Ophthalmology

Transcript-based notes on video lectures in Marrow by Dr Rajarathna Thangavel, M.S, D.N.B (Ophthalmology), FICO (UK)
About the Faculty

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Dr. Rajarathna completed her MBBS with a Gold medal in Ophthalmology. After doing an MS in Ophthalmology, she went on to do an Ocular Oncology fellowship from the University of Michigan. She is versatile in her clinical practice doing Phaco-surgeries, Retinal lasers for Diabetic Retinopathy, Retinopathy of Prematurity (ROP) and Oculoplastics. Above all, she enjoys being a passionate teacher sharing and her knowledge of Ophthalmology, empowering her students with concepts.
Instructions

- Notes are to be used in conjunction with Marrow videos.

- Please refer to the slides feature in the Marrow app for all the images discussed in the video. Only select images are included in the Notes.

- Blank spaces with ☐ are workbook spaces. Please fill it up with relevant content from the videos.

- Notes will not be made for Optional/ Image-based/ MCQ videos.

Please note:

- The information in this book is meant to complement Marrow videos. Content is to be used in conjunction with the videos, and not as a standalone material.

- In case of any discrepancy between videos and notes, you should consider the content in the videos as most accurate.

- The information contained in this book is strictly for educational purposes. Content is not intended as a substitute for professional medical advice, diagnosis or treatment.

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Disclaimer

- These Notes do not cover all the points, especially conceptual points, discussed in the videos. The information in this book is meant to complement Marrow videos and should be used in conjunction with the videos. Special emphasis on certain points and MCQ solving approach has to be understood from the videos only.
- Please refer to the slides feature in the Marrow app for all the images discussed in the video. Only select images are included in the Notes.
ANATOMY OF EYEBALL

INTRODUCTION

Sclera (white opaque)
Cornea (transparent)
Iris (has melanin)

Pupil (opening at centre)

COATS OF EYEBALL

Coats of eyeball

Outer

Ant 1/6th cornea

Post 5/6th cornea

middle

Uvea / uveal tract

a) Iris
b) Ciliary body
c) Choroid

Inner

Nervous layer

- Retina

Lamina cribrosa: It's a part of sclera through which optic nerve leaves eye ball

Choroid & Retina starts from the posterior end of pars plana from one end till other end.

Outside sclera, a loose layer extending from limbus

- Tenon's capsule / fascia

Potential space under tenon's capsule

- Subtenon's space / episcleral space
Foveola: most sensitive part of retina.
Cones are maximum in density

Structures absent: a) Rods
  b) Ganglion cell
  c) Blue cones

Portion of retina close to foveola = central retina.

Portion of retina close to pars plana = ora serrata.

Ora serrata: Peripheral termination of retina at pars plana.
  thinnest portion of the retina.

[Diagram of eye with labeled structures]
PATHWAYS OF AQUEOUS HUMOUR

Posterior chamber
Anterior chamber
Iris corneal angle
Trabecular meshwork
Schlemm's canal

Ciliary process
→
Produce aqueous humour
→
Moves to posterior chamber
→
Through pupil enters anterior chamber

At limbus at inner wall of sclera - Trabecular meshwork (present 360°)
Iris corneal angle / Angle of anterior chamber = 40°
Schlemm's canal - Endothelial lined cells

Aqueous Outflow

Trabecular outflow (90%)
Angle → TM → Schlemm's canal
→
Episceral vein

Uveoscleral outflow (10%)
Posterior chamber
→
Supra choroidal space
ORBIT

Pyramidal in shape
(Base anterior, apex behind)

Roof

Floor

Cross section

Eye ball

Apex

Floor

upper lid

Conjunctiva

Lower lid

Fornix

Conjunctiva - mucous lining inside inner surface of both eyelid
covers the exposed parts of sclera & stops at limbus
CONJUNCTIVA - VIRAL, BACTERIAL, NEONATAL
CONJUNCTIVITIS & TRACHOMA

ANATOMY OF CONJUNCTIVA

Parts of conjunctiva:
1) Palpebral conjunctiva.
2) Forniceal conjunctiva.
3) Bulbar conjunctiva.

Histology of conjunctiva:
1. Epithelium
2. Stroma
3. Fibrous tissue

Epithelium: Non-keratinising stratified squamous epithelium
specialised mucin containing cells in epithelium

↓

Goblet-cell (produce mucin of tear film)

Goblet cells:
- max in density at inferonasal bulbar conjunctiva.
- in fornices
- Decreased Goblet cells in vit -A deficiency & Diabetes mellitus

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Stroma (substantia propia)
Lymphoid tissue present (MALT / CALT)

↓
mucosa / conjunctiva associated lymphoid tissue
Lymphoid hyperplasia ⇒ "Follicle"

### CONJUNCTIVITIS

- Inflammation of conjunctiva
- Presents with conjunctival congestion
- more near the fornix when compared to limbus

**Discharge in conjunctivitis:**
- Watery / Serous - Viral
- Mucoid - Allergic
- Stringy / Ropy discharge - Vernal keratoconjunctivitis
- Mucopurulent - Bacterial, chlamydial
- Purulent - Gonococcal
- Bloody (sanguinous) - Gonococcal conjunctivitis

↓
when the exudates coagulates

it forms a pseudomembrane
- easy to peel
- peeling is painless
  - without bleeding

true membrane:
  adherent to underlying structure
  peeling - difficult
    - painful
    - with bleeding

associated with - c. diphtheriae
  - gonococcal / meningococcal

both true & pseudo membrane looks similar, can only be
differentiated by peeling

both can lead to fibrous scarring of conjunctiva.

---

**Symblepharon**

it's a complication of conjunctivitis

adhesions between palpebral & bulbar conjunctiva.

can be associated with:

  - thermal injuries
  - chemical injuries
  - ocular cicatrical pemphigoid
**CHEMOSIS**

It's a sign of conjunctivitis

Edema of conjunctiva.

Also seen in venous congestion of orbit

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**CAUSES OF CONJUNCTIVITIS**

**Bacterial conjunctivitis**

- **Acute**
  - mcC: staph. aureus

- **Hyperacute**
  - mcC: Gonococcus

- **Chronic (>3 months)**
  - mcC: staph. aureus
  - Can lead to blepharitis (inflammation of lids)

**Viral conjunctivitis:**

mcC: Adenovirus

Causes: Preauricular lymphadenopathy

- seen in viral conjunctivitis
  - Gonococcal conjunctivitis
  - Chlamydial conjunctivitis

---

**ADENOVIRAL CONJUNCTIVITIS**

Follicular conjunctivitis: Serotypes 1 → 11, 14

Pharyngo-conjunctival fever: Sero types 3, 4, and 7

Epidermic keratoconjunctivitis: Sero types 8, 19, and 37

(cornea affected on day 6)
Epidemic keratoconjunctivitis

Small, superficial, punctate keratitis
(Nummular keratitis / opacities)
Came photophobia

**ACUTE HEMORRHAGIC CONJUNCTIVITIS** 00:27:45

1. Enterovirus 70 (mcc) (Apollo conjunctivitis)
   (picorna virus)
2. Coxsackie virus A 24
3. Adenovirus serotypes 8 & 11
4. Str. Pneumoniae (petechiae)

**ANGULAR CONJUNCTIVITIS** 00:30:53

Caused by - Moraxella lacunata → produce proteolytic enzyme
   - Moraxella - Axenfeld diplococcus
   (hence also known as Diplobacillary conjunctivitis)
   - Staph. Aureus

Presents as redness at canthi
   & maceration of lid margins

Treatment:
1) Oxytetracycline eye ointment
2) D.O.C. 2% Zinc Oxide skin Lotion
**GONOCOCAL CONJUNCTIVITIS**

- Only conjunctivitis which must be treated when identified
- Gonococcus can penetrate the intact corneal intact corneal epithelium

**Treatment:**

1. Ciprofloxacin 500mg single dose
2. Rifampicin 600mg BD for 4 days

If systemic involvement of gonococcal infection
- IM Ceftriaxone

---

**NEONATAL CONJUNCTIVITIS**

- Also known as Ophthalmia neonatorum
- Presents with conjestion & papillary reaction
- Conjunctival scraping when stained
- Basophilic inclusions
- Chlamydia trachomatis (serotype D → K)
- (mcC of ophthalmia neonatorum)

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**Etiology of Ophthalmia Neonatorum**

- Day of birth chemical conjunctivitis (sterile inflammation)
- 24-48 hr gonococcal conjunctivitis
- 3-5 days other bacterial conjunctivitis
- 5-14 days chlamydial conjunctivitis
- 7-10 days viral conjunctivitis
- ↓
  - HSV type A
Chemical conjunctivitis:
- watery discharge
- Caused due to

1) Crede’s method:
   1% AgNO₃ solution to prevent gonococcal conjunctivitis

2) 5% povidone iodine eye drop
to prevent chlamydial infection

Gonococcal conjunctivitis can be treated with
- Aqueous penicillin eye drops 10,000 IV or 20,000 IV
  every 5 min x ½ hr
  ↓
  every hr

Chlamydial conjunctivitis + treatment
  erythromycin eye ointment
  +
  erythromycin syr. 50 mg / kg / day divided on 4 dose
  + treatment of parents

Q. mcg of neonatal conjunctivitis = chlamydial conjunctivitis
Q. mcg of neonatal blindness = gonococcal conjunctivitis

<table>
<thead>
<tr>
<th>TRACHOMA</th>
<th>00:48:29</th>
</tr>
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<tbody>
<tr>
<td>Also known as Egyptian ophthalmia.</td>
<td></td>
</tr>
<tr>
<td>mcg: Chlamydia trachomatis serotypes A, B, Ba, C</td>
<td></td>
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<tr>
<td>Transmitted by fingers, fomites, flies</td>
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WHO'S FISTO Staging

F - Follicle
I - Intense inflammation
S - Scarring
T - Trichiasis
O - Opacity

- Follicle: lymphoid hyperplasia
  Herbert's follicles at bulbar conjunctiva.
  - Pathognomonic of trachoma
  "Boiled sago grain" appearance of follicle
  Infective stage of trachoma.
  For endemicity ≥ 5 nos; ≥ 0.5 mm in diameter
  in children aged 0-9 yr

Histopathology of follicles:
  1) Necrosis in them
  2) multinucleated giant cells k/a leber cells

- Intense inflammation: epithelial hyperplasia with vascular core

- Scarring: horizontal whitish line (scar) at palpebral conjunctiva.
  of upper limb:
  - ARL'T's line

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Pannus:

Corneal vascularisation +

Cellular infiltration

Progressive Regressive

Cicatrical entropion of u/L: inward turning of lid margin

\[ \downarrow \]

TRICHIASIS (misdirection of eyelashes)

\[ \downarrow \] (corneal ulcers)

Corneal opacities

m/C infectious cause of preventable blindness: Trachoma.

WHO SAFE strategy for trachoma

S - Surgery

A - Antibiotic (DOC: Azithromycin 1g single oral dose 200mg / kg)

F - Face washing

E - Environment

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CONJUNCTIVA - VERNAL KERATOCONJUNCTIVITIS & MISCELLANEOUS DISORDERS

VERNAL KERATOCONJUNCTIVITIS

- Also known as spring catarrh
- Due to type I hypersensitivity (mainly) to endogenous allergens
  \[ \rightarrow \text{IgE mediated} \]
- Also type IV hypersensitivity (cell-mediated immunity)
- Young boys present in both eyes
- It's seasonal and recurrent
- Occur at the onset of summer
  - Hence known as "warm weather conjunctivitis"
- Spontaneous resolution at puberty
- Clinical features
  - Intense itching*
  - Vigorous eye rubbing*
  - Ropy/String discharge*
  - Redness
  - Burning

SIGNS OF VERNAL KERATOCONJUNCTIVITIS

1. Bulbar form:
   - Has "HORNER TRANTA DOTS"
   - Are gelatinous nodules at limbus
   - Composed of eosinophils & epithelial debris
Lid margins show
"COBBLE STONE
or
PAVMENT - STONE APPEARANCE"

"maxwell - Lyon sign"

Giant papillae repeatedly rubbing on cornea

(?) SHIELD COXED ULCER
when eosinophils infiltrate the cornea, just within limbus, it causes subepithelial scarring.

H/A (i) Pseudogerontoxon
   {pseudo = false; geron = senile; toxon = are}
   (false arcus senilis)
   Also H/A ‘cupid’s bow’

Thinning of cornea leads to its bulging

(ii) Secondary Keratoconus

MANAGEMENT OF VERNAL KERATOCONJUNCTIVITIS 00:10:53

1) Topical Antihistaminics
   a) Mast cell stabiliser
   b) Olopatadine (DOC)

3) Topical steroid (low potency)
   - Loteprednol
   - Fluocinolone

4) Topical immunosuppressants
   - Cyclosporine
   - Tacrolimus

PHLYCTENULAR KERATOCONJUNCTIVITIS 00:13:54

- Type IV hypersensitivity reaction against endogenous allergen
- m/c allergen: Staphylococcal protein
- 2nd m/c allergen: Tubercular protein
- Nodule at limbus / a phlycten with surrounding hyperaemia and corneal infiltration
- Clinical features: Photophobia
  - Foreign body sensation
  - Redness
- Treatment:
  - Dic: Steroids - topical
- Complication:
  - Fascicular corneal ulcer

(there is a bundle of blood vessels taken along with phlycten into cornea)

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**Limbus**

Limbus Anatomy:

- Sclero corneal junction
- Conjunctiva won't grow inside
- Hair pin like structures at limbus

> palisades of vogt
  
  (Limbal stem cells)

Prevents conjunctival growth into cornea.

Limbus stem cell marker:

1. ABCG2 - Universal LSC marker
2. P64
3. Cytokeratin 14 - exclusively at LSC
4. α2 enolase
Limbal stem cell deficiency

1. Conventional aniridia.
   (PA X6 gene mutation)
2. Riley - day familial
dysautonomia.

- Chemical Injury to eye
- Thermal injury to eye
- Pterygium

PTERYGIUM

Fibrovascular growth of conjunctiva & subconjunctival tissue over cornea.

Risk factors (all have limbal stem cell def)

- Sunlight
- Smoking
- PS3 mutation
- HPV infection
- Dry & windy weather

30° N to 30° S of equator of earth is pterygium belt

Clinical features of pterygium:
1) Cosmetic defect
2) Blurring of vision (Astigmatism
   \[ m/c \text{ with the rule astigmatism} \]
   \[ \rightarrow \text{Advanced cases irregular astigmatism} \]
3) Dry eye
4) Diplopia.
5) Blindness (if encroaches upon the pupillary area in visual axis)
parts of pterygium:

Iron deposition at the epithelium of cornea. Stocker's line

Management:

Surgical excision:

A) Bare sclera technique (complication: recurrence)

B) Bare sclera technique + graft

- Conjunctival limbal graft
- Buccal mucosa graft
- Amniotic membrane graft

- Superior bulbar conjunctiva used as conjunctival graft (since LSC are preserved due to less exposure)

- Graft secured by: a) Sutures
  b) Tissue glue (fibrinogen)
  c) Autologous serum (cut-paste technique)

PINGUECULA

- Elastotic degeneration of collagen
- Pterygium precursor
- Pterygium invades Bowman's layer of cornea
BITOT'S SPOT

Temporal part of bulbar conjunctiva

Id

Silvery spots

Due to vit A deficiency

Irreversible changes

SUPERIOR LIMBIC KERATO CONJUNCTIVITIS

- Associated with thyroid eye disease
- Usually hyperthyroid
- Middle aged females
- Filamentary keratopathy

[filaments are mucous strands + epithelial debris]

↓

are painful

Rx: Manage thyroid disorder

N - Acetyl cysteine (mucolytic)

Rabeperride

SUB CONJUNCTIVAL HEMORRHAGE

Bright red colour

May be associated with fracture of base of skull (if post, limit not visualised)

M/c in blunt trauma
Leukoplakia,

Gelatinous or keratinous mass / plaque

→ ACULAR SURFACE
SQUAMOUS NEOPLASIA

(m/c malignancy of conjunctiva)

Risk factors - sunlight

Smoking

HPV

HIV

Pterygium

Feeding vessel: k /a sentinel vessel

mX: surgical resection (wide)

adjacent topical:

1) mitomycin - C eye drops

2) Interferon α eye drops

Conjunctival lymphoma

- Salmon coloured mass

- m/c site of conjunctival lymphome

- inferior fornix

- management: Localised radiation

if systemic lymphome

- chemotherapy

- conjunctival melanoma

- treatment - surgical resection

- brachytherapy

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CORNEA

FUNCTIONS

a) Transmission of light  - Due to Transparency
b) Refraction (2/3 or 3/4)  - Due to curvature
c) Protection  - By multi layered structure of cornea.

LAYERS OF CORNEA

- Epithelium
- Bowman's layer
- Stroma
- Dua's layer
- Descemet's membrane
- Endothelium

EPITHELium

a) Non - keratinised stratified squamous epithelium

b) Types of cell:

- Tight junction
- Flat superficial cell
- Wing cell / umbrella cells
- Basal cells
- Basement membrane

Tight junctions prevents entry of tears into layers of cornea.

c) Layer that completely regenerate without scar
d) 7 days turn over time
**BOWMAN'S LAYER**

Acellular
Compacted layer of collagen type I
Never regenerate if damaged

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**STROMA**

- 90% of corneal thickness
- Thickest layer
- Has proteoglycan matrix - keratan sulphate
  
  Dermatan sulphate
- Collagen - type I, III, IV
- Regular arrangement of collagen
- Interfibrillar spacing is even
- Relatively dehydrated state of stroma
- Keratocytes / keratinocytes

↓

Produce new collagen if damaged, but won't be regular

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**DUA'S LAYER**

Also known as pre-descemet's membrane

Strongest layer of cornea.

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**DESCEMET'S MEMBRANE**

Secreted by endothelium - Basement membrane of endothelium

Collagen type IV, VIII

Peripheral termination of descemet's membrane at limbus

→ Schwalbe's ring / line

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ENDOTHELIUM

a) Single layer
b) Hexagonal shaped cells
c) Endothelial pumps
   $\rightarrow$ Na$^+$, K$^+$ ATPase pump
   $\rightarrow$ Bicarbonate pumps
   Pump out H$_2$O from stroma into aqueous
   $\downarrow$
   Keeping relatively dehydrated
d) Layer most important for maintaining corneal transparency
   - Endothelium
e) Endothelial density (cells/sq. mm)
   Normal at birth: 4000 cells/mm$^2$
   ↓ with age at 0.6% per year
   Normal young adult: 3500 cells/mm$^2$
f) Critical density: 500 cells/mm$^2$
   (density below which stromal edema occurs)
g) Normal endothelial pump function requires normal IOP

INVESTIGATIONS OF CORNEA

i) Keratometry (K) = measurement of corneal curvature
   (central, anterior)
   Normal: 42 to 46 D
   Higher K value: Keratoconus
   Lesser K value: Cornea plana
1. **Pachymetry**: Measurement of corneal thickness
   - Most accurate gold standard: Ultrasound pachymeter
   - CCT: Central corneal thickness
     \[ \text{CCT} : 500-600 \mu \]

2. **Esthesiometry**: Measurement of corneal sensation
   - COCHET - BONNET ESTHESIOMETER

3. **Specular Microscopy**: To visualise endothelium and count endothelium

4. **Confocal Microscopy**:
   - To visualise in vitro:
     - Any layer of the cornea
     - Corneal Nerves
     - Fungal hyphae
     - Acanthamoeba cysts

5. **Corneal Topography**: To access the surface elevation of cornea

6. **Gonioscopy**: To visualise the angle structure
   - Schwalbe's line
   - Very early KF ring

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**Corneal Innervation**

- Trigeminal nerve (V) → Ophthalmic division (V₁)
- Nasociliary nerve
- Long ciliary nerve

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Long ciliary nerves enters the cornea at stroma & creates nerve plexus at:
- Intrastromal
- Subepithelial
- Intraepithelial level

They enter cornea as radial arrangement & ends as unmyelinated free nerve endings

Corneal sensation decreased in:
- Viral Keratitis
- Leprosy
- Diabetes
- Neurotrophic Keratitis
  (Trigeminal nerve involvement)

Corneal nerves: Enlarged and visible:
1) Leprosy: Beaded corneal nerves
2) Acanthamoeba Keratitis: Radial perineuritis
3) Neurofibromatosis - I
4) Amyloidosis
5) Lattice stromal dystrophy
6) Fuch’s endothelial dystrophy
7) Ichthyosis
8) Trauma
9) Meniere’s syndrome
10) Refsum’s disease
FACTORS FOR CORNEAL TRANSPARENCY

1) Avascularity of cornea
2) Unmyelinated nature of corneal nerve
3) Absence of lymphatics
4) Absence of antigen presenting cells in the central cornea
5) Non-keratinising nature of epithelium
6) Tight junction between epithelium
7) Limbal stem cell
8) Intact Bowman's layer
9) Stroma-collagen-regular arrangement
10) Stroma-relatively dehydrated state
11) Endothelial pumps - ᴦ IOP
12) Adequate endothelial count / density

CORNEA-NUTRITION

Cornea's nutrition: Aqueous humor
Perilimbal capillaries

Cornea's metabolism: Aerobic glycolysis
With anaerobic glycolysis can survive up to 6-7 hr

O₂ supply to cornea: Atmospheric air
↓
Dissolves in tear film
↓
Diffuses into cornea.

DIMENSIONS OF CORNEA

V = 11mm
H = 13mm
adult: 12mm

At birth: 10mm

Cornea attains adult size at 2 yrs of age

Microcornea: Diameter ≤ 10mm
Megalocornea:
< 2yrs of age: ≥ 12mm
≥ 2yrs of age: ≥ 13mm

Grades of Corneal Opacities

NEBULA - Only up to or involving Bowman's membrane
MACULA - Basement membrane + < ½ stroma.
LEUCOMA - Basement membrane + ≥ ½ stroma.
ADHERENT LEUCOMA - Iris stuck behind leucoma.
MAX. Visually handicapped - Nebula.

Iron lines in cornea

Iron deposition occurs at epithelium
Stocker's line - Pterygium
Hudson Stahl's line - Old age
Ferry's line - Around filtering bleb of trabeculectomy
Fleischer's ring - Keratoconus

Vortex Keratopathy

Other abnormal deposits at epithelium
They are whorled deposits
Also known as Cornea Verticillata
Causes

1. Amiodarone
2. Chloroquine
3. Hydroxychloroquine
4. Indomethacin
5. Phenothiazine
6. Fabry's disease
CORNEAL ULCERS

INTRODUCTION

Definition: Breach / Discontinuity in epithelium with adjacent stromal involvement (cellular infiltration) / necrosis

C / F: • Photophobia.
• Lacrimation
• Redness - circum corneal / circum ciliary congestion
• Ciliary muscle spasm - pain
• ↓ in vision

INVESTIGATION

1) Corneal sensation → ↓ in viral keratitis
2) Staining of corneal ulcer →
   • fluorescein dye - orange dye (fills floor of the ulcer)
   → under cobalt blue light - green fluorescence
   • Rose Bengal dye(%) - pink colour (stains dead cells in ulcer margins)

3) Corneal scrapings →
   a) Staining -
      • Gram stain (bacterial)
      • 10% KOH (Fungal)
      • Gamoris methenamine
      • Lactophenol cotton blue
      • Acridine orange
   b) C/S: • Blood agar (bact)
      • SDA (Fungal)
      • Non nutrient agar t. e. coli overlay
**TREATMENT**

Non-specific Rx:
- Dark glasses
- Ciliary paralysis (cyclplegia) → Topical cycloplics

Cycloplics:
1) Atropine - 1 week
   - Homatropine - 3 days
   - Cyclopentolate - 1 day
   - Tropicamide - 4-6 hrs

Parasymp muscles → ciliary muscle → paralysis → pain relief
   - Spincter muscle → paralysis → Dilaator muscle overacting
   - (passive) mydriasis

*All cycloplics are mydriatics*

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**BACTERIAL CORNEAL ULCER**

Risk factors:
- Contact lens use
- Foreign body trauma
- Dry eye
- Diabetes
- Previous viral keratitis

**Streptococcus pneumoniae:**

→ Serpiginous corneal ulcer / ulcer
   serpens / creeping corneal ulcer

- Hypopyon (d/t asso. iridocyclitis)
  - Pus in Ant. Chamber
  - It is sterile

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Scanned with CamScanner
Strep pneumoniae → Mcc of Hypopyon bact. ulcer

- Pseudomonas aeruginosa:
  - "Biofilm" formation - Mcc of corneal ulcer in contact lens users
  - Mcc of perforating corneal ulcer
  - Mcc of rapidly spreading corneal ulcer
  - Asso. with - greenish discharge

- Staph. Aureus:
  - Mcc of peripherally located
  - Corneal ulcer

- Staph. Epidermidis:
  - Mcc of corneal ulcer following corneal transplant

Specific management: Tropical fortified antibiotics
1) 4th gen Fluoroquinolones (mono therapy)
2) Vancomycin + Cephalosporins
3) Vancomycin + Aminoglycosides

Bacteria that penetrate intact corneal epithelium:
1) Neisseria meningitidis
   - Gonorrheal
2) Shigella
3) Hemophilus influenzae
4) C. diphtheriae
5) Listeria monocytogenes
Fungal Corneal Ulcer

- mcc → Aspergillus fumigatus
- mcc in immuno compromised → candida Albicans
- Risk factors - 1) vegetable matter trauma
  2) Animal tail injury
  3) Farmers
  4) Chronic topical steroid use

C/F: • Long history - due to less pain
  • Signs are out of proportion to symptoms
  
  - Fungal keratitis:
    - Dry look - leathery appearance
    - ill-defined margins
      - Feathery margins
    - Satellite lesions ⊕
    - Stromal immune ring ⊕
      - aka → Immune ring of wesseley
        → represents deposition of fungal Ag - Ab

    - Fungal typhae can penetrate
      Intact DM / endothelium
    - Endothelial plaque →
    - Fungal hypopyon → infected
      → Immobile (d/t - jibrin)
      i.e: Rxed hypopyon / thick

  Specific Rx: - OCC: → Nataemycin (5% eye drops)
    - C. albicans - amphotericin (0.15% eye drops)
VIRAL KERATITIS

mcc → HSV type 1

Hall mark → ↓ / ⊕ of corneal sensation

Types of ulcers:

i) Epithelial involvement - ulcer
   → Active viral replication ⊕ @ epithelium

ii) Stromal involvement:
   → Hyper sensitivity to viral antigens

Viral corneal ulcer:

i) Dendritic ulcer - 2 types
   a) True dendrite: Terminal
      Knob / bulb ⊕ → HSV

   b) Pseudo dendrite: Terminal
      Knob ⊕ → VZV

ii) Geographic / Amoeboid ulcer:
    DOC → Acyclovir 3% eye ointment
    → 5 times a day

Stromal Keratitis:

i) Nummular Keratitis: coin like deposits of viral Ag - Ab complex @ stroma.
i) Disciform keratitis:

- Disc shaped stromal edema
- Endothelitis (type IV HS - CMV)
- Keratic precipitates

- Block trabecular meshwork
- Open angle glaucoma
- IOP ↑

DOC → Topical steroids + ↓ IOP

Herpes zoster ophthalmicus:

- Secondary reactivation of VZV
- Which is latent at Gasserian ganglion
- MC nerve involved at HZO - Frontal nerve
- Hutchinson’s sign / rule:
  - If there is a lesion at tips of nose

It is usually accompanied by corneal involvement

i.e., via - Nasociliary nerve → Long ciliary nerve supplying cornea
- MC cranial N palsy in HZO → III. Nerve
- Least common CN palsy in HZO → Facial N

TOC → Acyclovir (oral) → 800mg - 5 times a day \times 10-14 days

± Systemic steroids

In immune compromised → IV acyclovir

ACANTHAMOEBA KERATITIS

Risk factors: - Unhygienic practices of cleaning - Contact lens

- Cleaning in home made saline, tap water
- Swimming in public pools
unique C/F → Pain (d/t radial perineuritis)
- Symptoms out of proportion to signs

Corneal ulcer → starts as a limbitis
- Ring stromal infiltration
- Ring stromal abscess
- Satellite lesions
- Pseudo dendrites

DOC → PHMB (Poly hexa methyl biguanide)
Other drugs → I) Propamidone
  a) Chlorhexidine
  b) Neomycin
  c) Bacitracin

NON-INFECTIONOUS CAUSES OF CORNEAL ULCER

Exposure keratitis: - Lagophthalmos (in massive proptosis,
facial N. palsy) - Keratitis in inferior 1/3rd
of cornea.

Neuropasalytic Keratitis ← aka
- Treatment: Tarsorrhaphy (lateral)
  → preservative free lubricant
CORNEAL DEGENERATION AND DYSTROPHY

CORNEAL DEGENERATION

- **ARCUS SENILIS**:
  - Peripheral
  - Old Age-Physiological
  - Lipid degeneration-cholesterol gets deposit @ Bowman’s layer & Anterior stroma
  - Clear space b/w limbus & The deposit k/a Lucid interval of vogt
  - If this is seen in younger individual - Arcus Juvenilis
  - Work up - Blood sugar, fasting lipid profile
  - Arcus Juvenilis seen in diabetics, type II familial hypercholesterolemia

- **Band shaped keratopathy**:
  - Clear interval @ limbus
  - d/t Ca^++ @ Bowman’s layer
  - Calcific degeneration
  - Causes:
    - Dystrophic calcification
      - Chronic iridocyclitis
      - Phthisis Bulbi
      - Silicone oil inside eye
      - "Swiss cheese" Appearance
  - Metastatic calcification
    - Systemic hypercalcemia
management → * Topical EDTA → chelation of Ca$^{2+}$
* Excimer laser → Photo therapeutic Keratectomy

**KAYSER-FLEISCHER RING**

- d/t copper deposition @ DM
- very early KF Ring - Gonioscopy

*Causes:
* Wilsons disease
* Chalcosis

**KERATOCONUS**

It is B/L (asymmetrical), Progressive, non-inflammatory degeneration of central cornea resulting in:

- Ectasia (bulging) of inferior paracentral cornea
- Corneal thinning

Pathophysiology:
- d/t weak covalent bonds b/w stromal collagen

C/F: - Blurred vision

- Astigmatism, myopia (curvatural)
- Frequent change of glasses (teens to 30’s)

Signs:
* Scissoring reflex - seen on retinoscopy
* Earliest clinical sign
* Younig reflex on retinoscopy
* Oil droplet reflex on distant direct ophthalmoscopy
* Vogts striae - vertical tears of DM
* Fleischer ring - iron @ epithelium around base of cone
* Munson’s sig
munson’s sign

on downgaze; V-shaped in dentation of lower lid

Acute Hydrops d/t vogts striae

Apical scarring

Leucoma

management:

1) glasses-cylinders

a) Contact lens-Rigid gas permeable

3) ICRS placement (Intra corneal ring segments)

→ @ midperipheral (Cornea; made of PMMA, it is reversible

4) Corneal transplant (if apical scarring)

→ Keratoplasty

TOC → 5) C.L. - Corneal collagen cross linking using Riboflavin

→ Creating new covalent bonds / strengthen existing bond

∴ Further progression is stopped

Achieved by → Riboflavin + UV-A → Photo polymerisation
**CORNEAL DYSTROPHIES**

They are B/L inherited condition affecting particular layers of cornea. → Not a/w corneal inflammation

→ Not a/w corneal vasculatisaion

Classification of corneal dystrophies:

- **Epithelial:**
  - a) Cogan's dystrophy (meepidys)
  - b) Meesman's dystrophy
  - a) Bowman's:
    - a) Reis-Buckler dystrophy
    - b) Thiel-Behnke dystrophy

- **Stromal:**
  - a) Macular dystrophy (least common stromal)
  - b) Granular dystrophy
  - c) Lattice dystrophy (MC stroma)
  - d) Schnyder's crystalline dystrophy

- **Endothelial:**
  - a) Fuchs' endothelial dys (MC endothelial)
  - b) CHED (Cong Hereditary Endothelial Dys)
  - c) PPMD (Posterior polymorphus dys)

---

**COGANS DYSTROPHY**

- Aka Epithelial Basement membrane Dystrophy (EBMD)
- d/Abnormal hemidesmosomes
- ↑ shedding of epithelium
- Recurrent corneal erosion (painful)
- Shed epithelium looks like finger prints / Dots / maps
  → "finger print-map-dot-dystrophy"
  
  aka
STROMAL DYSTROPHIES

- macular Dystrophy:
  - Least common stromal dystrophy
  - AR in inheritance
  - Ab N deposition → MPS deposits @ stroma.
    → stain: Alcian Blue

- Granular Dystrophy:
  - AD
  - a/w TGF-β1 gene mutation
  - Hyaline deposits → stain: Masson’s Trichrome (Red)

- Lattice stromal Dystrophy:
  - MC stromal dys.
  - a/w TGF-β1 gene mutation
  - Ab N Deposit → Amyloid
    → Stain: Congo red

ENDOTHELIAL DYSTROPHY

1) Fuch’s Dystrophy:
   - MC endothelial dystrophy
   - 4th-5th decades of Life
   - “Guttae” in central endothelium
     → endothelial projections

   “Beaten metal” appearance of endothelium
- Stromal edema 
- Epithelial edema 
- Bullae @ epithelium

\[ \rightarrow \text{Bullous keratopathy} \]

3) CHED: - may a/w SNHL or Nystagmus

\[ \rightarrow \text{Congenital} \]

3) PPMD:
- maybe a/w Alport syndrome
- Congenital
- "Doughnut" shaped lesions in cornea

---

**CONGENITAL CLOUDY CORNEA**

Causes mnemonic - STUMPED

Sclero cornea.

Trauma (forceps delivery - DM tear)

Uterine infection (cong. syphilis - interstitial keratitis)

Mucopolysaccharidosis (except + hunters synd)

Peter's anomaly

ED - Endothelial Dystrophies (except Fuch's dys.)
UVEA-ANATOMY

Uvea:

1) Iris
2) Ciliary Body
   • Pars plicata / ciliary process
   • Pars plana
3) Choroid

IRIS

* Iris Root is the Thinnest portion of Iris
* COLLARETTE is the thickest portion of Iris
* Dilator pupillae muscle is radially arranged
Iris vasculature:

Radial vessel

minor Arterial circle of Iris

at pupillary border

→ major Arterial circle of Iris at iris root

Formed by
i) Anterior ciliary Artery
ii) Long posterior ciliary artery

Iris muscles

Sphincter pupillae/
Constrictor pupillae

• Circular fibres

• MiOsis
    (Constriction of pupil)

• parasympathetic through III C.N
    ↓
    Nerve to Inferior oblique
    ↓
    Reaches short ciliary nerve

Dilator pupillae

• Radial fibres

• MydriasIs
    (Dilatation of pupil)

• Sympathetic
    ↓
    Ophthalmic division of trigeminal nerve
- **Ciliary Body**
  
  - It has two parts  
    - i) Pars plicata / ciliary process  
      - a) Pars plana  
    - ii) Pars plicata / ciliary processes  
  
  - Around 60-70 ciliary processes are present in each eye  
  - Blood-Aqueous-Barrier  
    - Tight junctions between the pigmented ciliary epithelium  
    - Breakdown of blood-aqueous barrier occurs in inflammation  
  
  - a) Pars plana  
    - It is a region where we pass instruments & needles in vitreoretinal surgery / intravitreal injection  
    - It is 4.5 mm from the limbus
CILIARY MUSCLES

- Ciliary muscles receive innervation
  ↓
  Parasympathetic

Though III CN → Nerve to inferior oblique
  ↓
  Short ciliary nerve

- 3 groups of muscle fibres
  ↓
  Circular
  ↓
  Contraction results in accommodation

  Longitudinal
  ↓
  Contraction results in Trabecular Aqueous outflow

  Radial

CHOROID

1) Outer layer of large blood vessels → HALLER'S layer
2) Middle layer of medium-sized blood vessels
   ↓
   SATTLER'S layer
3) Inner layer of capillaries
   ↓
   CHORIOCAPILLARIS
• Venous drainage of choroid → VORTEX veins – four in number

• Equator of eyeball is at the level of vortex veins, 14mm from limbus

• Innermost layer of choroid → Bruch’s membrane

  ↓

  Which forms the basement membrane of RPE (Retinal pigment epithelium)

Bruch’s Membrane Rupture:

  1) LACQUER CRACKS → Seen in pathological myopia.

  2) ANGIOID STEAKS → Seen in pseudoxanthoma elasticum
Causes of physiological miosis

1) Bright light
2) Neonate
3) Elderly
4) Sleep
5) Near vision

Causes of physiological mydriasis
In dim light

Shapes of pupil:

- Key hole pupil
- Pear shaped pupil
- Tear shaped pupil
  Seen in iris coloboma.

Iris coloboma:

- Due to defective closure of embryonic fissure at day 35 of gestation
- Most common location: inferonasal
Ophthalmology

* D-shaped pupil

* Iridodialysis
  ↓
  Detachment of iris root from its ciliary body attachment

Causes
1) Blunt trauma
2) Iatrogenic (surgery)

Festooned pupil:

multiple posterior synechiae
↓
Festooned pupil
↓
Seen in iridocyclitis

Slit - shaped pupil:

seen in ectopia lentis et pupil
RUBEOSIS IRIDIS

- It is also known as Neovascularisation of iris (NVI)

Causes

1) Proliferative Diabetic Retinopathy (most common)
2) Ischemic type of CRVO
3) Ocular ischemic syndrome
4) Rale's disease
5) Retinopathy of prematurity (ROP)
6) Sickle cell Retinopathy
7) Retinoblastoma

- In all these conditions, there is Retinal Hypoxia.

  ↓

  Releases VEGF

  ↓

  Resulting in new vessel formation

- 1st Neovascularisation of iris

  ↓

  Occurs at the pupillary border of iris

  ↓

  It grows onto the surface of iris and reaches the peripheral iris and Angle → Neovascularisation of Angle (NVA)
Neovascularisation of Angle (NVG) 
↓
If infiltrates into the Trabecular meshwork 
↓
Causing Neovascular Glaucoma (NVG)

management of NVI / NVG:

- Pan Retinal photocoagulation (PRP) → Treatment of choice

  - Hypoxic retina.
    ↓ converted
  - Anoxic retina.
  - Macula spared

---

**HETEROCHROMIA IRIS** 00:14:59

---

Heterochromia Iris

↓

Heterochromia iridis
- Difference in color within the same iris

Heterochromia iridum
- Difference in color between the two irises
Congenital Horner's syndrome

- Melanocytes originate from Neural crest cell
- Neural crest cell migration requires sympathetic drive

- In congenital Horner's syndrome
  \[ \downarrow \]
  Sympathetic palsy
  \[ \downarrow \]
  Failure / Arrest of migration of neural crest cells
  \[ \downarrow \]
  Resulting in Hypochromia Iris (On involved side \& miosis)

Differentiating feature between congenital \& Acquired Horner's syndrome \( \rightarrow \) Hypochromia of Iris in congenital Horner's syndrome
Heterochromia Iridum

Heterochromia Iridum

- Hypochromia
  - Cause
    - Congenital Horner's syndrome
- Hyperchromia
  - Causes
    - Rubeosis iridis
    - Siderosis (iron)
      - Foreign body in eye
    - Prostaglandin analogues
    - Melanocytomas / melanomas of iris
IRIS NODULES

Non inflammatory iris Nodules

1) BRUSHFIELD spots
   - Seen in Down's syndrome

2) LISCH Nodules
   - Seen in NeuroFibromatosis
   - Lisch Nodules are Iris Hamartomas
     ↓
     Involving the Iris Pigmented epithelium
   - Lisch Nodules → one of the criteria for diagnosis of
     NeuroFibromatosis-1
Disclaimer

- These Notes do not cover all the points, especially conceptual points, discussed in the videos. The information in this book is meant to complement Marrow videos and should be used in conjunction with the videos. Special emphasis on certain points and MCQ solving approach has to be understood from the videos only.
ANTERIOR UVEITIS

- Inflammation of uveal tract → UVEITIS

ANATOMICAL CLASSIFICATION OF UVEITIS

- Uvea
  - Iris
    - Pars plicata
    - Pars plana
  - Ciliary body
  - Choroid
    - Posterior uveitis
      - Chorioiditis
    - Retino-choroiditis
    - Chorioiditis associated with retinal involvement
    - Chorio-retinitis
      - Retinitis followed by choroidal inflammation

- Iritis
- Cyclitis
- Iridocyclitis

Anterior uveitis

- Primary site inflamed is Aqueous Humor
- Entire uvea is inflamed → Panuveitis
- Principle site of inflammation on anterior uveitis → Aqueous humor
- Principle site of inflammation in intermediate uveitis → vitreous

PATHOLOGICAL CLASSIFICATION

- Uveitis
  - Suppurative
    - Pus formation
      - Panophthalmitis
  - Non-suppurative
    - Non-Granulomatous
    - Granulomatous
PANOPHTHALMITIS

- All the coats of eyeball including Tenon's capsule
  
  Cavities (Anterior chamber + posterior chamber, vitreous)
  
  Aqueous

- It is a pus filled blind eye

Treatment: EVISCERATION (FRILL)

↓

- Removal of contents of eyeball leaving behind only the scleral shell

- In panophthalmitis in place of scleral shell, only small part of sclera is left behind around the optic nerve

↓

This is known as FRILL EVISCERATION

- In panophthalmitis, Enucleation is contraindicated

- Because, in Enucleation

↓ after detaching the muscles from eyeball

we cut the eyeball with a small part of optic nerve

- Optic nerve is surrounded by meninges

- So, in panophthalmitis, Enucleation is not done due to risk of meningitis
### Non-Suppurative Uveitis

<table>
<thead>
<tr>
<th>Non-suppurative uveitis</th>
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</thead>
<tbody>
<tr>
<td>Non-granulomatous</td>
</tr>
<tr>
<td>- Lymphocytes are involved</td>
</tr>
<tr>
<td>E.g.: Fuch's Heterochromic Iridocyclitis</td>
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<tr>
<td>Granulomatous</td>
</tr>
<tr>
<td>- Macrophages are involved</td>
</tr>
<tr>
<td>E.g.: Lepra</td>
</tr>
<tr>
<td>1) Brucellosis</td>
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<tr>
<td>2) Phacoanaphylactic uveitis</td>
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<td>3) Tuberculosis</td>
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<td>4) Syphilis</td>
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<td>5) Sarcoidosis</td>
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<tr>
<td>6) Endophthalmitis</td>
</tr>
<tr>
<td>7) Hypothalamic</td>
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<tr>
<td>8) VHL syndrome</td>
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### Anterior Uveitis

<table>
<thead>
<tr>
<th>Anterior uveitis</th>
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<tbody>
<tr>
<td>Iridocyclitis</td>
</tr>
<tr>
<td>- Inflammation of iris and pars plicata</td>
</tr>
<tr>
<td>- Inflammation causes vasodilation</td>
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<tr>
<td>Patient presents with redness around the cornea</td>
</tr>
<tr>
<td>Circumcorneal / Circumciliary congestion</td>
</tr>
<tr>
<td>- Inflammatory mediators / Irritative toxins</td>
</tr>
<tr>
<td>Spasm of ciliary muscle → Pain, Blurring of vision</td>
</tr>
</tbody>
</table>
- Spasm of sphincter pupillae → miotic pupil
- Iris edema.
  ↓
  - Featureless iris
  ↓
  - In case of brown iris
    ↓
    "muddy iris"
  ↓
  - Breakdown of Blood-Aqueous Barrier
    ↓
    Leakage of proteins into Aqueous Humor
    ↓
    TURBID Aqueous (Decrease in vision)
  ↓
  - AQUEOUS FLARE → Presence of proteins in aqueous, when light is passed
    ↓
    Results in TYNDALL EFFECT
  ↓
  - It is the earliest sign of iridocyclitis
  ↓
  - Inflammatory cells in Aqueous → AQUEOUS CELLS
    ↓
    Earliest sign of ACTIVE iridocyclitis
  ↓
  - Aqueous cells → Brownian motion
  ↓
  - Inflammatory cells and debris blocking the trabecular meshwork
    ↓
    Results in 2° open angle Glaucoma
    ↓
    Aqueous outflow is reduced
    ↓
    Increased intraocular pressure
    (corneal endothelial pumps stops functioning)
    ↓
    Corneal edema
• Pus in the anterior chamber
  ↓
  Hypopyon

• In the uveitis of Behcet's disease
  ↓
  • FLEETING hypopyon (characteristic)
  • Transient hypopyon

• Inflammatory cells in the aqueous stick to the endothelium of cornea → Keratic precipitates (KP's)

Keratic precipitates - types

Fine / Granular KP's
  • Small in size
  • Lymphocytes
  • Seen in Non-granulomatous uveitis

Mutton - Fat KP's
  • Larger in size, waxy
  • Macrophages
  • Seen in Granulomatous uveitis
- MRed KP's seen in hemorrhagic uveitis caused by varicella zoster uveitis

- Pathognomonic sign of varicella zoster uveitis
  ↓
  SECTORAL iris atrophy

- One sector of iris shows atrophy i.e., lighter in color

- IRIS NODULES (Granulomas)
  - Composed of
    1) macrophages
    2) epitheloid cells

- Iris nodules are seen in Granulomatous uveitis

TYPES OF IRIS NODULES

Types of iris nodules
(Based on their location)

- Nodules present at pupillary margin
  ↓
  KOEPPE'S nodules

- Nodules present on body of iris
  ↓
  BUSACCA'S NODULES
TYPES OF POSTERIOR SYNECHIAE

- If entire posterior surface of the iris sticks to the lens and fibrinous exudates also covers the pupillary area.

  \[ \rightarrow \text{Total posterior synechiae} \]

  \( \downarrow \)

  This is known as total posterior synechiae

- The occluded pupil is known as \textit{OCLUSIO PUPILLAE}.

\[ \rightarrow \text{occlusio pupillae} \]

\( \)

2) If only pupillary margin is involved

If only certain areas of pupil stuck to the lens

\[ \rightarrow \text{FESTOONED PUPIL or irregular pupil} \]

If entire margin of pupil is stuck to lens

\[ \rightarrow \text{This is known as 360° posterior synechiae} \]

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Iris pearls (pathognomonic)
→ Iris Roseolas → seen in syphilis

Synechiae-Iris Adhesions

Types of synechia:

- Anterior synechiae
  * Iris sticks to cornea.

- Posterior synechiae
  * Iris sticks to the lens (anterior capsule).
360° posterior synechiae / Ring or Annular synechiae:

- The entire pupillary margin is stuck to the lens
- Posterior chamber has no connection with anterior chamber because at the pupil it is blocked

\[ \text{SECLUSIO} \]
\[ \text{PUPILLAE} \]

- But the ciliary process continue producing aqueous

\[ \rightarrow \]
Pressure in the posterior chamber increases

\[ \rightarrow \]
This pressure pushes the iris forward resulting iris bombe

\[ \rightarrow \]
Forward bowing of iris

- Shape of the anterior chamber becomes funnel shaped or irregular

Funnel shaped anterior chamber

\[ \rightarrow \]
Iris bombe

\[ \rightarrow \]
Peripheral

\[ \rightarrow \]
If the block is not relieved there will be adhesion of iris to cornea peripherally

- In iris bombe → 360° angle closure glaucoma
MANAGEMENT OF ANTERIOR UVEITIS / IRIDOCYCLITIS

* Drug of choice → Topical steroids
  +
  Topical cycloplegics
  ↓
  * Relieve pain by causing ciliary muscle paralysis
  * Increases blood supply (more antibodies)
  * Mydriatic effect
  ↓
  1) It can break newly formed posterior synechiae
  2) Prevent formation of synechiae because of dilatory action

COMPLICATIONS OF IRIDOCYCLITIS

1) Glaucoma - most common
   \( \alpha^\circ \text{ OAG} > \alpha^\circ \text{ ACG} \)

2) Complicated cataract → most common complication of Recurrent Anterior uveitis / Iridocyclitis

3) Band shaped keratopathy (BSK)

4) Macular edema

5) Phthisis Bulbi → Soft & shrunken eyeball
   * Ciliary body atrophy
   ↓
   Decreased aqueous production
   ↓
   Decreased IOP
**HLA ASSOCIATED UVEITIS**

- Uveitis can be caused by certain systemic conditions which are associated with HLA
  
  - **HLA B27** → most common association, causes anterior uveitis  
    → Seen in  
    1) Ankylosing spondylitis (most common)  
    a) Reactive arthritis  
    b) Psoriatic arthritis  
    c) Inflammatory bowel disease

- **HLA-DR5/DR4** → Behçet's Disease  
  * It causes posterior uveitis

- **HLA DR4** → VKH Syndrome  
  Juvenile idiopathic arthritis

- **HLA - A29** → Bird shot choroidopathy  
  It has the strongest association with uveitis

**FUCHS HETEROCHROMIC IRIDOCYCLITIS**

- Stellate KP's
- Diffuse distribution of KP's
• No posterior synechiae formation
• Poor response to steroids
• It resolves spontaneously
• It is usually unilateral
• Complicated cataract (posterior subcapsular cataract) ↓
  • It is the presenting symptom
• Amsler's sign ↓
  On doing paracentesis, results in hyphema. ↓
  Blood in anterior chamber ↓
  Bleeding is from the new vessels in anterior chamber
• It is associated with Rubella infection
**INTERMEDIATE UVEITIS**

- Inflammation of pars plana and presence of inflammatory mediators inside the vitreous

- Patient presents with pain, redness, decrease in vision
  + Floaters

**Signs:**

- Inflammatory cells present in the vitreous
  \[\downarrow\]
  - vitreous cells
  
  - when light passes, these vitreous cells form a shadow on retina → Floaters

- These cells clump together in vitreous gel forming snowball opacities

- These snow balls along with fibrinous exudates organised at the inferior pars plana
  \[\downarrow\]
  - snow banking at pars plana (pathognomonic of intermediate uveitis)

- macular edema
  \[\downarrow\]
  - most common cause of decreased vision in intermediate uveitis
MANAGEMENT OF INTERMEDIATE UVEITIS

- Drug of choice → PERIOCULAR steroids
  ↓
  Posterior sub-Tenon injection of triamcinolone

- Systemic / Intravitreal injection of steroids, if not responds next

- Cyclodestructive procedures
  → cyclophotocoagulation
  → cyclocryotherapy

- If it’s not responds
  ↓
  vitrectomy

- This is known as KAPLAN’S stepladder approach for intermediate uveitis

POSTERIOR UVEITIS

- It is characterised by painless decrease in vision
  ↓
  Due to macular involvement
  ↓
  metamorphopsia.
  central scotoma.

Toxoplasmosis:

- most common cause of posterior uveitis
- It is a Necrotising chorio-retinitis + Severe vitreitis
- Head light in Fog appearance on fundus examination
management:

- Drug of choice: TRIPLE THERAPY for ocular toxoplasmosis

  1. Systemic steroids
  2. Sulfadiazine +
  3. Pyrimethamine

- Toxoplasmosis heals and leaves behind the chorioretinitis scar

- Investigation for toxoplasmosis → Serology (Igm & IgG Antibodies)
CMV CHORIORETINITIS

- Most common cause of posterior
  in immunocompromised patients

- Occurs when
  \[ \text{CD}_4 < 50 \text{ cells} / \mu\text{L} \]

- Retinal hemorrhages
  +
  Retinal Necrosis

- "Sauce & cheese"
  
  or
  "pizza pie"

  +
  "Bushfire" appearance

- Drug of choice: HAART

SARCOIDOSIS

- It can cause panuveitis

- Chronic granulomatous anterior uveitis (most common)

- In this,
  mutton fat KP's
  Iris nodules
  Angle nodules → KP's & nodules at the angle

- In posterior uveitis
  CANDLE WAX DRIPPING
* when the retinal vessels gets inflamed, the exudates around the vessels appears as → Candle wax dripping
* LANDER's sign → Preretinal granulomas
Salt and pepper chorioretinitis seen in:
  1) Congenital rubella.
  2) Congenital

<table>
<thead>
<tr>
<th>SYMPATHETIC OPHTHALMIA</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Definition:</strong> Bilateral granulomatous panuveitis involving the non-injured eye following penetrating injury / intraocular surgery in the exciting eye (injured / operated eye)</td>
</tr>
<tr>
<td>* The non-injured eye is the sympathising eye</td>
</tr>
<tr>
<td><strong>Site of injury:</strong> 'Danger zone' of the eye ↓ Ciliary region</td>
</tr>
<tr>
<td>* Neo antigen / Sequestered antigen → Retinal 'S' antigen</td>
</tr>
<tr>
<td>* Timing of occurrence of sympathetic ophthalmia ↓ a weeks - 3 months following injury</td>
</tr>
<tr>
<td>* Earliest symptom: Blurred near vision in sympathising eye</td>
</tr>
</tbody>
</table>
Earliest sign:

- Presence of proteins in the vitreous
  ↓
- On passing light there will be flare due to Tyndall effect
  ↓
- Retrolental flare in the sympathising eye

Other signs:

- Mutton fat KP's
- Iris nodules
- DALKEN - FUCH'S nodules
  ↓
- Seen between RPE and choroid

Management:

- Prophylaxis:
  - If no visual potential in the injured eye
    ↓
  - Enucleation of the exciting eye
  - Within 2 weeks of injury

- Treatment of sympathetic ophthalmia:
  - Systemic steroids (High dose, long duration)
  - Or
    - Immunosuppressants
    +
    - Topical steroids
    +
    - Topical cyclopregics
* most common cause of sympathetic ophthalmitis
  ↓
  vitreoretinal surgery
  ↓
  Because, here we are giving three full thickness incisions in the ciliary region

---

**VOGT-KOYANAGI-HARADA SYNDROME (VKH SYNDROME)**

- Autoimmunity against melanocytes
- Common in Japanese population
- HLA-DR4
- 3 systems are affected
  1) Integumentary
     a) CNS
     b) Eye
  Integumentary:
     - vitiligo
     - alopecia
     - Poliosis (Premature graying / whitening of eyelashes)

  In CNS:
     - encephalitic like picture
     - meningismus
     - CSF pleocytosis
     - SNHL
     - Tinnitus

  Eye:
     - Bilateral Granulomatous panuveitis
     - multiple, small, serous retinal detachments
- Disc edema
- Dalen-Fuch's nodules
- "Sunset glow" appearance of fundus
- Sugihara's sign → Perilimbal vitiligo

Management:
- Treatment of choice → Systemic steroids or immunosuppressants
  + Topical steroids
  + Topical cycloplegics
SCLERA

- Sclera starts from the limbus and extends posteriorly
- It forms the posterior 5/6th of the outercoat of eyeball
- Sclera modified posteriorly at the region of optic nerve
  ↓
  To form lamina cribrosa.

Embryological origin of sclera:
- From Neural crest cells
- Temporal portion of sclera is from mesoderm

<table>
<thead>
<tr>
<th>LAYERS OF SCLERA</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Episclera</td>
</tr>
<tr>
<td>2) Stroma</td>
</tr>
<tr>
<td>3) Lamina fusca → it is close to the choroid</td>
</tr>
<tr>
<td>→ it contains melanocytes</td>
</tr>
<tr>
<td>→ it is the innermost layer of sclera.</td>
</tr>
</tbody>
</table>
- Sclera contains Type I Collagen
  ↓
  Irregular arrangement (opaque)

Blue sclera.
- Some abnormality in the collagen
  ↓
  Sclera gets thinned out
  ↓
  Underlying choroid is visible through it

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* which is apparently blue color sclera.

Causes:
1) Paget's Disease
2) Pseudoxanthoma elasticum
3) Osteogenesis Imperfecta - most common cause
4) Congenital glaucoma
5) Ehler-Danlos syndrome
6) marfan's syndrome
7) Scleritis
8) Nevus of ota

Hamartoma of melanocytes at lamina fusca.

---

**STAPHYLOMA**

**Definition:**

1) Ectasia of outer coats of eyeball (cornea / sclera)

Bulge

2) Lined by uveal tissue (iris / ciliary body / choroid)

**Types of staphyloma:**

1) ANTERIOR staphyloma.
2) INTERCILIARY staphyloma.
3) CILIARY staphyloma.
4) EQUATORIAL staphyloma.
5) POSTERIOR staphyloma.
<table>
<thead>
<tr>
<th>Type of staphyloma</th>
<th>Ectatic outer coat</th>
<th>Uveal lining</th>
<th>cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Anterior staphyloma</td>
<td>Cornea</td>
<td>Iris</td>
<td>Sloughing corneal ulcer / injury</td>
</tr>
<tr>
<td>2) Intercalary staphyloma</td>
<td>Limbus</td>
<td>Iris root</td>
<td>Peripherally located sloughing corneal ulcer / injury</td>
</tr>
</tbody>
</table>
| 3) Ciliary staphyloma | Sclera | Ciliary Body | 1. Scleritis  
2. Glaucoma  
3. Injury |
| 4) Equatorial staphyloma | Sclera | Choroid | Pathological myopia |
| 5) Posterior staphyloma | Sclera | choroid | Pathological myopia |
management:

- Image combination of intercalary +
  Anterior staphyloma

Treatment:
Staphylectomy +
Keratoplasty

- In case of intercalary staphyloma → scleral graft also used
- Treat the underlying cause
- Intercalary staphyloma is within 1 to 2mm of the limbus

Ciliary staphyloma ↓
It is beyond the 2mm of limbus

- Posterior staphyloma.
SCLERITIS

* Inflammation of sclera

Types of scleritis (with respect to Equator)

Anterior scleritis

Non-necrotising
- Diffuse
  - Redness
  - Thickening of sclera

Localised (Nodular)
- Redness
- Nodule - immobile
- Tender

Necrotising
- Diffuse
  - Inflammation

- Localised
  - Without inflammation
Causes of Anterior Scleritis:

1) Rheumatoid Arthritis (most common cause)
2) Wegener's granulomatosis
3) SLE
4) CTD (connective tissue diseases)

Management:
- Treat the underlying cause
  → systemic NSAID's
  → systemic steroids
  → Immunosuppressants

Necrotising Anterior Scleritis:

- With inflammation
  • Management:
    • Systemic steroids
    • Immunosuppressants

- Without inflammation
  SCLEROMALACIA PERFORANS
POSTERIOR SCLERITIS

- 20% of all scleritis cases
- Redness / pain may be present or absent
- Reduction of vision (85%)
  ↓
  Due to serous retinal detachment
- Sub tenons fluid
LENS ANATOMY

LENS

Anatomy:
- Shape of the lens: biconvex
- Anterior curvature is little lesser compared to the posterior surface of lens
- Radius of curvature $\propto \frac{1}{\text{Curvature}}$

![Anterior and Posterior Curvature Diagram]

- Anterior surface is flatter $\Rightarrow$ Higher radius of curvature of 10mm
- Posterior surface is more convex $\Rightarrow$ Lesser radius of curvature of 4mm
- Zonules are attached to lens

Frontal view of lens:

![Frontal View of Lens Diagram]

- Equator of lens
- Equatorial diameter
- Zonules or suspensory ligament of Zinn at 360°
• Frontal view of lens → Circular
• Circumference of lens is EQUATOR OF LENS
• Anterior surface & posterior surface of lens of different curvatures join at the EQUATOR
• Equatorial Diameter is 9mm (Normal)
• At the equator, zonules are attached 360° → zonules or suspensory ligament of zinn
• On the other end, the zonules are attached to the circular ciliary muscle which is parasymptomatically innervated
• Also known as ciliary Ring

LOCATION OF THE LENS

Aqueous humor → Patellar fossa, vitreous gel → Berger's space
- Shallow depression on vitreous gel to accommodate lens → patellar fossa
- Space between the posterior surface of lens and patellar fossa is Berger's space

Circular ligament between the posterior surface of lens & vitreous
Weiger's ligament
(Capsulohyaloid ligament)

PARTS OF THE LENS

- Lens has three parts
  1) Capsule
  2) Cortex
  3) Nucleus

Capsule → It is the basement membrane
  → Collagen Type IV
  → Thickest basement membrane in the body
  → Thinnest at the posterior pole
  → Thickest at Anterior pre-equatorial region
• Beneath the Anterior capsule, there is single layer of cells called Lens epithelial cells
• Lens epithelial cells produces lens fibres which forms substance of lens

**ZONES OF THE NUCLEUS**

- Depending upon which stage of life, these lens fibres are formed
- Embryonic Nucleus → portion of lens formed upto 3 months of gestation

- Infantile Nucleus
- Embryonic Nucleus
- Fetal Nucleus
- Adult Nucleus

- Fetal Nucleus → lens fibres formed from 3 months of gestation to birth
- Infantile Nucleus
- Adult Nucleus
- Embryonic Nucleus contains oldest fibres

**Lens sutures:**
- These are Y-shaped
- Anterior lens sutures → erect Y-shaped
- Posterior lens sutures → Inverted Y-shaped
- Embryonic Nucleus doesn’t has lens sutures
**COMPOSITION OF LENS**

- Lens has 65% of water
  - 35% of proteins

  - Soluble proteins
    - Crystallins
  - Insoluble proteins
    - Albummoids (10%)

**Crystallins:**

**Crystallins-Types**

- α
  - Largest in size
  - Chaeporons
- β
  - 55% (majority)
- γ
  - Least in number
  - Smallest in size
  - Mutation in cry γ gene
  - (γ-crystalline gene)

- Results in congenital cataract

- MIP-α6 (Major intrinsic protein-α6) also known as Aquaporin-0
  - Regulation of water movement in
  - Water out of lens

**LENS METABOLISM**

- 85% → Anaerobic Glycolysis
- 15% → Hmp shunt

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**Antioxidant Mechanisms**

1. Glutathione
2. Glutathione Reductase
3. Superoxide Dismutase
4. Catalase
5. Ascorbic Acid
6. β - Carotenoids
7. Vitamin-E

**Lens Development**

- Lens develops from surface ectoderm

* By week 3, optic vesicle reaches close to surface ectoderm
- Optic vesicle induces some changes in surface ectoderm
  
  Surface ectoderm becomes thickened
  Near the optic vesicle

  Lens placode formation
  (by week 4)

  ![Diagram of Lens Placode](image)

  - By week 5, lens placode develops a pit or invagination → lens pit

  ![Diagram of Lens Pit](image)

  - By week 6, it gets detached from the surface ectoderm forming lens vesicle

  ![Diagram of Lens Vesicle](image)

  - Lens vesicle pushes the optic vesicle Results in optic cup formation
  - Lens vesicle has cells all around
• In week 7, posteriorly located cells in lens vesicle becomes elongated and terminally differentiated.

![](Image of lens vesicle with annotations)

- Posterior cells in lens vesicle
- Forms primary lens fibres
- Which forms embryonic nucleus

• Embryonic nucleus do not have lens sutures because it is formed from primary lens fibres.

**Growth Factors**

• **PAX 6 gene has role in overall development**
  - It is involved in lens placode development
  - Mutation of PAX 6 gene results in congenital cataract

• **BMP - Bone Morphogenetic Protein**
• **FGF - Fibroblast Growth Factor**
• **IGF - Insulin like Growth Factor**
  - In week 7

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CONGENITAL CATARACT

CATARACT

- Opacification of lens or its capsule
  
  \[\text{Cortex} \quad \rightarrow \quad \text{Nucleus}\]

Classification of cataract

1. Etiological Classification

   \[\text{Congenital cataract} \quad \rightarrow \quad \text{Acquired cataract}\]

   Developmental cataract

   - Age related
   - Toxic
   - Systemic disease
   - Ocular Disease
   - Traumatic
   - Radiational

CONGENITAL CATARACT

Causes of congenital cataract

\[\begin{align*}
1/3^{rd} & \quad \rightarrow & \quad 1/3^{rd} & \quad \rightarrow & \quad 1/3^{rd} \\
\text{Inherited / hereditary} & \quad \rightarrow & \quad \text{Metabolic / infectious} & \quad \rightarrow & \quad \text{Idiopathic}
\end{align*}\]
• Hereditary congenital cataract

↓

Autosomal Dominant inheritance

Gene mutations associated with congenital cataract

• most common Cry G gene on chromosome 2q-40%

• Connexin gene – Cx 46 gene on chromosome 13q-25%

• AQP0 gene on chromosome 12q

• PITX 3 gene on chromosome 10q

Metabolic / Infectious causes

1) Hypocalcemia • maternal
   • Neonatal

2) Hypoglycemia

3) Galactosemia

4) Lowe's syndrome

TORCH Infections

Congenital Rubella

• Infection in 1st Trimester
• Involves fetal Nucleus
• 'pearly' in Appearance
• Live viruses present in the nucleus
BLUE DOT CATARACT

- most common type of congenital / developmental cataract
- it is also known as
  PUNCTATE cataract /
  cataracta punctata cerulea
- it is stationary / non-progressive
- visually insignificant

ZONULAR / LAMELLAR CATARACT

ZONULAR / Lamellar cataract

↓

- most common visually significant congenital cataract
- it involves ‘Foetal’ nucleus

Foetal nucleus is opacified
• RIDERS → Radial spokes of opacity coming from the main opacity

↓

"CART WHEEL" Appearance

• Zonular cataract is seen in Hypoparathyroidism

CATARACT PULVERULENTA

↓

Powdery deposits at Fetal Nucleus

→ Cataracta pulverulenta involving Embryonic Nucleus
ANTERIOR POLAR CATARACT

- Opacity localized to Anterior pole

- Occurs in conditions where there is delayed formation of Anterior chamber

  → Corneal ulcer in utero which is perforated

  a) Peter's Anomaly

  → Delayed separation of Lens placode from the surface ectoderm

POSTERIOR POLAR CATARACT

'Onion-ring' Appearance

- It is due to persistent attachment of Hyaloid Artery at the posterior pole

- It is associated with Mittendorf's dots in vitreous

  → Mittendorf dot in vitreous
**SUTURAL CATARACT**

- These are non-progressive
- Doesn’t affect the vision

---

**MANAGEMENT OF CONGENITAL CATARACT**

- In visually significant cataract
  - Centrally located opacity
  - If diameter of the opacity ≥ 3mm
  - Dense opacity
  - Risk of sensory deprivation amblyopia

**Timing of surgery**

- As early as possible within 4 months of age
- Because foveal maturation / fixation develops at 4 months of age.
• In unilateral cataract
  ↓
  • should operate with in 6 weeks of age
  • Because unilateral cataracts are more amblyogenic
• In bilateral cataract
  ↓
  with in 8 weeks of age

**TECHNIQUE OF SURGERY**

1. Lens Aspiration (through Limbal Route)
   
   + Primary posterior capsulotomy (PPC)
   
   Anterior vitrectomy (AV)
   
   + IOL implantation

2. Lensectomy (through pars plana)

   + PPC+AV+IOL implantation

**IOL Power**

- < 2 yrs of age: 20% undercorrection
- 2–6 yrs of age: 10% undercorrection
- > 6 yrs of age: Calculated IOL power

**IOL Material**

- Foldable Acrylic PC-IOL-Preferred
- PMMA (Poly methyl l methacrylate)
- Heparin coated IOL’s

  ↓

  In infectious cause → Congenital Rubella.
Sutures

- In children, sutures are must, what ever may be the incision length
CLASSIFICATION OF CATARACT

ANATOMICAL CLASSIFICATION OF CATARACT

I. Capsular cataract
   - Anterior capsular cataract
   - Posterior capsular cataract
      - Anterior subcapsular cataract
      - Anterior polar cataract
      - Posterior subcapsular cataract
      - Posterior polar cataract

II. Cortical cataract

III. Nuclear cataract

ANTERIOR CAPSULAR CATARACT

Anterior capsular cataract
   - Anterior subcapsular cataract
     - Diffuse opacity just beneath anterior capsule
     - Causes:
       1) Amiodarone
       2) Phenothiazine
       3) Pilocarpine
       4) SOLD
       5) Electric shock

   - Anterior polar cataract
     - Localised opacity at the anterior pole
     - Causes:
       Corneal perforation

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POSTERIOR CAPSULAR CATARACT

Posterior capsular cataract

Posterior subcapsular cataract
- Diffuse opacity just beneath Posterior capsule

Posterior polar cataract
- Localised opacity at the posterior Pole

Causes:
1) Complicated cataract
2) Steroids (systemic > topical)
3) Chloroquine → causes flaky posterior subcapsular cataract
4) Busulfan
5) Radiation

- Posterior subcapsular cataract lies close to the nodal point
  ↓
  So, it obstructs the refracted light (very visually handicapping)
  "Glare" especially at night

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CORTICAL CATARACT

- It is due to hydration of lens

Types

CUNEIFORM CATARACT

Wedge
- Peripheral wedge shaped opacities close to equator of lens
- Visually insignificant
- Most common found in Quadrant Inferonasal
- Most common type of senile cataract

CUPULIFORM CATARACT

- Disc like central opacity at posterior corlex
STAGES OF CORTICAL CATARACT FORMATION

1 Stage of lamellar separation:
- Layers of lens fibres are separated by water vacoules / cleft
- This water in the lens acts as prism and causes coloured halos

Tiny water droplets visible on Retroillumination using slit lamp

11 Incipient cataract:
- Opacities
- Refractive index of lens changes
- Lens will have multiple refractive index

\[ \mu \propto \sigma \propto \frac{1}{f} \]
* multiple refractive index (μ) results in multiple images resulting in uniocular polyopia.

III Intrumescence cataract:
* Water logged, swollen lens
  \[ \downarrow \]
  Displaces adjacent structures iris, aqueous humor

- Anterior chamber - shallow
- \( \alpha \) angle closure glaucoma
- Phacomorphic glaucoma
  \[ \downarrow \]
  Treatment of choice
  cataract extraction

IV Immature cataract:
* Clear and opaque lens fibres
  \[ \downarrow \]
  Greyish white color to lens
* Iris shadow
  \[ \downarrow \]
  When light focussed on pupil obliquely from the temporal site
  \[ \downarrow \]
  Pupillary margin casts a shadow on the lens behind
* Iris shadow requires clear subcapsular fibres
5. Mature cataract:
   - All fibres are opaque
   - "Pearly white" in color
   - Iris shadow absent

   ↓

   Because there are no clear subcapsular fibres

6. Hypermature cataract:

   Hypermature cataract

   Hm sclerotic cataract
   - Calcific plaques on capsule
   - Wrinkling of capsule
   - Complication: Lens Dislocation

   Hm morgagnian cataract
   - Liquifactive degeneration of cortex
     ↓
     - Milky white fluid

   Brown nucleus sinking
Hypermature Morgagnian cataract:

- Brown nucleus sinking and capsule leaks cortex particles
  - These particles leak out into aqueous and clog the trabecular meshwork
  - Impaired aqueous outflow
    - But the angle is open
      - $2^\circ$ open angle glaucoma
        - Phacolytic glaucoma

Treatment of choice $\rightarrow$ Cataract extraction
NUCLEAR CATARACT

- Due to denaturation of lens proteins (crystallins)

Risk factors:
1) Ageing
   a) Sunlight (UV)
   b) Smoking
2) Deficiency of vitamin A, C, E
3) Type 2 diabetes
4) Vitrectomy

Changes in Nuclear Cataract:
1) Nuclear sclerosis (hardening)
2) Discoloration - due to urochrome pigments deposited in lens
   - Lens becomes yellow
   - Amber
   - Brown - cataracta brunessence
   - Black - cataracta nigra

- This discoloration helps in grading of nuclear cataract

3) Increase in refractive index ($\mu$)

\[
\mu \propto \frac{D}{\ell} \propto \frac{1}{\ell^2} \text{ (decreases)}
\]

- Image moves in front of retina resulting in index myopia.

Retina

Image formed in front of retina.
- Second sight for near vision

↓

Improved near vision in a presbyopic

---

**ETIOLOGICAL CLASSIFICATION**

Etiological classification of cataract

↓

Congenital cataract

Acquired cataract

Acquired cataract:

Causes:

- Age-related
- Toxic - Drugs / smoking
- Systemic diseases
- Ocular diseases
- Metabolic conditions
• Traumatic
• Radiation

Drugs causing
• Anterior subcapsular cataract (ASC)
  • Amiodarone
  • Phenothiazine
  • Pilocarpine
  • Gold
• Posterior subcapsular cataract (PSC)
  • Steroids
  • Chloroquine (flaky type of PSC)
  • Busulfan
• Smoking causes nuclear sclerosis

Systemic conditions:
1) Myotonic dystrophy – Christmas tree cataract
2) Neurofibromatosis – a → posterior subcapsular cataract
3) Atopic dermatitis → Anterior subcapsular cataract "shield cataract"
4) Down’s syndrome

  ↓

  most common type is punctate cataract
  Stellate cataract

Metabolic conditions:
1) Diabetes – “snowflake / snowstorm” cataract
2) Galactosemia – “oil droplet” cataract
3) Hypoparathyroidism

• Ocular conditions causing cataract → Complicated cataract
Complicated cataract: Cataract due to ocular disease / degeneration

Causes:
- Chronic recurrent iridocyclitis - most common
- Extensive chorioretinitis
- Retinitis pigmentosa
- Retinal detachment
- Intraocular tumor
- Pathological myopia

Signs of complicated cataract:
- Posterior subcapsular cataract
- ‘Bread crumb’ appearance
- Polychromatic lustre

### TRAUMATIC CATARACT

Penetrating trauma.
- By copper foreign body
  - Chalcosis
- Sunflower cataract
- Also seen in Wilson’s disease
- It is an anterior subcapsular cataract

Blunt trauma:
- Rosette cataract
- Location of cataract → posterior subcapsular cataract
RADIATION CATARACT

IONISING RADIATION
β, γ, x-rays
↓
Affects the lens epithelial cells in the equator
↓
Results in posterior subcapsular cataract

NON IONISING RADIATION
• Infra-red rays
• Heat
↓
• Indirect affect on lens epithelial cells
• Heat absorbed by iris pigments transmitted to germinal cells of lens
↓
Posterior supcapsular cataract
Heat cataract
Or
Glass Blowers Cataract
Oil-droplet cataract

↓

Seen in Galactosemia

- CHRISTMAS TREE CATARACT
  
  Seen in myotonic Dystrophy

- Polychromatic lustre

  myotonic dystrophy patient with
  1) Frontal Bossing
  2) myogenic ptosis
  
  ↓

  which is compensated by overacting frontalis

  3) expression less face
Stellate cataract
seen in
1) myotonic dystrophy
2) Down's syndrome

Shield cataract
Seen in atopic dermatitis
It is an anterior subcapsular cataract
MANAGEMENT OF CATARACT - IOL

- Management of cataract is surgery
- In surgery, we remove the opacity, which means refractory power of eye
- Lens contributes to 1/3 or 1/4th of entire refraction
- So, we have to replace the refractory power
- In order to replace the refractory power of lens, we should know the power of intraocular lens
- Method to calculate IOL power
  ↓
  Biometry

Biometry

- SRK formula (Sanders, Retzlaff & Kraft formula)
  \[ \text{IOL power} = A - 0.9 - 2.5L \]
  
  \[ A \rightarrow \text{Constant} \]
  
  \[ K \rightarrow \text{Keratometry} \]
  
  \[ L \rightarrow \text{Axial length} \]

- Axial length of the eyeball is measured using A-scan

A-Scan

"A-Scan"
- Here we measure the distance between the corneal spike and Retinal spike → gives axial length
- Ocular / orbital ultrasound
  Probe frequency: 8-10 mega Hz
- Axial length can also be measured by IOL master
- IOL master → most accurate
  IOL power → PC-IOL in the capsular bag, to achieve Emmetropia, for distance
  - 1 mm change in corneal curvature → 6D change in IOL power
  - 1 mm change in Axial length → 3D change in IOL power

---

IOL (INTRAOCULAR LENS)  00:07:34

PC-IOL (Posterior chamber IOL)

↓

If it is placed behind the iris

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Types of IOL (Based on the material)

- RIGID PMMA
  - Poly methyl meth Acrylate

- FOLDABLE
  - 1. Acrylic
  - Hydrophobic
  - Hydrophilic (Hydrogel)

- ROLLABLE HYDROGEL

* Best IOL material: Hydrophobic Acrylic

Types of IOL (Based on their optics)

- MONOFOCAL IOL
- MULTIFOCAL IOL
  - Eliminates the need for glasses
  - multiple rings in the optics
  - Each ring has different refractory power
  - Two types
    1) Diffractive
    2) Refractive

- TORIC IOL
  - To correct pre-existing Astigmatism

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ACCOMODATING IOL

- After cataract surgery, patient cannot accommodate
- Because, in accommodation
  
  Anterior capsule bluges
  ↓
  Curvature increase
  near vision comes
to focus

- So, in cataract surgery, anterior capsule is opened
  ↓
  No Accommodation

- Accommodating IOL's are newer IOL's which themselves will accommodate
- Crysta lens

→ multifocal IOL
- Ideal location of IOL
  ↓
  Inside the capsular bag
  or
  on the posterior capsule

- In case of posterior capsular rupture
  IOL placed against the anterior capsule with haptics resting in the ciliary sulcus
  ↓
  Sulcus placement of PC-IOL
iris - fixated
PC-IOL

Scleral-fixated
PC-IOL

- Fixation can be dosing
  - Suture
  - Glue
MANAGEMENT OF CATARACT-SURGICAL TECHNIQUES

- There are two technique

  Intracapsular
  - Cataract / lens removed with the capsule intact
  - Indication:
    Lens dislocation
  - Surgery
    → ICCE (IntraCapsular Cataract Extraction)

  Extracapsular
  - Hallmark
    → Anterior capsulotomy
    - Leaving behind the capsular bag
    - Surgery’s
      1) ECCE (ExtraCapsular Cataract Extraction)
      2) SICS (Small Incision Cataract Surgery)
      3) Phacoemulsification
      4) FLECS (Femtosecond Laser-Assisted Cataract Surgery)

STEPS OF CATARACT SURGERIES

Anaesthesia

1) General Anaesthesia. → for children

2) Local Anaesthesia
   1) Retrobulbar Nerve Block
   2) Peribulbar Nerve Block
- Local Anaesthetic, mixture for block
  
  Lignocaine (short acting)
  +
  Bupivacaine (long acting)
  +
  Hyaluronidase (helps in diffusion of anaesthetic mixture into tissue)
  Adrenaline

3) Topical Anaesthesia → Lignocaine, Paracaine

- Only for phaco through corneal incision

- This Topical Anaesthesia should be supplemented with Intracameral 1% preservative free Lignocaine intra-operatively ↓
  Into the Anterior chamber

Scrubbing:

For periorbital skin → 10% povidone iodine eye drops (into conjunctival sac)

- 5% povidone iodine for 3 minutes ↓

Best way to prevent post-operative endophthalmitis
**INCISION**

- **ECCE (Extra Capsular Cataract Extraction)**
  
  ![](image)

  - Tenon's capsule & Conjunctiva
    
    → Inserted at the Limbus

  - In order to make Limbal incision, Peritomy is done
    
    Peritomy → cut and open the conjunctiva

  - After peritomy, Limbal incision is given

**SICS (Small Incision Cataract Surgery)**

![](image)
Sclerocorneal Tunnel → hallmark of SICS

→ self sealing incision

Phacoemulsification

Peripheral clear corneal incision (3.2-3.5 mm or 2.7-3.2 mm)

Peripheral clear corneal incision

AC (Anterior Chamber) entry

- After incision, instrument is within the AC entry

↓

Aqueous cones out

Cornea becomes flat

↓

To make AC (Anterior Chamber) inflated

↓

Irrigating solutions or viscoelastics

↓

Composition matches aqueous
Irrigating fluids

1) Normal saline
   a) Ringer's lactate
   2) BSS plus
      ↓
      * Balanced salt solution
         + Glutathione
      * Best irrigating fluid

viscoelastics

1) HPmC (Hydroxy Propyl methylcellulose)
   a) Sodium hyaluronate
   3) Chondroitin sulphate
   * Viscoelastics is described as OVDs - Oculo viscosurgical devices

<table>
<thead>
<tr>
<th>ANTERIOR CAPSULOTOMY</th>
<th>00:22:05</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>* Dye used to anterior capsule—Trypan Blue</td>
<td></td>
</tr>
<tr>
<td>* Two techniques</td>
<td></td>
</tr>
<tr>
<td>1) Can-opener Technique</td>
<td></td>
</tr>
<tr>
<td>2) CCC - Continuous Curvilinear Capsulorrhexis</td>
<td></td>
</tr>
</tbody>
</table>
In ECCE

Hydrodissection

(separation of capsule of cortex)

* Inject the fluid beneath the capsule
  ↓

This fluid cleaves the capsule and
the cortex
↓

Bring out the lens as a whole

In SICS

Hydrodissection
↓

Hydrodelineation
(separation of Nucleus & Cortex)
↓

Nucleus Delivery
↓

Cortex Aspiration
In phacoemulsification

- Nuclear fragmentation done using ultrasound energy

- Phacoprobe with Titanium needle at 40,000 Hz
  ↓
  Aspirate the nuclear fragment
  ↓
  Aspirate the cortex

IOL IMPLANTATION

- In both ECCE & SICS → Rigid IOL

- In phacoemulsification → foldable IOL

- Removal of Viscoelastics

- Reform the Anterior chamber → using Normal saline, Airbubble

- Incision closure
  ↓
  In ECCE → sutures must → risk of surgical - induced Astigmatism (SIA)

  SICS
  Phacoemulsification

  \[ \text{No sutures} \]

- Subconjunctival injection of Dexamethasone + Gentamycin

- Intracameral injection of CEFAZOLIN (Preservative Free)
  ↓
  Prevent post-operative endophthalmitis
FLACS

- Femtosecond laser Assisted cataract surgery

- Femtosecond laser → 10^{-9} seconds

- It works on the principle of

  ↓

  Photo Disruption

Steps

1) Capsulorrhexis

2) Nuclear fragmentation \{ Done by laser

3) Corneal inclusions

- Remaining steps are completed manually by surgeon
* Most common complication post-operatively
* It's also known as "AFTER-CATARACT"

**Two types of PCO**

- **Elschning's pearls**
- **Sommering's ring**

Peripheral ring-like opacities
In infants, this posterior capsulotomy during the cataract surgery itself

- Because, if PCO is formed
  ↓
  It will results in Amblyopia.

- Normally, Lens Epithelial Cells (LEC) are present on the Anterior capsule of lens

- But, After cataract surgery, thes LEC will cross the Equator and start growing on posterior capsule
  ↓
  Produces Aberrant lens fibres which are opaque
In children, PCO formation rate is very high
even if we remove posterior capsule

↓
because vitreous is firm in children
which is present behind posterior capsule, these LEC's again grow into the vitreous and forms PCO

So, Anterior vitrectomy is also done

In children
Along with cataract surgery

+ Primary posterior capsulotomy
+ Anterior capsulotomy

→ image of posterior capsulotomy
CONGENITAL ANOMALIES OF LENS

ANOMALIES OF LENS SHAPE

- Normal shape of lens: biconvex

1. Lenticous
   - Conical projection on surface of lens
   - 2 types:
     a) Anterior lenticous:
        - Projection on anterior lens surface
        - Seen in Alport’s syndrome
        - In a slit-lamp image, compare the corneal convexity to identify anterior surface of lens
     b) Posterior lenticous:
        - Projection on posterior lens surface
        - Seen in Lowe’s syndrome

- Other components of Lowe’s syndrome:
  * Congenital cataract
  * Congenital glaucoma
  * Renal tubular acidosis
  * Aminoaciduria

- Oil-globule reflex may be seen in either of the two lenticous
'Oil' in Ophthalmology

- Oil Droplet Cataract: Galactosemia
  - Nuclear cataract
  - Only reversible cataract

- Oil Droplet reflex: Keratoconus
  - Seen on distant direct ophthalmoscopy

- Oil Globule reflex: Lenticus

Anomalies of Shape and Size

- Normal equatorial diameter of lens: 8.8 to 9.2 mm

  - Microspherophakia.
  - Small and spherical lens
  - Seen in well-marchesani syndrome
  - Complication:
    - Lens dislocation - anteriorly and inferiorly

  - Components of well-marchesani syndrome
    - Short and stout
    - Mentally subnormal
    - Short and stubby fingers
      → Brachydactyly

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* Complication: Lens dislocation
  ↓
  If lens blocks pupil
  ↓
  Aqueous collection in pc
  ↓
  Pc pressure increases
  ↓
  Pushes iris forward and blocks angle
  ↓
  Secondary angle closure glaucoma
  also called phacotopic glaucoma

- Treatment of secondary ACG:
  * make patient lie supine
  ↓
  Administer mydriatic (Cycloplegic)

- Since cycloplegic is used, called inverse glaucoma
- Treatment of choice: Intracapsular lens extraction
ABNORMALITIES OF LENS POSITION

- Normal lens position: Patellar fossa.

ECTOPIA LENTIS:

- Lens displacement out of patellar fossa.
- Types:
  1. Subluxation: Partial displacement
     - On oblique illumination, jet black crescent seen at pupillary border
     - On retro-illumination, golden crescent at pupillary border seen
  2. Dislocation: Total lens displacement

CLINICAL FEATURES OF ECOPIA LENTIS

<table>
<thead>
<tr>
<th>Subluxation</th>
<th>Dislocation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Unicocular diplopia</td>
<td>1) Sudden painless marked decrease in vision</td>
</tr>
<tr>
<td>2) AC-irregular</td>
<td>2) AC-deep</td>
</tr>
<tr>
<td>3) Pupil-phakic and aphakic areas</td>
<td>3) Pupil-Jet black in color</td>
</tr>
<tr>
<td>4) Purkinje images</td>
<td>4) Purkinje images</td>
</tr>
<tr>
<td>1, 2, 3, 4 at phakic area</td>
<td>1 and 2 present</td>
</tr>
<tr>
<td>only 1 and 2 at aphakic area</td>
<td></td>
</tr>
<tr>
<td>Iris - Iridodonesis</td>
<td>Iridodonesis</td>
</tr>
<tr>
<td>phacodonesis seen</td>
<td></td>
</tr>
</tbody>
</table>
CAUSES OF ECTOPIA LENTIS

a) Congenital:

1. Familial ectopia lentis - autosomal dominant
2. Congenital aniridia
3. Weil-Marchesani syndrome - Anterior inferior dislocation
4. Marfan's syndrome - Supero temporal dislocation
5. Homocystinuria - Infero nasal dislocation
   - Due to cystathionine-β-synthetase deficiency
   - Hyperhomocysteinemia
6. Hyperlysinemia
7. Sulphite oxidase deficiency

b) Acquired:

1. Blunt trauma: m.c cause of acquired ectopia lentis
2. Pseudoexfoliation syndrome
3. Hypermature sclerotic cataract
4. Ciliary body tumours

MANAGEMENT OF ECTOPIA LENTIS

- Lens extraction (intra capsula)
  : if complete displacement

- Capsular Tension Rings
  - In case of subluxation
  - In the bag PCiol can be placed
REFRACTION - BASICS AND ACCOMODATION

- Total Refractive power of eyeball: +60 D (+58.6 D)
- Cornea (2/3 or 3/4): +45 D
- Lens (1/3 or 1/4): +15 D

![Diagram of refraction and accommodation]

ACCOMMODATION

Contraction of circular fibers of ciliary muscle

- Zonules
- Ciliary muscles

accommodation

- Zonules Relax
- Lens shrinks but volume is same
- Anterior curvature of lens ↑
- In diameter of ciliary ring
- Relaxation of lens capsule
- In equatorial diameter of lens
- In Dioptic power of lens
- ↓ in focal length of lens & eyeball
- No change in posterior curvature of lens
- ↑ in thickness (AP diameter) of lens → Ant. Chamber relative shallowing

---

**ACCOMMODATION TRIAD**

\[ \text{Near triad} \]

1) Convergence of both eyeballs (A/C, MR)
2) Constriction of pupil (A/C, miosis → ↑ depth of focus)
3) ↑ in Anterior Curvature of lens (A/L)

---

**PRESBYOPIA**

- Physiological loss of accommodation d/t Aging
- Results in Retraction of puncta proxima.
- Management → Convex (plus), spheres
- Multifocal IOL → Post cataract surgery

---

Accommodating IOL’s:

\[ \rightarrow ↑ the \ 'O' \ of its own self \]
REFRACTIVE ERRORS

INTRODUCTION

The refractive errors - Ametropia.

Types of Refractive errors:
1. Myopia – mc
2. Hypermetropia
3. Astigmatism

- Refractive errors are also called as Ametropia.

MYOPIA

- Near sightedness / short sightedness
- Image is infront of the Retina.

Types of myopia:
1. Axial
2. Curvatural
3. Index
4. Positional

AXIAL MYOPIA

- Seen when the Axial length of the ball is increased
- Causes:
  a. Pathological myopia
  b. Euphthalmos
  c. Posterior staphyloma
CURVATURAL MYOPIA

- Due to increased curvature
- Corneal causes:
  - Keratoconus
  - Keratoglobus
- Lens causes:
  - Lenticular:
    - Ant. Lenticular - Alport's syndrome
    - Post. Lenticular - Lowe's syndrome
  - Spherophakia - spherical lens
    - Seen in Weill Marchesani syndrome
  - Intumescent cataract - swollen lens
  - Diabetic cataract - accumulation of sorbitol and lens swells up
  - Pilocarpine - Accommodation spasm → Increased curvature of lens → Induced myopia.

INDEX MYOPIA

- Due to increased Refractive index of lens
- Seen in Nuclear sclerosis
- Nuclear sclerosis is the reason for second sight in the aged for Near vision

POSITIONAL MYOPIA

- It is due to Anterior subluxation or Anterior positioning of the lens
PATHOLOGICAL MYOPIA

<table>
<thead>
<tr>
<th>Refractive error</th>
<th>Axial length</th>
</tr>
</thead>
<tbody>
<tr>
<td>High myopia</td>
<td>-6 to -8 D</td>
</tr>
<tr>
<td>pathological myopia</td>
<td>≥ -8 D</td>
</tr>
</tbody>
</table>

- Eye findings in pathological myopia:
- Thin sclera, Blue sclera.
- Staphyloma – Equatorial and posterior – mcc is pathological myopia.
- Anterior chamber – Deep
- High risk of POAG
- Complicated cataract – post subcapsular cataract

Fundus finding:
- Temporal crescent around the optic disc
- Lacquer cracks – Ruptures of Bruch’s membrane

- Foster fuch’s spots – seen at macula
- Choroidal Neovascular Membrane

Peripheral Retinal findings:
- Lattice Retinal Degeneration – mc degeneration
- Retinal tears / breaks
- Rhegmatogenous Retinal Detachment
- Posterior vitreous Detachment – Weiss Rings

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6. Scan showing post. Staphyloma.

- Overall appearance of a myopic fundus is described as Tessellated /
- Tigroid appearance

### MANAGEMENT OF MYOPIA

1. Glasses: Concave (minus) spheres
   - Thinner at center, thicker at peripheries
   - Minified images
   - Images move in the same directions

2. Contact lenses

3. LASIK - Laser assisted Insitu Keratomileusis

   - Central stromal Ablation → Flattening the central cornea → K↓ D↓
   - Excimer laser (λ:193 nm) → Principle: Photo Ablation
   - Femtosecond laser → Principle: Photodisruption (λ=1053nm)
   - Any of the lasers can be used

**Prerequisites for LASIK:**
- Age ≥ 18 y
- Refraction should be stable at least from the past 1yr
- Residual stromal thickness - at least 250 μm
- No H/O viral keratitis in the past 6 months
- No H/O dry eyes

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4. SMILE PROCEDURE
- Small incision Lenticule Extraction
- Laser used: Femtosecond

5. Radial Keratotomy
- Radial cuts made on the cornea
  ↓
  Scar formation in the periphery
  ↓
  Scar contracts and that causes central corneal flattening
- It is unpredictable & undergo Regression
- May lead to Astigmatism

6. Clear Lens Extraction:
- K/a Fukala’s surgery
- removing upto 15 D of the lens
- patient remain Aphakic or put an IOL
- If IOL is implanted, the procedure is Known as Refractive Lens Exchange

7. Implantable collamer Lens:
- Collamer lens is made of porcine collagen
- This is phakic IOL - IOL is put on natural lens
HYPERMETROPIA

- Image is behind the Retina.

Components:

\[ \text{Total Hypermetropia} \quad \downarrow \quad \text{manifest H.M} \quad \downarrow \quad \text{Facultative H.M} \quad \downarrow \quad \text{Absolute H.M} \]

Latent Hypermetropia:
- Due to the inherent tone of the ciliary muscle
- It is found by doing cycloplegic Refraction

manifest H.M:
- Measuring Hypermetropia without cycloplegia.

Facultative H.M:
- When the patient tries to Accommodate
- Can be avoided by PEERING

Absolute H.M:
- The weakest convex lens which gives the patient a clear vision

TYPES OF HYPERMETROPIA

1. Axial H.M:
   - Small eye ball
     - New born Physiological H.m
     - Microphthalmos
     - Nanophthalmos

2. Curvature H.M:
   - Cornea plana

3. Index H.M:
   - Cortical sclerosis
4. Positional H.M:
   - Posterior dislocation / subluxation of lens
   - Aphakia

### MANAGEMENT OF HYPERMETROPIA

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Glasses: Convex / Plus spheres</td>
</tr>
<tr>
<td>2.</td>
<td>Contact lens</td>
</tr>
<tr>
<td>3.</td>
<td>LASIK - Flatten the peripheral corneal stroma.</td>
</tr>
<tr>
<td></td>
<td>- Excimer / Holmium laser are used</td>
</tr>
<tr>
<td>4.</td>
<td>Refractive lens exchange</td>
</tr>
</tbody>
</table>
ASTIGMATISM

In a sphere
  \[ D = \frac{1}{f} \]

\[ \Rightarrow \text{Curvature all around the sphere} \rightarrow \text{Same} \]
\[ \Rightarrow \text{All the 360° meridians} \rightarrow \text{Same curvature} \]
\[ \Rightarrow \text{Dioptic power is the same} \]

Since they have 1 dioptic power \[ \rightarrow \text{Single focal point/Len} \]

ASTIGMATISM

- Vertical & horizontal curvatures are different

  \[ \downarrow \]

  Different dioptic power \[ \Rightarrow \text{Different focal points} \]

- Light rays falling on the
  Vertical meridians

  \[ \downarrow \]

  Produce multiple focal points

  \[ \downarrow \]

  Focal line
  [Horizontal focal line]

- Vertical meridian produce a horizontal focal line
- Horizontal meridian produce a vertical focal line
- This refractory error is known as Astigmatism [‘stigma’ means point]
  because it does not produce a single focal point
ASTIGMATISM ON 2D

Surface:
1. \( \Rightarrow \) vertical curvature \( \rightarrow \) more curved / steep
   \( 90^\circ \) axis \( \rightarrow \) steep axis
   Horizontal curvature \( \rightarrow \) Flat

2. \( \Rightarrow \) horizontal curvature \( \rightarrow \) steep
   \( 180^\circ \) axis \( \rightarrow \) steep axis
   Vertical curvature \( \rightarrow \) Flat

REFRACTION OF LIGHT ON ASTIGMATIC SURFACE

- Causes of astigmatism:
  - Corneal (most common)
  - Lenticular
  - Macular / Retinal

- Sturm's Conoid

\[ H_y > H_x \]

\[ A - B \rightarrow \text{Focal interval of strum} \]

mid point of strum

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- $K_h$ - Curvature of horizontal meridian
- $K_v$ - Curvature of vertical meridian
- $A$ - Vertical focal line
- $E$ - Horizontal focal line
- $B$ & $D$ - Intermediary points
- $A - E$ distance $\Rightarrow$ Focal interval of sturm
- $C$ [midpoint of strum] $\Rightarrow$ mid point of focal interval of strum
- The cone formed by the light rays $\Rightarrow$ Sturm's conoid

Describes the optics of regular astigmatism

- When the following points lie on the retina:

```
A   B   C   D   E
1   0   0   0   0
↓   ↓   ↓   ↓   ↓
```

On the astigmatism clock,
12, 6 $\Rightarrow$ Seen clearly
3, 9 $\Rightarrow$ Out of focus

Vertical ellipse
Circle of least confusion
Horizontal ellipse
On the clock, $2, 9$ $\Rightarrow$ Clear
12, 6 $\Rightarrow$ Out of focus

All the meridians seen clearly
**TYPES OF ASTIGMATISM**

- Regular: Only 2 focal lines (because only 2 curvatures / Axis / Principal meridian)
- Irregular: More than 2 curvatures

**CORRECTION OF REGULAR ASTIGMATISM**

- Correction is done by using CYLINDERS

   - Convex (Plus)  \( \downarrow \) Along the steep axis  \( \downarrow \) Tese the power of the flatter axis
   - Concave (minus)  \( \downarrow \) Along the flat axis  \( \downarrow \) Tese the power of the steeper axis

- Power of cylinder acts perpendicular to its axis

**IRREGULAR ASTIGMATISM**

- More than 2 curvatures [Can be 3, 4 ... or infinite]
- Sometimes geometric assessment of the surface can be impossible
- Causes:
  - Keratoconus → Treated by Rigid gas permeable contact lens
  - Corneal transplant
  - Scarred cornea
- Cannot be corrected by cylinders
TYPES OF REGULAR ASTIGMATISM

- Described by strum's conoid

Simple \rightarrow Compound \rightarrow Mixed

I. Simple Astigmatism:

\( \frac{1}{f_p}, \frac{1}{f_a} \rightarrow \) a focal lines (One on the retina, & the other either infront of or behind the retina)

1. Simple myopic astigmatism

\( f_i \rightarrow \) On the retina.

\( f_a \rightarrow \) Infront of the retina.

2. Simple hypermetropic astigmatism

\( f_i \rightarrow \) On the retina.

\( f_a \rightarrow \) Behind the retina.

II. Compound Astigmatism

I. Compound myopic astigmatism

Both the focal lines are infront of the retina.
a. Compound hypermetropic astigmatism

Both the focal line are behind the retina.

iii Mixed astigmatism

One focal line infront of the other behind the retina.

---

**Other Types of Regular Astigmatism**

1. **With - the - rule astigmatism**
   (Rugby ball surface)

   - 90° vertical meridian: steep
   - Horizontal meridian: Flat

   Corrected by:

   - Convex cylinder (Plus) at 90° [Remember 'P' for plus & steep]
   - Concave cylinder (Minus) at 180°

2. **Against - the - rule astigmatism**
   (Egg against gravity surface)

   - Horizontal meridian (180°): steep
   - Vertical meridian: Flat

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* Corrected by:
  - Convex (plus) cylinder at 180°
  - Concave (minus) cylinder at 90°

111 Oblique astigmatism

- Surface has oblique axis:
  - 45°: Steep axis
  - 135°: Steep axis
RETINA-ANATOMY

DEVELOPMENT OF RETINA

- Derived from neuroectoderm
- From diencephalon
  ↓
  Optic bud
  ↓
  Elongates to form optic stalk
  ↓
  Forms optic vesicle
- Optic stalk eventually becomes optic nerve
- Optic vesicle gives rise to Retina.
- The lens vesicle near the optic vesicle cause it to form optic cup
  ↓
  Gives rise to all the layers of retina.
  - The outer lip of optic cup → Outer most layer of retina.
    (Retinal pigment epithelium)
  - The inner lip of optic cup → Inner 9 layers
    (Neuro sensory retina)

Sub retinal space: between retinal pigment epithelium + inner neurosensory retina.

Represents the cavity of optic vesicle
Site of retinal detachment

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10 → Retinal pigment epithelium  
9 → Layer of photo receptors  
8 → outer limiting membrane / External limiting membrane  
7 → outer nuclear layer  
6 → outer plexiform layer  
5 → Inner nuclear layer  
4 → Inner plexiform layer  
3 → Ganglion cell layer  
2 → RNFL (Retinal Nerve Fibre Layer)  
1 → Internal limiting membrane

* innermost layer of choroid → Bruch’s membrane

* Retinal pigment epithelium
  - Inner to the Bruch’s membrane
  - Contains microvilli
  - Single layer of cells containing melanin pigments
PHOTO RECEPTORS

- Retina
- Rods
- Cones

- Visual pigment from rods & cones

  ↓

  Phototransduction
  [Light energy → Electrical impulses]
  Occurs in the outer segment

INNER NUCLEAR LAYER

- INL
- Modulatory
  1. Bipolar
  2. Horizontal
  3. Amacrine
- Glial cells
  - Müller cell

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• Amacrine cells: Synapses only with the ganglion cells have only depolarizing action potentials

• Müller cell: Contains foot processes that form the outer limiting membrane & internal limiting membrane.

\[
\text{Light} \rightarrow \text{Cornea} \rightarrow \text{Aqueous} \rightarrow \text{Lens} \rightarrow \text{Vitreous} \downarrow \\
\text{outer segment} \downarrow \\
\text{Axons} \leftarrow \text{Ganglion cell} \leftarrow \text{Bipolar cell} \leftarrow \text{Electrical signals} \downarrow \\
\text{Optic nerve} \downarrow \\
\text{Visual pathway}
\]

---

**RETINAL PIGMENT EPITHELIUM**

• Functions of Retinal Pigment epithelium (RPE)

1. Melanin of RPE: Prevents back scattering of light

2. Villi of RPE: Phagocytosis of the shed outer segment of the photoreceptor

3. Recycling of vitamin A

4. Outer blood retinal barrier → Tight junctions between RPE

\[\downarrow\]

Keeps the subretinal space fluid free

5. Active RPE pump → Pumps out fluid from the subretinal space into the choroid.
* Bruch’s membrane → Basement membrane of RPE
* Pigments in RPE
  1. Melanin
  2. Lipofuscin (wear & tear pigment)
     - Auto fluorescent
     - Used in the investigation: Autofluorescence
* Shape of RPE → Cuboidal, but from surface → Hexagonal in shape

**MULLER CELL**

- It is a retinal glial cell
- Maintains the micromilieu of retina
- Contributes to the inner blood retinal barrier

**BLOOD RETINAL BARRIER**

Blood retinal barrier

- Outer BRB
  - Tight junctions between the retinal pigment epithelium

- Inner BRB
  - Tight junctions between the endothelial cells of retinal capillaries
Investigations:

Fundus Fluorescein Angiography:

* to assess the integrity of blood retinal barrier (BRB)

* Fluorescein dye injected (conc $\rightarrow$ 5%, 10% or 20%)

\[ \downarrow \]

Antecubital vein

\[ \downarrow \]

enters the circulatory system $\rightarrow$ choroid vessels.

Any defect is detected by the oozing out of the dye from the vessels.

Electroretinogram (ERG):

* To assess the functioning of retina.

* a wave: Photoreceptors function

* b wave: Muller cell origin
  
  Represents bipolar cell activity

* c wave: RPE Function:

Electro oculogram (EOG):

* Only the RPE activity

OCT- Optical Coherence Tomography

* In vivo imaging of retinal layers, choroid & the vitreous
**RETINAL BLOOD SUPPLY**

- a sources of blood supply
  - Outer 4 layers (1/3nd)
    - Choriocapillaries
  - Inner 6 layers (2/3nd)
    - Central Retinal Artery (CRA)
      - branch of ophthalmic artery
        (branch of ICA)

macula receives additional blood supply from Ciliary retinal artery
in 20% of normal population

**FUNDUS**

- The view of retina.
- Optic disc:
  * intra ocular portion of optic nerve
  * Diameter → 1.5 mm
    - Known as Disc diameter (DD)
    * DD = 1.5 mm
    * Thickness → 1 mm
    * Colour → pink

- Central retinal artery
  - Nasal branches
  - temporal branches

- macula: Part enclosed by the temporal branches of the central Retinal Artery
  (5.5 mm in diameter)

- Fovea: Centre of macula [1.5 mm in diameter]
  [fovea centralis]
- Foveola: Central portion of fovea (0.35mm)
- Macula is temporal to optic disc
- Foveola → 2 DD (3mm) temporal to the optic disc

**FUNDUS FLUORESCIN ANGIOGRAPHY**

- Dye injected via antecubital vein
- Foveal Avascular Zone (500μ in diameter)
  \[
  \downarrow \hspace{1cm}
  \text{Black coloured area in the center with no capillary}
  \]

**OPTICAL COHERENCE TOMOGRAPHY**

- Various layers of retina seen
  \[
  \rightarrow \text{RPE} \\
  \rightarrow \text{Bruch's membrane} \\
  \rightarrow \text{Choria capillaries}
  \]
- Depression: Foveola
  \[
  \downarrow \\
  \text{Floor of foveola: umbo}
  \]
RETINAL VASCULAR OCCLUSIONS AND INFLAMMATION

CENTRAL RETINAL ARTERY OCCLUSION

- Ischemia of inner six layers of retina
- Presentation: Sudden painless loss of vision
- Ophthalmic emergency
- Pupil: RAPD (Relative Afferent Pupillary Defect)

- Fundus findings:
  - Retinal arteriolar attenuation
  - Milky white retina - Due to edema
  - Cherry - Red spot at foveola
  - ‘Cattle - Truck’ appearance
    or ‘Box - Car’ appearance
    of retinal veins
  - Hollenhorst plaque - Composed
    of cholesterol emboli

- Complication: Consecutive optic atrophy
  (irreversible LOV) [loss of vision]

- Retinal anoxic time: 90 minutes

- 4-6 hrs time for revival of retina before irreversible
  loss of vision sets in
MANAGEMENT OF CRAO

- Treatment:
  1. Ocular massage
  2. Carbogen inhalation (5% CO₂) – vasodilation
  3. Decrease IOP:
     - I.V mannitol
     - Paracentesis
  4. Intravenous vasodilators
  5. Streptokinase / rtPA

- Investigations:
  - 1st inv ECG (to rule out arrhythmias)
  - IOCA Carotid doppler

- M.C. cause of CRAO: Atherosclerosis at CCA (common carotid artery)
  \[\text{Atherosclerotic plaque}\]

  Embolus

- M.C. component of embolus: Platelet – fibrin embolus

- M.C. site of occlusion: Site of entry of CRA into meningeal sheath of ON

- Fundus – fluorescein angiography:
  Dye passes from artery into the veins and fills all vessels
- In 20% of population, cilioretinal artery also supplies macula and foveola.
- In CRAO with cilioretinal artery sparing, vision is saved.

### Central Retinal Vein Occlusion

<table>
<thead>
<tr>
<th>Cause</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>m.c. cause</td>
<td>Atherosclerosis</td>
</tr>
<tr>
<td>2nd m.c. cause</td>
<td>DM</td>
</tr>
<tr>
<td>Other causes</td>
<td>Any factor resulting in Virchow’s triad</td>
</tr>
<tr>
<td></td>
<td>eg: Hyperhomocysteinemia</td>
</tr>
<tr>
<td></td>
<td>Factor C / Factor S deficiency</td>
</tr>
<tr>
<td></td>
<td>Antithrombin III deficiency</td>
</tr>
</tbody>
</table>

- m.c. site of occlusion: Lamina cribrosa

### Clinical features:
- Subacute decrease in vision

### Signs:
- Dilated, tortuous retinal veins
- Hemorrhages all over retina:
  - ‘Splashed Tomato’ or
  - ‘Blood Thunder’ appearance
- RNFL infarcts:
  - Cotton wool spots also
  - Called soft exudates
- Axoplasmic stasis causing disc edema
- Retinal hypoxia
  ↓
- VEGF production
  → macular edema
  (ma cause of decreased vision in CRVO)
  → Neovascularisation
  ↓
  Neo - vascularisation of iris (NVI)
  ↓
  Neo - vascular glaucoma (NVG)
  '100 day / 90 day glaucoma'

**TYPES OF CRVO**

1. Non - ischemic CRVO: < 10 DD of non - perfusion on FFA

2. Ischemic CRVO:
   - On FFA; shows: (Fluorescein Fundus angiography)
     a) ≥ 10 DD area of capillary non - perfusion (CNP)
   - b) Higher risk of NVI/NVG
   - c) Marked decrease in vision
   - d) On ERG, 'b' wave is absent
     (electro retinogram)

**MANAGEMENT OF CRVO:**
- CRVO: Treat the underlying cause
- Macular edema: Intra - vitreal steroids
- NVI / NVG: Pan - retinal photocoagulation (PRP)
CHERRY - RED SPOT - CAUSES

1. CRAO
   retinal edema present

2. Trauma

3. Niemann - Pick disease

4. Gangliosidoses

5. Tay - Sachs's disease

6. Farber's disease

7. Sandhoff disease

8. Sialidoses

9. Metachromatic leukodystrophy

‘Cherry Trees Never Grow Tall in Far Sandy Soil and Mud’

- Blunt trauma - Causes retinal edema.

   ↓

   Called Berlin's edema.

- Commotio Retinae:
  - Berlin's edema + CR. spot
  - Concussion injury
  - Contrecoup injury

RETINAL VASCULITIS

1. Eale's disease:
   - Also called Periphlebitis retinae
   - Inflammation around peripheral retinal veins
   - Type III hypersensitivity to tubercular proteins

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• Fundus findings:
  - Exudates around retinal veins - Sheathing
  - Retinal hemorrhages
  - Retinal hypoxia → VEGF → Neovascularization
  - Recurrent vitreous hemorrhage in apparently healthy young adult males: Typical presentation

• Complication:
  - Vitreous hemorrhage
  - Neovascular glaucoma
  - Tractional retinal detachment

• Management:
  - Systemic steroids + Empirical ATT

2. Posterior Uveitis:
  - Toxoplasmosis : Kyrieleis' Arteriitis
  - Sarcoidosis : Retinal veins involved, exudates around veins
    "Candle wax drippings"
  - Behcet's uveitis : veins > Arteriules
**DIABETIC RETINOPATHY**

- microangiopathy → involving retinal capillaries

- 1st Histopathological change
  \[\downarrow\]
  Thickening of basement membrane of retinal capillaries

- Hallmark Histopathological change
  \[\downarrow\]
  Loss of pericytes
Risk factors for Diabetic Retinopathy

1) Duration of Diabetes → most important

a) Glycemic control

b) Systemic factors - control (blood pressure, lipid levels, blood urea, serum creatinine)

4) Anemia

5) Smoking

6) Pregnancy

Diabetic Retinopathy (DR)

Non Proliferative
Diabetic Retinopathy (NPDR)

Proliferative
Diabetic Retinopathy (PDR)

Hallmark: Neovascularisation

NON PROLIFERATIVE DIABETIC RETINOPATHY (NPDR)

1) microaneurysm - earliest clinical finding of Diabetic retinopathy
   - Found at Inner Nuclear Layer
   - It's an outpouching of Retinal capillary wall
a) Dot-Blot Hemorrhages

- Due to rupture of microaneurysms at outer plexiform layer

3) Venous changes

- Beading
- Looping
- Sausaging

4) Cotton Wool spots

- Non specific finding
- Evidence of Retinal infarct
- Retinal Nerve fibre layer infarct (RNFL)
- Also known as soft exudates
5) Hard exudates
   - Lipoproteinaceous exudates / deposits at outer plexiform layer

6) IRMA (Intra Retinal Microvascular Anomaly)

   - Shunt develops between one Arteriole
     and the other Arteriole

7) Flame - shaped Retinal hemorrhages / Splinter hemorrhages
   seen at the level of Retinal Nerve Fibre layer

---

4-2-1 RULE

- 4-2-1 Rule → to grade the severity of
  
  Non proliferative Diabetic Retinopathy
  
  (NPDR)
• 4-2-1 Rule
  
  Retinal hemorrhages in 4 Quadrants
  Venous changes in 2 Quadrants
  IRMA in 1 Quadrant

  Any 1 change → Severe NPDR
  Any 2 change → very severe NPDR

  ![Retinal images showing hemorhages, microaneurysms, hard exudates, and new blood vessels.]

  ma-microaneurysms
  He-Hard Exudates
  F-Flame shaped hemorrhages
  Vb-Venous Beading
  Db-Dot Blot hemorrhages
  npdr-npdr

• Presence of New blood vessel → PDR

  ![Images showing new blood vessels.]

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Neovascularisation

1. Neovascularisation of Disc (NVO)
   ↓
   On Disc
   or
   1 DD around disc

2. Neovascularisation Elsewhere on retina (NVE)
   ↓
   beyond 1 DD

3. Vitreous

4. Neovascularisation in Iris (NVI)
Complications of PDR: Advanced Diabetic Eye Disease

1) Vitreous hemorrhage
2) Tractional Retinal Detachment
3) Neovascular Glaucoma

MANAGEMENT OF DIABETIC RETINOPATHY

Screening of Diabetic Retinopathy

* By dilated fundus examination

<table>
<thead>
<tr>
<th>Type of DM</th>
<th>1st screening</th>
<th>Subsequent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1 DM</td>
<td>5 yrs after Diagnosis of DM</td>
<td>Annually</td>
</tr>
<tr>
<td>Type 2 DM</td>
<td>Immediately upon Diagnosis of DM</td>
<td>Annually</td>
</tr>
</tbody>
</table>

For NPDR (Non proliferative diabetic Retinopathy)

- Glycemic control
- Systemic factor control
- Finothene \(\rightarrow\) delays progression of disease

For PDR (Proliferative Diabetic Retinopathy)

Gold standard treatment

\[\downarrow\]

Pan Retinal photocoagulation

\[\downarrow\]

Hypoxic Retina \(\rightarrow\) Anoxic retina
Pan Retinal photocoagulation:

Lasers that can be used

1) Argon Green laser - 514nm
2) Diode laser - 810nm
3) Double frequency Nd: YAG laser - 532nm

- Layer of Retina that is Targeted by the laser
  \[ \downarrow \]
  RPE (Retinal Pigment Epithelium)

- Retinal Blanching (greyish white color) - End point for laser

- 1st laser spot is Inferior Quadrant
Adjuvant Treatment:

- Intravitreal anti-VEGFs
  1) Bevacizumab
  2) Ranibizumab
  3) Afibercept
  4) Pegaptanib

- Surgical management for complications
  For vitreous hemorrhage
    \[
    \text{Vitreous Hemorrhage} \rightarrow \text{Vitreectomy}
    \]

- Neovascular Glaucoma
  \[
  \text{Neovascular Glaucoma} \rightarrow \text{Glucoma surgery}
  \]

\[
\text{months after laser photocoagulation} \rightarrow \text{Laser burns chorioretinal scars}
\]
A-V CROSSING CHANGES

1) BONNET sign

Due to compression of vein by the Arteriole

↓

Distal Banking of vein

↓

Bonnet sign

---

a) GUNN sign

* Tapering of Retinal veins
3) Salu's sign

Deflection of Retinal vein

Normally, Arteriole & venule separated by acute angle

with increased compression

Angulation increased

Deflection of Retinal vein

Salus sign

→ Grade 3

Hypertensive Retinopathy

Copperwiring of Arterioles
* Image $\rightarrow$ Bilateral papilloedema

$\downarrow$

Grade 4 Hypertensive Retinopathy

$\rightarrow$ in the macular

$\downarrow$

Hard exudates arranged in the form of star

$\downarrow$

macular star

Grade 3 Hypertensive Retinopathy
RETINITIS PIGMENTOSA

- Most common inherited Retinal Degeneration
- It's a Degeneration of Rods
- Earliest symptom → NYCTALOPIA
- Earliest sign → 1) Delay in 'a' wave of ERG
  2) Delay in Dark Adaptation test

INHERITENCES IN RP

- Most common → Autosomal Recessive
- Best prognosis → Autosomal Dominant
- Least common & worst prognosis → X-linked recessive
- DHA (Docosa Hexaenoic Acid) Decreased in Retinitis pigmentosa (RP)
  ↓
  mainly in X-Linked Recessive RP

Eye findings in RP:
1) Myopic
2) Secondary Keratoconus
3) POAG (Primary Open Angle Glaucoma)
4) Complicated cataract
5) Macular edema
FUNDUS TRIAD

1) Arteriolar attenuation (1st fundus finding)
2) Waxy yellow pallor of optic disc
3) Black pigmented bony corporcles at mid-peripheral retina.

↓
Because rods are maximum
Syndromic RPs

1) Usher Syndrome
   - Most common
   - RP + SNHL (Sensory Neural Hearing Loss)

2) Cockayne Syndrome
   RP + SNHL

3) Laurence-Moon-Biedl Syndrome
   RP + Obesity
   Polydactyly
   Mental Retardation
   Hypogonadism

4) Bassen-Kornzweig Syndrome
   - Abetalipoproteinemia
   - Steatorrhoea
   - Fat soluble vitamin deficiency
   - 'Acanthocytes' in peripheral blood smear
   - RP
5) KEARNS-SAYRE SYNDROME
   RP + mitochondriopathy
   Cardiac conduction defects
   CPEO (Chronic Progressive External Ophthalmoplegia)
   
   * 1st investigation if we suspect kearns-sayre syndrome

   \[ \rightarrow \]
   ECG → To identify cardiac conduction defect

6) NARP:
   Neuropathy (peripheral)
   Ataxia
   RP

7) REFSUM'S DISEASE:
   * Absence of phytanic acid oxidase
     * RP
     * Ataxia
     * Ichthyosis

<table>
<thead>
<tr>
<th>MANAGEMENT</th>
<th>00:13:33</th>
</tr>
</thead>
<tbody>
<tr>
<td>* Genetic counselling</td>
<td></td>
</tr>
<tr>
<td>* VORETIGENE → Gene therapy approved for RPE-65 gene associated RP</td>
<td></td>
</tr>
</tbody>
</table>
**RETINAL DETACHMENT**

Definition

* Separation of RPE (Retinal Pigment Epithelium) and inner Neurosensory Retina.

**Types of RD (Retinal Detachment)**

- **Primary RD**
  - Due to Retinal tear / break
- **Secondary RD**

- **RHEUMATOGEOUS RD**
  - Most common type

- **TRACTIONAL RD**
  - New vessels growing into the vitreous exert a pull on the retina.
  - Resulting in Tractional Retinal Detachment

- **SEROUS / EXUDATIVE RD**
  - Subretinal fluid in the subretinal space cause serous Retinal Detachment
**RHEGMA TOGENOUS RD**

- Most common type

**Causes:**

1. Myopia (most common cause)
2. Trauma
3. Marfan’s syndrome
4. Pseudophakic RD
5. Aphakic RD

**Clinical presentation:**

- Sudden painless loss of vision
  
- Flashes of light → photopsia.
  
- Floaters

**Signs:**

- Greyish-white detached retina

- Convex and corrugated

- Retinal tears at ora serrata

- Lincoff’s rules → To localize Retinal tears

- Light reflex: Abnormal

  ↓

  Results in RAPD

- SHAFFER’s sign: Tobacco dusting of vitreous
Investigations:
- Indirect ophthalmoscopy (IDO)
- B-Scan ultrasound (if media is opaque)

Management:

1. Identify the Retinal break
2. Seal the break using Laser or Cryo
3. Reattach the detached retina
   - External Tamponade
   - Internal Tamponade
     - Pars plana vitrectomy
     - Vitreous substitutes

By SCLERAL BUCKLE

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VITREOUS SUBSTITUTES

Vitreous substitutes

- Using gases
  - Pneumoretinopexy
    - Non-expansile gases
      - Air
    - Expansile gases
      - 1, SF (most commonly used)
      - 2, C₃F₈
      - Air travel is avoided

Gases get absorbed on its own

- Using liquids
  1) Silicone oil
     - Side effects:
       - Reverse hypopyon
       - Silicone oil enters the anterior chamber
     - Floats on the aqueous humor
       - Bullous keratopathy
       - Band shaped keratopathy
       - Secondary open angle glaucoma
       - a) PFCL (Perfluoro carbon liquid)

Liquids need second surgery for removal
Causes:

1) Proliferative Diabetic Retinopathy (PDR)
   * most common cause
2) Eale's Disease
3) Ischaemic type of CRVO
4) Retinopathy of prematurity
5) Sickle cell retinopathy
6) PHPV / PFV (Persistent Hyperplastic Primary Vitreous / Persistent Fetal Vasculature)

* In all these conditions, there is Hypoxia.
  ↓
  Producing VEGF
  ↓
  Neovascularisation
  which exerts pull
  on the Retina.
  ↓
  TRACTIONAL RD

Clinical Features:

* Concave RD
* Fibrous Bands in vitreous
* Neovascularisation
* Epiretinal Membrane (ERM) on macula.
management

1) Pre-operative injection of Anti-VEGF intravitreally
   a) vitrectomy
   b) ERM peeling
   c) Endo laser PRP (Pan Retinal Photocoagulation)
   d) vitreous substitutes

SEROUS / EXUDATIVE RD

* Due to break down of outer Blood Retinal Barrier
  \[\rightarrow\]
  Resulting in sub Retinal fluid

Causes:

1) Malignant melanoma of choroid
2) Exophytic type of RD
3) Central serous choroidopathy
4) PHH Retinopathy (Pregnancy Induced Hypertension)
5) Posterior Scleritis
6) VMH (Vogt Koyanagi Harada) syndrome
Clinical features

- Subacute / Sudden decrease in vision
- Detached Retina → Convex & smooth
- SHIFTING Fluid - Hallmark

Investigation:

- Indirect ophthalmoscopy
- OCT (Optical Coherence Tomography) - most sensitive

Management:

- Treatment of underlying cause
RETINOBLASTOMA

- Most common primary malignant intraocular tumor of children
- Average age of presentation → 18 months
- Most common presentation of retinoblastoma.
  ↓
  Leukocoria.
- 2nd most common presentation
  ↓
  Squint

- Most common cause of intraocular calcification
- Most common route of metastasis through optic nerve (CSF)
- Most common site of metastasis → Brain
- "Cottage Cheese" Appearance of Tumor

Growth patterns of Retinoblastoma:

1) Endophytic → most common
  ↓
  Grows into the vitreous

2) Exophytic
   - It grows towards the choroid
   - It is associated with Serous Retina Detachment

3) Diffuse → Retinal thickening
  ↓
  Retinocytoma
  - Presents at 5-7 yrs of Age
HEREDITARY RETINOBLASTOMA

- Both alleles of RB (Retinoblastoma) gene (13q) have germline mutation
- Autosomal Dominant inheritance
- 40% of all RB's are hereditary (60% → sporadic)
- Trilateral Retinoblastoma
  - It is a combination of
    - Bilateral Retinoblastoma
    - Pinealoblastoma
- Second malignancy: most common → osteosarcoma
- Investigation of choice
  ↓
  MRI of orbits + Brain
- A-scan shows V-Y pattern
- FNAC / FNAB (Fine Needle Aspiration Biopsy) are contraindicated

TREATMENT

- Retinoblastoma classified based on ICRB in to
  - Group A, B, C, D, E

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• ICRB → International classification of retinoblastoma

• Group A → Focal Treatment 1) laser
  a) TTT (Transpupillary Thermotherapy)
  3) Cryotherapy
  4) Brachytherapy

• Groups B, C, D → Chemotherapy → Treatment of choice for Retinoblastoma.
  ↓
  "VEC" Regimen
  1. Vincaistine
  2. Etoposide Systemic (i.v)
  3. Carboplatin

  In vitreous seeds
  ↓
  Intra vitreal injection of 1) Topotecan
    a) Topotecan + melphalan

  Groupe E → Enucleation of eyeball
  ↓
  (minimum of 15mm length of optic nerve is cut along with eyeball)
- To rule out metastasis
- Enucleated eyeball
  ↓
  Histopathology
  ↓
  Rosettes
  ↓
  True rosettes
  PLEXNER-WINTERSTEINER rosettes
  • Specific for retinoblastomas
  • Indicates well differentiated tumor
  • Prognosis for life—Good
  ↓
  Pseudo-rosettes
  Homer-wright rosettes
  Fleurettes
  Can be seen in other PNET tumors (Primitive Neuro Ectodermal Tumors)
ADJUVANT CHEMOTHERAPY

- If the enucleated specimen show infiltration into the optic nerve
  
  ↓

  We have to give Adjuvant Chemotherapy

  - High dose VEC
    
    +
    
    Radiation

- In Retinoblastoma,

  Aqueous humor has high LDH (Lactate Dehydrogenase) levels

- Tumor cells can be seen in Anterior chamber
  
  ↓

  Appears as Hypopyon
  
  ↓

  Pseudo Hypopyon

- It's the 1st cancer to be associated with gene
  
  ↓

  Knudson 2-hit hypothesis is based on the Retinoblastoma gene located on 13q chromosome
macula

- Portion of retina which is enclosed by the Temporal branches of Central Retinal Artery (CRA)
- It measures around 5.5mm in diameter
- The central 1.5mm area of macula → Fovea.
- The central 0.35mm is called Foveola.

Symptoms of macular pathologies
1) Visual acuity < 6/6 (Does not improve with pinhole)
2) Metamorphopsia → Distorted vision
3) Central scotoma
MACULAR FUNCTION TEST

1) Visual Acuity
2) Color vision
3) 2-point Discrimination Test
4) Entoptic phenomenon
5) Amsler's Grid
6) Maddox Rod Test
7) Photostress Test
8) Laser Interferometry

Amsler Grid in Macular Degeneration

* Patient with macular pathology
  ↓ metamorphopsia.
  Lines in amsler grid appear wavy with central scotoma.
MACULAR EDEMA

- Edema at the level of outer plexiform layer
  (also known as Henle's layer)

- Extracellular fluid accumulates ↓ sometime forms cysts
  Also known as cystoid macular edema.

Causes:
1) Cataract surgery
2) Diabetic maculopathy
3) Uveitis - (Anterior / Intermediate / Posterior)
4) CRVO (Central Retinal Vein Occlusion)
5) Retinitis pigmentosa.
6) Vitreomacular Traction (vMT)
7) Drugs → 1) Prostaglandin Analogue
   a) Epinephrine & Dipivefrine
   b) Nicotinic acid

Ophthalmoscopy:

'Honey comb' Appearance

FFA: Fundus Flourescence

Angiography

↓

'Flower-petal' Appearance
due to leakage of dye
due to leakage of dye
in cystoid macular edema.
Optical coherence Tomography → Investigation of choice for
cystoid macular edema.

- Cyst at the level of outer plexiform layer

Management:
- Intravitreal steroids
- Intravitreal anti-VEGFs
- Macular grid laser-diode laser (680 nm)
- Vitrectomy (for refractory macular edema)
- Topical carbonic anhydrase inhibitors → In Retinitis pigmentosa
  with cystoid macular edema
  
  Brinzolamide
dorzolamide
BULL'S EYE MACULOPATHY

- In the region of macula

   \[\downarrow\]

   Atrophy of RPE (Retinal Pigmented epithelium)

   \[\downarrow\]

   Resembles Bull's eye

Causes:

1) Chloroquine

2) Hydroxychloroquine (HCQ)
   - Most sensitive test for HCQ Toxicity
     \[\downarrow\]
     Multifocal ERG

3) Congenital cone deficiency

4) X-linked Retinoschisis

→ Bull's eye macula

\[\downarrow\]

Alternate hyperpigmented & hypopigmented zones
CENTRAL SEROUS CHOROIDOPATHY

Pathophysiology:

Abnormal Hyperpermeable Choriocapillaries
↓
Resulting in subretinal fluid at macula
↓
Localised serous Retinal Detachment at macula

Risk factors:
- Type A personality
- Emotional stress
- Endogenous Hypercortisolism
- Exogenous steroids

Clinical feature:
- Ring reflex on ophthalmoscopy
* Investigation of choice → optical coherence tomography

↓

Subretinal fluid

Other investigations

* On Fundus Fluorescence Angiography
  1. Dye leaks into the subretinal space
      ↓
      ‘Smokestack’ Appearance
      Or
      mushroom-shaped Appearance

2. ‘Ink-blot’ Appearance
   Or
   ‘Enlarging dot’ sign

Management: Conservative

* Treatment of choice → Reassurance - it resolves spontaneously
* For fast recovery → Laser photocoagulation
   (only if it is away from foveal region)
BEST'S DISEASE

- It is also known as vitelliform macular dystrophy
- Due to mutation of VMD2 gene (Bestrophin gene)
- Autosomal Dominant inheritance
- Slow & progressive decrease in central vision
- 'Egg Yolk' Appearance (Normal vision) of fundus
- 'SCRAMBLED EGG' Appearance of fundus
  ↓
  Decrease in vision

- Electrooculogram is Abnormal but Electoretinogram is Normal

→ 'Egg Yolk' Appearance of fundus
STARGARDT'S DISEASE

- Mutation of ABCA4 gene
- Decrease in central vision
- 'Yellow flecks' at RPE (Retinal Pigment Epithelium)
- Described as pisciform lesion (appear like fishtail)
- 'Beaten Bronze' appearance of macula
- FUNDUS FLAVIMACULATUS → variant of stargardt's disease
- On FFA
  - 'DARK' or 'SILENT' choroid

ARMD-AGE RELATED MACULAR DEGENERATION

Risk factor:

1. Ageing
2. Sunlight
3. Smoking
4. Deficiency of vitamin - A, C, E
**Types of ARMD**

**Dry ARMD**
- Slow, progressive decrease in vision
- 'GLARE' during daytime
- Most common type

**Hallmark:**
- 'DRUSEN'

**Accumulation of lipofuscin between RPE & Bruch's membrane**
- Complications
  - Geographic Atrophy of RPE
- Treatment:
  - Antioxidants

**WET / Exudative ARMD**
- Rapidly progressive decrease in vision
- Poor visual prognosis

**Hallmark:**
- CNVM

**Choroidal Neovascular Membrane**
- Complications
  - Disciform scar at macula
- Treatment:
  1) Intra-vitreal anti-VEGF
  2) Photo Dynamic Therapy (PDT)

**Verteporfin Dye IV**
- Laser
  - photo thrombosis
RETINA-INVESTIGATIONS

1) FFA (Fundus Fluorescein Angiography)
2) FAF (Fundus Auto Fluorescence)
3) OCT (Optical Coherence Tomography)
4) ERG
5) multifocal ERG
6) EOG

FUNDUS FLUORESCINE ANGIOGRAPHY - FFA

- Fluorescein dye injected intravenously → Antecubital vein
- 10%, 20% & 25% of concentration of dye is used
- It reaches retinal vessels by 10-15 sec (Normal Retinal filling time)

1) To Assess the Integrity of BRB (Blood Retinal Barrier)
2) To visualise the Retinal Vasculature
3) To Assess the Foveal Avascular Zone (FAZ)

↓
To Diagnose Ischemic Maculopathy
FAF-FUNDUS AUTO FLUORESCENCE

- Auto fluorescence
  ↓
  Due to the presence of lipofuscin at the level of RPE (Retinal Pigment Epithelium)
  
- Lipofuscin fluoresces on its own, resulting in Auto-fluorescence

- In RPE Atrophy → No lipofuscin
  ↓
  The areas appear black

- In Drusen → High amount of lipofuscin
  ↓
  Hyper-fluorescence

- No dye injected in FAF

OCT-OPTICAL COHERENCE TOMOGRAPHY

- It visualises:
  1) All the layers of Retina:
     a) Choroid layers
     3) Vitreous (useful in vitreomacular Traction)

- To measure macular thickness

- Investigation of choice for:
  1) Subretinal fluid
     a) Macular edema.

- AS-OCT (Anterior Segment OCT)
  ↓
  For visualisation of Anterior segment structures
Fundus Fluorescence
Angiography

↓

Image → Retinal vessels filled with the dye
a-wave → Originates from photoreceptors
b-wave → From Muller Cell but represents Bipolar cell
c-wave → Originates from RPE (Retinal Pigment Epithelium)

- In early Retinitis pigmentosa → Delay in 'a' wave
- In advanced Retinitis pigmentosa
  ↓
  Extinguished / Flat ERG
- In ischemic type of CRVO
  ↓
  'b' wave is Absent
- In siderosis (ocular Foreign Body)
  ↓
  Negative ERG,
  ↓
  b wave becomes smaller
- To follow up the ocular toxicity due to iron Foreign Body → Serial ERG.
multifocal ERG

64 points on macula (hexagonal area)

- multiple Areas on the macula

- 64 ERG's

- It is one of the macular function test

- multifocal ERG sensitive investigation for HCQ (Hydroxychloroquine) Toxicity

---

**EOG—ELECTRO-OCULOGRAM**

- measures the resting potential of macula

- Indicator of RPE (Retinal Pigment Epithelium) activity

- ARDEN'S ratio (or index) = \( \frac{\text{Light peak}}{\text{Dark trough}} \)
GLAUCOMA: DEFINITION AND CLASSIFICATION

DEFINITION

- multifactorial, chronic and progressive OPTIC Neuropathy
due to death of retinal ganglion cells
- Characteristic optic disc changes and visual field defects

CLASSIFICATION OF GLAUCOMA

<table>
<thead>
<tr>
<th>Developmental</th>
<th>Acquired</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Congenital: Birth → 6 months</td>
<td>1. Open angle → primary (POAG)</td>
</tr>
<tr>
<td>2. Infantile: 1m → 3 yrs</td>
<td></td>
</tr>
<tr>
<td>3. Juvenile: 3 yrs → 13 yrs</td>
<td>2. Angle closure → Primary (PACG)</td>
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</tbody>
</table>

RISK FACTORS

POAG

1. Ageing
2. Black races
3. Smoking
4. Diabetes
5. Family history
6. Grave's disease
7. Treatment of HTN
8. myopia
9. migraine

PACG

1. Ageing
2. Family history
3. Females
4. Hypermetropes

IOP - only modifiable risk factor
**PROVOCATIVE TESTS**

- Difference between pre-test and post-test IOP ≥ 8mm Hg → suggestive of glaucoma.
- POAG: water drinking test
- PACG: 1. Dark-room test
  2. Prone test
  3. Prone-dark room test
  4. Mydriatic test
  5. Mydriatic-miotic test

**OPTIC DISC VISUALISATION**

Normally: neuro-retinal rim: Inferior > Superior > Nasal > Temporal

- Changes in disc:
  - Cup increase in size
    - Vessels move nasally
    - Baring of circumlinear vessels
    - Over all enlargement of cup
    - Bayoneting sign
    - Pores of lamina cribrosa visible – laminar dot sign
OPTIC DISC CHANGES

- Visualisation of these changes can be done using slit lamp along with a +78 D or +90 D lens
- Goldmann three mirror: central +60 D lens used to visualise posterior pole

Ocular hypertension (OHT): Increased IOP with normal optic disc and visual fields

Optic disc changes present and visual field defects present:

- IOP increased
  - POAG

- IOP normal
  - Normal tension glaucoma (NTG)

- OHT and NTG can progress to POAG, so should be treated by decreasing the IOP
PRIMARY OPEN ANGLE GLAUCOMA

RETINAL NERVE FIBRE ARRANGEMENT

- Peripheral nasal fibres - most resistant to glaucoma.

OPTIC DISC

- Central cup - horizontally oval
- Peripheral neuro-retinal rim-axons from all over retina converge here
- Thickness of rim - Inferior > Superior > Nasal > Temporal
  (ISNT rule)
- Cup - disc ratio (CDR) : normal → upto 0.6
  and difference between 2 CDR's → upto 0.1
  (ie difference between CDR of rt. & lt. eye)

OPTIC DISC CHANGES DUE TO GLAUCOMA

- Inferior ganglion cells of Bjerrum's area - die first
- Inferior rim thinning
- ISNT rule is broken
- Rim notching - due to localised ganglion cell death
- vertically oval cup–inferior rim loss
- Increased CDR (CDR ≥ 0.7) or difference between ≥ 0.2
- Enlargement of cup
- Deepening of cup – ‘Bean pot Cupping’
- Laminar dot sign
- Cavernous optic atrophy – primary atrophy

Changes on vessels of disc
- Nasalisation of vessels
- Baring of circumlinear vessels
- BAYONETTING sign – sharp angulation of the vessel

---

**VISUALISATION OF FUNDUS / OPTIC DISC**

- Slit lamp – combines advantages of direct (magnification) and indirect (stereopsis) ophthalmoscopy
- Lenses used with slit lamp

A. Concave: attached to slit lamp
   - MRUBY lens: plano-concave; − 58.6 D

B. Convex: hand-held

1. Contact type: touches cornea
   - Goldmann 3 – mirror – lens: central retinal visualisation lens of +60 D

2. Non-contact:
   - High power lenses
   - +78D or +90D
VISUAL FIELD DEFECTS OF GLAUCOMA

Earliest defect: isopter contraction leads to baring of blind spot

visually insignificant

PROGRESSION OF VISUAL FIELD DEFECTS

1. Earliest visually significant glaucomatous VF defect: Paracentral scotoma

![Diagram of Paracentral scotoma]

2. Seidel's
   - Sickle-shaped Scotoma

3. Ejerrum's Arcuate Scotoma:
   - Sicedel's scotoma extends to opposite side and stops at the horizontal raphe

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4. Ring scotoma - Advanced Glaucoma

Nasal step of Roenne

Patient has tubular / tunnel vision

5. Small peripheral Temporal island of vision is spared

Nasal Temporal

- Seen in end stage glaucoma.

PERIMETRY

- Visual field charting

1. Kinetic perimeter
   a) Goldmann's perimeter - for both central and peripheral field
      - Gold standard
   b) Lister's perimeter - only peripheral field
   c) Bjerrum's tangent screen - only central 30° VF.
      - Campimetry
   d) Confrontation test - VF of patient compared to examiner's VF
II static perimetry:
- Automated perimeters
  - E.g.: Humphrey's visual field analyser
- Newer perimeters:
  1) SWAP - Short wavelength automated perimetry
  2) FDP - Frequency doubling perimetry

Goldmann perimeter
- Normal visual field:
  max : Temporal
  Least : Superior
GONIOSCOPY AND TONOMETRY

GONIOSCOPY:
- To visualise angle structures
- Angle structures not seen on slit lamp due to total internal reflection (TIR)
- Critical angle of air - cornea interface - 46°

Types of gonioscopy:

Gonioscopes

Direct
used in surgery (goniotomy)
1. Koepepe's
2. Swan Jacob

Indirect
For diagnostic purpose
1. Goldmann's gonioscope (1 / 2 / 3 mirrors)
2. Zeiss 4-mirror gonioscope

* Gonioscope eliminates the TIR.
* Depending on whether light is seen directly or reflected by a mirror, there are a types of gonioscopy - Direct and Indirect
ANGLE STRUCTURES:

- From posterior to anterior
  1. Iris root
  2. Ciliary body band
  3. Scleral spur
  4. Trabecular meshwork
  5. Schwalbe’s line

‘I can see till Schwalbe’

- From anterior, if up to scleral spur is visible – open angle

Other uses of gonioscopy:

- Very early K-F ring
- Peripheral anterior synchiae (PAS)
- Any foreign body at the angle

Miscellaneous:

- Angle recession $\Rightarrow$ Enlarged / widened ciliary body band
- Sampaolesi’s line $\Rightarrow$ Prominent ( pigmented) Schwalbe’s line in pigment dispersion glaucoma.

ASSESSMENT OF ANGLE

1) Indirect: by AC depth on slit-lamp
   Van Herrick’s method
a) Objective methods:
   a) Ultrasound biomicroscopy
      by high frequency (35-50 MHz) anterior segment is visualised
   b) Anterior segment – OCT

Goldmann's gonioscopes

3 mirrors:
Smallest : To visualise angle
2nd biggest : Peripheral retina
Biggest : Equatorial / mid peripheral retina

Centre lens : +60 D
            : To see posterior pole

Zeis – 4-mirror gonioscope:
* All four quadrants can be visualised at once
TONOMETRY

- Measurement of intraocular pressure (IOP)
- Normal IOP: 10-21 mm Hg (16 mm Hg)
- Due to resistance to aqueous outflow → max at juxtacanalicular portion of trabecular meshwork
- Diurnal variation <inline>8 mm Hg
- Highest in the early morning

TYPES OF TONO METERS:

1. Indentation tonometer
   - Schiotz tonometer:
     IOP is affected by scleral rigidity

2. Applanation tonometer
   - Flatten the cornea
   - 2 types: Contact and noncontact type

A. Contact type
   a) Goldmann’s tonometer:
      - Gold standard
      - IOP is affected by pachymetry (Central corneal thickness)

b) Perkin’s hand-held tonometer
   - In congenital glaucoma
   - Also useful in bed-ridden patients
   - Used in children

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c) Tonopen
   → in older children
   → scarred corneas

B. Non-contact type
   * Non contact air - puff tonometer
     → post-op
     → also used for glaucoma screening

NEWER TONOMETERS

1. Rebound tonometer
   - Self - use

2. Dynamic contour tonometer
   - Called PASCAL
   - Not affected by central corneal thickness
   - Used in post - LASIK IOP measurement

3. Ocular response analyser (ORA)
   - Measures ocular pulse amplitude (OPA)

TONOGRAPHY

- Measurement of facility of aqueous outflow
- Normal: 0.22 - 0.28 μL/min/mm Hg
Disclaimer

- These Notes do not cover all the points, especially conceptual points, discussed in the videos. The information in this book is meant to complement Marrow videos and should be used in conjunction with the videos. Special emphasis on certain points and MCQ solving approach has to be understood from the videos only.
ACUTE CONGESTIVE GLAUCOMA

- Acute attack of angle closure glaucoma (ACE)
- Cause: when pupil is mid-dilated
  ↓
  Peripheral bunching of iris
  ↓
  Relative pupillary block due to increased resistance to aqueous outflow
  ↓
  AC pressure increased
  ↓
  Iris pushed forward
  ↓
  Angle closure

- Ophthalmic emergency
- IOP increase is rapid

CLINICAL FEATURES

1. Redness → circumciliary congestion
2. IOP increases to 40-60 mm Hg → Pain
   → Vagal stimulation
   ♦ Abdominal pain and vomiting
3. Endothelial pump failure → Corneal edema
   ♦ Decreased vision
   - Coloured halos
4. A.C. - Shallow
5. Angle - Closed
a. Pupil: Vogt's triad
   a) mid-dilated
   b) vertically oval
   c) Non-reactive to light

* Dilated, unreactive, vertically oval pupil

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

MANAGEMENT

- Immediate treatment: Decrease IOP
  1) I.V. mannitol
  2) Acetazolamide

DOC: Pilocar pine (miotic)

TOC: - Peripheral laser iridotomy
   - Using Nd-YAG laser (1064 nm)
   - As treatment for affected eye and prophylaxis of opposite eye

CHANGES AFTER AN ATTACK OF ACGL

Vogt's triad: Evidence of past angle closure attack

1) Glaucomflecken:
   Anterior subcapsular opacities on lens

2) Iris atrophy (sphincter muscle)

3) Pigment deposition on endothelium
### Natural Progression of Primary Angle Closure

<table>
<thead>
<tr>
<th></th>
<th>Gonioscopy</th>
<th>IOP</th>
<th>Optic disc</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary angle closure suspect (PACS)</td>
<td>Irido-trabecular contact (ITC) ≥ 3 quadrants</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Primary angle closure (PAC)</td>
<td>ITC ≥ 3 quadrants ± Peripheral anterior synechiae</td>
<td>Increased</td>
<td>Normal</td>
</tr>
<tr>
<td>Primary angle closure glaucoma (PACG)</td>
<td>ITC ≥ 3 quadrants ± Peripheral anterior synechiae</td>
<td>Increased</td>
<td>Optic disc changes and visual field defects</td>
</tr>
</tbody>
</table>

- **DOC for acute angle closure attack**: Pilocarpine
- **DOC for primary angle closure glaucoma**: Prostaglandin analogues
MEDICAL MANAGEMENT OF GLAUCOMA

mechanisms to decrease IOP

1) Decrease aqueous production

2) Increase aqueous outflow by:
   a) Increase trabecular outflow
   b) Increase uveoscleral outflow

3) Dehydrate vitreous

1) DECREASE AQUEOUS PRODUCTION

- Called aqueous suppressants
- Include:
  A. α - Agonists
  B. β - Blockers
  C. Carbonic anhydrase inhibitors (CA inhibitors)

A. α - agonists:

1. selective α-2 agonists
   - Contra indicated in children - Cause CNS suppression
     that manifests as sleep apnea, in children and drowsiness in adults
   - Drugs:
     a) Apraclonidine: Prevent IOP spike during laser iridotomy
     b) Brimonidine: Increase uveoscleral outflow
       Neuroprotectant
     c) Clonidine
a. Non-selective α-agonists
  - Contra-indicated in hypertensives
  - Drugs:
    d) Dipivefrine
  e) Epinephrine: Blackish discolouration of conjunctiva as side-effect
  - Both drugs cause macular edema;
    so contra-indicated in aphakic glaucoma

β - BLOCKERS

1. Selective β1 blockers
   Betaxolol
   - Used in asthmatics
   - Increase blood flow to optic nerve

2. Non-selective β-blockers:
   Timolol
   Levobunol
   - Contra-indicated in asthmatics
     A-V blocks
     Congestive heart failure
     Diabetics

CARBONIC ANHYDRASE INHIBITORS

1. Systemic - Acetazolamide

2. Topical - Brinzolamide
   Dorzolamide
Side effects of CA inhibitors:

1. Allergy
2. Aplastic anemia
3. Acidosis (metabolic)
4. Bone marrow suppression
   - Can be: a) Dose related
   - b) Idiosyncratic
5. Renal calculi
6. Dyspepsia
7. Glaucoma
8. Hypokalemia
9. Paresthesias in extremities and peri-oral region

II A) INCREASE TRABECULAR OUTFLOW

1. Pilocarpine
   - DDC for acute congestive glaucoma for miotic action
   - Side effects:
     a) Punctal stenosis
     b) Anterior subcapsular cataract
     c) Iris cyst
     d) Accommodation spasm → Induced myopia.
        :: Contra-indicated in young myopes
     e) Retinal detachment
        :: Contra-indicated in high myopes
     f) Contra-indicated in inflammation

2. Rho kinase inhibitors
   - Nedasuril
   - Side effect: Cornea verticillata.
8) INCREASE UVEO-SCLERAL OUTFLOW
- prostaglandin (PGF2α) analogues → DOC for decreasing IOP in primary open angle and angle closure glaucoma.

1. Latanoprost (most commonly used)
2. Travoprost
3. Bimatoprost (increase both trabecular and uveoscleral outflow)
4. Unoprostone
   - Once daily administration (good compliance)
   - Side effect:
     a) Trichomegaly
     b) Periocular hyperpigmentation
     c) Hyperchromia of iris
     d) Macular edema
     e) Contra-indicated in inflammatory glaucomas

II) DEHYDRATION OF VITREOUS
- Called hyperosmotics
  1) IV mannitol (20%)
  2) Oral glycerol syrup: contra-indicated in diabetics
  3) Isosorbide
  4) 50% urea.
Surgical Management of Glaucoma

Trabeculectomy

- Gold standard surgery for both open and closed angle
- Surgical resection of one clock hour of trabecular meshwork
- Adjacent peripheral iridectomy
- Aqueous drains into subconjunctival space by forming a filtering bleb and into episcleral veins

Antifibrotics

- Used to maintain patency of the filtering bleb
- Substances used:
  1) Mitomycin - C (most common)
  2) 5 - Fluorouracil
  3) Ologen (collagen implant)
- Augments effects of trabeculectomy
GLAUCOMA DRAINAGE DEVICES (GDD)

- No filtering bleb → less chance of fibrosis and failure
- Sutured onto sclera between superior and lateral rectus
- Also called seton or aqueous shunt
- Devices used:
  1. Ahmed glaucoma valve (most common)
  2. Miotto implant
  3. Baerveldt’s valve
  4. Krupin’s valve

MINIMALLY INVASIVE GLAUCOMA SURGERY

1. Viscocanalostomy
2. Express shunt — MRI compatible stainless steel
3. L-stent — titanium
4. Sol X shunt — Gold — drains into suprachoroidal space

The ExPRESS™ Mini Glaucoma Shunt

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CONGENITAL GLAUCOMA

INTRODUCTION

- Presents at birth to 6 months of age
- Mutation of CYP1B1 gene
- Incidence: 1 in 10,000 live births

SYMPTOMS:

Triad:
- Photophobia.
- Watering - most common
- Blepharospasm

SIGNS

- Buphthalmos
- Axial myopia
- Blue sclera.
- Megalocornea
- Haab’s striae:
  - Horizontal or circumferential tears of Descemet’s membrane
- Acute hydrops
- Deep anterior chamber
- At angle:
  - Barkan’s membrane
  - High / Anterior insertion of iris
- Lens:
  - Flat
  - Tendency for dislocation

- Optic disc:
  - Cupping (reversible if IOP is controlled by yrs of age)

---

**MANAGEMENT**

- Surgical management
  
  1. Goniotomy
     - Direct gonioscopes used
     - Done if cornea is clear

  2. Trabeculotomy
     - Done when cornea is hazy

  3. Trabeculotomy - Trabeculectomy
     - Best surgical outcome
     - Surgery of choice for congenital glaucoma.
GLAUCOMA: MISCELLANEOUS

SECONDARY ANGLE CLOSURE GLAUCOMAS

- No relief of angle closure post laser peripheral iridotomy, causes could be:
  1. Malignant glaucoma.
  2. Plateau iris.

MALIGNANT GLAUCOMA

- Not associated with malignancy.
- Also called Aqueous misdirection syndrome.
- Precipitating factor → Ciliary body inflammation and edema.
  ↓
  Aqueous flows from PC. To vitreous
  ↓
  Pushes lens and iris forward
  ↓
  Secondary angle closure occurs.

- Drug of choice: Cycloplegic – atropine (also a mydriatic).
  Also called inverse glaucoma, because cycloplegics are usually contra-indicated in angle closure glaucoma.

- Surgery:
  - Anterior hyaloidotomy (using Nd: YAG laser).

- Patient usually has history of trabeculectomy.
PLATEAU IRIS

- Natural contour of iris is like a plateau.
- Peripheral iridotomy does not relieve angle closure
- Treatment:
  * Laser iridoplasty: Using Argon laser (photocoagulation)
    - Causes scar tissue formation → Opens up the angle

SECONDARY OPEN ANGLE GLAUCOMAS

- mc cause of a°OAG: Pseudoxflliation syndrome

Pigment dispersion glaucoma
- myopia males
- Secondary OAG due to iris pigment deposition
- Sampolesi's line:
  - Prominent, pigmented schwalbe's line
  - Deposition of melanin
- Krukenberg spindle:
  - melanin at endothelium
HYPERSECRETORY GLAUCOMA
- Increased production of aqueous humor
- Occur in epidemic dropsy
- Argemone contamination of mustard oil
- Sudden increased IOP → Pain
- Corneal edema → colored halos

<table>
<thead>
<tr>
<th>GENETICS OF GLAUCOMA</th>
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<tbody>
<tr>
<td>Congenital glaucoma</td>
<td>CYP1B1 gene</td>
</tr>
<tr>
<td>Juvenile glaucoma</td>
<td>MYOC gene (myocillin)</td>
</tr>
<tr>
<td>NTG (Normal Tension Glaucoma)</td>
<td>TBR1 gene</td>
</tr>
<tr>
<td>Pseudo exfoliation glaucoma</td>
<td>LOXL1 gene</td>
</tr>
<tr>
<td>Steroid induced glaucoma</td>
<td>TGR gene</td>
</tr>
<tr>
<td>Congenital aniridia</td>
<td>PAX6 gene</td>
</tr>
<tr>
<td>Axenfeld-Reiger anomaly</td>
<td>PITX3 gene</td>
</tr>
<tr>
<td>ICE syndrome (Anterior Segment Dysgenesis)</td>
<td>FOXC1 gene</td>
</tr>
</tbody>
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VISUAL PATHWAY

* 4 order neuron pathway:

1st order: Photoreceptors (Rods and cones)
2nd order: Bipolar cells
3rd order: Ganglion cells (forming optic nerve)
4th order: Optic radiations (starts from LGB)

Optic tract: Ipsilateral temporal fibres + contralateral nasal fibres

- Nasal retina looks at temporal fields and superior retinal fibres look at inferior field
  and vice versa.
LESIONS OF VISUAL PATHWAY

1. Optic chiasmal lesions:
   - Fibres affected: B/L crossing nasal fibres ⇒ B/L temporal field
   - Field defect: bi temporal hemi-anopia
   - Caused by: Pituitary tumors cranio pharyngioma

2. Lesion of 🟠 optic tract:
   - Fibres affected: 🟠 Temporal 🟠 Nasal
   - Fields affected: 🟠 Nasal 🟠 Temporal
   - Field defect: 🟠 homonymous hemianopia

3. Lesion of 🟧 optic radiation:
   - Optic radiations:
     - Superior fibres: BAUM's loop - Pass via parietal lobe
     - Inferior fibres: MEYER's loop - Pass via temporal lobe
- In optic radiation lesion:
  a) Lesion of Temporal lobe (Meyer's loop):
     - Fibres affected: ☒ Inferotemporal, ☐ Inferonasal
     - Fields affected: ☐ Superonasal, ☒ Supero temporal
     - Field defect:
       ☒ Homonymous superior quadrantanopia.
       ‘pie in sky’ VF defect
  b) ☐ Parietal lobe lesion (Baum’s loop):
     - Fibres affected Fields affected
       ☐ Supero temporal → ☐ Inferonasal
       ☐ Supero nasal → ☐ Infero temporal
     - Field defect:
       ☒ Homonymous inferior quadrantanopia.
       ‘Pie on the floor’ VF defect

4. LESION OF VISUAL CORTEX:

- Visual cortex:
  - Primary visual cortex: Brodmann Area 17
    or Area V1
  - Secondary visual cortex: Brodmann Area 18, 19
    or Area V₂ and V₃
- Extensive representation of macula.
- Dual blood supply to visual cortex:
  1. MCA (Middle Cerebral Artery)
  2. PCA (Posterior Cerebral Artery)

Therefore, lesion of visual cortex → macular sparing

Lesion of ◯ visual cortex:
- Fibres affected
  ◯ temporal fibres → ◯ nasal field
  ◯ nasal fibres → ◯ temporal field

- Field defect: ◯ homonymous hemianopia with macular sparing
  ‘Cookie cutter defect’

S. Lesion of LGB:

- Contralateral homonymous sectoranopia
  `Keyhole visual field defect`
- Inferonasal fibres enter the substance of opposite ON
  before crossing over forming von willibrandt knee.

- ① von willibrandt knee - ② Inferonasal fibres

- ② Junctional lesion:

  - Involves ① optic nerve + ② vw knee (③ Inferonasal fibres)
  - Causes:
    1. ② Optic neuritis
    2. Mass lesions - Tuberous meningioma
       Pituitary tumor
- **Optic neuritis:**
  - superotemporal quadrantanopia + central scotoma
  - superotemporal quadrantanopia + centrocaecal scotoma

- **Mass lesion:**
  - Pressure atrophy of ON
    - superotemporal quadrantanopia + complete anopia

- All these field defects are called Junctional scotoma of Traquair
OPTIC NERVE AND LESIONS

OPTIC NERVE

• Each optic nerve has 1.2 million axons of ganglion cells
• 5cm in length
• 4 parts:
  1. Intraocular / Optic disc / Papilla:
     • Shortest (1mm)
     • Physiological blind spot (In temporal visual field)
     • Unmyelinated
  2. Intra. orbital:
     • Longest (4.5mm)
     • Myelinated
  3. Intra. canalicular:
     • Within optic canal (In lesser wing of sphenoid)
     • Traumatic optic neuropathy common in this segment
     • Accompanied by ophthalmic artery
  4. Intra. cranial:
     • Susceptible to pressure atrophy
• Blood supply of optic nerve:
  - Short posterior ciliary artery (SPCA)
  - SPCA along with pial vessels form circle or plexus
    Of Zinn - Holier
**OPTIC NEURITIS**

- Inflammation of optic nerve
- Sudden, profound, progressive decrease in vision
- Red desaturation
- Phosphenes: Light sensation with eye movement
- Uthoff symptom:
  - Transient decreased vision with increased body temperature
- Pulfrich phenomenon:
  - Stereo-illusion false sensation of depth
- Earliest sign: Relative afferent pupillary defect (RAPD)
  - Also called Marcus-Gunn Pupil
  - Elicited by swinging flashlight test

- In right optic neuritis results of swinging flashlight test is as follows:

```
<table>
<thead>
<tr>
<th></th>
<th>Right pupil</th>
<th>Left pupil</th>
</tr>
</thead>
<tbody>
<tr>
<td>Light</td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td>Aff: 2 II nerve</td>
<td></td>
<td>Aff: 2 II nerve</td>
</tr>
<tr>
<td>Eff: 2 II nerve</td>
<td></td>
<td>Eff: 1 II nerve</td>
</tr>
<tr>
<td></td>
<td>O</td>
<td>O</td>
</tr>
<tr>
<td></td>
<td>No constriction</td>
<td>Light</td>
</tr>
<tr>
<td>Aff: 1 II nerve</td>
<td></td>
<td>Eff: 1 II nerve</td>
</tr>
<tr>
<td>Eff: 2 II nerve</td>
<td></td>
<td>Eff: 1 II nerve</td>
</tr>
<tr>
<td>Light</td>
<td></td>
<td>O</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Recovery to normal size.</td>
</tr>
</tbody>
</table>
```

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CLINICAL TYPES OF OPTIC NEURITIS

I. Retrobulbar neuritis:
   - Inflammation of nerve anywhere behind lamina cribrosa
     (upto lateral geniculate body)
   - Most common type
   - Painful on eye-movements.
   - Most common cause: multiple sclerosis.
   - Fundus examination looks normal

II. Papillitis:
   - Inflammation of optic disc
   - Painless
   - Usually seen in children
   - Hyperemic disc edema on fundus examination.

III. Neuro retinitis:
   - Inflammation of RNFL (Retinal nerve fiber layer)
   - Least common type
   - Most common cause:
     Cat- scratch disease
   - Fundus examination:
     - Disc edema
     - Macular star
• Overall most common cause of optic neuritis: multiple sclerosis

• Investigation of choice (IOC)
  - Gadolinium – enhanced MRI of orbit and brain

• Treatment of choice:
  - Systemic steroids
  - Protocol:
    1st three days – Intravenous methyl prednisolone
    Dose: 1g/day

    Following 11 days – Oral prednisolone
    Dose: 1mg/kg/day

    Then taper the dose

  - Only oral steroids are contra – indicated because of increased risk of recurrence

• Visual field defects of optic neuritis:
  1. Central scotoma
  2. Centrocaecal scotoma

---

ANTERIOR ISCHEMIC OPTIC NEUROPATHY (AION)

• Occlusion of SPCA

• Types of AION:
  1. Non arteritic AION (NA-AION)
  2. Arteritic AION (A-AION)
Non-arteritic AION:
- most common type of AION
- mc. cause: Atherosclerosis
- Sudden painless loss of vision
- Field defect: inferior altitudinal hemianopia.
- Fundus finding: hyperemia with disc edema.
- management:
  - Treat underlying cause
  - Levodopa + carbidopa

Arteritic AION:
- mc. cause: Giant Cell Arteritis (GCA)
- Sudden painful loss of vision
- Other symptoms:
  - Amaurosis fugax
  - Scalp tenderness
  - Jaw claudication
- Associated with polymyalgia rheumatica
- Fundus finding:
  - Pale disc edema.
- IOC-Temporal Artery Biopsy
- ESR, CRP increased
- TOC- Systemic steroids.
  3 + 11 days protocol followed
- Considered as a emergency
- Steroids protect the other eye from inflammation
PAPILLEDEMA

- Bilateral disc edema due to raised intracranial tension (ICT) causing axoplasmic stasis

- Clinical features:
  - Raised ICT symptoms - Headache, diplopia (6th nerve palsy), projectile vomiting
  - Pupil normal

- Visual field defect:
  - Bilateral enlarged blind spot

- Fundus findings: Depends on stage

I. Early stage:
  - Blurring of disc margin-earliest (nasal margin first)
  - Veins: dilated and tortuous
  - Absent spontaneous venous pulsation
  - Hyperemia of disc

II. Established stage:
  - Obliteration of cup
  - Elevation of disc surface
  - Peripapillary retinal edema - PATON'S lines
  - Cotton wool spots
  - Disc hemorrhages

III. Chronic stage:
  - Champagne cork appearance of optic disc

IV. Atrophic stage:
  - Secondary optic atrophy
**IDIOPATHIC INTRACRANIAL HYPERTENSION**

- Also called - Benign intracranial hypertension (BIH)
  - Pseudotumor cerebri

- Bi-lateral disc edema due to raised ICT
- Clinical features of raised ICT
- MRI or CT of brain normal (ventricle size-N/small)
- Hallmark: Raised CSF opening pressure on lumbar puncture
- Risk factors: Obesity
  - Perimenopausal
  - Females

- Causes: Drugs like:
  1. Vit. A toxicity
  2. Tetracyclines
  3. Chronic steroid use / steroid withdrawal
  4. OCPs (oral contraceptives)
  5. Phenothiazines
  6. Nitrofurantoin
  7. Nalidixic acid

- Management:
  - Stop drugs (if any)
  - Weight Reduction
  - Medical management:
    1. Acetazolamide
    2. Furosemide
    3. Topiramate

- Surgical management:
  1. Ventriculo peritoneal shunt
  2. Optic nerve sheath fenestration
**OPTIC ATROPHY**

- Types:
  1. Primary optic atrophy:
     - optic atrophy with no preceding disc edema
  
  Causes:
  a) Hereditary optic neuropathies
  b) Toxic optic neuropathies
  c) Glaucoma
  d) Retrobulbar neuritis

2. Secondary optic atrophy:
   - optic atrophy following disc edema
  
  Causes:
  a) Papilledema
  b) IIH (Idiopathic Intracranial Hypertension)
  c) Papillitis
  d) Neuroretinitis

3. Consecutive optic atrophy:
   - due to extensive conditions affecting retina
  
  Causes:
  a) CRAO
  b) Retinitis pigmentosa
  c) Extensive chorioretinitis
  d) Following PRP
Fundus Findings:

<table>
<thead>
<tr>
<th></th>
<th>Primary OA</th>
<th>Secondary OA</th>
<th>Consecutive OA</th>
</tr>
</thead>
<tbody>
<tr>
<td>OPTIC disc color</td>
<td>Chalky white</td>
<td>Dirty white</td>
<td>Waxy yellow pallor</td>
</tr>
<tr>
<td>margins</td>
<td>well-defined</td>
<td>Blurred</td>
<td>Well-defined</td>
</tr>
<tr>
<td>Cup</td>
<td>Normal/deep</td>
<td>Obliterated</td>
<td>Normal</td>
</tr>
<tr>
<td>Vessels on disc</td>
<td>Normal</td>
<td>Sheathing</td>
<td>Attenuated</td>
</tr>
</tbody>
</table>

Hereditary Optic Neuropathies

- Most common: Kjer's autosomal dominant hereditary optic neuropathy
- Leber's Hereditary optic neuropathy (LHON)
  - Due to mitochondrial mutation
  - Mitochondrial inheritance
  - Maternal transmission
  - Males affected
- Bilateral, sequential, subacute loss of vision in young males (3\textsuperscript{rd} - 4\textsuperscript{th} decade)

- Fundus findings:
  - Juxta-papillary telangiectasias - Do not leak on FFA

---

**TOXIC OPTIC NEUROPATHY**

- Toxins that "CLAIMED" vision are:
  
  C - Chloramphenicol / cyanide (Toxic agent of Tobacco amblyopia)
  
  L - Lead
  
  A - Aspirin / Arsenic / Alcohol
  
  I - Isoniazid
  
  M - Methanol (Formic acid toxic to ganglion cells)
  
  E - Ethambutol
  
  D - Digoxin (Can cause xanithopsia)

  Deficiency of vit. B1 and B12

---

**BI-LATERAL CENTRO-CAECAL SCOTOMA**

- Selective damage to papillomacular bundle

- Causes:
  
  1. vit B 12 deficiency
  
  2. Tobacco amblyopia.
  
  3. Alcohol amblyopia.
**PUPIL AND ITS ABNORMALITIES**

**POPUILLARY LIGHT REFLEX PATHWAY**

- **Sphincter**
  - → Nasal fibers
  - → Temporal fibers
  - → Sphincter
  - → Relative Afferent pupillary defect
- **III N**
  - → Distal optic Tract lesion
  - ▼ Wernicke's Hemianopic pupil
  - ▼ PTN

Afferent → Optic Nerve (L/R)
Centre → Pretectal Nucleus @ midbrain
Edinger westphal Nucleus: (Parasympathetic subnucleus of III CN)
  ▼ @ midbrain

**WERNICKE'S HEMIANOPIC PUPIL**

Right @ → • Temporal ½ of Retina.
  ▼ No pupillary constriction
  • Nasal ½ of Retina.
  ▼ P. Constriction
  • Nasal ½ of side
  ▼ Pupil constriction
  • Temporal ½ of Retina.
  ▼ P. Constriction

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DORSAL MIDBRAIN SYNDROME

- Fibers of accommodation reflex are spared
  aka Parinaud's DMS
  - Light reflex absent & near reflex is retained

Causes:
- Neurosyphilis
- Tabes Dorsalis
- DM (mcc)

This pupil is k/a. - Argyll-Robertson pupil

.: This can be called as Light-Near Dissociation

ADIE'S TONIC Pupil

If:
- III N palsy → Dilated pupil
  * Ciliary Gang palsy → Dilated pupil

Adies tonic pupil - lesion @ ciliary ganglion
  - Post ganglionic lesion
  - Dilated pupil
  - Sluggish to both light & near reflex
  - Pupil constricts to 0.25% Pilocarpine
  - d/t Denervation Hypersensitivity

Adie's syndrome (Adie-Holmes's synd):
  - Adie's pupil + Absent Deep Tendon Reflex
    - Knee Jerk (Ab) + Ankle jerk (Ab)

Hutchison's pupil @ uncal herniation
  - Constriction start with → Later dialation of pupil
  - d/t III N involvement
DISORDERS OF GAZE

GAZE CENTRES

1. Vertical gaze centres: * Interstitial Nucleus of Cajal (INc)  
   * Rostral Interstitial medial Longitudinal fasciculus (ri mlF)
2. Horizontal gaze centres: Paramedian pontine reticular formation

PATHWAY FOR HORIZONTAL GAZE

* Horizontal gaze ⇒ either Left gaze / Right gaze
  
  * Eg: Right gaze:

  ![Diagram of eye movement](image)

  Right eye: Abduction
  ↓
  ○ Lateral rectus supplied by ○ 6th nerve

  Left eye: Adduction
  ↓
  ○ Medial rectus supplied by ○ 3rd nerve

That is the ○ 6th nucleus & the ○ 3rd nucleus should be connected

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LESIONS OF HORIZONTAL GAZE

- Lesion of frontal eye field:
  - Contralateral horizontal gaze palsy
  - Gaze preference to the same side

- Lesion of PPRF:
  - Ipsilateral horizontal gaze palsy
  - Gaze preference to opposite side

- Lesion of VI nucleus:
  - Ipsilateral horizontal gaze palsy
LESION OF MLF

Eg: ⊗ MLF lesion

On ⊡ gaze

⊗ eye abduction + ⊙ eye adduction deficit
(/± impulse is not passed onto
⊙ medial rectus subnucleus)

- Ipsilateral Adduction deficit + contralateral abduction with nystagmus

- Since it is a lesion of the structure (MLF) connecting
two nuclei (VIth nucleus + medial Rectus subnucleus)

↓

Known as internuclear opthalmoplegia.

↓

most common cause: multiple sclerosis

LESION OF BILATERAL MLF:

⇒ Bilateral adduction deficit

⊗ Bilateral divergent squint
[Wall – eyed condition]

⇒ Bilateral internuclear opthalmoplegia.
[most common cause: multiple sclerosis]

* Also known as WEBINO syndrome

- Wall Eyed condition
- Bilateral
- INO – inter nuclear opthalmoplegia.
Lesion of MLF + PPRF

Eg: R MLF + R PPRF

\[ \downarrow \quad \downarrow \]

R adduction deficit \quad R gaze palsy \quad \Rightarrow R abduction deficit

\[ + \]

L adduction deficit

\[ \begin{array}{l}
\text{R} \\
\text{L}
\end{array} \]

One-and-a-half syndrome

Lesion of MLF \& VI nucleus \quad \text{Produce the same effect as above}

Lesion of MLF \& PPRF + VI nucleus

Eight \& a half syndrome; one \& a half syndrome

\[ + \]

VII nerve palsy

Disclaimer

- These Notes do not cover all the points, especially conceptual points, discussed in the videos. The information in this book is meant to complement Marrow videos and should be used in conjunction with the videos. Special emphasis on certain points and MCQ solving approach has to be understood from the videos only.
**ANATOMY OF EOM**

- **eom** - 6 in number

  - 4 Recti
    - a. Horizontal
      - • Medial
      - • Lateral - 6th N
    - a. Vertical
      - • Superior
      - • Inferior
  - 2 oblique
    - Sup oblique
      - 4th Nerve
    - Int oblique

- All EOM are supplied by 3rd N except 5th & 6th

- Origin of all recti muscles = Annulus of Zinn

- Spiral of Tillaux: Imaginary line obtained by connecting the insertions of all recti

  - **m.R.** (5.5mm) from limbus
    - Superior Rectus (S.R.) - 7.7 mm from limbus
    - Limbus
  - LR - 6.9 mm from limbus
    - Spiral of Tillaux
  - LR - 6.6 mm from limbus

- Distance of Recti from limbus: S.R > L.R > I.R > M.R

- Superior Rectus - Elevation

- Inferior Rectus - Depression

- Lateral Rectus - Abduction

- Medial Rectus - Adduction

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Anatomy of obliques:
- Superior oblique:
  - Origin - Apex
  - Functional origin - Trochlea
  - Muscle with longest tendon
- Superior oblique on contraction - intorsion of eyeball
- Inferior oblique Anatomy:
  - Only muscle with NO tendon
  - It arises from the Anteromedial part of orbit (Maxillary part)
  - On contraction: Extorsion of eye ball
  - Muscle whose insertion is closest to the macula - Inferior oblique

<table>
<thead>
<tr>
<th>ACTIONS OF EOM'S</th>
<th>00:13:32</th>
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<tbody>
<tr>
<td>M.R</td>
<td>ABDUCTION</td>
</tr>
<tr>
<td>L.R</td>
<td>ABDUCTION</td>
</tr>
<tr>
<td>S.R</td>
<td>ELEVATION</td>
</tr>
<tr>
<td>I.R</td>
<td>DEPRESSION</td>
</tr>
<tr>
<td>S.O</td>
<td>INTORSION</td>
</tr>
<tr>
<td>I.O</td>
<td>EXTORSION</td>
</tr>
</tbody>
</table>

"SINRAD" - Superior Intorsion
Recti Adduct

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GAZE

- Rt. gaze - R. LR + L. MR ⇒ Yoke muscles

\[
\downarrow
\]

Helps in version / Binocular conjugate movement

Hering's law: Equal innervation of yoke muscles for motor correspondence

- Violation / Exception of Hering's law: Dissociated vertical Deviation

SOLE / BEST ACTION OF MUSCLE

- Superior oblique - Depression of Adducted eye
- Inf. oblique - Elevation of Adducted eye
- Sup. rectus - Elevation of Abducted eye
- Inf. rectus - Depression of Abducted eye

GAZES

- Total - 9 gazes
- 1° gaze - 1
- 2° gazes - 4
- 3° gazes - 4
- Cardinal gazes - 6

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Sherrington's law: Reciprocal inhibition of V/L Antagonist muscle. This is for uniconal movement → DUCTION

6TH NERVE PALSY - RIGHT

→ Rt.lat.Rectus palsy

* Rt. lat. rectus palsy - V/L medial rectus - overactive
  - Rt. eye in Adducted position
  - Squint in Rt.eye - Rt. convergent squint / Rt. Esotropia

* ESO - Convergent squint
* EXO - Divergent squint
* TROPIA - manifest squint
* PHORIA - Latent squint

Binocular Diplopia: Diplopia only when BOTH eyes are opened

↓

Patient turns Head on same side of palsy, this is to avoid Diplopia.
K/a compensatory Head posture. This is to avoid Binocular Diplopia.

Eg: In LR Palsy → Head / Face turns to the same side
   In S.O Palsy → Head tilt towards contralateral shoulder

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TESTS TO DETECT PHORIA

Orthotropia.

Dissociative tests / Breaking the fusion of eyes

Lt. Exophoria.

Tests:
1. Cover – uncover test
2. Alternate cover test
3. Maddox Rod test
4. Red – green glasses test – Least Dissociative test

MEASUREMENT OF SQUINT

Hirschberg test
2. Krimsky’s test
3. Prism Bar cover test (PACT)
4. Double Maddox rod test
5. Synaptophore

Requires patient’s cooperation
units for squint:
1. Degrees
2. Prism Diopters

One Degree = 2 prism Diopters

Primary Deviation: The angle of deviation when the normal eye is fixated
2° Deviation: The angle of squint being measured when the paralysed eye is fixated
* In paralytic squint, 2° Deviation >> 1° Deviation – explained by Hering's law

COMITANT & NON COMITANT SQUINT

1. Comitant squint: Angle of squint measuring is same in all gazes
2. Non comitant / Incomitant squint: Angle of squint measuring are different in different gazes

Eg: 1. Paralytic squint
   2. Restrictive squint – in # of floor of orbit
   3. A.V Pattern squint – occurs because of obliques overactivity
      - Eyes - more convergent in upgaze – ‘A’ pattern
      - more divergent in Downgaze – ‘V’ pattern

FORCED DUCTION TEST (FDT):

• It's a test to differentiate paralytic and Restrictive squint
• No Resistance to the movement • Feel tug / Resistance to movement

↓
FDT Negative
↓
Paralytic squint

↓
FDT Positive
↓
Restrictive squint

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1. Correction of Refractive errors
   
   * There is more tendency for Hypermetropes to develop Esotropia
     
     Hypermetropia: Esotropia
     Myopia: Exophoria > Exotropia
   
2. Prisms in spectacles

3. Botulinum toxin 'A' injection into the overacting muscle \Rightarrow I/L Antagonist
   
   * Eg: In RLR palsy \Rightarrow Injection given to RMR
   
   * This avoids Diplopia in the patient

4. Surgeries:
   
   * Strengthening the paralysed muscle
     
     \begin{align*}
     \downarrow \\
     \text{RESECTION}
     \end{align*}
   
   * Weakening the overacting I/L Antagonist
     
     \begin{align*}
     \downarrow \\
     \text{RECESSION}
     \end{align*}

   * Eg: In RLR palsy \Rightarrow RLR Resection \pm RMR Recession
VITREOUS

- Volume - 4ml
- 99% of vitreous: Water
- Consistency due to: Hyaluronic acid
- High ascorbate levels
- Type II collagen
- Hyalocytes present in vitreous

Syneresis: Collapse of vitreous
Synchysis: Liquefaction of vitreous

VITREOUS DEGENERATIONS

1. Asteroid hyalosis
   - Calcific degeneration
   - Whitish calcium soaps seen

2. Synchysis scintillans
   - Cholesterol crystals in vitreous
   - Golden shower appearance
   - Sequelae of vitreous hemorrhage

VITREOUS HEMORRHAGE (VH)

- Floaters: in tiny bleeds
- Sudden painless loss of vision - in massive bleeds

Most common cause:
- in children: Blunt trauma
- Older adults: Proliferative diabetic retinopathy
- Young adults
  (Recurrent VH): Eale's disease
management:
- Observation
- Vitrectomy: Immediately, for vision in single eye
  - After 3-6 months, in diabetics (type II)
  - After 1 month, in type I DM

Complication:
- Secondary open angle glaucoma - block of trabecular mesh work by the blood cells

Intravitreal injections or vitrectomy instruments are passed through pars plana because the strongest attachment is at vitreous base

---

**INTRAVITREAL INJECTIONS**

* Drugs given:
  1. Antibacterials except Gentamicin → Retinal toxicity
  2. Antifungals
  3. Antimetabolites
  4. Steroids
  5. Anti-VEGF's

Warning: Not all points are covered in the notes, especially conceptual explanations. Please use the notes in conjunction with Marrow Edition 4 videos.

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**VITRECTOMY**

- 3 ports: → For light source
  → For irrigating fluid
  → For instruments

- Instruments are usually 23G or 25G, 27G (newer)
- Intravitreal injections - 25G or 30G needles
PERSISTANT HYPERPLASTIC PRIMARY VITREOUS

Embryology of vitreous:

1° vitreous: Hyaloid artery
   - Arises from mesoderm

2° vitreous: vitreous gel
   - Derived from neuroectoderm

3° vitreous: Zonule
   - Derived from neuroectoderm

Primary vitreous usually regresses at birth
If persistent, causes Persistant Hyperplastic primary vitreous or persistant fetal vasculature

Clinical features:

1) Leucocoria
2) microphthalmos
3) Congenital cataract
4) Elongated ciliary processes

Complications:

1) Vitreous hemorrhage
2) Tractional retinal detachment
3) Secondary angle closure glaucoma
**ORBIT**

**ORBIT - ANATOMY**

- Shape → Pyramidal or Pearshaped
- Volume → 30ml
- There are four walls formed by seven bones
  - Roof of the orbit formed by
    1. Orbital plate of frontal bone
    2. Lesser wing of sphenoid
  - Lateral wall of orbit
    1) Frontal Bone
    2) Zygomatic Bone
    3) Greater wing of sphenoid
  - Floor of the orbit
    1) Maxillary Bone
    2) Zygomatic Bone
    3) Palatine Bone

---

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Scanned with CamScanner
• medial wall of orbit (from Anterior to Posterior)
  1) Maxillary Bone \{ forms the Lacrimal fossa
  2) Lacrimal Bone \ to accommodate Lacrimal sac
  3) Ethmoid Bone
      ↓
      Lamina papyracea
  4) Body of sphenoid

• Roof of the orbit is related to the Frontal sinus
• medial wall is related to the Ethmoid sinus
• Floor is related to Maxillary sinus

**ORBITAL CONTENTS**

1. Eyeball
2. Optic Nerve
3. Extra ocular muscles
4. Levator palpebrae superioris
5. Ciliary ganglia
   ↓
   Lies posteriorly between the lateral Rectus and optic Nerve
6. Blood vessels
7. Lymphatics & Lymphoid Tissue
8. Lacrimal Gland & Lacrimal sac
9. Other cranial Nerves
10. Orbital fat
**ORBITAL FLOOR FRACTURE**

- mc wall to be fractured
- Occurs due to Blunt trauma.
- Size of the object ≥ 5cm
- mc site of fracture
  ↓
  Posteromedial aspect of the floor
- It is also known as Blow out Fracture
  ↓
  Because orbital contents are prolapsing out

Clinical features
- Periorbital ecchymosis
- If Inferior Rectus muscle gets entrapped in the floor fracture
  ↓
  When the patient is looking up
  ↓
  Involved eye is not moving up
  ‘Restricted elevation’
'Restricted elevation'

\[ \downarrow \]
Squint $\rightarrow$ Diplopia.

- Loss of sensation over the ipsilateral cheek

\[ \downarrow \]
Due to infraorbital nerve involvement

- Investigation of choice

\[ \downarrow \]
Plain CT of orbit coronal section

x-ray $\rightarrow$ Water's view $\rightarrow$ 'Teardrop'

or

'Waterdrop' sign
management

* Observe and short course of systemic steroids
  ↓
  To settle down orbital edema.

* Orbit Floor Repair
  Indications:
    1) Enophthalmos ≥ 2mm
    a) Persistent Diplopia for > 2 weeks

Blow out Fractures (BOF)

↓

Pure BOF

↓

Impure BOF associated with rim fractures

---

**THYROID EYE DISEASE**

* most of proptosis in adults

* It may be unilateral or bilateral

* Exophthalmos
  ↓
  Proptosis due to Thyroid eye Disease
Fibroblast Activation result in formation of

- Adipocyte
  - Inflammation
  - Releases inflammatory cytokines

- Myofibroblast
  - Secretes Glycosaminoglycans GAGs which are hydrophilic
  - Volume increases
  - Displaces eyeball outwards

- Later this myofibroblast

  - Causes fibrosis of muscle

  - Restriction of muscle movement → Restrictive squint

- Order of muscle involvement

  - Both enlargement & fibrosis

  - 1st muscle → Inferior Rectus
    - Medial Rectus
    - Superior Rectus
    - Lateral Rectus
    - Obliques

EOM enlargement with tendon sparing
Dalrymple sign
Due to
1) Overactivity of muller's muscle
2) Fibrosis of LPS → Pulls the lid up

VON GRAEFE'S SIGN
Lid lag on down gaze

* upper lid retraction and Exophthalmos
  ↓
  Staring look
  Kocher's sign

* Investigation of choice for TED
  ↓
  CT orbit
1. Stop smoking
2. Normalise the Thyroid Function Test
3. Systemic steroids – Treatment of choice
4. Immunosuppressant
5. Radiotherapy
6. Orbital wall Decompression
   ↓
   Order:
   Medial wall
   ↓
   Inferior wall
   ↓
   Lateral wall
   ↓
   Superior wall

- MCC of Decreased vision in TED
  ↓
  Compressive optic Neuropathy

- Restriction of eye movements in TED
  ↓
  1st muscle $\rightarrow$ Inferior Rectus
  So, 1st movement $\rightarrow$ Elevation of eyeball
PROPTOSIS

- Forward protrusion of the eyeball

Axial proptosis

- Eyeball is pushed forward and outward

1) Optic Nerve Tumors
2) Retrobulbar hemorrhage
3) Orbital cellulitis
4) Cavernous hemangioma

Non-Axial Proptosis / Eccentric proptosis

1) Down & out proptosis
   1) Meningoencephalocele
   2) Frontal sinus mucocele
   3) Rhabdomyosarcoma
      ↓
      me location
      superonasal
      orbit
II) Down & In proptosis
   i) Lacrimal Gland tumor
      a) Dermoid cyst
   III) upward proptosis
       • Carcinoma maxilla
   IV) Proptosis outwards → Ethmoid sinusitis (very rare)

DIFERENT PRESENTATIONS

• Acute proptosis
  Cause → Orbital hemorrhage
  orbital cellulitis

Orbital cellulitis

Unilateral proptosis, pain, fever, decreased ocular motility, erythema, and edema of the eyelids

MC CAUSE OF UNILATERAL PROPTOSIS IN CHILDREN

Intermittent proptosis

• Orbital varix
• Orbital lymphangioma

Proptosis increases during
   upper Respiratory Tract infection

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Pulsating proptosis

- Carotico cavernous fistula (CCF)

\[\rightarrow\] Abnormal communication between carotid artery and cavernous sinus

\[\rightarrow\] 'Bruit' can be heard

\[\rightarrow\] Eye \rightarrow Pulsating

- Meningoencephalocoele

- Congenital AVM (Arterio venous malformations)

Unilateral proptosis

- MCC

In Adults \[\rightarrow\] TED's

In children \[\rightarrow\] Orbital cellulitis
BILATERAL PROPTOSIS

Bilateral proptosis

* MCC
  In Adults → TED
  In Children
  ↓
  i) Orbital metastasis
     from Neuroblastoma
     (Adrenal medulla)
     ↓
     Also causes Bilateral periorbital

ii) AML (Acute Myeloid Leukemia)
    ↓
    CHLOROMA of orbit
    Granulocytic sarcoma

measurement of proptosis

i) Hertel's
   exophthalmometer

ii) In children
    ↓
    Luedde's Exophthalmometer
3) Naegel’s Exophthalmometer

4) CT

Pseudophtosis

1) High Axial myopia

2) Contralateral enophthalmos

3) Upper lid retraction

Complications of proptosis

1) Exposure Keratopathy – Due to lagophthalmos

2) Diplopia
   * Due to cranial nerve involvement
     ↓
     CN palsy

3) Optic nerve compression
   ↓
   Pupil shows RAPD
ORBITAL CELLULITIS

• Inflammation of soft tissue behind the orbital septum

• Orbital septum extends from rim of the orbit into the eye lids

Clinical features

• Unilateral painful proptosis

• Decreased ocular motility

↓

Diplopia

• Incidence of unilateral proptosis in children → Orbital cellulitis

↓

Due to ethmoidal sinusitis

• Investigation of choice → Orbital CT

  • Proptosis

  • Subperiosteal Abscess

Treatment: Intravenous Antibiotics

Complications of orbital cellulitis

1) Compression optic Neuropathy → Blindness, RAPD

2) Cavernous sinus thrombosis

3) Cranial neuropathy

4) Brain Abscess

5) Death
BLUNT TRAUMA

Common causes:
1. Fist injury
   a. Cricket ball injury
   b. Road traffic accidents

RACOON'S EYE / PANDA'S EYE

⇒ Due to periorbital ecchymosis
⇒ Blackish discolouration around orbit

SUB-CONJUNCTIVAL HEMORRHAGE

⇒ Bright red appearance due to accumulation of fresh blood beneath conjunctiva.
⇒ Look for posterior limit of the redness to rule out base of skull fracture.

CHEMOSIS

⇒ Edema of the conjunctiva.
SCLERAL RUPTURE

⇒ Ruptures at the thinnest area: Posterior to insertion of recti
⇒ and most thinnest area: At the limbus
⇒ Contra-co coup injury:
  rupture occurs opposite to direction of force

BLOW-OUT FRACTURE

⇒ Pure blow-out fracture: Involving only the floor, at the posterior-medial aspect
⇒ Impure fracture: Involves the rim of orbit
⇒ Medial wall fractures:
  ⇒ Air from ethmoidal sinus leak into orbit
  ⇒ Emphysema on palpation of orbit

HYPHEMA

⇒ Blood in anterior chamber
⇒ Complication:
  1. Blood can block trabecular meshwork
     ⇒ Raised IOP
  2. Corneal blood staining in the stromal layer
⇒ Source of blood:
  ⇒ Major arterial circle of iris at iris root
**IRIDO DIALYSIS**

- Separation of iris-root from the ciliary body attachment
- Iris root: Thinnest part of iris
- Results in a D-shaped pupil

**TRAUMATIC MYDRIASIS**

- Due to sphinter tears
- On gonioscopy:
  - Widened ciliary body band
  - Suggestive of angle recession
  - (separation between circular and longitudinal ciliary muscles)
- Can result in angle-recession glaucoma
  - (secondary open-angle glaucoma)

**VOSSIUS RING**

- Blunt trauma → sudden increase in IOP
  - Spasm of sphinteric muscles
  - Miotic pupil hits anterior lens capsule
  - Iris leaves behind imprint of melanin pigments
  - Vossius ring

*Cited from Ophthalmology v.2.0 / Marrow 4.0 / 2020*
**ROSETTE CATARACT**

⇒ Flower-shaped cataract
⇒ Posterior - subcapsular cataract

**SUBLUXATION OF LENS**

⇒ Due to excess stretch on zonules, they can break and lead to dislocation of lens
⇒ Most commonly, lens dislocates posteriorly into the vitreous

**VITREOUS HEMORRHAGE**

• Bleeding into the vitreous
• In children, mc cause of vitreous hemorrhage
  ↓
  Blunt trauma.
VITREOUS BASE AVULSION

- Peripheral vitreous overlying the pars-plana (strongest attachment of vitreous)
- Pathognomonic sign of blunt trauma

BERLIN'S EDEMA

⇒ Increased IOP due to trauma
  ↓
  Ganglion cell apoptosis
  ↓
  Cytotoxic edema
  ↓
  Appears as pale (milky-white retina)
  ↓
  At foveola: Cherry-red spot

⇒ Commotio retinae: Berlin's edema + cherry-red spot
⇒ Type of contra-coup injury

OPTIC NERVE AVULSION

⇒ in severe trauma, especially decelerating injury in road-traffic accidents
LIDS

ANATOMY OF EYELIDS

- Upper lid covers 1-2mm of cornea.
- Lower lid rests just at the inferior limbus
- Both meet at the medial / inner canthus and lateral / outer canthus

Cross-section:

- Orbicularis oculi (VII N)
- Skin
- Tarsal plate
- Meibomian / Tarsal gland
- Gland of Zeis and Moll
- Cilia / eyelash

- Tarsal plate: D-shaped in upper lid
  Crescent shaped in lower lid.
- Meibomian gland: Modified sebaceous glands
- Zeis gland: Modified sebaceous glands
- Gland of Moll: Modified sweat glands
INFLAMMATION OF LID GLANDS

I. Acute inflammation:
   - Acute suppurative inflammation - Hordeolum
     
     a) Hordeolum externum (Stye):
        - Gland of Zeis inflamed
        - Painful swelling of lid margin
        - Pus pointing at base of eyelash
        - Rx: Epilation

     b) Hordeolum internum:
        - meibomian gland
        - Painful swelling away from lid margin
        - Rx: Incision and drainage (vertical incision after evertong lid margin)

II. Chronic inflammation
   
   - Chalazion:
     - Chronic lipogranulomatous inflammation of meibomian gland
     - Painless swelling away from lid margin

   Rx: Incision and curettage
   - Intralesional steroid injection
     (to prevent recurrence)
   - Triamcinolone acetonide used
- Recurrent hordeola:

Causes:
1. In children : Refractive errors
2. In adults : Undiagnosed DM
3. In elderly : Rule out Sebaceous gland carcinoma

---

**DISORDERS OF LID MARGINS**

1. Ectropion:
   - Outward turning of lid margin
   - Signs: Epiphora (overflow of tears)
   - Causes:
     - Ageing (Involutional ectropion)
     - Facial nerve palsy (paralytic)
     - Scar on skin near eye-lid (cicatricial)
   - Treatment / Surgeries
     1. Canthoplasty: Lateral canthal tendon tightening
     2. Conjectivo-plasty: For medial ectropion
        - Spindle resection of small area of conjunctiva → Scar formation → pulls lid inwards
     3. Kuhnt Szymanowski procedure:
        - For massive ectropion
        - Hexagonal area of skin resected and sutured
ENTROPION

- Inward turning of lid margin
- Misdirection of lashes - trichiasis
- Hyperlacrimation: Due to reflex lacrimation caused due to irritation of cornea.

Types:
1. Involutional: Over-riding of pre-septal portion of orbital over the pre-tarsal portion
2. Cicatricial: eg: in trachoma, cicatricial entropion of upper lid occurs

Surgical procedures for entropion

‘Quick Jones is a wise Entrepreneur’
1. Quickert’s sutures
2. Jone’s procedure
3. Weis procedure
4. Tarsal wedge resection - usually for cicatricia of trachoma

LID COLOBOMA

- Congenital absence of tissue in the lid
- Usually in upper lid
- Associated with Goldenhar Syndrome
S-Shaped lid margin:

Causes:
1. Dacryoadenitis
2. Neuro fibromatosis
   - Plexiform neurofibroma of upper lid

BLEPHARITIS
- Inflammation of lid margin
- Types:
  1. Squamous: Flaky material at base of eyelash
  2. Seborrheic: Oily crusts at base of lash
  3. Ulcerative: Tiny bleeding ulcers

Complication of ulcerative blepharitis:
1. Tylasis
2. Cicatricial entro / Ectropion

BLEPHAROPTOSIS

- Causes:
  1. Neurogenic: a) Third nerve palsy
     b) Horner's Syndrome
  2. Myogenic: a) Congenital ptosis (m.c. cause)
     - Absent lid crease
     - Lid-lag on downgaze
     - Poor LPS function

- Marcus Gunn Jaw Winking phenomenon:
  Seen in certain cases of congenital ptosis
- Patient with ptosis → Opens jaw → Lid elevation; → Closes jaw → Ptosis returns

Cause:
- mandibular division of trigeminal nerve – innervates Lateral pterygoid and LPS

  b) myasthenia gravis

- most common cause of bilateral ptosis

- Ptosis varies depending on time of day and the activities of patient

- Tests
  1. Ice-pack test
  2. Edrophonion test

- Cogan’s lid twitch sign: Over-shoot of the eye, on looking from down-gaze to primary gaze

  c) myotonic dystrophy
d) Oculo – Pharyngeal dystrophy
e) CPEO – Chronic progressive external ophthalmoplegia

- Bilateral ptosis with motility defect in all gazes

- If associated with retinitis pigmentosa, called Kearn-Sayre Syndrome
3. Involutional ptosis:
   - Also called aponeurotic ptosis; idiopathic ptosis
   - Overall most common cause of ptosis
   - Dehiscence of LPS → Poor contraction → Ptosis

4. Mechanical ptosis:
   - Can be due to tumour of lid, repeated chalazion, edema

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**MANAGEMENT OF PTOSIS**

1. In Horner’s Syndrome: Fasanella-Servat procedure
   (tarso-mullerectomy)

2. In myasthenia: usually medical management
   - Fasanella-Servat procedure can be done

3. LPS resection:
   a) Blascovics’ Surgery: Conjunctival approach
   b) Everbusch’s Surgery: Skin approach

4. LPS advancement procedure:

5. Tarso-Frontalis sling / Frontalis Suspension procedure:
   - Subcutaneous sling from tarsal plate to the
     frontalis muscle
   - Materials used for sling:
     - Fascia lata of patient (best)
     - Supramid sutures
     - Silicone rods
BLEPHAROPHIMOSIS SYNDROME

- Height and width of palpebral fissure is decreased
- Epicanthus inversus: Extra fold of skin from lower lid to upper lid
- Lateral ectropion
- Telecanthus: medial canthal distance is more than half of inter-pupillary distance
- Superior orbital rim hypoplasia.
- Flat and wide nasal bridge
- Autosomal dominant myogenic ptosis

DISORDERS OR EYE-LASHES

1. Distichiasis:
   - Extra row of abnormal lash arising from meibomian glands
   - Lid margin normal

2. Trichiasis:
   - Misdirection of lash due to inturning of lid

3. Madarosis:
   - Loss of eyelash or eyebrow
   - Causes: hypothyroidism, leprosy, syphilis, SLE
4. Trichomegaly:
   - Thin, long lashes
   - Seen with use of prostaglandin analogues, cyclosporin

5. Poliosis:
   - Premature greying / whitening of eyelashes
   - Seen in Vogt–Koyanagi–Harada Syndrome
LACRIMAL APPARATUS

LACRIMAL GLAND

- 2 glands → main and accessory

- main lacrimal gland:
  - Situated in superotemporal part of orbit
  - 2 lobes: Palpebral and orbital. Separated by LPS aponeurosis (vertical fibrous part of LPS)
  - Function: Reflex secretion of tears
  - Biopsy always taken from deeper orbital lobe to avoid cutting ductules

- Accessory glands:
  - Basal secretion of tears

  1) Gland of Krause: Situated at superior and inferior fornices of palpebral conjunctiva

  2) Gland of Wolfring: At superior or free border of tarsal plate

LACRIMAL DRAINAGE SYSTEM

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1) Lacrimal punctum: At lid margin
   lower punctum medial to upper punctum

2) Lacrimal canaliculi:
   - vertical part: 2mm
   - horizontal part: 8mm

3) Common canaliculi

4) Lacrimal sac: Opening of common canaliculi here is guarded by valve of Rosenmüller

5) Nasolacrimal duct (NLD):
   - Runs downwards, backwards and laterally

6) Inferior meatus of nose:
   - Opening of NLD here is guarded by valve of Hasner

---

**Dacryocystitis**

- Inflammation of lacrimal sac

**Congenital dacryocystitis:**

- **Cause:** Imperforate Hasner’s valve
- **Clinical features:**
  1) Tearing / watering
  2) Infection and enlargement of lacrimal sac
     - Swelling at medial canthus
  3) Pressure at medial canthus
     \[\rightarrow\] Regurgitation

- Regurgitation of:
  - Clear fluid: Congenital NLD obstruction
  - Mucopurulent material: Congenital dacryocystitis
MANAGEMENT OF DACRYOCYSTITIS

- Depends on age of child
  - < 9 months : Crigler’s lacrimal sac massage
    Topical antibiotics
  - 9 months to 4 yrs : Probing and syringing
  - > 4 years : DCR surgery

Encysted mucocele:
  - Swelling at medial canthus, fluctuant, but no regurgitation
  - Bluish color due to venous engorgement

- In adult:
  - Acute : Systemic antibiotics + NSAIDs
  - Chronic : Treatment of choice: DCR surgery

DACRYO-CYSTO-RHINOSTOMY (DCR)

- Lacrimal sac connected to middle meatus of nose through a bony opening in the lateral wall of nose

- Contraindications:
  1. Age < 4 yrs
  2. Lacrimal sac malignancy - Dacryocystectomy done
  3. Atrophic Rhinitis

SCHIRMER’S TEST

- To check lacrimal gland function
- Schirmer’s strip at junction of lateral and middle third of lower lid
Test I: Without topical anesthesia.
   checks total secretion (basal + reflex)

Test II: Topical anesthesia used
   only basal secretion
   - Whatman filter paper no. 41 used

---

TEAR FILM

- 3 layers

1. Outer lipid layer:
   - Sebum from meibomian gland and gland of Zeis
   - Prevent evaporation of tears

2. Middle aqueous layer
   - Thickest layer
   - Lacrimal glands

3. Inner mucin layer
   - Secreted by goblet cells of conjunctiva

Goblet cells: Specialized epithelial cells of conjunctiva.
   - Maximum density at inferonasal bulbar conjunctiva

   - Density assessed by conjunctival impression cytology
   - PAS positive cells
   - Density decreased in diabetes and vit. A deficiency

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DRY EYE

- Lipid deficient dry eye:
  - In meibomian gland dysfunction

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* Aqueous deficient:
  - Keratoconjunctivitis sicca (KCS)
    - Mc cause: Sjogren's syndrome
    - Primary Sjogren's: Idiopathic
    - Secondary Sjogren's; mc cause: Rheumatoid arthritis
      [most common type]

* Mucin deficient
  - Diabetes
  - Vit. A deficiency

---

**TEAR-FILM BREAK UP TIME**

00:27:23

* Assess mucin components of tears
* Wettability of tear film
* Fluorescein dye on conjunctiva.
  ↓
  Ask patient to blink
  ↓
  Observe under blue light
  ↓
  Time between last blink and appearance of first dark spot - Tear break-up time

* Normal: 15 seconds
RETINOPATHY OF PREMATURITY

DEVELOPMENT OF RETINAL VASCULATURE

- Starts from optic disc and grow towards periphery
- Reach nasal ora by 36 weeks and temporal ora by 40 weeks
- Babies delivered before 36 weeks (pre-mature) have hypoxic retina that releases VEGF causing abnormal vasculature

RISK FACTORS FOR ROP

- Gestational age - most important
- Low birth weight
- Exposure to Oxygen (PO₂)
- Sepsis
- Blood transfusions
- Hyaline membrane disease
- Twin delivery

ROP SCREENING

- Revised guidelines for Indian babies:
  1st ROP screening to be done at:
  a) 4 weeks after birth if:
    - Gestational age: 28-34 weeks
    - Birth-weight: < 2000g

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b) 3 weeks after birth if:
   - Gestational age : < 28 weeks
   - Birth - weight : < 1200g

ICROP ZONES:

* International classification of ROP divides fundus into
  3 zones

1. Zone 1: Area with optic disc as centre and
   radius as twice the distance between disc and
   foreola. ROP changes here are most severe

2. Zone 2: distance between nasal border of zone 1 to
   nasal ora serrata (all around zone 1)

3. Zone 3: Extreme periphery outside zone 2 up to
   temporal ora serrata

<table>
<thead>
<tr>
<th>STAGES OF ROP</th>
<th>00:10:00</th>
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</thead>
<tbody>
<tr>
<td>Stage 1: Clear demarcation line between vascular and Non-vascular zone</td>
<td></td>
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<tr>
<td>Stage 2: Line progresses to a ridge</td>
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<tr>
<td>Stage 3: Vessels form on the ridge</td>
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<tr>
<td>(Extra-retinal neovascularization)</td>
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<tr>
<td>Stage 4: Subtotal tractional RD</td>
<td></td>
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<tr>
<td>Types: 4a: Extra-foveal</td>
<td></td>
</tr>
<tr>
<td>4b: Foreal involvement</td>
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<tr>
<td>Stage 5: Total RD - funnel shaped RD</td>
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</tbody>
</table>

* Clinical presentation of RD: Leuco-coria (if screening not done)

* If screening done - Treat at or before stage 3 to save vision
- Treatment: At stage 3
  - Laser photo-coagulation of avascular retina
  - Bevacizumab (intra-vitreal)

At stage 4: Can repair vitreo-retinal surgery

---

**PLUS DISEASE**

- Venous dilation and arteriolar tortuosity in at least 2 quadrants of posterior pole
- Neovascularization in iris, hazy vitreous
- Presence of plus disease mandates treatment

Criteria to start treatment:
- Threshold disease: 5 contiguous or 8 non-contiguous clock hours of stage 3 disease in zone 1 or 2, with plus disease
  - Terminology not in use now

- Type - 1 ROP:
  1) Any stage in zone 1 with plus disease
  2) Stage 3 disease in zone 1 (without plus)
  3) Stage 2-3 in zone 11 with plus disease

Type 1 ROP requires urgent treatment within 48-72 hrs of diagnosis

- Type - 2 ROP:
  1) Stage 1-2 disease in zone 1
  2) Stage 1 in zone 11

- Gold standard treatment: Laser photoocoagulation of avascular retina
- Aggressive posterior ROP
- Plus disease in zone I or II out of proportion to the peripheral neovascularization

* Early screening and appropriate treatment can prevent ROP

Cicatricial ROP:
* Partial or non-treatment can cause scar
* Scar drags macula (ectopic macula)
* Cause RD

Examination of fundus:
- Dilated fundus exam with 20D or 25D lens along with indirect ophthalmoscope
- Zone I: If nasal border of disc visible in field of view
- To visualize ora serrata - scleral indentation