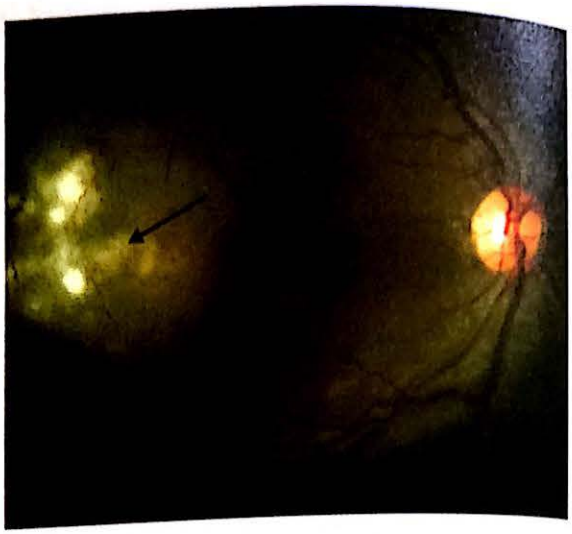
7. **This is the fundus photo of a 50 year old patient with history of diabetes mellitus for the past 8 years. The fundus in both eyes shows diabetic retinopathy. What is indicated by the black arrow in the photograph?**
   a. Hard exudates
   b. Cotton wool spot
   c. Neovascularisation
   d. Microaneurysm



8. **This is the fundus photo of a 6-year-old boy who presented with decrease in vision in the left eye. The right eye was normal. The left eye fundus showed areas of exudation as seen in the photograph. Telengiectatic vessels were also seen. What is the possible diagnosis?**
   a. Eales' disease
   b. Coat's disease
   c. PHPV
   d. Retinal artery macroaneurysm

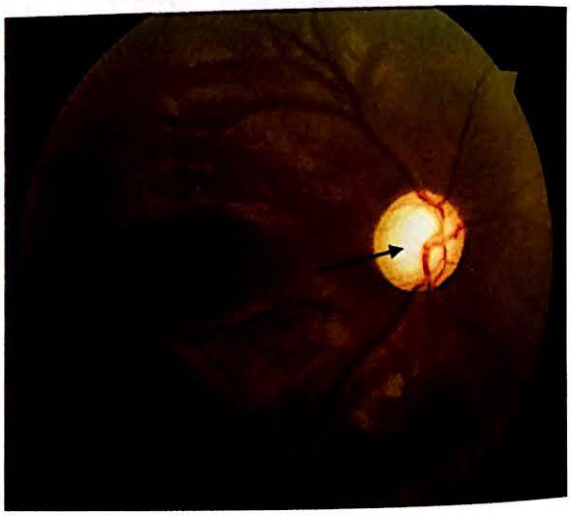


9. **A 25-year-old man presents with complains of pain, redness and decrease in vision in the right for the past one week. On examination, the visual acuity in the right eye is 6/18. The anterior segment is normal. The posterior segment shows vitritis and the fundus photograph is given above. The left eye is normal. What can be the possible diagnosis?**



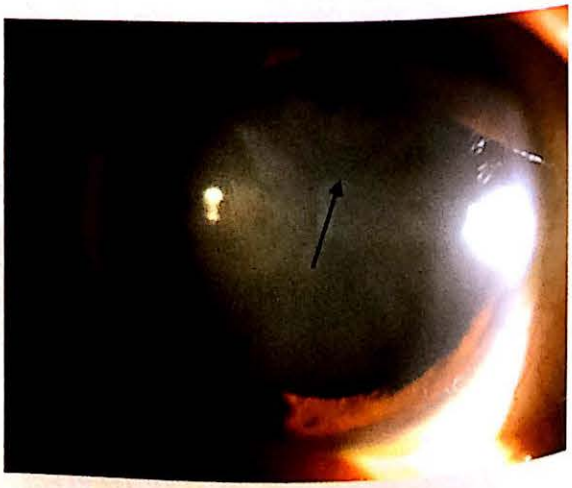
a. Retinal detachment
b. Coat's disease
c. Posterior uveitis
d. Eales' disease

10. **This is the fundus photo of a 55-year-old male who presented with complaints of headache, eye pain and fatigue. What are the investigations to be done in this patient?**



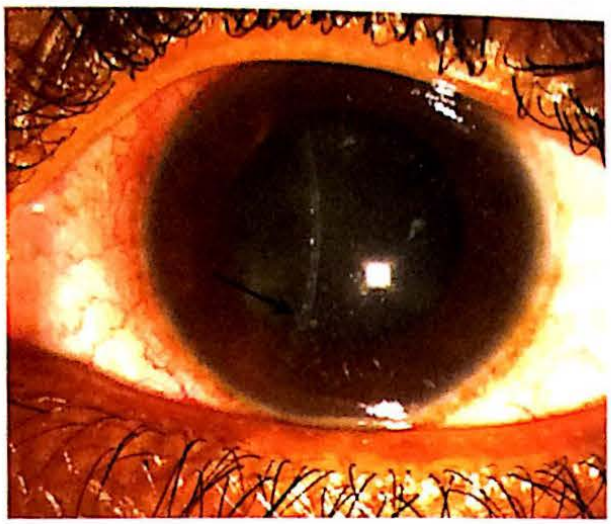
a. Fundus Fluorescein angiography and OCT
b. Gonioscopy, Tonometry and Visual fields
c. Retinoscopy and refraction
d. Indirect ophthalmoscopy

11. **The lens in the photograph shows a typical type of cataract. What history should be elicited from the patient?**

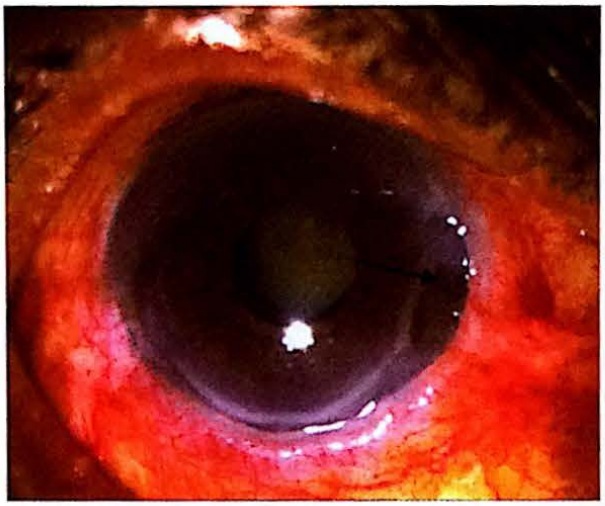


a. Diabetes mellitus
b. Trauma
c. Steroid use
d. History of using glasses

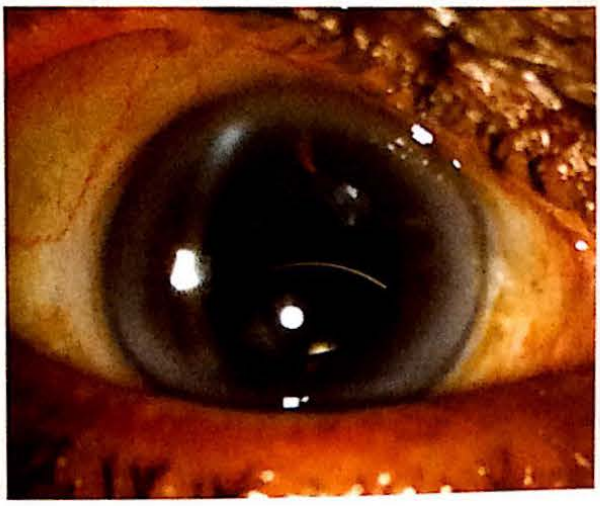
12. **This is the anterior segment photograph of a 20-year-old man who presented with acute anterior uveitis. Looking at the picture, which one of the following seems the least likely diagnosis?**
    a. Tuberculosis
    b. Ankylosing spondylitis
    c. Sarcoidosis
    d. Leprosy



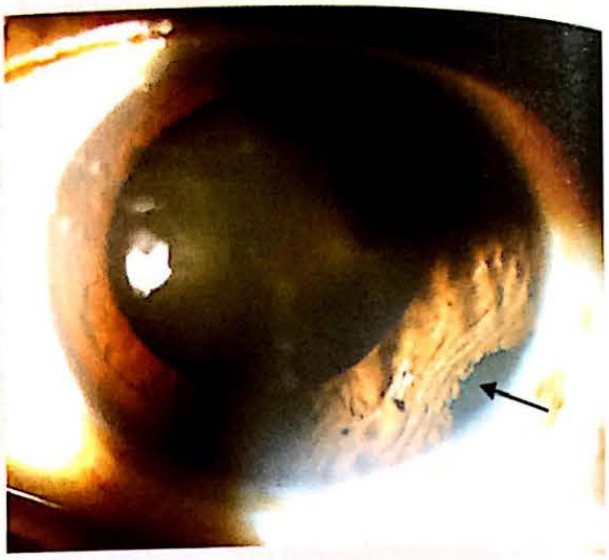
13. **A 50-year-old male patient presented with severe pain and redness in the right eye for the past two weeks. On examination, an ulcer as shown in the photograph above was seen. Scraping and culture from the ulcer bed showed no organism. Systemic work up was also normal. What may be the likely diagnosis?**
    a. Marginal keratitis
    b. Terrien's marginal degeneration
    c. Mooren's ulcer
    d. Fungal corneal ulcer



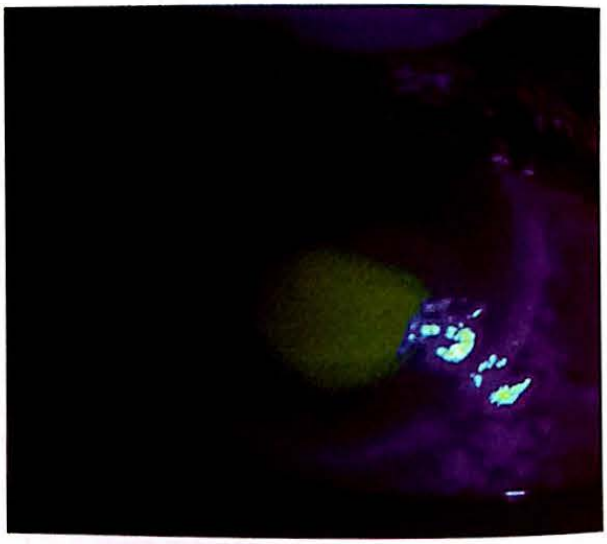
14. **This is the anterior segment photograph of a patient who has history of cataract surgery with IOL done many years ago. He came with complaints of decrease in vision in the pseudophakic eye. What is the diagnosis?**
    a. Sunrise syndrome
    b. Sunset syndrome
    c. Dislocated IOL
    d. Windshield wiper syndrome



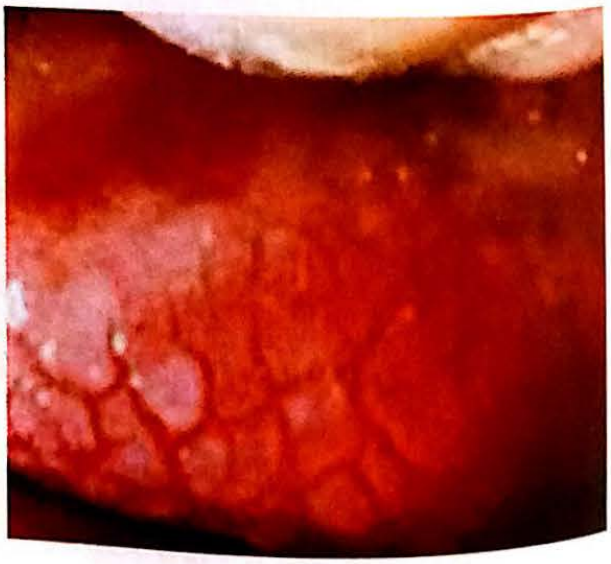
15. **This is the anterior segment photograph of a patient who presented with history of tennis ball injury to the right eye. What does the arrow in the photograph indicate?**
    a. Iris hole
    b. Iridodialysis
    c. Sphincter tear
    d. Multiple pupil



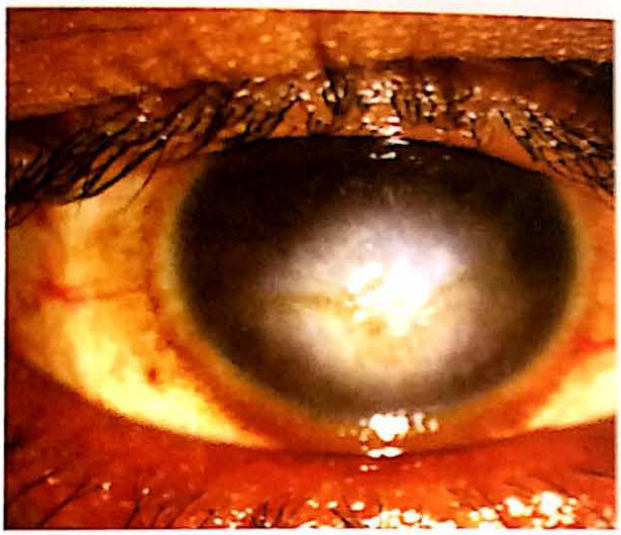
16. **A 10-year-old boy presented with history of cricket ball injury. He had complaints of pain, redness and watering since then. On examination, a large area of epithelial defect was seen on the cornea. It became clearly visible after staining and examination under cobalt blue filter as shown in the photograph above. What was the stain used?**
    a. Rose Bengal
    b. Sodium Fluorescein
    c. Masson's trichrome
    d. Trypan blue



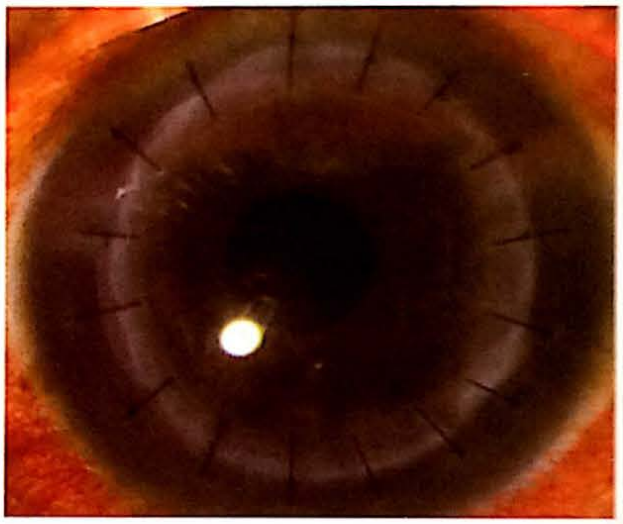
17. **A 10-year-old boy presented with complaints of recurrent episodes of itching and redness in both eyes. The symptoms are worse in summer. The photograph shows the appearance of the tarsal conjunctiva. What is the diagnosis?**
    a. Trachoma
    b. Vernal keratoconjunctivitis
    c. Phlyctenular conjunctivitis
    d. Viral conjunctivitis



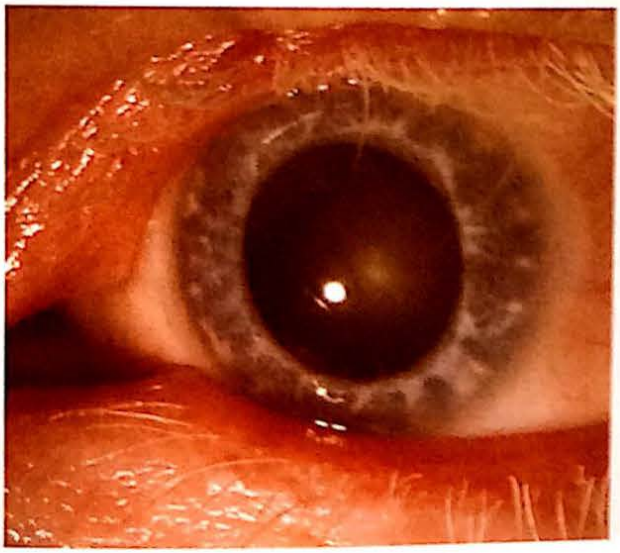
18. **Identify the type of corneal opacity:**
    a. Nebular
    b. Macular
    c. Leucomatous
    d. None of the above



19. **This is the photograph of a patient who has undergone a certain corneal surgery. What is the name of the surgery?**
    a. LASIK
    b. Radial keratotomy
    c. Keratoplasty
    d. Photorefractive keratectomy



20. **This is the anterior segment photograph of a 6-year-old boy presenting with decreased vision in both eyes, intolerance to bright light since early childhood. The child has diffuse hypopigmentation of skin and hair all throughout the body. One other sibling has the same history. Which of the following features about the condition is false?**
    a. Translucent iris
    b. Foveal hypoplasia
    c. Good stereopsis
    d. Nystagmus



ANSWERS AND EXPLANATIONS

1. **Ans. b. Central retinal vein occlusion**
   This is the typical splashed tomato appearance of CRVO where the entire fundus is scattered with haemorrhages.
2. **Ans. b. Branch retinal artery occlusion**
   The fundus photo shows whitening of the superior part of the retina whereas the lower half of the retina is normal. Hence, the answer is branch retinal artery occlusion.
   In central retinal artery occlusion, the whole retina appears white due to ischaemia with red foveal reflex or cherry red spot.
3. **Ans. d. Branch retinal vein occlusion**
   In this fundus picture, haemorrhages are seen only along one arcade of vessels whereas the rest of the fundus is normal. Hence, the answer is BRVO.
   In CRVO, the entire fundus is scattered with haemorrhages.
4. **Ans. c. Age-related macular degeneration**
   The yellowish dots seen at the macula in the fundus picture are drusens (Dry ARMD)
5. **Ans. b. Retinitis pigmentosa**
   The fundus photo shows classical triad of retinitis pigmentosa with pale disc, attenuated blood vessels and bony spicules or pigments in the mid peripheral retina. The blackish dots seen are the pigments or bony spicules.
6. **Ans. c. Morning glory syndrome**
   This is an anomaly of the optic disc which is associated with disc excavation and absence of lamina cribrosa. The disc looks large with the vessels emanating from it like the spokes of a wheel.
7. **Ans. b. Cotton wool spot**
   The photograph shows diabetic retinopathy with intraretinal haemorrhages, hard exudates and cotton wool spots. The small, well circumscribed white dots indicated by the white arrow are hard exudates. The larger ill-defined white areas indicated by the black arrow are cotton wool spots.
8. **Ans. b. Coat's disease**
   Coat's disease is classically described in boys between 5–10 years of age. It is a unilateral condition presenting with telangiectatic vessels and exudation, usually in the inferotemporal quadrant of the retina.
9. **Ans. c. Posterior uveitis**
   The patient presents with pain, redness and decrease in vision. Examination shows vitritis. The arrow in the photograph points to the patch of choroiditis. Hence, the answer is posterior uveitis or choroiditis
10. **Ans. b. Gonioscopy, Tonometry, Visual fields**
    The depression at the centre of the optic disc is called the optic cup. The normal ratio of the area of the cup to the disc is 0.3:1. Here, the fundus picture shows an increased cup: disc ratio with thinning of the surrounding neuroretinal rim. (C: D ratio is roughly 0.7:1). This, along with symptoms of headache and eye strain is suggestive of glaucoma. Hence, the answer.

11. **Ans. b. Trauma**
The typical cataract seen in the picture is rosette cataract (looks like petals of a flower). Hence, the relevant history is that of blunt trauma.

12. **Ans. b. Ankylosing spondylitis**
The black arrow in the picture points to brownish deposits on the corneal endothelium. These are called keratic precipitates. Here, these KPs are large and greasy (mutton- fat KPs) suggestive of granulomatous uveitis. Ankylosing spondylitis causes non-granulomatous uveitis and hence is the answer.

13. **Ans. c. Mooren's ulcer**
The picture shows a corneal ulcer extending to the limbus. This is called as peripheral ulcerative keratitis (PUK). PUK is usually immunological and associated with systemic diseases like SLE, Rheumatoid arthritis etc. Mooren's ulcer is a type of PUK with no systemic association. It is a very painful condition and presents with an indolent ulcer

14. **Ans. b. Sunset syndrome**
In the photograph it can be seen that the IOL is decentred inferiorly. This is called sunset syndrome.

15. **Ans. b Iridodialysis**
Iridodialysis means tearing of the iris from its root.
Features of blunt trauma in the eye
- Lid ecchymosis
- Conjunctival laceration, subconjunctival haemorrhage
- Corneal abrasion, corneal epithelial defect
- **Hyphaema**[Q]: Blood in the anterior chamber
- **Angle recession**[Q]: Trauma to the angle leads to angle recession and **glaucoma**
- Iridocyclitis or traumatic uveitis
- **Iridodialysis:** Tearing of iris from its root. It may give rise to **D-shaped pupil**[Q]
- Traumatic mydriasis
- Sphincter tear leading to irregular pupil
- Lens: **Vossius ring**[Q], **Rosette cataract**[Q]
- Cyclodialysis: Tearing of ciliary body from its root
- **Vitreous haemorrhage**
- Choroiditis, choroidal rupture
- **Berlin's oedema**[Q]: Concussion oedema of the nerve fibre layer of the retina which presents as **cherry red spot**[Q]
- Retinal tears and rhegmatogenous retinal detachment
- **Traumatic optic neuropathy**[Q]
- **Avulsion of the optic nerve**[Q]
- **Globe rupture**
- **Blow-out fracture of the orbit**

16. **Ans. b. Sodium Fluorescein**
Fluorescein stain is used to detect epithelial abrasion or epithelial defect in the cornea because it stains the areas where epithelium is absent. It is best examined under cobalt blue filter. The green area in the photograph is the epithelial defect.

17. **Ans. b. Vernal keratoconjunctivitis**

The photograph shows flat topped elevations on the conjunctiva which are known as papillae. The typical appearance is called cobblestone appearance.

18. **Ans. c. Leucomatous**

The different types of corneal opacity are:

- Nebular: This involves < 1/3rd of the corneal stroma. So the iris details can be seen quite clearly through the opacity
- Macular: This involves >1/3rd to half of the corneal stroma. So iris details can be seen hazily through the opacity.
- Leucomatous: This involves more than 1/2 to full thickness of the stroma. Hence, iris details are not visible through the opacity.
- Adherent leucoma: This is a leucomatous opacity with iris attached to it

19. **Ans. c. Keratoplasty**

Keratoplasty means cornea transplantation. The photograph shows a corneal graft sutured to the host bed by multiple sutures.

20. **Ans. c. Good stereopsis**

The diagnosis is Oculocutaneous albinism.

Albinism is a disorder of melanin synthesis which may affect either the eye alone (ocular albinism) or the eyes, hair and skin (oculocutaneous albinism).

Oculocutaneous albinism is of two types:

- Tyrosinase negative or complete albinos—The features are:
  - Autosomal recessive inheritance
  - **Translucent iris** giving rise to pink eye appearance. This leads to light intolerance
  - Nystagmus
  - Hypopigmented fundus with **foveal hypoplasia**Q
  - **Absence of stereopsis**Q
  - Optic chiasma has very few uncrossed fibres
  - Strabismus may also be present
- Tyrosinase positive or incomplete albinos—The feature are:
  - Less common
  - Autosomal recessive
  - Similar features but with less severity
  - Associated with syndromes like **Chediak-Higashi, Hermanasky-Pudlak**

Self Assessment and Review of

OPHTHALMOLOGY

Salient Features

- Concise text referenced from Clinical Ophthalmology: Kanski, Ophthalmology: Yanoff-Duker and Comprehensive Ophthalmology: AK Khurana
- Important points to remember have been highlighted with superscript Q
- Collection of representative recent MCQs from AIIMS/AIPG/PGI/COMEDK/DNB and State PG entrance examinations
- Photographic quiz has been included keeping in mind the recent trend of image-based questions.

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